RISK FACTORS FOR PERINATAL DEATH
IN NEW SOUTH WALES

by

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Except where it is indicated otherwise, this thesis represents my original work.

Stephen A. Buetow
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ABSTRACT

This is a case-control study of risk factors for perinatal death in New South Wales during 1985-86. The study draws on the social and medical sciences in formulating a conceptual model from which hypotheses are generated for testing. Attempts to verify these hypotheses involve analyses of two main sets of data, each comprising information that was collected from separate sources and subsequently merged through record linkage. The first data set is for the complete State of New South Wales, and contains registration and Health Department data. The second set of data is derived from the obstetric patient records and interviews with patients of six large public hospitals.

Quantitative methods employed include measurements of prevalence and risk, mainly from contingency tables. The studied risk factors relate most proximally to the newborn’s physiological maturity and to obstetric factors, to health care factors, and to women’s pregnancy health. More background influences on perinatal death are considered last. Among identified risk factors for perinatal death are a greater propensity of boys than girls for preterm birth, little or no help with housework for women in the paid labour force, doing the same exercise in the last but not the first trimester, and a maternal birthplace in most selected regions outside Australia.
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**GLOSSARY OF MEDICAL TERMS**

*Abruptio placentae* (accidental haemorrhage): bleeding from the placental site owing to the premature separation of a normally situated placenta after 22 weeks' gestation and before the birth.

*Abdominal viscera*: the internal organs of the abdomen.

*Accoucheur*: A person who conducts a birth.

*Active phase of labour*: period of active dilatation of the cervix during the first stage of labour.

*Amelia*: the total congenital absence of the arms or legs.

*Amenorrhoea*: the absence of menstruation.

*Amniocentesis*: piercing of the amniotic sac through the abdominal wall to withdraw, for testing, a sample of the amniotic fluid surrounding the baby.

*Amnioscopy*: direct examination of the inside of the amniotic sac using an instrument (an amnioscope) that is passed through the abdominal wall.

*Amniotomy*: the surgical rupture of the amniotic membranes surrounding the baby in the uterus.

*Anaemia*: a reduced number of red blood cells or a lack of haemoglobin in them.

*Anaesthetic*: an agent capable of removing sensation.

*Analgesic*: an agent capable of removing pain.

*Anencephalus*: a developmental defect characterized by absence of some or all of the bones of the rear of the skull, and of the cerebrum, the largest and most highly developed part of the brain.

*Antenatal*: occurring before birth.

*Antenatal screening*: this identifies from a population of apparently healthy women those who are sufficiently at risk of a specific disorder to justify a subsequent diagnostic test or procedure, or primary preventive action.

*Antepartum*: occurring late in the pregnancy and before the onset of labour.

*Apgar score*: an assessment of the condition of the infant at birth, based on five objective signs: heart rate, respiratory effort, muscle tone, response to stimulation, and colour. At one and five minutes after the birth, each sign is given a score of 0, 1 or 2, and the sum of the five scores is the Apgar score.

*Apnoea*: a cessation of breathing.
Appropriate for gestational age: infants with birthweights between the tenth and ninetieth percentiles for their gestational ages.

Augmentation: the stimulation of uterine contractions after labour begins spontaneously. This can be achieved by amniotomy and, if necessary, oxytocin stimulation.

Auscultation: the process of listening, usually with the aid of a stethoscope, to sounds produced by the movement of gas or air within the body.

Autonomic nervous system: controls bodily functions that are not consciously directed.

Hyperbilirubinaemia: an excess of bilirubin (a bile pigment) in the bloodstream.

Birthweight: the first weight of the foetus or infant obtained after delivery.

Bishop score: assesses the ripeness of the cervix on the basis of five criteria: dilatation, consistency, length, position and the station of the head in relation to the ischial spines. Each criterion is given a score of 0, 1 or 2 and the total is the Bishop score.

Breech presentation: presentation by the buttocks (frank breech) or more rarely the feet or knees (footling breech). Normally, the head presents first (cephalic presentation), but the side, shoulder or placenta can also be the first parts to appear.

Caesarean section: surgical operation for delivering the infant, placenta and membranes through the abdominal and uterine walls.

Cannulae: small hollow tubes inserted into the uterus to achieve evacuation.

Carboxyhaemoglobin: formed when carbon monoxide combines with haemoglobin, a pigment in the red blood cells. It reduces the oxygen carrying capacity of the blood by up to 12 per cent (Abel, 1983).

Cardiotocography: an automatic graphic correlation of foetal heart rate patterns and uterine contractions.

Catecholamines: chemical substances affecting the functioning of the sympathetic and central nervous systems.

Cephalopelvic disproportion: the head is too large for the pelvis through which it must pass.

Cerebral palsy: a developmental abnormality of the brain producing weakness and incoordination in the limbs.

Cervical os: the opening of the cervix.

Chorioamnionitis: inflammation of the foetal (outer chorionic and inner amniotic) membranes.

Chorionic villus sampling: the sampling of cells from the fingerlike projections that arise from the membrane surrounding the embryo after implantation.
Chromosomal disorder: abnormality of chromosome number or structure.

Cleft lip and palate: there is a fissure in the upper lip and palate (roof of the mouth) resulting from the failure of the two sides to fuse in embryonic development.

Cocaine (also 'coke', Benzoyl Methylecgonine): is a central nervous system stimulant producing mood-altering effects.

Collagen: a protein that is the main constituent of white fibrous connective tissue. Although relatively inelastic, it has a high tensile strength.

Conceptus: the viable product of conception.

Congenital anomalies: all disorders present at birth, whether they are caused by genetic or environmental factors.

Contraction stress test: relies upon the foetal heart rate response to induced or spontaneous contractions.

Curettage: an operation that involves scraping tissue from the internal surface of the uterus.

Depolymerization: the initiated or accelerated breakdown of polymers, substances formed by the linkage of many smaller molecules (monomers).

Diabetes: disorder characterized by a deficiency of the hormone insulin.

Diastolic blood pressure: the lower and more important pressure of blood in the arteries when the ventricles (the two lower chambers of the heart) relax and refill.

Disorders of complex genetic aetiology (also multifactorial aetiology): those attributed to the combined effects of many genes and other, nongenetic factors.

Dystocia (also dysfunctional labour): abnormal labour resulting from deviations from normal patterns in the latent or active phases of labour.

Early neonatal death: a liveborn infant weighing at least 500 grams at delivery (or when the birthweight is unavailable, of at least 22 weeks' gestation) that dies before seven completed days of infant life.

Edward's syndrome (also trisomy 18): a congenital anomaly characterized by delicate facial features including low set, malformed ears and smallness of the jaw and mouth, as well as overlapping fingers, cardiac and central nervous defects and low birthweight.

Elective caesarean section: a caesarean section performed before the onset of labour.

Emergency caesarean section: a caesarean section performed after the onset of labour.
Epidural block: the continuous infusion of an anaesthetic agent into the epidural space between the outermost membranes covering the spinal cord and the bony column of the spine. The intended effect is to numb the sensory nerve fibres from the uterus, and eliminate pain and sensation during childbirth.

Essential hypertension: abnormally high blood pressure in an individual who is otherwise healthy.

Evacuation: the removal through suction of the unwanted product of conception.

Failure to progress: the inability of the foetus to progress through the birth canal. This can result from cephalopelvic disproportion and uterine inertia.

False positivity: the frequency of good outcomes in patients with positive test results.

Flexion: the limbs and head of the foetus are flexed or bent over its trunk.

Foetal alcohol syndrome: set of related conditions including symmetrically retarded foetal growth, central nervous system dysfunction and facial anomalies which arise in many infants born to women that are alcoholics or that drink alcohol excessively.

Foetal distress: suspected from certain changes in the foetal heart rate (F.H.R.), the passage of meconium, excessive foetal movements and the funic souffle - a high pitched sound synchronous with the foetal heart rate. Confirmation of foetal distress during labour may depend on foetal scalp pH determination (Goodlin, 1981).

Foetal position: the relation of the foetal presenting part to the woman’s pelvis.

Foetal transfusion: Rhesus negative blood compatible with the pregnant woman is transfused either into the foetal peritoneal cavity or, since 1981, into an umbilical vein with the help of foetoscopy or ultrasound (Rodeck et al., 1981; Chamberlain, 1984; Berkowitz et al., 1986).

Forceps: instruments used to help deliver the baby toward the end of the second stage of labour.

General anaesthetic: removes feeling from all parts of the body through achieving total unconsciousness.

Gestational age: dated usually in completed gestational weeks from the last normal menstrual period.

Glucocorticoids: a group of corticosteroids that mainly affect carbohydrate metabolism, and that can be used to stimulate foetal lung maturity.

Haematoma: a solid swelling produced by the clotting of blood which has accumulated in the tissues.

Heroin (also Diacetylmorphine): a central nervous system depressant producing mood altering effects.
Hydramnios: excess amniotic fluid surrounding the foetus in utero.

Hydrocephalus: an excess of cerebrospinal fluid in the ventricles (cavities) of the brain.

Hypertensive disorders of pregnancy: there are three broad groups: chronic hypertension predating the pregnancy; pregnancy-induced hypertension (pre-eclampsia and eclampsia) superimposed on chronic hypertension; and pregnancy-induced hypertension alone (Carlson, 1988).

Hyperventilation: breathing at an abnormally rapid rate which lowers the carbon dioxide concentration in the blood.

Hypocalcaemia: a low level of calcium in the blood.

Hypoglycaemia: an abnormally low blood sugar.

Hypospadias: a congenital anomaly characterized by the opening of the urethra on the underside of the penis.

Hypotension: an abnormally low arterial blood pressure.

Hypoxaemia: an abnormally low concentration of oxygen in the blood.

Hypoxia (also asphyxia): a deficiency of oxygen in the tissues.

Hysterotomy: an operation that involves an incision into the uterus to remove its nonviable contents.

Iatrogenic mishap: ill-fortune resulting from the unforeseen or inevitable side-effects of a physician’s words or actions.

Idiopathic: of unknown cause.

Immature infants: strictly speaking, birthweights between 500 and 1 000 grams. However, the term immaturity is sometimes used in the more general sense of not physiologically mature.

Incompetent cervix: a cervix that is unable to hold the foetus in the uterus because it dilates prematurely.

Induction of labour: the deliberate initiation of uterine contractions before their spontaneous onset.

Intestinal atresia: a congenital absence or abnormal narrowing of an intestinal body opening.

Internal electronic foetal monitoring: a foetal electrocardiogram from a spiral electrode attached to the foetal presenting part; the procedure first requires rupture of the membranes.

Intervillous space: placental space between the tiny chorionic villi (branching structures which arise from the outer membrane surrounding the foetus) in which maternal blood circulates.

Intracranial haemorrhage: bleeding within the skull.

Intrapartum: occurring during labour and delivery.
Intrauterine growth retardation: birthweights below the tenth percentile for the gestational ages. These infants may be designated small for gestational age, or small for dates.

Intraventricular haemorrhage: a cerebral haemorrhage which begins on the lateral ventricle of the brain in infants below 34 weeks' gestation.

Laminaria tents: rod-shaped structures which, having been prepared from the dried stems of the seaweed 'laminaria', are inserted into the cervix where, by absorbing water and so expanding, they cause the cervix to dilate.

Large for gestational age: infants with birthweights above the ninetieth percentile for their gestational ages.

Late neonatal death: a liveborn infant weighing at least 500 grams at delivery (or, when the birthweight is unavailable, being of at least 22 weeks' gestation) who dies after seven, but before 28, completed days of infant life.

Latent phase of labour: period of slow dilation of the cervix following the onset of labour. The latent phase ends when the cervix begins to dilate rapidly.

Life at birth: evidence includes breathing, a heart beat, pulsation of the umbilical cord or definite movement of the voluntary muscles.

Lithotomy position: the mother delivers her baby while lying on her back with flexed thighs and legs abducted and held in place with lithotomy poles.

Low amniotomy: rupture of the forewaters with a forceps such as Kocher's.

Low birthweight (also light-weight): of infants weighing less than 2 500 grams at birth.

Malpresentation: any presentation other than the vertex (the top of the head).

Marfan's syndrome: characterized by excessive height, abnormally long, slender fingers, and congenital defects of the heart and eyes.

Meconium: the first stools of the conceptus, the passage in utero of which suggests foetal distress.

Medical induction of labour: the initiation of labour using drugs, namely syntocinon given intravenously or prostaglandin administered vaginally.

Microcephaly: a nervous system defect characterized by arrest of brain growth and an abnormally small head.

Moderately overweight woman: enters pregnancy weighing at least 20 per cent above the ideal weight for her height and age.

Monogenic defects: defects in a single gene pair.
Neonatal death: the sum of early neonatal deaths and late neonatal deaths.

Narcotic withdrawal syndrome: includes hyperflexia, tremors, irritability, excessive high-pitched crying and disturbed sleep. Other symptoms involve the gastrointestinal tract.

Neural tube defects: a group of congenital anomalies caused by failure to close of the neural tube, the embryological structure from which the brain and spinal cord develop.

Nonstress test: uses unstressed foetal heart rate tracings to detect abnormalities.

Normotensive: the blood pressure is 'normal.'

Oblique lie: the lie of the foetus in utero is transverse; that is, the long axis of the foetus lies across, not parallel to, the long axis of the uterus.

Occipito-posterior position: the back of the foetal head faces the back of the pelvis.

Oedema: an excess of fluid.

Oestriol tests: permit assessment of placental function and foetal growth.

Osteomyelitis: an inflammation of the bone marrow due to infection.

Paracervical block: infiltration of a local anaesthetic immediately lateral to the cervix to block sensation from the uterus toward the end of the first stage of labour.

Parenteral fluid therapy: the administration of fluid intravenously.

Patau's syndrome (also trisomy 13-15): a congenital anomaly characterized by cerebral and cardiac defects, ocular anomalies, malformed ears and cleft lip and palate.

Pathogenic: causing disease.

Perfusion: the passage of fluid through a passage, as with the flow of maternal blood through the intervillous space.

Perinatal: refers in this study to stillbirths and neonatal deaths, but outside Australia, usually to stillbirths and early neonatal deaths.

Phenylketonuria: a rare metabolic disease characterized by the presence in the urine of phenylketones.

Phocomelia: a congenital absence of the upper arm(s) and/or upper leg(s).

Placental abruption: a translation of abruptio placentae.

Placental insufficiency: abnormally decreased placental function depriving the foetus, for example, of an adequate supply of oxygen and nutrients.
Postmaturity (also post-term): infants born after 41 weeks' gestation.

Postmaturity syndrome: a set of related conditions associated with decreased placental function.

Postperinatal deaths: infant deaths after seven completed days of life. In Australia, postperinatal death are infant deaths after 28 days of life.

Post-term: from 42 weeks of pregnancy (294 or more days).

Pre-eclampsia (also toxaemia of pregnancy): disorder characterized by high blood pressure, protein in the urine and, according to some prescriptions, the swelling of certain body parts.

Premature rupture of the membranes: evidence before the 37th week of pregnancy of leaking amniotic fluid (the fluid contained in the amniotic sac forming the 'bag of waters').

Prematurity: a descriptive term generalizing the non-specific process by which a foetus or infant is born of low birthweight because it is born preterm, growth retarded or both.

Presentation: the part of the infant born first.

Preterm: before 37 weeks of pregnancy (less than 259 days).

Primary prevention: the prevention of the occurrence of a disorder by removing its causes.

Primary sex ratio: the sex ratio at conception.

Primigravidae: women pregnant for the first time.

Progesterone: an important hormone for the maintenance of pregnancy.

Prostaglandins: hormone-like substances, one effect of which is to cause uterine contractions.

Proteinuria: at least 300 mg. of protein per litre in either a clean-catch, mid-stream specimen of urine, or a 24 hour collection of urine.

Psychoneuroendocrinal responses: hormonal and nervous system responses to psychological stress.

Psychoprophylaxis: the use of breathing and relaxation techniques rather than drugs to cope with the pain of labour contractions.

Pulmonary hypertension: raised blood pressure in the blood vessels supplying the lungs.

Pyloric stenosis: a narrowing of the muscular outlet of the stomach.

Regional or local anaesthesia: relieves sensation from a limited area or region of the body.
Respiratory distress syndrome (also hyaline membrane disease): incomplete expansion of the newborn infant’s lungs (atelectasis) owing to a lack of surfactant, a wetting agent needed for the air sacs to slide apart for the next breath, and not stick together.

Retrolental fibroplasia (also retinopathy of pregnancy): abnormal proliferation of fibrous tissue behind the lens of the eye which can cause blindness.

Rh erythroblastosis: a rare but severe form of congenital haemolytic anaemia usually caused by incompatibility of the Rhesus blood groups of the mother and baby. Prophylaxis of Rh-sensitization involves giving anti-D gamma globulin to Rhesus negative women.

Second stage labour: this begins with complete dilatation of the cervix and ends with delivery of the infant.

Secondary prevention: the prevention of overt disorders through early detection and appropriate therapeutic intervention.

Secondary sex ratio: the sex ratio at birth.

Sensitivity: ability of a test to detect abnormal outcomes.

Sex chromosome aneuploidies: these arise when the chromosome number of a cell is not an exact multiple of the normal number.

Short maternal stature: a maternal height below 152 centimetres.

Shoulder dystocia: the shoulder is too large for the pelvic outlet through which it must pass.

Small for gestational age: see intrauterine growth retardation.

Specificity: the probability of a negative test result when the outcome is good.

Splanchnic area: the abdomen and its internal organs.

Spontaneous abortion (also miscarriage): the expulsion from the uterus of an embryo or a nonviable foetus.

Stillbirth: for statistical purposes, a stillbirth is the birth of a foetus weighing at least 500 grams (or, when the birthweight is unknown, of at least 22 weeks’ gestation) and showing no evidence of life at birth.

Subcutaneous: beneath the skin.

Sudden infant death syndrome (also cot death): the death of an infant, usually overnight in its cot, from an unidentifiable cause. Such deaths occur from three weeks of age, but most often between one and four months.

Surgical induction of labour: see amniotomy.

Sympathetic nervous system: part of the autonomic nervous system that controls involuntary responses to alarm, deciding on ‘fight or flight’.
Nun will die Sonn' so hell aufgehn
als sei kein Unglück die Nacht geschehn!
Das Unglück geschah nur mir allein
Die Sonne, sie scheinet allgemein!
Du mußt nicht die Nacht in dir verschränken,
mußt sie ins ew'ge Licht versenken!
Ein Lämplein verlosch in meinem Zelt
Heil sei dem Freudenlicht der Welt

Now the sun will rise again
as if no misfortune had occurred during the night.
The misfortune was mine alone;
but the sun shines for everyone.
You must not become entwined with darkness,
but be immersed in eternal light.
A little lamp went out in my dwelling.
Hail to the light that cheers the world!

Friedrich Rückert, adapted in Gustav Mahler's Kindertotenleider (Songs on the Death of Children), Edition Peters.
1.0 INTRODUCTION

Perinatal death\(^1\) ends life, tragically almost before life begins;\(^2\) hopes for a future are denied like the absent consolation of a past. In Australia, including its largest State, New South Wales, perinatal death is 'statistically rare',\(^3\) but even so, there can be no complacency in seeking further to salvage life. The successes of regions including, most notably, Scandinavia, testify to this fact. Moreover, although Scandinavian health services and funding systems may not be reproducible in Australia,\(^4\) reductions in perinatal mortality are likely sequelae, in all developed countries, of recent research developments.

The most exciting of these advances is perhaps the discovery of the aetiological importance of the maternal environment during pregnancy. To date, progress in lowering perinatal mortality has resulted mainly from improvements in diagnostic and therapeutic methods; that is, in treatment. Realization of the aetiological contribution of maternal lifestyle factors promises that new lower limits of perinatal mortality will follow a commitment of research and resources to

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1 See section 1.1.
2 From a biological point of view, human life begins at conception. It is the value to be attributed to that life, particularly in its incipient stages, which is questioned in the debate on induced abortion (Royal Commission of Inquiry, 1977).
3 To be statistically rare, incident cases of an event must occur in less than 2 per cent of the study population over a specified time period (Shelley, 1988). In Australia the incidence of perinatal death registered in 1985-86 was 1.17 per cent of all births (Australian Bureau of Statistics, 1988a).
4 For example, in contrast to the Australian situation, the private health care sector is largely nonexistent in Scandinavia.
primary prevention. Specifically, continued progress depends upon elucidating the nexus between perinatal mortality risks and social as well as clinical factors, and this imperative compels the present interdisciplinary investigation of risk factors for perinatal death.

The present chapter has several functions. The term 'perinatal death' is defined, and difficulties affecting its use, especially in a comparative context, are discussed. Then, historical trends in perinatal and postperinatal mortality are described at the international and Australian State and Territorial levels. Overall declines in perinatal mortality are accounted for in terms of sociodemographic changes, with reference specifically to Australia, and general medical advances. Subsequent discussion centres on the causes of perinatal deaths registered in Australia in 1986, these causes also being compared with those for 1979. Some attention is paid to lacunae in recent perinatal mortality research, and against this total background, the aims of the thesis are stated.

1.1 PERINATAL TERMINOLOGY AND ITS APPLICATION

Perinatal events are presented in Figure 1.0. The conceptualization is the product of attempts, described below, to resolve differences experienced over time and between regions in perinatal terminology, and in the quality of birth and perinatal death registrations. These problems have required solution for meaningful comparison of internally valid and reliable perinatal death statistics to be undertaken.

Historical difficulties in defining perinatal events resulted, for example, from the Eighth Revision of the International Classification

---

5 Primary prevention involves preventing the occurrence of a disorder by removing its causes. Secondary prevention involves preventing overt cases through early detection and appropriate therapeutic intervention.
of Diseases (1967-69). Although it defined the perinatal period as 'extending from the 28th week of gestation to the seventh day of life' (World Health Organization, 1969: 763), some countries were known to collect data down to and below the twentieth completed week of gestation and up to the 28th completed day of infant life. Moreover, the criterion of a minimum gestational age was unsatisfactory: missing or unreliable data were produced by difficulties in determining the first day of the last normal menstruation, and by late booking for antenatal care (Beischer et al., 1983; and see section 5.1.1).

Figure 1.0
Gestational Periods, Stages and Ages

<table>
<thead>
<tr>
<th>Name for Conceptus When Born Dead</th>
<th>Fertilization</th>
<th>Stage of Viability</th>
<th>Term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abortus Early</td>
<td>Late</td>
<td>Stillbirth</td>
<td>Neo-inatal Death</td>
</tr>
<tr>
<td>Preivable</td>
<td>Preivable</td>
<td>Viable</td>
<td></td>
</tr>
<tr>
<td>Birthweight (g)</td>
<td>6 50 100 200</td>
<td>500 750 1000 2000</td>
<td>3000</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physiological Maturity</th>
<th>Embryo</th>
<th>Preivable Premature Foetus</th>
<th>Viable Premature Foetus</th>
<th>Immature Foetus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biologic Periods</td>
<td>Embryonic Period</td>
<td>Foetal Period</td>
<td>Neo-natal Period</td>
<td></td>
</tr>
<tr>
<td>Fertilization Age-weeks</td>
<td>2 4 6 8 10 12 14 16 18 20 22 24 26 28 30 32 34 36 38 40 42</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Menstrual Age-weeks</td>
<td>2 4 6 8 10 12 14 16 18 20 22 24 26 28 30 32 34 36 38 40 42 44</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Menstrual Age-Lunar Months</td>
<td>1 2 3 4 5 6 7 8 9 10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Menstrual Age-Calendar Months</td>
<td>1 2 3 4 5 6 7 8 9 10</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trimesters</td>
<td>First</td>
<td>Second</td>
<td>Third</td>
<td></td>
</tr>
</tbody>
</table>

(Source: Developed from Valdes-Dapena and Huss, 1983: 5)

6 Gestational ages and intervals are measured, usually in completed weeks, from the first day of the last normal menstrual period (World Health Organization, 1967-69, 1977-78).
Consequently, the Ninth Revision of the International Classification of Diseases (1977-78), following the Ninth Revision Conference (1975), recommended that a minimum birthweight of 500 grams be made the primary criterion for including foetuses and newborn infants, whether alive or dead, in national perinatal statistics. When the birthweight was unavailable, the corresponding gestational age (22 completed weeks) or the body-length (25 centimetres crown-heel) was to suffice. The perinatal period was to extend up to the end of the seventh completed day of infant life, thus aggregating stillbirths and early neonatal deaths. These recommendations, among others, were adopted by the 27th World Health Assembly, but comparisons of perinatal events have continued to be affected by definitional differences.

In Australia, for example, national perinatal statistics have always included late neonatal deaths. Yet these statistics have incorporated the other recommendations of the Ninth Revision which has been less true of New South Wales's own separate publications of perinatal statistics since 1969. This State has applied to stillbirths a minimum birthweight criterion of 400 grams or a gestational age of 20 weeks, and to neonatal deaths no criterion of

---

7 The birthweight is the first weight of the foetus or infant obtained after delivery. It should be measured within the first hour of life, before significant postnatal weight loss occurs.

8 These criteria define the viability of the foetus or infant; that is its theoretical ability to survive extraterine life.

9 For statistical purposes, a stillbirth is the birth of a foetus weighing at least 500 grams and showing no evidence of life at birth. Evidence of life would include breathing, a heart beat, pulsation of the umbilical cord or definite movement of the voluntary muscles. The Ninth Revision Conference recommended that the term stillbirth be replaced with the term foetal death. But since, strictly speaking, the foetus vis-à-vis the embryo exists from at most ten weeks, whereas by definition a stillbirth cannot take place until usually 22 weeks, the term stillbirth is retained in this study.

10 An early neonatal death relates to a liveborn infant weighing at least 500 grams at delivery (or when the birthweight is unavailable being of at least 22 weeks' gestation) who dies before seven completed days of infant life.

11 A late neonatal death relates to a liveborn infant weighing at least 500 grams at delivery (or, when the birthweight is unavailable, being of at least 22 weeks' gestation) who dies after seven, but before 28, completed days of infant life.
viability other than live birth followed by death within 28 days of
this birth (see section 3.4). These different practices have
complicated comparisons of the statistics reported by New South Wales
with those prepared at the national level, whilst also contributing,
at least in the Hunter Region of New South Wales, to the under­
reporting of babies weighing less than 500 grams\textsuperscript{12} (Lawson, 1987; and
see section 1.3).

Fortunately, in all countries, under-reporting of birthweights up to
1000 grams dates only to the World Health Organization’s (1975)
revision of criteria for including conceptuses\textsuperscript{13} in perinatal
statistics. Serving also to undercount stillbirths has been the
practice when calculating rates, of applying a lower limit of maturity
to stillbirths but not to all live births\textsuperscript{14} (World Health Organization, 1977; United Nations, 1987). However, attenuating this problem has
been the likelihood that some neonates, dying before their births are
registered, have been reported as stillbirths rather than as live
births and neonatal deaths.\textsuperscript{15}

To help resolve these problems, the Ninth Revision Conference
recommended that countries prepare solely for international
comparisons ‘standard perinatal statistics’. These statistics were to
be restricted to foetuses and infants weighing at least 1000 grams
(or having the corresponding gestational age of 28 weeks or
body-length of 35 centimetres crown-heel) for both the numerators and
denominators of all rates, and to infants dying at ages under seven
completed days (World Health Organization, 1977).

\textsuperscript{12} Ignorance of the State definition has been partly responsible, but also
problematic has been defining perinatal events using a minimum gestational
age.
\textsuperscript{13} The term conceptus relates here to viable products of conception.
\textsuperscript{14} See footnote 9 for a definition of life at birth.
\textsuperscript{15} The sum of early neonatal deaths and late neonatal deaths. See footnotes
10 and 11.
Broad comparisons of perinatal statistics have usually been possible, the concept of a perinatal outcome having helped to resolve problems of incomplete registration and geographic differences in defining vital events. This is because it overcomes different legal requirements and administrative practices for setting 'signs of life' criteria, solving the problem of deliberate or inadvertent misclassification of perinatal deaths as either stillbirths or neonatal deaths.

However, problems have remained in distinguishing spontaneous abortions from stillbirths. For instance, stillbirths may be miscounted as abortions to avoid registration of the death and burial of the body; although in Japan, for instance, foetal deaths after twelve weeks of pregnancy must be reported (Kondo, 1985). Also, abortions may be registered as stillbirths if the foetus being viable entitles receipt of a maternity benefit (Edouard, 1985), or if the registration is welcomed as a tangible reminder of the baby's existence (Condon, 1987 and see section 3.7.2).

Of course, separating out the gestational periods, stages and ages that define perinatal events can be important. This permits consideration of components' distinctive epidemiological features, as well as those defining their essential commonality as perinatal phenomena.

1.2 INTERNATIONAL PERINATAL AND POSTPERINATAL STATISTICS

Three measures of success in reducing perinatal and postperinatal mortality since the early-mid 1950s are examined for selected countries in the developed world. These measures are change over time.

16 The term spontaneous abortion or miscarriage relates to the expulsion from the uterus of an embryo or a nonviable foetus.
in perinatal death ratios, in postperinatal death ratios and in the proportion of all perinatal and postperinatal deaths occurring during the perinatal period. Cross-country differences in the percentages of total live births occurring at low birthweights are suggested to account in part for these changes in perinatal and, to a lesser extent, postperinatal mortality.

1.2.1 PERINATAL DEATH RATIOS

Figure 1.1 shows perinatal death ratios from 1952 to 1985 for five developed countries: Australia, Canada, the Federal Republic of Germany, Japan and Sweden. Following Shryock and Siegel (1981), the perinatal death ratio is calculated as the number of perinatal deaths per 1,000 live births. This measure is preferred here to the perinatal death rate (see section 1.3), which is defined as the number of perinatal deaths per 1,000 total births (live births plus stillbirths) all registered in the same year. This preference is due to the United Nations Statistical Office having prepared perinatal death ratios for most countries, including those selected. The numerator in these ratios is the sum of foetal deaths of at least 28 weeks’ gestation, including deaths at unknown gestational ages, and infant deaths in the first week of extrauterine life.

The use of live births rather than total births in the denominator is of little consequence since the numerator is much the more sensitive to change (although see section 1.3). Only the stillbirth component, that is, neither the early neonatal death component of the numerator of perinatal deaths nor the denominator of live births, is restricted

17 In fact, the Statistical Office reports perinatal death ratios only for countries reporting more than 1,000 perinatal deaths in a given year. However, numbers of perinatal deaths below this threshold are still reported, so that the researcher was still able to calculate ratios as, for example, for Sweden since 1977.
to foetuses and infants weighing at least 1 000 grams at birth - as recommended by the Ninth Revision Conference for international comparisons. However, this problem would not be resolved by the calculation of rates; it reflects the type of data available.

Specifically, the named countries were selected on the basis of four criteria for international comparisons: appropriateness as units of analysis, completeness, coverage, and uniformity of measurement over the stated time period. The appropriateness of the countries as units of analysis is the most difficult criterion to define, however, following Parsons and Shils (1951), an ideal-typical index seems appropriate. That is, throughout the period the countries furnished reliable perinatal data exemplifying national success in reducing levels of perinatal mortality. Moreover, these areas were broadly representative of other developed countries. For instance, each population comprised many millions of people who operated a complex social system including distinct urban and rural subareas (Hartford, 1985).

![Figure 1.1 Perinatal Death Ratios in Selected Countries, 1952-85](Source: United Nations, Demographic Yearbook, 1962-87)
Data from civil registers were assessed for completeness by National Statistical Offices (United Nations, 1962-87). Data were estimated to be at least 90 per cent complete for Canada, the Federal Republic of Germany and Japan over the total time period, and for Australia since 1976. Before 1976, Australia gave no specific information regarding the completeness of its data (United Nations, 1962-87), but sources within Australia (e.g., Australian Bureau of Statistics, 1953) generally confirm, for example, the completeness of stillbirth notifications. Even in Queensland and possibly in Tasmania, where notification of stillbirths was not made compulsory until 1959 and 1966 respectively, substantial under-reporting was unlikely. This is reassuring because 'since foetal deaths are less completely registered than infant deaths the reliability of perinatal deaths is equal to or better than the quality of foetal death registration' (United Nations, 1987:75). A minor limitation of Australian data has, however, been the practice of tabulating events by their year of registration rather than of occurrence.

The United Nations applied no quality codes to Swedish perinatal data since they did not come from civil registers. The data were derived from special hospital records which have formed the basis of an efficient and highly reliable medical birth registration system (Karlberg, 1985; Meirik and Lindmark, 1985).

Incomplete data for live births and infant deaths were not a serious problem for the selected countries. However, in Japan until at least the late 1970s, first-day infant deaths were classified as foetal deaths because the clinical application of definitions was not uniform (World Health Organization, 1977; Hartford, 1885).

The criterion of coverage relates to the inclusion of all population subgroups within each country in the enumeration system. A few
deviations from complete coverage are unlikely to have had a noticeable effect. Among minor changes in Japanese territorial coverage was the exclusion of Okinawa before 1973; and data for Japan related to Japanese nationals in Japan only, in contrast, for instance, to Canadian statistics which also included Canadian residents temporarily in the United States. Australian perinatal figures before 1966 failed to include full-blooded Aboriginals. Although this subgroup was small in number, its members have always borne a much higher mortality risk than have white Australians (Bates and Linder-Pelz, 1987). Figures relating to the Federal Republic of Germany included the relevant data for West Berlin, for which separate data are not supplied.

Figure 1.1 shows, for all five countries, a steady decline and accompanying convergence of perinatal death ratios from 1952 to 1985. Sweden, which represents Scandinavia's success in preventing perinatal deaths, recorded the lowest ratios throughout this period. Indeed by 1978 Sweden had achieved a level of perinatal mortality lower than the Australian level five years later. By 1985, the perinatal death ratio in Sweden was 7.3 per 1,000 live births, which raises the question of an absolute lower limit beyond which perinatal mortality cannot fall.

Despite the remarkability of the Swedish ratios, perhaps most exceptional were the experiences of Japan and, to a slightly lesser extent, of the Federal Republic of Germany. In both countries, the startling feature was the magnitude of the fall over the period 1952-85 in perinatal death ratios: 83.1 per cent in Japan, and 78.3 per cent in the Federal Republic of Germany. Japan was the slower of the two countries to begin its substantive decline, but sustained decreases, especially after the early 1960s, yielded a ratio of 7.7 per 1,000 by 1985. The Federal Republic of Germany, despite
experiencing periodic fluctuations in its rate of decline, attained
the respectable ratio of 10.6 per 1 000 by 1984.18

Australia and Canada began the period with ratios approximating 30 per
1 000, and a third of a century later had levels of perinatal
mortality 69.4 per cent and 75.6 per cent lower respectively. The
decreases were most erratic for Australia, the data problems described
above possibly accounting for the very temporary fluctuations of 1958,
1964 and 1972.

**Distribution of low birthweights among live births:** These differences
in perinatal mortality should, in large part, be explained by
variations in the prevalence of low birthweights (Stubblefield, 1984)
and, less superficially, by the factors underlying birthweight
derivatives among the five countries. In terms merely of
differences in birthweight, Figure 1.2 presents for Canada, the
Federal Republic of Germany, Japan and Sweden, percentages of all live
births occurring during 1972-1985 in different categories of low
birthweights. This information has not been collected nationally in
Australia, but it has been collected, for example, in New South Wales
since 1975, so that this State is also represented in Figure 1.2.

However, the birthweight categories applied to the countries differ
slightly from those used for New South Wales. The birthweights for
the countries are classified by the United Nations Statistical Office
according to the Eighth Revision of the International Classification
of Diseases (for example 501 to 1 000 grams). The birthweight
groupings used for New South Wales are those recommended by the Ninth
Revision Conference (for example, 500 to 999 grams), which are also
used subsequently in this study (for example, see Table 3.0).

18 The 1985 figure is not available.
Figure 1.2
Percentages of Total Live Births Occurring at Low Birthweights,\(^1\) in Selected Regions, 1972-85

1 Low birthweights for New South Wales are classified according to the Ninth Revision (1977) of the International Classification of Diseases; that is, 500-999 grams, 1 000-1 499 grams, 1 500-2 499 grams.

2 Note that the graph for birthweights 1 501-2 500 grams uses a different vertical scale from the graphs for birthweights 501-1 000 grams and 1 001-1 500 grams.

This difference can create problems. As shown by Macfarlane and Mugford (1984) for total births in England and Wales during 1980, 'digit preference' for round numbers can lead to slightly higher proportions of low birthweights (and indeed birthweights less than 3000/3001 grams) being recorded according to the Eighth compared to the Ninth Revision. Applying the results of their study to the data grouped in Figure 1.2, suggests that the bias may be greatest at birthweights '1 501 to 2 500 grams'. Yet the key point is that the percentages for New South Wales would be slightly higher than those reported if the classification system of the Eighth Revision rather than the Ninth Revision had been used. Conversely, the countries' percentages would be reduced a little by classification according to the Ninth rather than the Eighth Revision.

A second caveat relates to the exclusion of foetal deaths before 28 weeks from the perinatal death ratios discussed in section 1.2.1. Since almost all these omitted babies are likely to have birthweights of 1 000 grams or less, the data for extremely low birthweight babies (501 to 1 000 grams) in Figure 1.2 can account for very little of the foetal component of the perinatal death ratios in Figure 1.1. However, the discussion of extremely low birthweights (501 to 1 000 grams) should retain some relevance to the perinatal death ratios because the early neonatal component of these ratios does not invoke any birthweight criterion (see section 1.2).

Figure 1.2 shows that during the complete period, 1972-85, Japan recorded the lowest percentages of births at 501 to 1 000 grams, remarkably just 0.06 per cent in 1972 before rising to 0.15 per cent in 1985, and at 1 001 to 1 500 grams. These low percentages may

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19 The birthweight categories are represented in Figure 1.2 as 501 to 1 000 grams, 1 001 to 1 500 grams and 1 501 to 2 500 grams respectively, even though the corresponding birthweight categories for New South Wales are 500 to 999 grams, 1 000 to 1 499 grams and 1 500 to 2 499 grams respectively.
reflect genetic factors, the exceptional growth of the Japanese economy, and Japan's significant reliance on induced abortion as a method of fertility control. The latter development may help to account for the low proportion of infant deaths in Japan that have been certified as due to congenital anomalies (Macfarlane and Mugford, 1984). Sweden recorded the next lowest percentages of both birthweights until 1983, when Canada recorded a lower percentage of infants weighing 1 001 to 1 500 grams. The relative difference in the percentages for Japan and Sweden was greatest at birthweights 501 to 1 000 grams.

Japan's continued success in lowering its perinatal mortality is likely, therefore, to reflect in part, at least from the early 1970s, this country's comparatively small percentage of babies weighing 1 500 grams or less. Why Sweden maintained even lower levels of perinatal mortality is suggested by its low percentages at birthweights 501 to 1 000 and 1 001 to 1 500 grams, and its very low percentages at birthweights 1 501 to 2 500 grams. At these last birthweights, which are much less hazardous but considerably more numerous than birthweights of 1 500 grams or less, the percentages for Sweden, which averaged about 3.6 per cent, were about three-quarters of those for Japan. Indeed the percentages of birthweights 1 501 to 2 500 grams for Japan, which were the second lowest of those reported by the five regions for most of the 1970s, increased slightly after 1979 to become the highest for these regions by 1985. Increases in percentages were also noted for Japan and, particularly in recent years, Sweden at birthweights 1 500 grams and below.

Comparison of the experiences of Canada, the Federal Republic of Germany and New South Wales reveals, first, that for most of the period, Canada recorded the highest percentages of births at
birthweights below 1 001 grams and, of the three regions, the Federal Republic of Germany recorded the lowest percentages. Stability characterized the percentages for these countries, between which the figures for New South Wales tended to fluctuate.

At birthweights 1 001 to 1 500 grams, the percentages for the three regions were close together, and so small changes magnified their distributional differences in birthweight. The highest percentages of live births at birthweights 1 001 to 1 500 grams were recorded by the Federal Republic of Germany until 1977 and in 1984-85, but for most of the intervening period by New South Wales.

At birthweights 1 501 to 2 500 grams, percentages of births were conspicuously higher in Canada than in the other two regions until 1981, even though the perinatal death ratios depicted in Figure 1.1 were consistently lower in Canada. The experience of New South Wales is interesting particularly after 1978, from which time, except for 1979 and 1983, only Sweden recorded lower percentages of live births at birthweights 1 501 to 2 500 grams.

1.2.2 POSTPERINATAL DEATH RATIOS

Improvements in perinatal outcomes are viewed in the context of parallel efforts to prevent postperinatal deaths; that is, infant deaths after seven completed days of life. Thus postperinatal death ratios, calculated as postperinatal deaths per 1 000 live births, are depicted in Figure 1.3 for the same five countries represented in Figure 1.1 for the period 1952-1985.

Throughout this period, postperinatal death ratios were lower in each country than were perinatal death ratios. However, from the start of the period, in response partly to the birthweight distributions just
described and more importantly to falling mortality from infectious diseases, postperinatal death ratios continued to fall, except in the Federal Republic of Germany during the late 1960s and early 1970s. This widespread decline, which was greatest in Japan and then Canada, slowed with the achievement of low levels of postperinatal mortality (less than about 10 per 1,000) in all five countries by the mid 1960s. Japan continued to show the greatest improvements.

After recording the highest postperinatal death ratios until 1966, Japan achieved the lowest levels of the five countries from 1982 onwards. Indeed by 1970 Japan had achieved a lower ratio of postperinatal death than the lowest 1985 perinatal death ratio, 7.3 per 1,000 in Sweden, shown in Figure 1.1. The Federal Republic of Germany achieved impressive reductions in postperinatal mortality until 1967, but its progress then remained disappointing: after 1970, it recorded the highest ratios of the five countries being studied.

Figure 1.3
Postperinatal Death Ratios in Selected Countries, 1952-85

(Source. United Nations, Demographic Yearbook, 1962-87)
Sweden stands out as the nation that maintained the consistently lowest postperinatal death ratios - less than 4.0 per 1,000 since 1964, yet the Australian example is also impressive. Australia's ratios stayed only slightly higher than Sweden's, showing a small but, relative to Sweden, parallel decline over time.

1.2.3 DISTRIBUTION OF PERINATAL DEATHS AMONG COMBINED PERINATAL AND POSTPERINATAL DEATHS

All these statistics, although helpful, do not show change in levels of perinatal mortality relative to change in levels of postperinatal deaths. Yet this is needed to compare differences in the timing and pace of the declines. For this reason, Figure 1.4 shows, for the same five countries, historical change in the proportion of perinatal deaths among all perinatal and postperinatal deaths. Although it is difficult to generalize the experiences of these countries, broad cycles of change can be identified.

First, from 1956 until the mid-late 1960s, proportions of perinatal deaths tended to increase, mainly in Japan, Canada and the Federal Republic of Germany: improvements in survival after the first week of life surpassed those associated with the perinatal period. Of the countries represented in Figure 1.4, Japan recorded easily the largest increase: 15 percentage points between 1956 and 1967. Substantive changes are not apparent for Australia and Sweden.

Around 1970, except in Japan, there emerged a second pattern of decreasing proportions of perinatal deaths, the decline being especially marked in the Federal Republic of Germany and in Sweden. This pattern indicates slowing of the decline in postperinatal death ratios as they became progressively low relative to the range of
values they had historically assumed, and continuing gains in lowering perinatal mortality.

1.3 AUSTRALIAN PERINATAL DEATH RATES BY STATE OVER TIME

Perinatal death ratios give stability to international comparisons, but perinatal death rates, which express the number of perinatal deaths per 1 000 total births (see section 1.2.1), are preferred in Figure 1.5 for depicting perinatal mortality changes in selected Australian States from 1936 to 1986. This is because the calculation of perinatal death rates has become standard practice in Australia. In fact, the idiosyncratic reporting of perinatal statistics by individual States and Territories has made necessary the use of annual rates published nationally, and hence uniformly, for each region; this has been done, in accordance with the recommendations of the Ninth Revision Conference, since 1975.

Figure 1.4
Perinatal Deaths as a Proportion of Perinatal and Postperinatal Deaths in Selected Countries, 1952-85

Figure 1.5
Perinatal Death Rates for Australian States, 1936-86

(State
- N.S.W.
- Queensland
- S. Australia
- Tasmania
- Victoria
- W. Australia)

Although of little consequence, perinatal death rates are also slightly less susceptible to bias in their denominators than are perinatal death ratios. Thus, notwithstanding that both of these measures are more prone to inaccuracy in their numerators than in their denominators, it is worth mentioning the refinements of denominator values achieved by the use of rates. The first of these advantages occurs because the definition of perinatal death in Australia differs from the World Health Organization recommendation on which the foregoing discussion was based (see section 1.1). Specifically, including late neonatal deaths in the perinatal period increases the need to accommodate all 'at risk' individuals in the denominator as well as in the numerator; although late neonatal deaths are much fewer than late foetal and early neonatal deaths, which are themselves rare events. Secondly, perinatal death rates have the small theoretical advantage over perinatal death ratios, although it is of no especial benefit here, of approximating probabilities more closely (Shryock and Siegel, 1981).

It is true that the denominator in perinatal deaths rates is more susceptible to incomplete reporting especially of stillbirths than is the denominator in the perinatal death ratios, as previously defined. However, under-reporting of stillbirths has not been a problem for State notifications in Australia, and the bias would be small anyway for such rare events as stillbirths.

Nevertheless, some difficulties in comparing perinatal statistics for different States are not remedied by the use of rates. As noted above, perinatal statistics are available for the period since 1975 that accord with the recommendations of the Ninth Revision Conference of that same year. But statistics for earlier years are based on Numerators are normally smaller than denominators, and are often subsets thereof.
different definitions of perinatal events. Until 1969, reported statistics for stillbirths related to infants not born alive after at least the 28th week or, as in New South Wales, the seventh month of pregnancy. Statistics for neonatal deaths related to any liveborn child dying within 28 days of its birth. Since 1969 in New South Wales, and for a brief period nationally during the early 1970s, perinatal statistics have been reported only in accordance with amended legislation, introduced in all States and Territories between 1964 and 1969 to provide for the registration of stillbirths of at least 20 weeks' gestation or 400 grams birthweight. In sum, perinatal statistics reported before and since 1975 are not strictly comparable, although historical differences between the regions still should be.

Against this background, two features emerge from Figure 1.5. First, State perinatal death rates fell by approximately 60 per cent from 1936 to 1974, while from 1975 to 1986, the reduction was at least one-third in all States except Tasmania (where rates fell by 28.0 per cent). The apparent decline in most States during 1936-86 was about 80 per cent, in Tasmania the reduction being less impressive at 73 per cent. Secondly, fairly small differences in rates occurred among the States from the mid 1940s. Nevertheless, certain States dominated particular periods with relatively low or high rates.

Two States in particular stand out for their low rates of perinatal death. First, excluding the years 1966-67, Tasmania recorded the lowest rates of any Australian State from 1960 to 1972. This is remarkable given Tasmania's high rates less than a decade earlier. Secondly, South Australia maintained an excellent record of reducing perinatal death rates. Along with Western Australia, it dominated improvements from 1938 until 1946. It also made its presence felt thereafter until 1974 without ever dominating in the manner of earlier
years, and since 1975, has been the State most often recording the lowest rate of perinatal deaths. Only New South Wales and Queensland have never achieved the lowest rate in any year.

Also interesting are the States reporting the poorest perinatal death rates. Conspicuous by its high rates until 1954 was Tasmania, while from 1955 to 1981, New South Wales and Queensland recorded the highest death rates nineteen and seven times respectively. In the twelve years from 1975 to 1986, when rates are directly comparable, Tasmania and New South Wales recorded the highest rates of perinatal death seven and four times respectively. In New South Wales, these comparatively high rates, although only marginally so, have been compounded by this State having the largest population and hence always recording the most perinatal deaths in Australia. By contrast, in Tasmania, the smallest Australian State, recently relatively high rates have reflected comparatively few deaths.

1.4 UNDERSTANDING THE RECENT CHANGE IN PERINATAL MORTALITY

The foregoing discussion exemplifies the successes during recent decades of developed countries, including Australia, in reducing their levels of perinatal mortality (Foster, 1981; Grant and Lapsley, 1985; Australian Bureau of Statistics, 1987). The reasons for this success need to be explored because they affect current and future research directions. In sections 1.4.1 and 1.4.2, the decline in perinatal mortality is related to sociodemographic changes, which are examined specifically for Australia, and to general medical improvements.

1.4.1 SOCIODEMOGRAPHIC TRENDS AND PERINATAL MORTALITY IN AUSTRALIA

The sociodemographic factors that might have contributed to change in perinatal mortality rates are many and varied. They include
improvements in women's status, associated, for example, with increases in female education and improved work conditions for women; although, to the extent that progress remains to be achieved, these areas can still be viewed as posing risks for perinatal death (see chapter 2). Change in fertility is a more distinctively demographic phenomenon attending the overall decline in perinatal mortality. This relationship is now discussed using Australian data, but the results are likely also to apply in other developed countries.

Table 1.0 shows that Australia's perinatal death rate declined by 7.5 per 1 000 after 1975 to reach 11.1 per 1 000 in 1986, and that these rates were affected only minimally by change in the ages of childbearing during this period. Standardizing to the 1975 distribution of births by maternal age, the 1986 perinatal death rate would have been just 0.2 per 1 000 higher had the distribution of births by maternal age not changed.

Table 1.0: Age-Specific Perinatal Death Rates, and Perinatal Death Rates Unstandardized and Standardized for Maternal Age (Per 1 000), 1975-86

<table>
<thead>
<tr>
<th>Year</th>
<th>Perinatal Death Rates</th>
<th>Age-Specific Perinatal Death Rates Maternal Ages (Years)</th>
<th>Total Low-Risk1 Ages</th>
<th>Total High-Risk Ages</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Unstd.</td>
<td>Std.</td>
<td>&lt;20</td>
<td>20-24</td>
</tr>
<tr>
<td>19752</td>
<td>18.6</td>
<td>18.6</td>
<td>22.1</td>
<td>17.9</td>
</tr>
<tr>
<td>1976</td>
<td>18.3</td>
<td>18.4</td>
<td>22.0</td>
<td>16.4</td>
</tr>
<tr>
<td>1977</td>
<td>16.1</td>
<td>16.1</td>
<td>20.9</td>
<td>14.8</td>
</tr>
<tr>
<td>1978</td>
<td>15.8</td>
<td>15.9</td>
<td>20.1</td>
<td>14.8</td>
</tr>
<tr>
<td>1979</td>
<td>14.6</td>
<td>14.7</td>
<td>18.3</td>
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<td>13.7</td>
<td>13.7</td>
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<tr>
<td>1986</td>
<td>11.1</td>
<td>11.3</td>
<td>14.4</td>
<td>11.3</td>
</tr>
</tbody>
</table>

1 The total low-risk ages are 20-34 years. The total high-risk ages are <20 and >35 years.
2 The standard is the 1975 age distribution.
The data needed to calculate 1952-72 perinatal death rates standardized for the maternal age distribution of total births are not available, and the data for 1973-74 are not used because they incorporate definitions that differ from those pertaining to subsequent years. However, if changes in definitions before and after 1975 are assumed not to exist, which should not bias results too greatly given the relative stability of trend lines for individual States in Figure 1.5, it is possible to estimate how much of the 1952-1975 change in overall perinatal death ratios, as defined previously, resulted from change in the distribution of live births by maternal age. The perinatal death ratio in 1975 was 20.2 per 1000, whereas, standardizing to the 1952 distribution of live births by maternal age, it rises to 21.4 per 1000. Given a 1952 perinatal death ratio of 34.0 per 1000, this again indicates the very small extent to which the decline in perinatal mortality resulted from change in the maternal age distribution of live births. Of a total decline of 13.8 per 1000 (34.0 minus 20.2), only 1.2 per 1000 (8.7 per cent) could be attributed to this factor.

However, this exceeds Khawaja's (1970) estimate that 6.6 per cent of the decline in perinatal mortality between 1935 and 1966 was attributable to change in the distribution of births by maternal age. This earlier study found that change in the distribution of births by parity, and by age and parity together, respectively accounted for 10.8 and 12.9 per cent of the decline in perinatal mortality during the same period, 1935-66.

Returning to the present analysis, change in the maternal age distribution of childbearing therefore also contributed comparatively little to the overall decline in perinatal mortality 1952-86. Yet it is important also to know the extent to which such change in the
distribution of childbearing affected perinatal mortality in different maternal age groups, or affected at least the maternal age distribution of perinatal deaths.

Age-specific perinatal death rates for single ages are needed to answer the former question using direct standardization for women aged, say, less than 20 years. These data are not available, but it is possible to determine the extent to which change in the distribution of perinatal deaths by maternal age from 1975 to 1986 resulted from change in the distribution of births by maternal age and from change in the relativities between perinatal mortality rates in the age group of interest and all other reproductive age groups. For individual years, this has been done by adjusting the percentage of total perinatal deaths occurring to women in different reproductive age groups by the ratio of the proportion of total births to women in an age group in the base year (1975) and in the year of interest.

The resultant adjusted and unadjusted percentages for each reproductive age group are portrayed graphically in Figure 1.6, with a horizontal line being added through the base year plot. This last line may (a) represent no change either in the distribution of births by maternal age or in the level of perinatal mortality in a given maternal age group relative to levels in all other reproductive age groups, or (b) indicate that these components cancel out, as may only occur when the unadjusted trend line touches the horizontal line but the adjusted trend line does not also do so.

Deviations of the unadjusted trend line from this horizontal line indicate actual change in the contribution of a specified age group to total perinatal mortality. Deviations of the adjusted from the unadjusted trend line indicate how much of the actual change resulted from shifts in the maternal age distribution of births. Finally,
Figure 1.6
Percentages of Australian Perinatal Deaths Occurring at Different Maternal Ages, 1975-86, Adjusted for the 1975 Maternal Age Distribution of Australian Total Births

deviations of the adjusted line from the horizontal line indicate how much of the actual change was due to change in the perinatal mortality rate for the specified maternal age group relative to change in rates for all other reproductive age groups. Table 1.0 aids in the interpretation of change in these relativities of perinatal mortality rates between maternal age groups.

However, before describing change over time in the described percentage distributions, Figure 1.6 shows that the percentage distribution of perinatal deaths by maternal age takes an inverse U shape. Perinatal death least commonly involves adolescents (less than age 20 years) and mature women (35 years of age and over) who collectively accounted for 20.8 per cent and 18.9 per cent of perinatal deaths to women of known age in 1975 and 1986 respectively. This is because there are comparatively small proportions of births to women at adolescent and mature ages, notwithstanding that these ages yield the highest rates of perinatal death as shown in Table 1.0. The safest ages for childbearing are 25-29 years, while most hazardous are the ages 40 years and over: the age-specific perinatal death rates for these two groups in 1986 were 9.9 per 1 000 and 23.9 per 1 000 respectively. Rates for the total group of women at the ages at highest risk of perinatal death, namely less than 20 years and 35 years and over, were about 1.5 times those for women aged 20-34 years from 1975 to 1986.

Against this background is shown change over time in the distribution of perinatal deaths by maternal age. Figure 1.6 shows that unadjusted percentages of all perinatal deaths occurring to women aged less than 20 and women aged 20-24 years declined between 1975 and 1986. However, again, when 1975 is taken as the base year, these declines are demonstrated to have resulted from a declining proportion of
births to women in these age groups after 1975, and particularly after 1982.

Without this change, the unadjusted percentage of all perinatal deaths occurring to women aged less than 20 and 20-24 years would have increased marginally between 1975 and 1986. Furthermore, the declines in the unadjusted percentages of perinatal deaths occurring in these early reproductive age groups would have been even greater had not perinatal mortality rates for both age groups shown an overall slight increase relative to perinatal mortality rates at all other maternal ages. Table 1.0 reveals that this increase occurred because perinatal mortality rates fell less strongly at maternal ages less than 24 years than at older ages, specifically 30 years and over.

Among women aged 25-29 years, the unadjusted and adjusted percentages fluctuate yet stay fairly close together. Nevertheless, before adjustment, increases are noted in the relative contributions to perinatal death of this maternal age group, and of maternal ages 30-34 and 35-39 years. These increases resulted in part among 25-29 year-olds and among 30-34 and 35-39 years-olds from increases in the proportions of total births occurring to women at these ages. Adjustment for these increases yields percentages of perinatal deaths that rise less strongly at ages 25-29 years from 1974 to 1986, and that actually fall at maternal ages 30-34 and 35-39 years.

Increases in the unadjusted percentages of all perinatal deaths occurred in the last two groups despite perinatal death rates falling more strongly in these groups than at most other ages. At ages 25-29 years, increases occurred because the perinatal death rates for this age group increased marginally over the full period 1975-86 relative to the change in rates at all other reproductive ages. In practical
terms, these rates fell a little more slowly at ages 25-29 years than at other ages, although fluctuations distort this trend.

Women aged 40 years and over contributed decreasing unadjusted percentages of perinatal deaths for most of the period 1975-86. This was due to a decline in the proportion of all births occurring to these women until 1982, and to perinatal death rates falling more strongly at these oldest ages than at other reproductive ages. The former factor dominated until 1981, the latter thereafter. Because, in this mature age group, the decline in unadjusted percentages of perinatal deaths exceeded the fall in proportions of births, adjusted percentages of perinatal deaths tended likewise to fall.

This discussion of individual age groups helps to explain why, overall, changes in the distribution of births by maternal age had very little effect on total perinatal mortality rates during 1975-86 (see Table 1.0). The distribution of births changed quite a lot at some ages, but these changes tended to cancel out. In particular, a declining proportion of births at ages less than 25 years and an increasing proportion of births at ages 30-39 years between 1975 and 1986 offset each other. A de-emphasizing of one high-risk age group, adolescent women, and one low-risk age group, women aged 20-24 years, was counterbalanced by increased emphasis on second high-risk, 35-39 years, and low-risk, 30-34, maternal age-groups.

The overall shift described can also be observed from change in percentages of perinatal deaths at the maternal ages at lowest risk (20 to 34 years) and highest risk (below 20 years, and 35 years and above) of this outcome (see Figure 1.7). During 1975-86, the

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21 However, the conspicuous decline for 1985 in the relativities between rates at ages 40 years and over and all other ages is an artefact of small numbers exacerbated by abnormal delays in the registration process in New South Wales.
unadjusted percentage of perinatal deaths occurring at low-risk ages tended to increase while the percentage occurring at high-risk ages tended, therefore, to fall. However, this pattern is less clear after adjustment for an increase in the proportion of total births occurring to women aged 20-34 years and for a decrease in the proportion occurring to women at higher-risk ages.

Adjusted percentages still rise at low-risk ages and fall at high-risk ages, owing to perinatal death rates falling at high-risk ages relative to the change in these rates at low-risk ages, but the extent of these shifts in adjusted percentages of perinatal deaths is diminished. Indeed, the adjusted trend lines for the two age groups show little deviation from the 'no change' situation, indicating convergence of the relativities between perinatal mortality rates at high-risk and low-risk maternal ages.

Figure 1.7
Percentages of Australian Perinatal Deaths Occurring at High-Risk and Low-Risk Maternal Ages, 1975-86, Adjusted for the 1975 Maternal Age Distribution of Australian Total Births

Influence of fertility rates and age structure: Two questions now arise which are answered by adopting a similar approach to that taken above. The questions are, to what extent were changes in the maternal age distribution of births the result of movements in fertility per se rather than of shifts in the age structure of women of reproductive age? And, how can changes in fertility in different age groups be related back to total and maternal age-specific perinatal mortality rates during 1952-86?

To help answer the former question first, Figure 1.8 shows percentages of all live births occurring to women of reproductive age in different age groups from 1952 to 1986 before and after adjustment for change in the age structure since the base year of 1952. The adjustment factor is the ratio of the proportion of women of reproductive age in a given age group in 1952 and in the year being studied. In Figure 1.8, horizontal lines are again drawn, this time through the 1952 plots, to indicate (a) no change either in age structure or in the relativities between fertility rates for the age group under scrutiny and all other reproductive age groups, or (b) that these components cancel out. The age-specific fertility rates displayed in Figure 1.9 aid the interpretation of Figure 1.8.

According to Figure 1.8, unadjusted percentages of all births occurring at ages less than 20 years and 20-24 years increased until the early 1970s, and then fell. The increases resulted, first, from increased proportions of adolescent women of reproductive age after 1952, and 20-24 year-olds after the mid 1950s. Indeed, increased proportions of 20-24 year-olds from 1964 account almost entirely for attendant changes in the distribution of births. Adjustment for these changes in age structure yields percentages of births that, overall, from 1952 until the early 1970s, rise less markedly at maternal ages
Figure 1.8
Percentages of Australian Live Births Occurring at Different Maternal Ages, 1952-86, Adjusted for the 1952 Reproductive Age Structure of Australian Women

24 years and below.

With reference to this same period, increases in unadjusted percentages of births resulted, secondly, from rises in adolescent fertility rates from 1962 to 1973, and fertility rates among 20-24 year-old women principally during the 1950s, relative to the changes in fertility rates at all other reproductive ages. Figure 1.9 helps to explain this shift in the relativities between rates. For adolescents, fertility rates increased at ages 15-19 years until 1971, but generally fell at other ages after 1961. Relative to increases in fertility among women aged 20-24 years 1952-61, fertility rates, at ages other than 25-29 years, showed minimal change during the 1950s.

Figure 1.9
Age-Specific Fertility Rates for Australia, 1952-86

![Age-Specific Fertility Rates for Australia, 1952-86](image)


22 Fertility rates are expressed both for 15-19 and 40-44 year-olds rather than, as with reported percentages of births, respectively for women aged less than 20 years, and 40 years or more. This is done out of convenience since, for practical purposes, the two measurements for each age group are broadly equivalent.
Falling unadjusted percentages of all births occurring to women aged 15-24 years since the early 1970s resulted from fertility rates falling at maternal ages 15-19 years after 1973, and at ages 20-24 years after 1976, relative to the change in fertility rates at all other reproductive ages; this is most salient at ages 20-24 years during the 1980s. As shown by Figure 1.9, this change in the relativities between fertility rates for women aged 15-24 years and all other reproductive age groups resulted from two sets of forces: between 1971 and 1976, fertility rates declined more rapidly at maternal ages 15-24 years than at other reproductive ages. Thereafter, until 1986, fertility rates continued to decline at ages 15-24 years but fertility rates at other ages either showed little change or increased. In general, proportionate increases in women aged 15-24 years retarded the relative decline in births to these women, although the extent to which this occurred diminished over time.

At maternal ages 25-29 years, unadjusted percentages of births increased after 1961 because, during 1955-86, fertility rates rose at maternal ages 25-29 years relative to the change in fertility rates at other reproductive ages. These increases in percentages of births to 25-29 year-old women occurred despite, and are augmented after adjustment for, proportionately fewer women of reproductive age aged 25-29 years after 1955, and especially until about 1970.

Among 30-34 year-old women, unadjusted percentages of births fell between 1956 and 1974. Most of this period of decline, 1959-74, was promoted by a declining proportion of 30-34 year-old women (this latter reduction being smaller, however, than the percentage decline in births), and by a slight fall in fertility rates for this maternal age group relative to the change in fertility rates at other
reproductive ages. Increases in unadjusted percentages of births to 30-34 year-olds after 1974 resulted, except during the late 1970s to early 1980s, from fertility rates that increased relative to changes in these rates at other reproductive ages. As shown by Figure 1.9, at ages 30-34 years, fertility rates rose from 1975 to 1986, but overall, declined, showed minimal change or increased more gradually than at other ages.

Unadjusted percentages of births occurring to women aged 35-39 years and 40 years and over fell respectively between the late and early 1950s and the mid-late 1970s. This decline took place despite these age groups accounting for higher proportions of all women of reproductive age until the early 1960s for 35-39 year-olds, and the mid 1960s for women aged at least 40 years. Thereafter, the decline was facilitated by opposite changes in age structure and, most saliently from the early to mid 1960s until the late 1970s, principally by a decline in fertility at these mature ages relative to fertility change at other reproductive ages. Since the late 1970s, relative stability has characterized the contribution of both mature-age groups, although especially women at reproductive ages 40 years and over, to total fertility and to proportionate changes in the distribution of births by maternal age.

In sum, from 1952 to 1966, women at adolescent and mature ages, for example, who, other things being equal, are at elevated risk of perinatal death, accounted for an increased unadjusted percentage of all births. As shown by Figure 1.10, this resulted from increased proportions of women at high-risk ages, and from fertility rates that rose at high-risk ages during the early 1960s relative to the change in rates at low-risk ages. However, the importance of the latter effect, which resulted from fertility rates falling more strongly at
low-risk than at high-risk maternal ages (see Figure 1.11), diminished over time; and led to the same relativities between fertility rates in 1965 as in 1952. Accordingly, at high-risk ages, adjusted percentages of births increased from 1960 to 1966 after having fallen from 1952 to 1960. This reversal occurred in response to a shift in the relativities between fertility rates at high-risk and low-risk maternal ages.

After 1966, there was a decline in unadjusted percentages of births occurring to women at high-risk ages. This overall decline was retarded, briefly during the late 1960s, by proportionately more women at these high-risk maternal ages. Thereafter, it was promoted by proportionately fewer women at these ages, this decrease being less, however, than the percentage decline in births, and especially since 1973 by greater declines in the age-specific fertility of high-risk than low-risk maternal ages: from 1973 to 1986, the fertility rate for high-risk ages fell by 28.7 per cent to 15.4 per 1000, while that for low-risk ages fell by 14.8 per cent to 107.8 per 1000.

Figure 1.10
Percentages of Australian Live Births Occurring at High-Risk and Low-Risk Maternal Ages, 1952-66, Adjusted for the 1952 Reproductive Age Structure of Australian Women

The second question posed above relates to linking shifts in fertility back to changes in total and age-specific perinatal mortality. The basic point is that, other things being equal, greater declines in fertility at, and in percentages of all births to, high-risk vis-à-vis low-risk reproductive ages would have acted to lower total rates of perinatal death. However, this effect has been shown to have been slight for the period 1952-86. The reasons for this are probably as follows: (a) births at the high-risk maternal ages have accounted for relatively small proportions of total births, and (b) the perinatal mortality risks of these higher-risk births have not sufficiently exceeded those of other births for a reduction in fertility at the high-risk ages relative to that at the low-risk ages to have substantially reduced perinatal mortality. Nevertheless, changes in age-specific fertility, most conspicuously at ages over 40 years, could have made important contributions to the size of reductions in age-specific perinatal mortality rates.

Figure 1.11
Fertility Rates Specific for High-Risk and Low-Risk Maternal Ages in Australia, 1952-86

Reasons for fertility change: The following discussion seeks to explain the described changes in fertility, first for adolescent and then for mature-aged women. With adolescents, it is helpful to distinguish between the nuptial and ex-nuptial components of the recent fertility decline, and so Figure 1.12 shows these components of the age-specific fertility rate for women aged 15-19 years. The nuptial component fell consistently after 1971 by a total of 83.1 per cent to reach 6.2 in 1986. This decline, which mainly affected nuptial births conceived ex-nuptially (Siedlecky, 1985), occurred in response to an emerging libertarian social conscience that reduced pressures to legitimize premarital conceptions by marriage and, by encouraging educational and career aspirations for economic and ideological reasons, also discouraged the formation of marriages that, of course, might have precipitated pregnancy.

Figure 1.12
Nuptial and Ex-Nuptial Components of Adolescent Fertility Rates for Australia, 1971-86

Facilitating fewer nuptial births to adolescents in the 1970s were changes, therefore, that help to explain, and may be partly mediated by, rising ages of women at first marriage, decreasing proportions marrying and an increase in the age of women at the birth of the first nuptial child (McDonald, 1982; Choi and Ruzicka, 1988). These changes include a substantial rise in teenage and young adult unemployment after 1974 (Caldwell, 1982), a probable shift in some childbearing to informal consensual unions, and a possible shift to retaining children by sole mothers, particularly after the introduction of the Supporting Mother’s Benefit in July 1973 (Carmichael, 1988).

Moreover, there became available improved methods of fertility control (Caldwell, 1982), including easier access to induced abortion (Siedlecky, 1985). Carmichael (1988) has suggested that the liberalizing of access to abortion might actually have triggered the decline in fertility among adolescents. Specifically, the late 1971 Levine judgement in R. v. Wald et al. in New South Wales was perhaps the watershed, from a national perspective, in establishing broader grounds for abortion as a method of controlling family formation (Carmichael, 1988). The decision, which followed a legal ruling in Victoria and earlier South Australian legislation, both in 1969, made clear that a woman’s social and economic circumstances were relevant to evaluating whether the danger to her physical or mental health, which by law gave legal access to abortion, in fact existed.

This importance attached to the 1971 New South Wales decision is supported by the timing of the fertility decline among adolescents, and indeed also older women, depicted in Figures 1.9 and 1.11. This figure indicates that in the age-groups below age 40 years, fertility rates peaked in 1971 and fell from 1972 onwards, which could represent an expected lag effect following the New South Wales ruling. Although
the concurrence of these events could be coincidence, abortion offers an attractive explanation at least for the decline in adolescent fertility.

According to Figure 1.12, ex-nuptial births became a larger proportion of total births to adolescents. The ex-nuptial component of adolescent fertility rates fell during 1971-76 but not as greatly as the nuptial component. Indeed the ex-nuptial component increased from 14.0 in 1977 to 16.8 in 1983 before declining until 1986. These changes are biased upwards by the inclusion in the denominator of an increased proportion of adolescent women unmarried, and hence at risk of having an ex-nuptial birth. However, adolescent ex-nuptial confinement rates (per 1 000 non-married adolescent women) also increased - from 15.8 in 1976 to 17.0 in 1981 - before falling to 16.2 in 1986 (Khoo and McDonald, 1988).

The decline in adolescent ex-nuptial fertility, except therefore during the late 1970s and possibly the early 1980s, corresponds with the increased availability of contraceptive methods and, especially after 1971, induced abortion. Moreover, there was a growing acceptance by both society and families of adolescents using these methods (Caldwell, 1982; Hugo, 1986). Although falling adolescent ex-nuptial fertility rates acted to depress rates of perinatal death, it is uncertain to what extent an opposite effect on perinatal survival attended the increase in ex-nuptial births as a proportion of all adolescent births.

Facilitating this increase has been the changing context of ex-nuptial birth. Women who in earlier times would have married to legitimize ex-nuptial conceptions, chose increasingly instead to become single mothers or to cohabit. In recent years, the women giving birth ex-nuptially have frequently come from disadvantaged backgrounds (Khoo
and McDonald, 1988), and possibly, this tendency has been accentuated over time - assuming that women of low socioeconomic status have been less able or less inclined than have other women to use increasingly accessible abortion facilities. This 'deprivation hypothesis' would not portend well for antenatal care among adolescent unmarried women, or for expectant single women at other ages. Indeed it supports the view that ex-nuptiality is a risk factor for perinatal death (see section 2.11.2).

Among mature-aged women, and indeed women aged 30-34 years, the decline in fertility, until at least 1976, was associated with increasingly efficient methods of fertility control. In Australia, the 1960s brought widespread acceptance of the oral contraceptive and intrauterine device (Lavis, 1975). With the 1970s and concern over the side-effects of the pill came the increased use of sterilization, especially among women (Young and Ware, 1979), and of legal abortions, in particular among the small group of women becoming pregnant over 45 years of age (Yusuf and Briggs, 1979, Caldwell, 1982).

This increase in abortion among mature-aged women was a response not only to the juridical and legislative developments described above, but also to the more widespread screening23 of these women, for example by amniocentesis,24 to identify 'handicapped' foetuses and pregnancies for which termination may be offered. Also of note is that increased recourse to induced abortions in such circumstances would have discounted the number of perinatal deaths.

Other factors contributing to the decline in fertility at mature ages might have included some waiving of childbearing by women for whom

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23 Antenatal screening identifies from a population of apparently healthy women those who are sufficiently at risk of a specific disorder to justify a subsequent diagnostic test or procedure, or primary preventive action.

24 Amniocentesis involves piercing the amniotic sac through the abdominal wall to withdraw, for testing, a sample of the amniotic fluid surrounding the baby.
institutional and value structures militated against combining child-rearing with work outside the home (Hugo, 1986). The gradual emergence of new structures,25 entry into new sexual unions following marriage dissolution, and a concomitant recouping of births deferred from earlier ages may aid explanation of the recent increase in fertility rates among women at advanced maternal ages.

Irrespective of this increase, the increased obstetric risks associated with pregnancies at mature ages might have been mitigated by other, salutary characteristics of these women. For example, women who deferred childbearing are likely to have been highly educated and their knowledge and experience might have helped to reduce the risks of perinatal complications. Besides the underlying changes in the roles and statuses of women, mature-aged women, and also women at other ages, have benefited from a general expansion of health services and medical supervision. The latter developments can be traced particularly to the medical benefits scheme introduced under the 1953 National Health Act, and revitalized under the Whitlam Government in the second half of 1975 (Palmer, 1979).

Finally, the recent increase in the fertility of mature women seems broadly to coincide with an increase since the late 1970s in first births, which are at elevated risk of perinatal death, at ages 35-39 years (Choi and Ruzicka, 1987). However, first births are comparatively rare at these ages, and the increase, or a substantial part of it, may be artefactual. Carmichael (1986) has observed that Australian birth order statistics are based only on previous issue of

25 These include increased acceptance by families and society of combining family formation with labour force participation. This change in values has been reflected in the improved provision of child care either in the family or external to it (Duncan and Morgan, 1975), and facilitated by technological innovations such as labour-saving devices in the home (Kupinsky, 1977).
the current marriage, and so higher parity births in second and subsequent marriages, which are most likely to occur to older women, can be misclassified as first births (or, more generally, to lower parities than they actually are). At least partly artefactual would therefore also be any extension to mature ages of the overall decline in high order births during 1971-85 (Choi and Ruzicka, 1987), a decline, ceteris paribus, that would help to reduce perinatal mortality risks.

1.4.2 MEDICAL ADVANCES

Despite these changes, the best explanation for the decline in perinatal death rates is technological advance, especially over the last thirty years, in the ability of medicine to diagnose and treat disorders, or their symptoms, in particular after birth (Budetti and McManus, 1982; Bakketeig et al., 1984; Saling and Arabin, 1988). For example, the increased survival of the highest risk infants, weighing less than 1 000 grams, has usually been attributed mainly to three areas of neonatal intensive care: ventilatory support, improved understanding of neonatal nutritional requirements and better resuscitation in the delivery room (Bhat and Zikos-Labropoulon, 1986). Identification of the need for care has been enhanced by the increased ability to assess the condition of neonates at birth by means of Apgar scores and the additional use of biochemical methods.

Consequently, stillbirth rates have fallen less strongly than have rates of neonatal death, although considerable gains have been made in

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26 This is on the assumption that people answer the question(s) on previous issue strictly as asked.
27 This is not to diminish the importance of earlier developments such as infection control - sulphonamides and antibiotics became available in New South Wales in 1936 and 1946 respectively (Boland, 1984) - and immunization and serum treatments. But the period since the 1960s, with its concentration of manifold achievements, is thought to mark the real beginning of perinatal medicine (Saling and Arabin, 1988).
systematic exploration of the intrauterine space and in consequent accessibility to the foetus. One result has been the termination of more handicapped foetuses, identified through improved and more widespread antenatal screening, which has contributed to reductions in the perinatal deaths certified as due to congenital anomalies such as the neural tube defects (see section 1.5).\textsuperscript{28} Notwithstanding this, the size of the contribution of terminations to these reductions is uncertain because medical care has kept malformed babies alive, for example during the perinatal period, that formerly would have died at earlier ages (Alberman, 1985).

More generally, the initial, real breakthroughs in antenatal diagnostic and therapeutic developments have come with 'the advent of the techniques of amnioscopy\textsuperscript{29} and foetal blood sampling and of amniocentesis and foetal transfusion'\textsuperscript{30} (Dobbs and Gairdner, 1966: 453). Achievements have also included the prevention of Rh erythroblastosis,\textsuperscript{31} ultrasonic diagnosis, lung maturation diagnostics and therapy,\textsuperscript{32} and the use of prostaglandins for cervical ripening.\textsuperscript{33} Antenatal and intrapartum applications have attended landmarks in cardiotocography\textsuperscript{34} and tocolysis\textsuperscript{35} (Saling and Arabin, 1988).

\textsuperscript{28} The neural tube defects are a group of congenital anomalies caused by failure to close of the neural tube, the embryological structure from which the brain and spinal cord develop.

\textsuperscript{29} Amnioscopy is direct examination of the inside of the amniotic sac using an instrument (an amnioscope) that is passed through the abdominal wall.

\textsuperscript{30} Rhesus negative blood compatible with the pregnant mother is transfused either into the foetal peritoneal cavity or, since 1981, into an umbilical vein with the help of foetoscopy or ultrasound (Rodeck et al., 1981; Chamberlain, 1984; Berkowitz et al., 1986). Even intracardial transfusions can now be performed (Saling and Arabin, 1988).

\textsuperscript{31} Prophylaxis of Rh-sensitization involves giving anti-D gamma globulin to Rhesus negative women. Rh erythroblastosis is a rare but severe form of congenital haemolytic anaemia usually caused by incompatibility of the Rhesus blood groups of the mother and baby.

\textsuperscript{32} Drugs, including most prominently the glucocorticoids, can promote lung maturation when, for example, a lecithin-sphingomyelin ratio of less than 2 gives cause for concern about the risk of respiratory distress in the newborn.

\textsuperscript{33} See section 6.6.

\textsuperscript{34} Cardiotocography provides an automatic graphic correlation of foetal heart rate patterns and uterine contractions.
1.5 AUSTRALIAN CAUSE OF PERINATAL DEATH STATISTICS

The sociodemographic and medical trends just discussed underlie recent national perinatal data for events registered in 1986, this year being the main time frame in which this study operates. The 1986 perinatal death rate for Australia was 11.5 per 1 000, which, representing a continuation of the historical decline in these rates, was due mainly to the 1986 neonatal death rate falling to 5.0 per 1 000 live births; rates for neonates are expressed per 1 000 live births, whereas rates for stillbirths and perinatal deaths are expressed per 1 000 total births. Slightly over half the neonatal deaths (52.5 per cent) were to newborn36 less than one day old; and 80.2 per cent took place within the first six days of infant life. The 1986 stillbirth rate was 6.5 per 1 000, two-thirds of the stillbirths occurring before labour commenced (Australian Bureau of Statistics, 1988).

The following discussion focuses first on cause of death components of 1986 rates of perinatal death, stillbirth and neonatal death. Then, selected cause of death components of maternal age-specific perinatal death rates are compared for two years, 1979 and 1986. Cause of perinatal death statistics are based on the recommendations of the Ninth Revision Conference in distinguishing between the main condition in the foetus or infant, and the main condition in the mother affecting the foetus or infant (see sections 2.1 and 4.2).

Figure 1.13 decomposes death rates for stillbirths, neonatal deaths and perinatal deaths into additive components37 defined by these two

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35 Tocolytics, or betamimetics (for example, Salbutamol which is better known by the proprietary name of Ventolin) relax smooth muscles. They may be given antenatally to try to inhibit uterine contractions. During labour, betamimetics can be used for intrauterine resuscitation; that is, to relieve intrapartum foetal distress.

36 This study resists the movement toward use of the term 'newborns', using instead the term newborn both as a noun in the singular and plural senses, and as an adjective.

37 These components are sometimes referred to as cause-specific death rates.
Figure 1.13
Cause of Death Components of Australian Stillbirth, Neonatal Death and Perinatal Death Rates, 1986

Main Condition in the Foetus or Infant
- Hypoxia, Birth Asphyxia and Other Respiratory Complications
- Other Conditions Originating In the Perinatal Period
- Congenital Anomalies
- Slow Foetal Growth, Foetal Malnutrition and Immaturity
- Foetal and Neonatal Haemorrhage
- All Other Causes

Main Condition In the Mother Affecting the Foetus or Infant
- Maternal Conditions which may be Unrelated to the Present Pregnancy
- Maternal Complications of Pregnancy
- Complications of Placenta, Cord and Membranes
- Other Complications of the Labour and Delivery
- No Maternal Condition Reported

sets of main conditions. With reference to the main condition in the foetus or infant, three leading causes of perinatal death accounted for 9.3 per 1 000 of the 1986 perinatal death rate (80.9 per cent). The most important cause of perinatal deaths, and especially of stillbirths, was hypoxia, birth asphyxia and other respiratory conditions.\[38\] This accounted for 4.4 per 1 000 of the perinatal death rate, slightly over two-thirds (68.2 per cent) of this component being composed of stillbirths attributable mainly to anoxia or asphyxia occurring before or during labour. Put differently, respiratory complications accounted for 38.3 per cent of the perinatal death rate, and 46.2 per cent of the stillbirth rate.

The second leading cause of perinatal death, and the most important cause of death among neonates, was congenital anomalies,\[39\] which accounted for 2.6 per 1 000 of the perinatal death rate (22.6 per cent). The corresponding component of the neonatal death rate certified as due to congenital anomalies was 1.8 per 1 000, or 36.0 per cent of this rate. Of these anomalies, only anencephalus and similar anomalies,\[40\] and hydrocephalus,\[41\] were more frequently reported as a main cause of death among stillbirths. The third most important cause of perinatal death was other conditions originating in the perinatal period.\[42\] This accounted for 2.3 per 1 000 of the perinatal death rate (20.0 per cent), and 2.0 per 1 000 of the stillbirth rate, (30.8 per cent).

\[38\] The terms hypoxia and asphyxia describe here a deficiency of oxygen in the tissues.
\[39\] Congenital anomalies include all disorders present at birth, whether they are caused by genetic or environmental factors.
\[40\] Anencephalus is a developmental defect characterized by absence of some or all of the bones of the rear of the skull, and of the cerebrum, the largest and most highly developed part of the brain.
\[41\] Hydrocephalus describes an excess of cerebrospinal fluid in the ventricles (cavities) of the brain.
\[42\] These other conditions include particularly infections specific to the perinatal period.
Two points emerge from Figure 1.13’s decomposing of perinatal death, stillbirth and neonatal death rates for 1986 into cause of death components defined by the main condition in the mother affecting the foetus or infant. First, no maternal condition accounted for 4.2 per 1000, or 36.8 per cent of this rate. For neonatal deaths, the corresponding percentage was 44.0, higher than for any identifiable cause. Secondly, complications of the placenta, cord and membranes was the leading reported condition in the mother producing perinatal death, although this condition was important only among stillbirths, accounting for 2.8 per 1000 of the stillbirth rate (42.5 per cent).

The following discussion uses Table 1.1 to compare selected cause of death components of total and maternal age-specific perinatal death rates in 1979 and 1986, while for the same period, Table 1.2 compares percentages of all perinatal deaths in different maternal age groups in these cause of death categories. Data before 1979 are not reported because causes of perinatal death in Australia were then tabulated according to the underlying cause of death based on the Eighth Revision of the International Classification of Diseases; the subsequent Ninth Revision classifies the cause of death, as shown above, according to the main condition in the foetus or infant, and the main condition in the mother.

Table 1.1 shows a decline in most cause of death components of the 1979 and 1986 perinatal death rates, most strongly at maternal ages 35 years and over. The largest reduction per 1000 births was in deaths due to congenital anomalies, which resulted, in part, from a decline in mortality from deformities of the central nervous system. At mature ages, these latter defects accounted for a decline in the perinatal death rate of 0.7 (1.4 minus 0.7) per 1000 during 1979-86.
Table 1.1: Selected Cause of Death Components of Total and Maternal Age-Specific Perinatal Death Rates (Per 1,000), 1979 and 1986

<table>
<thead>
<tr>
<th>A. Main condition in the foetus or infant</th>
<th>Maternal Age (Years)</th>
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<tr>
<td>Congenital Anomalies</td>
<td>4.0</td>
<td>2.3</td>
<td>3.3</td>
<td>2.5</td>
<td>4.2</td>
<td>2.5</td>
<td>3.5</td>
<td>2.6</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Deformities of central nervous system</td>
<td>1.7</td>
<td>1.0</td>
<td>1.4</td>
<td>0.7</td>
<td>1.4</td>
<td>0.7</td>
<td>1.4</td>
<td>0.9</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Hypoxia, birth asphyxia and other respiratory conditions</td>
<td>6.0</td>
<td>5.6</td>
<td>4.8</td>
<td>4.0</td>
<td>7.5</td>
<td>6.2</td>
<td>5.0</td>
<td>4.3</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Foetal death from asphyxia or anoxia before onset of labour or at an unspecified time</td>
<td>2.3</td>
<td>2.5</td>
<td>2.1</td>
<td>2.1</td>
<td>4.1</td>
<td>3.5</td>
<td>2.3</td>
<td>2.3</td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Other conditions originating in the perinatal period</td>
<td>4.2</td>
<td>3.5</td>
<td>2.9</td>
<td>2.1</td>
<td>6.8</td>
<td>3.8</td>
<td>3.0</td>
<td>2.3</td>
<td></td>
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</tr>
<tr>
<td>B. Main maternal condition affecting the foetus or infant</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal conditions which may be unrelated to present pregnancy</td>
<td>2.6</td>
<td>2.4</td>
<td>1.3</td>
<td>1.0</td>
<td>3.5</td>
<td>2.0</td>
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<td>1.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal hypertensive disorders of pregnancy</td>
<td>1.4</td>
<td>1.0</td>
<td>0.7</td>
<td>0.6</td>
<td>2.5</td>
<td>1.2</td>
<td>0.9</td>
<td>0.6</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal complications of pregnancy</td>
<td>1.6</td>
<td>2.6</td>
<td>1.7</td>
<td>2.1</td>
<td>3.8</td>
<td>3.6</td>
<td>1.9</td>
<td>2.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premature rupture of membranes</td>
<td>0.8</td>
<td>0.3</td>
<td>0.6</td>
<td>0.5</td>
<td>1.0</td>
<td>0.7</td>
<td>0.6</td>
<td>0.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complications of the placenta, cord and membranes</td>
<td>4.4</td>
<td>4.3</td>
<td>3.9</td>
<td>3.0</td>
<td>6.1</td>
<td>5.0</td>
<td>4.1</td>
<td>3.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placental separation and haemorrhage</td>
<td>2.0</td>
<td>1.9</td>
<td>1.8</td>
<td>1.2</td>
<td>2.9</td>
<td>2.2</td>
<td>1.9</td>
<td>1.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No maternal condition reported</td>
<td>9.4</td>
<td>4.8</td>
<td>6.3</td>
<td>4.1</td>
<td>8.1</td>
<td>4.9</td>
<td>6.6</td>
<td>4.0</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Total(^2)</td>
<td>18.3</td>
<td>14.3</td>
<td>13.8</td>
<td>10.5</td>
<td>22.2</td>
<td>15.9</td>
<td>14.6</td>
<td>11.1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1 Total rates exclude women of unknown ages.
2 Because only selected causes of death are shown, perinatal death rates represent the sum of the components associated with each separate set of main conditions (A and B) plus an unrecorded residual component.

### Table 1.2: Percentages of All Perinatal Deaths Attributable to Selected Causes of Death, 1979 and 1986

<table>
<thead>
<tr>
<th>A. Main condition in the foetus or infant</th>
<th>Maternal Age (Years)</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;20</td>
<td>20-34</td>
<td>&gt;34</td>
<td>Total</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>1979</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>1986</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Congenital Anomalies</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deformities of central nervous system</td>
<td>21.9</td>
<td>16.1</td>
<td>23.9</td>
<td>23.8</td>
<td>18.9</td>
<td>15.7</td>
<td>24.0</td>
</tr>
<tr>
<td>Hypoxia, birth asphyxia and other respiratory conditions</td>
<td>32.8</td>
<td>39.2</td>
<td>34.8</td>
<td>38.1</td>
<td>33.8</td>
<td>39.0</td>
<td>34.2</td>
</tr>
<tr>
<td>Foetal death from asphyxia or anoxia before onset of labour or at an unspecified time</td>
<td>12.6</td>
<td>17.5</td>
<td>15.2</td>
<td>20.0</td>
<td>18.5</td>
<td>22.0</td>
<td>15.8</td>
</tr>
<tr>
<td>Other conditions originating in the perinatal period</td>
<td>23.0</td>
<td>24.5</td>
<td>21.0</td>
<td>20.0</td>
<td>30.6</td>
<td>23.9</td>
<td>20.5</td>
</tr>
<tr>
<td><strong>B. Main maternal condition affecting the foetus or infant</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal conditions which may be unrelated to present pregnancy</td>
<td>14.2</td>
<td>16.8</td>
<td>9.4</td>
<td>9.5</td>
<td>15.8</td>
<td>12.6</td>
<td>10.3</td>
</tr>
<tr>
<td>Maternal hypertensive disorders of pregnancy</td>
<td>7.7</td>
<td>7.0</td>
<td>5.1</td>
<td>5.7</td>
<td>11.3</td>
<td>7.5</td>
<td>6.2</td>
</tr>
<tr>
<td>Maternal complications of pregnancy</td>
<td>8.7</td>
<td>18.2</td>
<td>12.3</td>
<td>20.0</td>
<td>17.1</td>
<td>22.6</td>
<td>13.0</td>
</tr>
<tr>
<td>Premature rupture of membranes</td>
<td>4.4</td>
<td>2.1</td>
<td>4.3</td>
<td>4.8</td>
<td>4.5</td>
<td>4.4</td>
<td>4.1</td>
</tr>
<tr>
<td>Complications of the placenta, cord and membranes</td>
<td>24.0</td>
<td>30.1</td>
<td>28.3</td>
<td>28.6</td>
<td>27.5</td>
<td>31.4</td>
<td>28.1</td>
</tr>
<tr>
<td>Placental separation and haemorrhage</td>
<td>10.9</td>
<td>13.3</td>
<td>13.0</td>
<td>11.4</td>
<td>13.1</td>
<td>13.8</td>
<td>13.0</td>
</tr>
<tr>
<td>No maternal condition reported</td>
<td>51.4</td>
<td>33.6</td>
<td>45.7</td>
<td>39.0</td>
<td>36.5</td>
<td>30.8</td>
<td>45.2</td>
</tr>
<tr>
<td><strong>Total Percentages</strong></td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

1 Percentages exclude women of unknown age.
2 Because only selected causes of death are shown, the totals represent the sum of the percentages associated with each separate set of main conditions (A and B) plus an unrecorded residual percentage.

This indicates a reduction of 50 per cent in the size of this component of the perinatal death rate, yet, because of its small size, a decline of only 1.9 in the percentage of all perinatal deaths attributable to this cause (see Table 1.2).

Similarly, at maternal ages less than 20 years, all congenital anomalies and the deformities of the central nervous system showed impressive declines per 1,000 births, respectively 1.7 per 1,000 (a decrease of 42.5 per cent) and 0.7 per 1,000 (a decrease of 41.2 per cent), between 1979 and 1986. However, declines in the percentages of all perinatal deaths certified as due to these causes were larger than at older maternal ages, the reductions being 5.8 per cent for congenital anomalies and 2.3 per cent for deformities of the central nervous system. After collapsing over maternal age, the size of these same components of the perinatal death rate declined respectively by about one-third and one-quarter during 1975-86, these components accounting for only slightly smaller percentages of all perinatal deaths in 1986 than in 1979.

The available data do not permit cause of death components of stillbirth and neonatal death rates to be calculated by maternal age. However, without regard to the age of the mother, a decline of about 25 per cent during 1979-86 occurred in the congenital anomalies component of both the stillbirth and neonatal death rates. The percentage of all stillbirths and neonatal deaths attributable to congenital anomalies respectively fell by 1.5 per cent and increased by 2.7 per cent. These findings compare with Alberman's (1985) observation for England and Wales of a marked decline in the rate of stillbirths certified as due to congenital anomalies, but greater declines in the rates of neonatal death attributed to other causes.
In Australia, and England and Wales (Alberman, 1985), there are two main explanations for the decline in perinatal mortality attributable to congenital anomalies, although the relative importance of each is uncertain. These explanations are the more widespread screening of women, leading to the therapeutic abortion of affected foetuses, and the increased ability of neonatal intensive care to keep alive some malformed foetuses, for example with spina bifida, beyond the perinatal period (see section 2.2.1). In addition, environmental factors, such as decreased exposure to heavy metals as in copper-bearing intrauterine devices (Graham et al., 1980), might have contributed to the decline in lethal neural tube defects (see section 2.8.4); these defects form the main component of deformities of the central nervous system.

Table 1.1 further shows a large reduction from 1979 to 1986 in perinatal deaths attributable to other conditions originating in the perinatal period. The decline in this component of the perinatal death rate by 23.3 per cent (to 2.3 per 1 000) was only slightly lower than the reduction for congenital anomalies; indeed, except among adolescents, these 'other conditions' were associated with a greater reduction in mortality than were congenital anomalies. However, Table 1.2 shows almost no change in percentages of all perinatal deaths attributable to these 'other conditions', except at maternal ages 35 years and over.

The group of respiratory complications accounted for a comparatively small decline in the perinatal death rate (see Table 1.1). Indeed when these complications occurred before the onset of labour or at an unspecified time, the size of this component increased at maternal ages below 20 years. Table 1.2, moreover, shows increased percentages of all perinatal deaths attributable to hypoxia, birth asphyxia and
other respiratory conditions, especially when occurring before the onset of labour or at an unspecified time.

Of the main maternal conditions, reductions of about one-fifth are shown in the size of two components of the perinatal death rate: maternal conditions which may be unrelated to the present pregnancy and complications of placenta, cord and membranes. The leading cause of death in each of these categories, namely the hypertensive disorders of pregnancy and antepartum haemorrhage, showed overall declines per 1 000 births of about one-third, the latter complication being the slightly more hazardous. The reductions associated with these general and specific complications were largest at mature ages and among women aged 20 to 34 years. Both sets of complications accounted for similar proportions of all perinatal deaths in 1979 and 1986.

The decline in the contribution of maternal hypertensive disorders to perinatal mortality is especially interesting given uncertainties about when to treat hypertension (Trudinger and Parik, 1982; Lubbe, 1987). The decline appears to support the basic management of severe hypertension through lowering blood pressure and timely delivery (Gallery, 1984; Korda and Horvarth, 1984; and see section 5.3.1). Among women with antepartum haemorrhage, reductions in perinatal mortality have attended modern management practices including earlier and improved diagnosis especially of placenta praevia, but also, for example, of abruptio placentae when it occurs.

Except among women aged 35 years and over, an increase occurred in the component of the perinatal death rate described as maternal complications of pregnancy. Yet most conspicuous is that at all maternal ages, deaths reported not to be associated with any maternal condition showed a substantial fall per 1 000 births. In addition,
there was a decline of 9.2 in the percentage of all perinatal deaths attributable to this 'cause'.

1.6 RESEARCH PRIORITIES

Broad and specific research priorities derive from the foregoing examination of current Australian perinatal death statistics, and from preceding sections which have sought to describe and account for historical falls in levels of perinatal mortality in Australia and elsewhere. The broad priorities relate to what is required to reduce rates of perinatal death further, including the need to sustain improvements in treatment regimes and to prevent aetiological conditions developing in the first place.

Only recently has much attention been paid to the latter priority. Earlier neglect stemmed from the belief that perinatal death results mainly from conditions originating before or during birth, including especially monogenic defects,\textsuperscript{43} chromosomal disorders\textsuperscript{44} and placental insufficiency,\textsuperscript{45} the prevention of which largely resists scientific progress. Consequently, medical researchers channelled their energies into developing effective forms of treatment.

However, during the last fifteen years, these professionals have undertaken increased research into the primary prevention of perinatal complications. This is due in large part to their realization that, while demographers continue to learn that 'the circumstances of prenatal life and the birth process are endogenous and so relatively

\textsuperscript{43} Monogenic defects are defects in a single gene pair.
\textsuperscript{44} Chromosomal disorders describe an abnormality of chromosome number or structure.
\textsuperscript{45} Placental insufficiency refers to abnormally decreased placental function, depriving the foetus, for example, of an adequate supply of oxygen and nutrients.
unpreventable causes of death' (Shryock and Siegel, 1971, 1980: 405),
this is not quite accurate.

Some important stated causes of perinatal death are exogenous whilst
others are endogenous, so that collectively, these causes are probably
best categorized as both endogenous and endogenous (see section 2.0).
Cleland (1989: 7) describes variation in 'the incidence of deaths from
endogenous causes such as prematurity and congenital anomalies', yet
deaths from prematurity are not strictly endogenous. It is true that
premature rupture of the membranes and/or preterm labour, may resist
tocolysis, but the frequently non-medical precursors of these
complications, which occur antenatally, are preventable and probably
justify description of prematurity as an exogenous cause of perinatal
death. Most congenital anomalies are endogenously defined, but
environmental factors are known to influence, for example, the neural
tube defects, these being genetic disorders of multifactorial origin.

Specifically, medical researchers, such as Gruenwald (1975), have
awakened to the important aetiological contribution of the woman's
environment during, and even before, pregnancy. The pregnant woman's
lifestyle has been observed to influence not only foetal physiological
immaturity but also, as noted above, congenital anomalies of complex
genetic aetiology. Of further interest are the iatrogenic risks
associated with health care especially during the confinement, so
that in all these areas, the promise of primary prevention has
stimulated epidemiological research.

46 The prophylactic effect of tocolysis is also controversial.
47 Disorders of complex genetic aetiology are those often attributed to the
combined effects of many genes and other, nongenetic factors; this is called
multifactorial aetiology.
48 The issue, in fact, is much larger than merely health risks; there are
other considerations such as the individual's freedom to choose. But this
choice must be based on an appropriate intellectual persuasion founded in
evidence produced scientifically and dispassionately.
This is not, however, to suggest that demographers have completely neglected perinatal research. A seminal contribution was made by Bourgeois-Pichat's (1951a, 1951b) biometric analyses of infant mortality,49 and in recent years, perinatal research by demographers, although still comparatively rare, has continued to be undertaken. Inquiries, for example by Leridon (1973) and Bongaarts (1983) who have estimated the risks of intrauterine mortality, have tended to be descriptive and analytic rather than explanatory in nature; yet this approach is characteristic generally of formal demography as distinct from population studies (Newell, 1988). Some demographers have undertaken explanatory perinatal research particularly in terms of the relation between birth intervals and perinatal or infant mortality (see Hobcraft, 1983; Miller, 1989).

An appreciation, therefore, of the large and diverse number of known or suspected risk factors for perinatal death, and of their different degrees of responsiveness to primary prevention, leads specifically to three research approaches. The first involves elucidating the aetiological effects of small numbers of risk factors both in the environment of the pregnant woman, including her attitudes and behaviour, and external to this environment. To date, this imperative has been achieved by randomized controlled trials and especially by observational studies that seek usually in discrete, clinical disciplines to answer specific research questions.

Less common is the second approach whereby large-scale investigations, through their considerations of the separate and joint effects of diverse risk factors, produce a panoramic sense of the tapestry of multiple aetiologies for perinatal death in particular populations.

49 For example, Poston and Rogers (1985) recently attributed neonatal mortality to so-called endogenous causes of death during the first eighteen days of life, not the first 28 days.
This approach, which is essentially eclectic and holistic, best describes the present investigation. In seeking to elucidate risk factors for perinatal death, the study will attempt to draw on the materials and methods of diverse disciplines - because the answers to aetiological questions are presumed to be also interdisciplinary. It will develop a conceptual model of proximate and background determinants of perinatal death that can guide, integrate and focus the succeeding empirical investigation. The study will touch on, but not aspire fully to adopt, the third approach of assessing especially through multivariate analyses the interactive effects of proximate and background determinants of perinatal death in and between environmental and nonenvironmental contexts.

From this broad agenda emerge other, specific research priorities that bear on the stated causes of perinatal death, and on the physiological maturity of the conceptus with which perinatal death is integrally bound. Specifically, medical advances have salvaged increasing numbers of low and even very low birthweight babies, yet these advances have not proved able to lower significantly the proportion of perinatal deaths with low birthweights (Behrman, 1985). Physiological immaturity remains a principal determinant of perinatal death.

The problem is not simply to understand why immaturity occurs, for instance by studying the causes of preterm deliveries\(^50\) such as premature rupture of the membranes,\(^51\) and of intrauterine growth

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\(^50\) Dated in completed gestational weeks from the last normal menstrual period and in accordance with the most recent recommendations of the World Health Organization, preterm is before 37 gestational weeks (less than 259 days). Term is from 37 to 41 weeks (259 to 293 days), and post-term is from 42 weeks (294 or more days) (World Health Organization, 1977).

\(^51\) Premature rupture of the membranes is defined as evidence of leaking amniotic fluid (the fluid contained in the amniotic sac forming the 'bag of waters') before the 37th gestational week.
The research priorities are to identify whether the causes of the immaturity differ between the babies that die and those that survive, and then, if appropriate, to investigate first for the former group, the causes of low birthweight. It is important also to assess how adjustment for birthweight affects the contributions to perinatal death of suspected risk factors.

This research priority, which could help to reduce, for example, the incidence of deaths attributed to hypoxia, birth asphyxia and other respiratory complications, guides the subsequent development of the thesis. Two other research priorities, which are beyond the scope of this study, are also in particular need of study. Both internalize the occurrence of some causes of perinatal and especially neonatal death, such as congenital anomalies, independently of birthweight.

The first issue concerns the causes of congenital anomalies of complex genetic aetiology such as the neural tube defects. Despite recognition of their multifactorial origins, the specific environmental contributions of teratogenic agents have not effectively been studied. The second research issue relates to the unexplained intrauterine deaths of mature foetuses near term. Although there may exist some underlying abnormality of pregnancy such as accidents to the umbilical cord, chorioamnionitis and low maternal blood volume (Derom, 1985; Van Geijn et al., 1985), such factors

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52 Intrauterine growth retardation is usually indicated by birthweights below the tenth percentile (the third or fifth percentile is sometimes used) for their gestational ages. These infants may be designated small for gestational age, or small for dates. Likewise, infants with birthweights above the ninetieth percentile for their gestational ages may be considered large for gestational age. Infants with birthweights between the tenth and ninetieth percentiles for their gestational ages may be considered appropriate for gestational age.

53 Teratogenic effects are those inducing the formation of developmental abnormalities.

54 Chorioamnionitis is inflammation of the foetal (outer chorionic and inner amniotic) membranes.
cannot fully explain these deaths, and it remains unclear why complications suddenly develop (see section 2.5).

1.7 AIMS

In accordance with the framework for research set in section 1.6, this thesis has the following broad aim: to elucidate for New South Wales (see section 3.2) the separate and joint effects of environmental and nonenvironmental risk factors for perinatal death. A subsidiary aim is to pay particular attention to understanding why physiological immaturity is sometimes, but not always, associated with perinatal death. These aims invoke three specific objectives:

(a) To develop a conceptual framework specifying linkages among the proximate and background factors causing perinatal death;

(b) To formulate hypotheses about risk factors for perinatal death in New South Wales; and

(c) To collect and analyse retrospective data for the purpose of testing these hypotheses in a case-control framework.

1.8 CONCLUSION

This chapter has provided general background information on historical changes in perinatal terminology and measurement, and in levels of perinatal mortality outside and in Australia. The reasons for the recent decline in perinatal mortality were presented in terms of sociodemographic changes, with particular reference to Australia, and medical improvements. A discussion followed of 1986 Australian
perinatal death statistics pertaining to the stated causes of perinatal death in this year, which were then compared with the causes reported in 1979. This led in turn to a consideration of future research directions as pursued in chapter 2.
CHAPTER 2.0
CONCEPTUAL MODEL AND LITERATURE REVIEW

2.0 INTRODUCTION

The first stage in any research is observation, initially involving sense-perceptual data. This is usually followed by a critical reading of the literature leading to an image of real world structure. In this study, such an image is formally represented by an a priori model (Figure 2.0), although for the sake of clarity, presentation of the model precedes a discussion of the underlying literature.

Figure 2.0 is a conceptual model which seeks to structure existing knowledge. The model visually simplifies complex interactions between known and suspected risk factors for perinatal death, acting as a device for explanation while permitting in turn the deduction of basic hypotheses requiring testing.

Figure 2.0 unifies categories of risk factors for perinatal death by specifying recursive linkages within a time-dependent framework. Each category is therefore classified as a truly dependent, proximate or background variable, whilst also as mainly endogenous, mainly exogenous, or endogenous and exogenous in approximately equal proportions. The mainly endogenous variables are relatively unpreventable and tend to be the most intrinsic characteristics of the pregnancy. The mainly exogenous variables are environmental, often behavioural, factors or events strongly influenced by such factors, where both possibilities are amenable to change.

Perinatal death and its stated cause(s) are conceptualized as dependent variables although, in chapters 5 to 8 - for operational
Figure 2.0
A Conceptual Model of Risk Factors for Perinatal Death

<table>
<thead>
<tr>
<th>Background Variables</th>
<th>Proximate Variables</th>
<th>Dependent Variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mainly Macro-level</td>
<td>Mainly Micro-level</td>
<td></td>
</tr>
</tbody>
</table>

**Institutional Factors**
- a. Ethics and The Law
- b. Medical Technology
- c. Social Philosophy and Action
- d. Political Economy

**Family Medical History**
- a. Family and Maternal General Medical Histories
- b. Obstetrical History

**Family Social Factors**
- a. Sociobiological Factors
- b. Sociocultural Factors
- c. Socioeconomic Factors
- d. Geographical Factors

**Pregnancy Health**
- a. Psychological Stress
- b. Physical Activity
- c. Physical Environment
- d. Chemical Factors
- e. Nutrition

**Antenatal Care**
- Remaining factors

**Chromosomal and Genetic Factors**
- Remaining factors

**Obstetrical Factors**
- a. Physiological Factors
- b. Maternal Disease

**Newborn's Physiological Maturity**
- a. Birthweight
- b. Newborn's Condition at Birth
- c. Newborn's Growth and Development

**Perinatal Death**
- Stated Cause(s) of Perinatal Death

**Postnatal Care**
- a. Postnatal Health Care
- b. Postnatal General Care

**Intrapartum Health Care**
purposes - explanation is sought only for perinatal death per se. In the conceptual model, both variables, which are jointly endogenous and exogenous, are shown to result from the direct or low-order indirect effects of eight groups of proximate factors. Of these factors, which are discussed in Part A of this chapter, two groups relate only to the neonate, and ipso facto have the most proximate effects on its survival. These factors are postnatal care, which is exogenously defined, and newborn physiological maturity which is indistinguishably endogenous and exogenous. The remaining six groups of factors describe the maternal-foetal unit, and comprise four sets of mainly exogenous factors: intrapartum and antenatal health care, pregnancy health and paternal factors, and two groups of mainly endogenous factors: obstetrical factors and genetic and chromosomal factors.

Through and only through these variables' direct or indirect effects, do low-order background variables exert the most remote, always indirect, influences on perinatal death. At the micro-level, background variables describe family social factors, which are mainly exogenous to the pregnancy and birth, and the mainly endogenous family medical history. These micro-level background variables are discussed in Part B of the chapter. They operate, with other, micro-level proximate variables, within a larger framework of macro-level institutional factors. However, background variables at the macro-level are integrated thematically into the complete chapter rather than being examined separately.

This chapter focuses on all these categories of risk factors and their interactions in the context of developed countries. Categories conceptualized at the micro-level are discussed in a logical temporal sequence, moving from the most proximate to potentially the most indirect influences; this sometimes differs from the order depicted in
Figure 2.0 which takes into account an endogenous-exogenous classification. In each category, the discussion of risk factors advances forward in time. Most of the risk factors pertain to only a very small proportion of the maternity population. They also relate to long-term infant morbidity, but that is beyond the scope of this thesis.

2.1 THE DEPENDENT VARIABLES

Two variables, perinatal death and its stated cause(s), are identified as dependent variables, or more specifically, as truly dependent variables since they have no predictive powers themselves. These dependent variables are perinatal death, which is described in section 1.1., and the stated cause(s) of perinatal death.

Following a recommendation of the World Health Organization’s Ninth Revision Conference (1975), categories of the stated cause(s) of perinatal death distinguish between whether aetiological conditions originate in the foetus or infant, or in the mother (World Health Organization, 1977). The most common conditions in the mother causing perinatal death are maternal hypertensive disorders;1 antepartum haemorrhage including particularly abruptio placentae;2 and premature rupture of the membranes. In 1986, no maternal condition was reported in more than one-third of perinatal deaths in Australia, but in less than one-quarter of perinatal deaths in New South Wales (Australian Bureau of Statistics, 1988a, 1988b). The conditions in the foetus or infant that most often cause perinatal death are hypoxia, especially

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1 Maternal hypertensive disorders can be divided into three groups: chronic hypertension predating the pregnancy; pregnancy-induced hypertension (pre-eclampsia and eclampsia) superimposed on chronic hypertension; and pregnancy-induced hypertension alone (Carlson, 1988).

2 Abruptio placentae, or accidental haemorrhage, is bleeding from the placental site owing to the premature separation of a normally situated placenta after 22 weeks’ gestation and before the birth.
in utero, other respiratory complications and congenital anomalies

PART A: PROXIMATE VARIABLES

Eight groups of proximate variables affecting the pregnant woman and
her baby are postulated to contribute to perinatal death and its
stated cause(s). These effects may be direct or indirect, so that
interactions occur among the proximate variables themselves. The
proximate variables are discussed in retrospective order of their
likely, temporal causal effects, that is, moving backwards in time.

2.2 POSTNATAL CARE

The causes of perinatal death are mainly prenatal in origin. Yet
survival possibilities for neonates can also be influenced by
postnatal factors pertaining to health care - that is, appropriate
paediatric evaluation and management - and other non-medical aspects
of caring for neonates such as the nutrition and hygiene of babies.

2.2.1 POSTNATAL HEALTH CARE

Standard aspects of postnatal care, most importantly in the period
immediately following delivery, include clearing the airway and
thermal protection. However, severely asphyxiated infants may benefit
only from intensive resuscitation and special observation and
treatment, both of the causes of their depression and of anticipated
sequelae. The main difficulty for specialist caregivers and parents
can be to decide if, or, in terms of currently accepted practice, more
usually when, some infants should not be treated or should have their
treatment withdrawn.
Three situations in which withholding treatment may be considered reasonable were agreed on by two international symposia that convened in Sydney in November 1985 and November 1986 to consider the problems of very low birthweight infants. These situations were that treatment be unable to prevent, first, death that is inevitable and imminent, or secondly, suffering by infants that is believed intolerable. The third situation is that major disability is expected to result from continued treatment (Buchanan, 1987).

The last situation is probably the most polemical, and the decision not to begin treatment, or to discontinue it, is the result of a complex of medical, ethical, legal, philosophical and economic concerns. Since, in theory, treatment should permit more infants to survive at least the neonatal period, discussion is needed of how these concerns affect decisions of whether to treat.

At the base of medico-ethical consideration is the difficulty that many treatment regimes are experimental, and the birthweight and other diagnostic indicators at birth can then make accurate prognoses extremely difficult (Yu, 1985). Outcomes of neonatal morbidity are often not known until years after birth (Braunack-Mayer, 1986). This may be interpreted as a strong justification for treatment, yet experimental regimes have produced some disastrous consequences such as retrolental fibroplasia, and every day doctors watch the distress to families produced by the intensive care of neonates whose prospects for survival or minor disability are uncertain or believed poor. On the other hand, the outcomes of intensive care, viewed retrospectively, are not discouraging.

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3 This is defined by Yu (1985: 353) as 'cerebral palsy, blindness, sensorineural deafness, developmental delay on psychometric testing, epilepsy and hydrocephalus'.

4 Blindness results from this abnormal proliferation of fibrous tissue behind the lens of the eye, which has been attributed most often to an overadministration of oxygen.
Advances in neonatal intensive care technology, and attendant improvements in the survival of the smallest infants, have led to increased incidences of major disabilities, such as cerebral palsy (Stanley and Watson, 1988), in very low birthweight infants that might previously have died. However, the same technology has permitted a normal childhood for other infants that might otherwise have survived with a handicap or died (Yu, 1985). In developed countries the handicap rate for very low birthweight infants has remained stable and relatively low (Stewart et al., 1981). Cerebral palsy rates actually fell in Western Australia for neonatal survivors who were born between 1960 and 1982 with birthweights less than 2,000 grams (Stanley and Watson, 1988).

Legal and philosophical considerations may also influence decision-making. According to Lipman (1986), at common law and under current Australian state criminal codes, a conviction of murder could be laid against medical practitioners who fail to provide infants with 'the necessaries of life ... or [to] do any act undertaken to be done, the omission of which would be dangerous' (p. 291). Lipman adds, however, that prosecutions are likely to be few because it may, in practice, be difficult to prove causation and neglect of legal duty with respect to the requirement of the Actus Reus.6

Philosophical approaches, based on definitions of personhood, view the right to care in terms of the moral and ontological statuses of premature infants. Disagreement within this paradigm has prevented the development of moral norms for use in resolving neonatal dilemmas. The utilitarian perspective resolves these dilemmas in terms of what is best for the greatest number (Braunack-Mayer, 1986). In this

5 Cerebral palsy is a developmental abnormality of the brain producing weakness and incoordination in the limbs.
6 The Actus Reus is the physical element of an act forbidden under criminal law, as distinguished from the mental element of the mens rea.
calculus, the interests of the infant are important, but less so than those of the family and society.

Families may, for example, be emotionally and financially incapable of caring for a severely disabled child, and so they may withhold consent for treatment. In these circumstances, support for parental authority argues that the baby should be allowed, or helped, to die - not, for example, by failing to provide nourishment but perhaps by not undertaking a possibly life saving surgical operation. Doctors who feel unable to accept this position may threaten legal action for authority to treat, although in Australia, recourse to the law has been rare (Buchanan, 1987). Utilitarian approaches further recognize societal interests, which in recent years have constrained economically the care neonatalogists can provide.

Limited resources and financial claims from other medical and non-medical agencies may require practitioners, in theory, to balance the expected benefits of intensive care for any individual neonate against the very large financial costs of care. In section 6.2, the increased demands for treatment are shown to have sometimes exceeded in New South Wales the supply of beds and of attendant specialist care. In these circumstances, choices can become necessary between infants according to their expected, comparative abilities to benefit from the treatment they need.

When the expected outcome is promising but suitable care is not available on site, neonates may be transferred to a (different) regional centre where the necessary intensive care can be given. Although this has been found to improve outcomes (see section 6.3), despite risks including intracranial haemorrhage, postnatal transfers

7 This authority was supported by the Australian Medical Association in its submission to the Law Reform Commission of Western Australia in 1982.
8 Intracranial haemorrhage is bleeding within the skull.
have diminished in number. This is because complications can usually be foreseen antenatally and postnatal transfer allegedly increases birthweight-specific perinatal death rates compared with in utero transfer (Lobb et al., 1983; Crowley, 1985). In fact, comparing survival rates for babies transferred antenatally vis-à-vis after birth is of 'little value - because babies selected for neonatal transfer are generally more ill' (Chiswick, 1982: 83). There is also a limit to the number of postnatal transfers that can be undertaken. In part this is because of the emphasis on the antenatal transfer of babies, of which only a small proportion subsequently require definitive ventilatory support (Chiswick, 1982; and see section 6.2).

In the final analysis, the views and recommendations of physicians necessarily dominate decisions about how best to treat infants in need of intensive care. However neutrally health care providers give parents available information about the likelihood of severe disability, and involve them in decision-making, it is the former who make the threshold decisions about viability and outcome chances on which participants usually need to act (Fleischman and Rhoden, 1988).

For the doctors, each case is considered separately, and different doctors, especially in disimilar hospitals, may not respond in the same way to the same case - since there may not be right or wrong answers. Nevertheless, as a broad guiding statement of principle, it may be helpful to recall the position of Ian Kennedy,9 at the Sydney 1985 Symposium, that 'it may well be necessary in most cases to "treat for living" [when] there is as yet no evidence to treat for dying' (Buchanan, 1987: 184). Not to give treatment in these circumstances is perhaps the risk factor for perinatal death.

9 At the time of the symposium, Ian Kennedy was Professor of Medical Law and Ethics, Kings College London.
2.2.2 POSTNATAL GENERAL CARE

In the infant, non-medical aspects of postnatal care include adequate nutrition, hygiene and sleep. In societies such as Australia, the contribution of these factors to perinatal mortality is likely to be minimal, but deserving of some discussion nonetheless. For example, developed societies tend to perpetuate the belief that although breastfeeding is best, artificial substitutes are almost as good. However, this view is mistaken, except for the very smallest babies (Widdowson, 1987), if Minchen (1985: 29) is correct in arguing that 'the risks of formulae [for disease and inadequate growth] are unacceptable except where truly unavoidable.'

These risks result from differences between human milk and artificial formulae in nutrient composition and in immunological and food tolerance factors. Only in rare situations, as with exposure to toxins, is formula feeding preferable to breastfeeding (Berglund et al., 1985; Minchen, 1985). In recent years, about four-fifths of Australian mothers were practising breastfeeding upon discharge from hospital, but rapid weaning occurred soon after the return home (Palmer, 1984).

Bathing the neonate further reduces risks for neonatal morbidity and death. Bathing promotes cleanliness and permits both exercise without clothing and assessment of levels of growth and development. Washing clothes and bedding is important to prevent infection by viruses which have been implicated, for example, as a partial cause of the sudden infant death syndrome\(^\text{10}\) (Knight, 1983). Also associated with the syndrome are complications of sleep. Infants who experience periods

\(^{10}\) Sudden infant death syndrome, also called cot death, is the death of an infant, usually overnight in its cot, from an unidentifiable cause. Such deaths occur from three weeks of age, but most often between one and four months, that is, after the perinatal period.
of prolonged sleep apnoea\textsuperscript{11} may enter a descending spiral of respiratory activity which can lead to sudden infant death (Knight, 1983).

2.3 NEWBORN'S PHYSIOLOGICAL MATURITY

A strong relationship exists between perinatal death and the endogenous, physiological condition of the newborn. The premature conceptus\textsuperscript{12} is at the greatest risk because of the relative immaturity of its different organ or physiological systems. Difficulties in surviving the delivery and extrauterine life increase directly with the degree of this immaturity. Postmature infants\textsuperscript{13} also face increased dangers, for instance of the postmaturity syndrome.\textsuperscript{14}

2.3.1 BIRTHWEIGHT

Although the largest infants face increased perinatal risks, low birthweight is a much more frequent cause of perinatal death. Proportions of low birthweight infants have fallen only slightly in countries reporting substantial declines in perinatal mortality, and with social and medical progress having reduced numbers of perinatal deaths among infants of normal birthweight in particular, the contribution of low birthweight to perinatal mortality has increased. This is despite the successes of recent decades in salvaging through newborn intensive care the lives of infants even with very low birthweights. Thus in the United States, for instance, low

\textsuperscript{11} This is a cessation of breathing.
\textsuperscript{12} Prematurity is a purely descriptive term, generalizing the non-specific process by which a foetus or infant is born of low birthweight because it is born preterm and/or growth retarded.
\textsuperscript{13} Postmaturity, except when associated with the postmaturity syndrome (see footnote 14, chapter 2) is a synonym for post-term, which refers to neonates born after 41 weeks’ gestation.
\textsuperscript{14} The postmaturity syndrome is a set of related conditions associated with decreased placental function.
birthweight babies still accounted for two-thirds of neonatal deaths in the early 1980s whilst infants weighing less than 1 500 grams accounted for one-half (Behrman, 1986; and see section 3.5).

Low birthweight is often ascribed to 'prematurity'. However, this designation is unsatisfactory when it is important to know whether an infant is small because of a shortened gestation, retarded foetal growth in utero, or both complications. A distinction is then required between these mechanisms.

Preterm births are discussed in sections 2.6.1 and 5.1.1. Intrauterine growth retardation (see also section 5.1.1) produces high-risk pregnancies, mainly because foetuses that are growth-retarded, or small for gestational age, are more likely to suffer from intrauterine hypoxia or birth asphyxia than are normal or preterm babies (Adams, 1983). These complications are merely compounded if the gestational period is short. Numerous factors are responsible for foetal growth retardation, the most common including vascular problems in the woman such as those associated with the hypertensive disorders; maternal smoking; placental insufficiency of unknown aetiology; chromosomal abnormalities such as trisomies 13 and 18; and twins after about 32 weeks' gestation (Milstein, 1981).

Increased risks affecting large for gestational age infants are those, for example, characterizing the postmaturity syndrome. This can cause infants, because of placental dysfunction, to have acute hypoxaemia and aspirate meconium (Milstein, 1981).

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15 Trisomies 13 and 18 are characterized by the presence in each cell of three chromosomes 13 and 18 respectively, instead of the usual two.
16 Hypoxaemia defines an abnormally low concentration of oxygen in the blood.
17 Meconium is the first stools of the conceptus, the passage in utero of which suggests foetal distress.
2.3.2 NEWBORN’S CONDITION AT BIRTH

Apgar scoring\(^{18}\) provides a simple measurement of the physiological condition and viability of infants during their first minutes after birth. Scores permit comparison of the responsiveness of different populations of infants to resuscitation and other forms of treatment, and provide basic, cross-sectional measures of infants’ well-being and perinatal mortality risks. Low Apgar scores are a risk factor for perinatal death, but whether low scores at one or five minutes best portend perinatal mortality requires investigation (see section 5.1.3).

Apgar scores have sometimes been criticized for their inability to reflect the degree of acidosis at delivery, and hence to indicate asphyxia, and to predict long-term neurological handicap (Nelson and Ellenberg, 1981; Sykes et al., 1985; Low et al., 1985). However, the risk factor for perinatal death is less the Apgar score itself, which was never intended for these purposes, than the undue importance at times attached to it. Optimal evaluations of infants’ conditions after delivery require combining Apgar assessment with other methods including electronic foetal heart rate monitoring (although see section 2.4.2) and biochemical parameters including blood gas measurements and cord blood pH testing (Page et al., 1986; Saling and Arabin, 1988).

2.3.3 NEWBORN’S GROWTH AND DEVELOPMENT

After birth, perinatal risks improve with the physical development of

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\(^{18}\) Virginia Apgar’s (1953) widely used assessment of the condition of the newborn at birth is based on five objective signs: heart rate, respiratory effort, muscle tone, response to stimulation, and colour. At one and five minutes after the birth, each sign is given a score of 0, 1 or 2, and the sum of the five scores is the Apgar score.
light-weight\textsuperscript{19} and, in particular, immature\textsuperscript{20} infants. This necessitates close surveillance of neonatal growth and development, something which also applies, of course, if less urgently, to normal newborn. Disturbances in growth and development outside normal variability may indicate inadequate nutrition or disease in the infant, these states requiring correction to increase survival chances.

\textbf{2.4 INTRAPARTUM HEALTH CARE}

Perinatal deaths, specifically intrapartum\textsuperscript{21} and postnatal deaths, and their immediate precursors, postnatal care and the physiological condition of newborn, are affected, for example, by exogenous factors in the pregnant woman’s environment. These factors include intrapartum health care, the effects of which operate singly and in combination with other proximate factors. Risk factors in intrapartum care are mainly iatrogenic mishaps,\textsuperscript{22} which may be influenced by patients’ social class and place of residence (see sections 2.11.3 and 2.11.4), but which reflect, most importantly, the possibly inappropriate use of medical technology.

Medical advances, especially over the last quarter of this century, have helped to eliminate uncertainty in the diagnosis and treatment of high-risk pregnancies and, in doing so, to contribute to global reductions in perinatal mortality (Foster, 1981; Bowes, 1981; Budetti and McManus, 1982). However, the new technologies have also led to widespread medical intervention especially in intrapartum care to

\textsuperscript{19} Light-weight infants are the same as low birthweight infants; that is, they weigh less than 2 500 grams at birth.
\textsuperscript{20} Strictly speaking, immature infants have birthweights between 500 and 1 000 grams (see Figure 1.0), although the term immaturity is sometimes used in the more general sense of not physiologically mature.
\textsuperscript{21} Intrapartum events are those occurring during labour and delivery.
\textsuperscript{22} An iatrogenic mishap is ill-fortune resulting from the unforeseen or inevitable side-effects of a physician’s words or actions.
avoid unanticipated problems. Some of the technologies, such as the use of analgesia and anaesthesia, have been routinely used even though they can produce serious complications. Perhaps more disconcerting, in terms of the likelihood of complications, has been the extension to low-risk patients of procedures intended originally for use only in high-risk circumstances.

Critics of this practice of 'just-in-case' obstetrics have argued that in routine cases the potential dangers of specialized procedures such as internal foetal heart rate monitoring and caesarean section outweigh the benefits. However, the rejoinder is that the 'low-risk' categorization is a retrospective diagnosis in a litigious age that places increasing emphasis on the survival and health of the baby and its mother. In high technology medicine, obstetricians have sought protection against the possibility that they could have done more to prevent any mishap.

The following discussion considers the value and hazards of intrapartum technologies, and of the hospital birth settings to which medical technologies are conducive. The concerns of the literature, rather than necessarily of the writer, yield an overall picture that is negative, and sometimes polemical. It is important to appreciate that intervention causes complications only very rarely, and, as stated above, is used to diminish risks in women whose risk status is not clear. The need remains, nevertheless, to assess critically the outcomes of invasive procedures such as caesarean section particularly for the women who are identifiably low-risk - among whom the incidence of these procedures should be able to be safely reduced. The key difficulty is assessing the risk status of individual women.

23 The primary function of analgesia is to remove pain; that of anaesthesia is to remove sensation.

24 The caesarean section is a surgical operation for delivering the infant, placenta and membranes through the abdominal and uterine walls.
2.4.1 ANALGESIA AND ANAESTHESIA

Analgesic and anaesthetic drugs have long been used to relieve pain and anxiety during labour and delivery. This is important not only for the comfort of the woman, but because maternal distress increases the release of catecholamines which, among other things, cause vasoconstriction of the uterine vasculature and foetal deoxygenation. However, commonly used modern drugs can also produce these same complications which they were designed to eliminate.

Regional anaesthesia such as the epidural block exemplifies this point. Its arterial vasoconstrictive properties decrease uterine blood flow, and consequently hypotension, which can result either directly from the anaesthesia or from the supine position, diminishes the foetal oxygen supply. Under such conditions, spontaneous uterine contractions can evoke foetal distress by compressing the uterine vessels. Pain relief and the supine position can weaken the

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25 Catecholamines are chemical substances affecting the functioning of the sympathetic and central nervous systems. The sympathetic nervous system controls involuntary responses to alarm, deciding on 'fight or flight'.

26 Vasoconstriction means a decrease in the diameter of the uterine blood vessels, especially the arteries.

27 Regional or local anaesthesia relieves sensation from a limited area or region of the body. In contrast general anaesthesia, through achieving total unconsciousness, removes feeling from all of the body.

28 The epidural block involves the continuous infusion of an anaesthetic agent into the epidural space between the outermost membranes covering the spinal cord and the bony column of the spine. The intended effect is to numb the sensory nerve fibres from the uterus, and eliminate pain and sensation during childbirth.

29 Hypotension occurs when the arterial blood pressure is abnormally low.

30 In the supine or lithotomy position, the mother delivers her baby while lying on her back.

31 Foetal distress is suspected from certain changes in the foetal heart rate (F.H.R.), the passage of meconium, excessive foetal movements and the funic souffle - a high pitched sound synchronous with the foetal heart rate. Worrisome changes in the F.H.R. include the baseline F.H.R. (recorded between contractions) falling below, or exceeding, 100 and 160 beats per minute respectively; loss of beat-to-beat variability; and, especially, either late F.H.R. decelerations following the onset of contractions, or delayed recovery of the F.H.R. to its baseline at the end of contractions. Confirmation of foetal distress during labour may depend on foetal scalp pH determination (Myles, 1968; Goodlin, 1981).
contractions, but uterine stimulants, which may then be needed to augment labour,\textsuperscript{32} further impair oxygen delivery to the baby.

To reduce the amount of analgesia and anaesthesia required during labour and delivery, psychoprophylactic methods\textsuperscript{33} have been devised and popularized over recent decades. However, they have brought their own potential problems such as maternal hyperventilation,\textsuperscript{34} which has been associated with foetal acidosis (Albright, 1982).

2.4.2 FOETAL MONITORING

Internal electronic foetal heart rate monitoring\textsuperscript{35} can facilitate the early identification of foetal distress: amniotomy\textsuperscript{36} can show meconium staining and the monitoring per se will reveal an abnormal foetal heart rate pattern which, in the presence, for example, of a low foetal scalp pH, may be considered pathognomic of foetal asphyxia. The diagnosis of foetal distress allows intervention to be quickly initiated, so that, according to LaSala and Strassner (1986), internal foetal heart rate monitoring is a key reason for the decline in intrapartum foetal mortality. Yet the procedure, including rupture of the membranes and the attachment of an electrode to the foetus, is not without risk.

Amniotomy allows bacteria to enter the uterus, and can induce or

\textsuperscript{32} Augmentation is the stimulation of uterine contractions after labour begins spontaneously. This can be achieved by amniotomy and, if necessary, oxytocic stimulation.
\textsuperscript{33} Psychoprophylaxis uses breathing and relaxation techniques rather than drugs to cope with the pain of labour contractions.
\textsuperscript{34} Hyperventilation, or breathing at an abnormally rapid rate, lowers the carbon dioxide concentration in the blood.
\textsuperscript{35} Internal electronic foetal monitoring requires rupturing the membranes in order to obtain a foetal electrocardiogram from a spiral electrode attached to the foetal presenting part.
\textsuperscript{36} Amniotomy is the surgical rupture of the amniotic membranes surrounding the baby in the uterus.
augment labour\textsuperscript{37} - which must not be contraindicated (see section 6.6).

A major risk of low amniotomy\textsuperscript{38} is prolapse of the cord when the presenting part is a poor fit. In addition, uterine decompression, when excessive, can lead to placental abruption in patients with polyhydramnios, and amniotomy can elicit changes in the foetal heart rate. Foetal distress appears to increase delivery by caesarean section (Eddington et al., 1975; Haverkamp et al., 1976; Kelso et al., 1978), although monitoring plus foetal blood sampling to confirm hypoxia has been observed to reduce caesarean incidence (Haverkamp et al., 1979).

The attachment of electrodes to the foetal scalp can lead to scalp infection, haematoma\textsuperscript{39} and osteomyelitis\textsuperscript{40} (Atlas and Serr, 1976; Haverkamp et al., 1976; Okada et al., 1977); indeed Ashkenazi et al. (1985) reported scalp complications in 41 per cent of monitored neonates. Further potential dangers include uterine perforations when introducing the catheter into the uterus, plus injuries to the foetus resulting from errors in attaching the electrode (Abouleish, 1977; Petty, 1979).

These risks of internal foetal monitoring raise questions about its safety in identifiably low-risk patients, particularly when the technique is used without foetal blood analysis. Cardiotocography alone cannot permit a reliable diagnosis of intrauterine hypoxia (Saling and Arabin, 1988), comparable results having been obtained through auscultation\textsuperscript{41} (Haverkamp et al., 1976).

\textsuperscript{37} Induction of labour is the deliberate initiation of uterine contractions before their spontaneous onset.
\textsuperscript{38} Low amniotomy involves rupture of the forewaters with a forceps such as Kocher's.
\textsuperscript{39} A haematoma is a solid swelling produced by the clotting of blood, which has accumulated within the tissues.
\textsuperscript{40} Osteomyelitis is an inflammation of the bone marrow due to infection.
\textsuperscript{41} Auscultation is the process of listening, usually with the aid of a stethoscope, to sounds produced by the movement of gas or air within the body.
2.4.3 CAESAREAN SECTION AND INSTRUMENTAL DELIVERY

Generally, caesarean section is the appropriate mode of delivery when the risks it imposes on the woman or baby are smaller than those of vaginal delivery (Chamberlain, 1984a). Unfortunately, this decision is more often evaluative than based on absolute indications, and a plethora of studies has arisen to lament the continued increase over recent decades in delivery by caesarean section.

These increases typify modern obstetric practice in many developed countries, two exceptions being the Netherlands and Czechoslovakia. The incidence of caesarean section is conspicuously high in the United States where it has increased from 5 per cent of deliveries in 1970 to 20 per cent in 1983 (Taffel et al., 1985). Many have expressed concern at this rise, and at the convergence of rates in most countries toward this upper level, mainly because the effects of caesarean section on perinatal mortality remain unclear, while the risks to the woman are increased: reported risks of maternal death associated with caesarean section range from two to 26 times, depending on the risk status of the group of women being studied (Evrard and Gold, 1977; Cotton, 1981; Biggs, 1984).

Increased rates of caesarean section have paralleled the decline in rates of perinatal death, but the association may be incidental rather than causal since excellent perinatal outcomes have been reported when caesarean section rates fall below 10 per cent (Bergsjo et al., 1982; O'Driscoll and Crowley, 1983; Porreco, 1985). For specific groups of infants, results are often also inconclusive. Doyle et al. (1985) describe the beneficial effect of caesarean delivery for the very low birthweight singleton breech. Yet other research (for example Olshan

42 In about 4 per cent of infants the presentation, which is the part of the infant born first, is the buttocks (frank breech) or more rarely the feet or knees (footling breech). Normally, the head presents first (cephalic
et al., 1984) shows delivery by caesarean section to confer no benefit to infants weighing 700 to 1,500 grams at birth, regardless of the presentation (see section 6.8).

There are, moreover, the known risks to the baby of the caesarean operation even under epidural anaesthesia. Caesarean sections involve major abdominal surgery which can lead to serious complications such as postoperative infection of the infant (Nielsen, 1986). Caesarean sections predispose to the respiratory distress syndrome43 (White et al., 1985) and persistent pulmonary hypertension44 (Heritage and Cunningham, 1985).

Why then do high caesarean section rates persist in so many countries? Identified reasons are social and obstetric factors, the social and demographic characteristics of maternity patients being discussed first. Higher rates of caesarean section have sometimes been observed among private patients than among public patients (Biggs, 1984, Chalmers, 1985; Porreco, 1985), which has been explained by the additional financial burden of caesarean delivery for women without health insurance, and by higher fees paid to physicians for this method of delivery (National Institutes of Health Taskforce, 1981).

However, in Australia, such arguments have become less tenable over time. Since February 1984, the Medicare system, which is Australia’s universal health insurance scheme, has reduced the cost of health care for patients in private hospitals who do not own separate health presentation), but the side, shoulder or placenta can also be the first parts to appear.

43 The respiratory distress syndrome, also known as hyaline membrane disease, is characterized by incomplete expansion of the newborn infant’s lungs (atelectasis). This results from a lack of surfactant, a wetting agent needed for the air sacs to slide apart for the next breath, and not stick together. 44 With pulmonary hypertension, there is raised blood pressure within the blood vessels supplying the lungs.
insurance. Moreover, since August 1987, the same obstetric fee has been charged for all types of deliveries (Commonwealth Department of Health, 1989). The effects of these changes on national rates of caesarean section is unclear. But in Tasmania, where the caesarean section rate has continued to be lower than in the mainland States, the effect is negligible, the caesarean section rate having increased from 12.6 per cent in 1987 to 12.9 per cent in 1988 (Marsden and Correy, 1989).

Alternatively, reduced birth rates might have contributed to rising caesarean section rates by giving increased emphasis to the healthy survival of newborn, and by leading to more births to women at mature ages, especially older primigravidae. Caesarean sections give hospitals and doctors increased control over time and resources, and have sometimes been partly explained as defensive medicine prompted by a fear of litigation.

This may be most important in the United States where malpractice suits have seen juries award large sums against doctors as damages for negligence, usually for failure to perform an allegedly necessary caesarean section. Only part of the cost of professional liability insurance can be passed on in fees to clients, so that to prevent litigation, caesarean deliveries might have occurred both earlier during labour and more frequently (Meehan, 1988). However, a major effect of this type was rejected in the United States by a caesarean birth national task force which claimed that appropriate informed consent practices were protective for doctors (National Institutes of Health Task Force, 1981).

45 Patients in a private hospital who do not own health insurance receive from Medicare the standard 85 per cent reimbursement on antenatal care and a 75 per cent reimbursement on care received during the confinement. However, these patients pay the full cost of accommodation in a private hospital.

46 Primigravidae are women pregnant for the first time.
The second set of reasons for rising incidences of caesarean section relates to obstetric factors. Doctors have readily invoked indications for caesarean section including prior caesarean section; failure to progress;\textsuperscript{47} malpresentations,\textsuperscript{48} especially breech; and foetal distress (Cotton, 1981; Anderson and Lomas, 1984). At least in second stage labour,\textsuperscript{49} this liberalization has occurred because the caesarean operation, made safer and easier by, among other things, the increased use of epidural anaesthesia and electronic foetal heart rate monitoring, has been seen to present a lesser risk to the foetus than delivery with instruments.

The forceps operation,\textsuperscript{50} for example, brings the risk of intracranial haemorrhage and other bodily damage to the infant, although any such haemorrhage may precede rather than follow the application of forceps. Increased foetal and maternal morbidity has been associated particularly with the use, or perhaps the misuse, of Kielland’s rotational forceps (Cotton, 1981, O’Driscoll et al., 1981; Chow et al., 1987), whilst a disconcerting association exists between use of the vacuum extractor\textsuperscript{51} and both shoulder dystocia\textsuperscript{52} and retinal haemorrhage (Benedetti and Gabbe, 1978; Egge et al., 1981).

However, until the 1980s, rates of forceps operations increased in concert with many of the same developments in medical technology, such

\textsuperscript{47} The inability of the foetus to progress through the birth canal results from two main causes: the head is too large for the pelvis through which it must pass (cephalopelvic disproportion) or the contractions of the uterus are weak, irregular and poorly coordinated (uterine inertia).

\textsuperscript{48} A malpresentation is any presentation other than the vertex (the top of the head).

\textsuperscript{49} Second stage labour begins with complete dilation of the cervix and ends with delivery of the infant.

\textsuperscript{50} Forceps are instruments used to help deliver the baby toward the end of the second stage of labour.

\textsuperscript{51} The vacuum extractor or ventouse is a metal suction cup that is attached to the baby’s scalp in order to aid delivery. In European countries, excluding the United Kingdom, this device has often been preferred to forceps.

\textsuperscript{52} With shoulder dystocia the shoulder is too large for the pelvic outlet through which it must pass (see footnote 56, chapter 2).
as epidural anaesthesia,\textsuperscript{53} that have acted to elevate rates of caesarean sections (Ratten, 1985); and Need (1987) reports increased use of the ventouse since 1983 at the Flinders Medical Centre in South Australia. Yet it has not been proved that instrumental delivery is more hazardous, and less effective, than many caesarean sections and, as is now discussed, there is experimental and population-based evidence suggesting that caesarean deliveries may be safely reduced for some indications.

The dictum 'once a section, always a section' no longer holds good because most modern caesareans are performed with a low transverse incision. This makes safe a subsequent trial of labour\textsuperscript{54} in selected patients as the risk of uterine rupture is small. A trial of labour is also relatively efficacious. Despite the relatively high proportion of repeat caesareans in Norway, 43 per cent of Norwegian women with a previous caesarean section delivered vaginally in 1980 (Notzon et al., 1987). Still higher levels may be feasible. Between 54 and 82 per cent of patients with a prior caesarean section in various studies have proved able to deliver \textit{per vaginam} successfully (Lavin et al., 1982; Martin et al., 1983; Jarrell et al., 1985; Paul et al., 1985; Ngu and Quin, 1985). When syntocinon is used to induce or augment labour, the safety of a trial of labour is not so clear. Some researchers, for example Horenstein and Phelan (1985), perceive it as an acceptable alternative to caesarean section. Others, such as Kishor et al. (1986), question the use of syntocinon to improve prospects for vaginal delivery.

Similarly, the breech presentation \textit{per se} may not usually warrant caesarean delivery. In Hungary, delivery \textit{per vaginam} was performed on

\textsuperscript{53} Need (1987) notes, however, that an epidural 'cocktail' of pethidine and bupivacaine appears to reduce the need for rotational forceps delivery.

\textsuperscript{54} A trial of vaginal delivery or a 'trial of scar' may be given to women who have had prior caesarean section for a nonrecurrent cause.
61.3 per cent of breech babies in 1983 while the comparable percentage for Sweden in 1981 was just 6.2 per cent (Notzon et al., 1987). However, for the premature breech, or when one or more babies of multiple gestation present abnormally, the safety of vaginal delivery is much less certain (Chevernak, 1984; Buekens et al., 1985; and see section 6.8).

Some caesarean deliveries performed for foetal distress may be eliminated by selective use and more careful interpretation of electronic foetal heart rate monitoring during labour (Cotton, 1981). In 1982, in Scotland, one in five babies experiencing foetal distress was delivered by caesarean section, whereas the ratio in the United States was 1 in 1.4.

Much smaller international differences have characterized caesarean delivery for failure to progress or dystocia. Yet research from Ireland and, more recently, from Canada (O’Driscoll et al. 1984; Akoury et al., 1988), suggests that the active management of dystocia in nulliparous women can safely and effectively reduce the need for operative delivery (see sections 6.7 and 6.7.2). Paul et al. (1985) also report the efficacy and safety of a trial of labour in women with a prior caesarean section for dystocia, although this previous indication was associated with a reduced success rate of vaginal delivery both when oxytocin was administered and compared to the other previous indications.

55 Perinatal mortality rates specific for delivery type are not available to the writer, but Sweden, as noted in section 1.2.1, has the lowest rate of perinatal death in the world.
56 Dystocia is abnormal labour resulting from deviations from normal patterns in the latent or active phases of labour (see chapter 6 footnote 38).
57 This involves amniotomy, once labour is established, followed by oxytocic augmentation if labour is nonprogressive.
58 Syntocinon, a synthetic form of the naturally-occurring hormone, oxytocin, is given to augment labour (see footnote 32, chapter 2) by causing the uterus to contract. The same drug can be used to induce or start labour artificially.
Particularly caesarean sections, but also instrumental deliveries, might have been, and may still be, used too often when intervention is not medically justified. Some women are at recognizably high risk of complications, and for these women the greatest risks may attend failure to deliver by caesarean section; indeed, an interesting ethical and legal dilemma concerns mandatory delivery by surgical means on nonconsenting women when such treatment is necessary to save the life of the foetus (Fleischman and Rhoden, 1988).

For other women, the risks of mortality (and morbidity) may be identifiably low, making little intervention necessary since, at the collective level, the side-effects in ‘low-risk’ women appear to outweigh the possible benefits. When the degree of risk is not clear, the appropriateness of intervention is debatable. However, Myers and Geicher (1988) report one way of tempering the need to avert the risk of unforeseen complications: all obstetricians in a New England hospital sought second opinions before undertaking non-urgent procedures; followed objective criteria for the most common indications; and participated in a detailed peer review. During the two-year period of these changes, the caesarean section rate fell by one-third to 11.5 per cent without a detectable effect on perinatal mortality or morbidity.

2.4.4 PLACE OF BIRTH

Sophisticated medical technologies, which are essential for high-risk deliveries but which sometimes do not improve outcomes and at worst cause complications, encourage and indeed require births in hospitals (Goodlin, 1980). Concern over this medicalization of childbirth helps to explain the growth of support for alternative childbirth settings, namely women’s own homes, or birthing centres that are freestanding or
attached to existing maternity units. Support for these alternative settings further derives from the psychological benefits they afford: increases in control over the birth experience and in family participation; relaxed surroundings; strong, perhaps 'therapeutic', relationships between women and their own midwives; and improved mother-newborn relationships - all of which, in a primary sense, may improve outcomes for low-risk parturients.

Opponents, especially of home birth, view this setting rather than hospital birth as a risk factor for perinatal death. Complications can arise suddenly and unexpectedly during the labour and delivery, and even among carefully selected, low-risk patients there is a persistent incidence of complications such as birth asphyxia which require obstetric intervention obtainable only in a hospital (Permezel et al., 1987). The avoidance of intervention can itself create complications. For example, if labour is allowed to progress beyond 24 hours without augmentation or analgesia, there is an increased likelihood of non-spontaneous delivery and meconium aspiration (Eggers et al., 1985). In Australia in 1986, 11.7 per cent (N=127) of the women with total planned home births were transferred to hospital: two babies died59 (Homebirths Australia, 1987).

The reality is that each birth setting brings its own iatrogenic risks of perinatal mortality and morbidity, some deaths occurring irrespective of where birth takes place (Spurrett, 1988). In Australia at least, studies, such as by Howe (1988), lack the statistical power to quantify the outcome risks peculiar to home and hospital delivery per se, that is, by controlling for the characteristics of the women who give birth, and who intend to give

59 Both babies died postnatally: a waterborn baby could not be resuscitated and the second baby was of low birthweight with a chromosomal deficiency not identified to the writer.
birth, in these settings. Consequently, the literature does not substantiate claims about the comparative safety of home birth (National Health and Medical Research Council, 1987, 1989). Nor does it demonstrate conclusively that, for the baby, the iatrogenic risks of institutional delivery exceed the benefits, or that, at least in England and Wales, the shift to hospital delivery accounts for the decline in crude perinatal mortality (Campbell and Macfarlane, 1986).

International, cross-sectional comparisons, although fraught with problems including differences in health care delivery, suggest that, at the aggregate level, the place of delivery is possibly of negligible importance for carefully screened low-risk women. Countries with almost 100 per cent hospitalization may have comparatively high rates of perinatal death, as in England and Wales, or low rates as most notably in Sweden. The Netherlands, with approximately 35 per cent of total births occurring at home since 1979, has one of the world’s lowest perinatal death rates (World Health Statistics Annual, 1986; Hingstman and Boon, 1988), although especially in urban districts, which have high levels of hospitalization (Hingstman and Boon, 1988).

Based on these facts, as well as increasing acceptance that all women should have the right to choose their own places of confinement, and evidence that a small but growing number of women are determined to exercise this right (National Health and Medical Research Council, 1987), the safety of home delivery will depend on the cooperation of all groups involved in the birth experience. Alternative birth settings do not appear to constitute a major risk factor for perinatal death for low-risk parturients who receive appropriate supervision including, if necessary, transfer to a suitable hospital (National Health and Medical Research Council, 1987).
ANTENATAL HEALTH CARE

A direct nexus rarely occurs between antenatal health care and perinatal death. For instance, the likelihood of an amniocentesis and more recently of chorionic villus sampling\(^{60}\) precipitating a spontaneous abortion is about one per cent and less than one per cent respectively (Hogge et al., 1986; Green et al., 1988). The connection between antenatal care and perinatal complications is thus usually mediated by other variables.

When assessing in these terms the causal contribution of antenatal health care, it is especially important to consider the amount of health care received as well as its quality or type. For two reasons, investigators have found a positive association between the quantity of care, as measured by the number of antenatal visits, and perinatal outcome (Fraser, 1983). First, preterm births suffer high perinatal mortality, but necessarily fewer clinic attendances. Secondly, low-risk women are the most likely to receive care early and regularly. The women in greatest need of care are thought to receive the least care because they lack access to, and acceptance of, available health services; this reflects these women’s lack of knowledge and financial resources. However, section 6.4 shows a different relationship between the amount of antenatal care and perinatal mortality.

Few researchers have related the quality of antenatal care to perinatal outcomes. However, whereas the most sophisticated and ostensibly highest quality intrapartum care may too often act against the interests of, in particular, high status women (see section

\(^{60}\) This is the sampling of cells from the fingerlike projections that arise from the membrane surrounding the embryo after implantation.
2.11.3), the best antenatal health care affects perinatal risks favourably.

This is because obstetric interventions before the onset of labour are typically safer than those during labour and delivery and they are perhaps more often associated with diagnostic and preventive action than with the treatment of disease or disorders. The role of health professionals during pregnancy *per se* is often advisory, for example about pregnancy health, and there may be greater opportunities than during the confinement for physicians to respond to what patients actually want. The literature is replete with evidence (for example, Behrman, 1985) of the benefits of appropriate antenatal care.

More appropriate, therefore, is evidence that high quality antenatal care is received by too few women, and that the receivers of the least care are frequently the ones who need the most. In general, medical practitioners appear to have failed to educate women adequately about pregnancy health (Fraser, 1983), and sometimes to have identified women at risk of complications. In part this reflects the unpredictable nature of many obstetrical emergencies. Some physicians might not have taken account of all available information (Chng et al., 1980; Hall et al., 1980), and have neglected to perform antenatal investigations with important screening and diagnostic roles.

An example of an investigation undertaken less frequently than perhaps it ought to be is glucose tolerance testing between 28 and 30 weeks' gestation. Routine performance of this test in order to identify gestational diabetics may, according to a report from Victoria, Australia, reduce the incidence of unexplained deaths *in utero* near term (Consultative Council on Obstetric and Paediatric Mortality and Morbidity, 1986). More generally, the Council identified inadequate
antenatal monitoring as the main, potentially avoidable cause of stillbirths.

One reason physicians do not more often undertake antenatal tests, such as foetal surveillance, is that critics continue to question their safety and efficacy. The last decade has seen the growing replacement of biochemical tests, for instance oestriol tests, with biophysical monitoring of foetal risk based on foetal heart rate tracings. However, the value of cardiotocographs, including the nonstress test and the contraction stress test, has now also been challenged. Thacker and Berkelman (1986), after reviewing published studies on both methods and on foetal movement monitoring, found the tests to have limited diagnostic accuracy and efficacy. The tests demonstrated high rates of false positivity and low sensitivity, and their specificity, whilst usually over 80 per cent, exceeded 90 per cent in less than half of the reported studies. Biophysical profile scoring (see Manning, 1985), has become the most successful method currently available for appraising the status of the high-risk foetus.

2.6 OBSTETRICAL FACTORS

Obstetrical factors, being the clinical parameters of pregnancy, fall into two groups. The first group is physiological factors, while the second group, labelled maternal disease, emphasizes pathological risk factors. Both groups are mainly endogenous variables, although, for

61 Oestriol tests permit assessment of placental function and foetal growth.
62 The nonstress test uses unstressed F.H.R. tracings to detect abnormalities.
63 The contraction stress test relies upon the F.H.R. response to induced or spontaneous contractions.
64 False positivity relates the frequency of a good outcome to patients with positive test results.
65 Low sensitivity refers to the poor ability of a test to detect abnormal outcomes.
66 Specificity is the probability of a negative test result when the outcome is good.
example, physiological factors such as preterm birth could in some individuals be defined exogenously. Obstetrical factors produce perinatal death or its stated cause(s) directly or through the mediating effects of other proximate variables.

2.6.1 PHYSIOLOGICAL FACTORS

The physiological characteristics of five obstetrical factors influence perinatal survival. The first factor is the sex of the foetus or infant. The remaining factors, which can be associated with pregnancy difficulties, are pregnancy plurality; relations of the foetus to the uterus; maternal weight change; and the period of gestation.

Sex of the Foetus or Infant: The sexes have differential perinatal mortality. The higher intrauterine loss of male foetuses reduces the primary sex ratio\(^67\) of about 110, to a birth, or secondary, sex ratio of approximately 104. In part this reflects the tendency for boys to deliver spontaneously at earlier gestational ages than girls (Hall and Carr-Hill, 1982; and see section 5.2.2). This greater physiological maturity of girls outweighs presumably the more rapid growth of male foetuses after the 24th gestational week; so that by term, boys weigh 150 to 200 grams more than girls (Aubry et al., 1978; Brar and Rutherford, 1988).\(^68\)

In addition, there is uncertain evidence of a preponderance of male foetuses in pregnancies complicated by hypertensive disorders of pregnancy (Chesley, 1978; MacGillivray, 1983), placenta praevia (MacGillivray, 1986; Mills et al., 1987) and premature rupture of the membranes (MacGillivray and Davey, 1985; Seki and Kato, 1987) (see

\(^{67}\) The primary sex ratio is that found at conception.

\(^{68}\) Of course, this range will vary, for example, with the race, parity and physical stature of the pregnant woman.
sections 5.3 to 5.3.3). Instrumental and operative deliveries are performed most commonly for boys (Hall and Carr-Hill, 1982), and during the neonatal period, male mortality exceeds that in females (Chen, 1983).

By contrast, female foetuses are at increased risk for some complications. Girls are more likely than boys to be breech births, to have their mother's labour induced (Hall and Carr-Hill, 1982) and to be anencephalic.69 Such risks are most salient (although, relative to total births, numerically least important) in multiple pregnancies because as the number of infants born at one time increases, so does the proportion of females (Green, 1970).

Pregnancy Plurality: The multiple pregnancy increases the risk of perinatal death, mainly by diminishing foetal growth and maturity. One reason for this is that the gestational period tends to be shortened. Few triplets are carried to term and a study in Aberdeen, Scotland, found preterm deliveries to be associated with 28.2 per cent of twin pregnancies (N=624) from 1968 to 1977 (MacGillivray et al., 1982). A second reason is that after 32 weeks, twins generally are small for gestational age, their weights dropping to about the tenth percentile (Milstein, 1981). This early onset of growth deceleration is ascribed by McKeown and Record (1952) to strains on the maternal blood supply, but reduced growth rates can also result from the altered conduction through the intervillous space70 produced by the lowered mass of separate placentas (Adamsons and Myers, 1975).

69 Anencephaly is characterized by poor development of the cranial vault and cerebral hemispheres.
70 The intervillous space is the placental space between or among tiny chorionic villi (branching structures which arise from the outer membrane surrounding the foetus) in which maternal blood circulates.
In fact almost every risk associated with a single pregnancy, for example toxaemia, hydramnios, antepartum and postpartum haemorrhage and dysfunctional labour is increased with a multiple pregnancy (Cotton, 1981). Yet, paradoxically, despite the higher incidence of prematurity among multiple births than singletons, mortality among preterm twins is lower than among singletons of the same gestational age (Rydhstrom, 1985). According to Fabre et al. (1988), the survival advantage for twins (about two times) was only apparent at birthweights 1 500 to 2 499 grams (see section 5.2.1).

**Foetal Relations:** Abnormal relations of the foetus to the uterus include a transverse and oblique lie, attitudes other than flexion, malpresentations including most commonly the breech (see section 6.8), and positions associated with these malpresentations or an occipito-posterior position of the vertex (Myles, 1968; Green, 1970).

**Maternal Weight Change:** Since the early 1970s, pregnant women have been advised to 'eat to appetite' and to gain at least eleven kilograms. Weight gains below seven kilograms at term have typically been associated with foetal growth retardation and low birthweights, gains greater than eighteen kilograms bringing the problems associated with large babies (see section 2.3.1) (Brunel, 1981).

The recommended weight gains usually help to increase birthweights, especially for underweight women who tend to deliver smaller babies than do women with larger prepregnant body weights (Brown et al.,

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71 Hydramnios means excess amniotic fluid surrounding the foetus in utero.
72 Dysfunctional labour is defined by aberrations from the normal patterns of cervical dilatation and foetal descent.
73 When the lie of the foetus in utero is transverse, the long axis of the foetus lies across, not parallel to, the long axis of the uterus.
74 An attitude of flexion means that the limbs and head of the foetus are flexed or bent over its trunk.
75 The foetal position is the relationship of the foetal presenting part to the mother’s pelvis. An occipito-posterior position occurs when the back of the foetal head faces the back of the pelvis.
Moderately overweight women also achieve increased birthweights with maternal weight gains (Luke et al., 1981, Abrams and Laros, 1986), but, among very overweight women, total weight gains during pregnancy have been found to produce no significant effect on birthweight (Abrams and Laros, 1986). According to Pitkin (1976), more important than total weight gain is the pattern of weight accumulation. For example, women failing to achieve a minimal weight gain of one kilogram or less during the first trimester face increased risks of growth retardation in utero and of a low birthweight baby.

**Period of Gestation:** Gestational age strongly influences health care and perinatal outcome. Most worrisome are preterm deliveries since they best explain low birthweights in developed countries and contribute from 50 to 70 per cent of perinatal deaths in most data sets (Hoffman and Bakketeig, 1984). The main problem is the relative immaturity of the different physiological systems of preterm babies, which leaves these infants poorly equipped to withstand the hazards of the labour and extrauterine life. Difficulties such as poor control of body temperature, difficult respiration and an inability to handle infections are compounded when preterm births are also small for gestational age (see sections 2.3.1 and 5.1.1).

Almost three-quarters of preterm births have been found to occur spontaneously, almost half of these resulting from a maternal or foetal complication including especially a multiple pregnancy (Rush, 1976). Other spontaneous preterm births, which are of unknown cause, have been hypothesized to originate with the effect of behavioural stressors (see section 2.8) that cause the autonomic nervous system\(^7\) to release catecholamines, specifically norepinephrine. This can

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\(^7\) The autonomic nervous system, of which the sympathetic division is a part (see Footnote 25) controls bodily functions that are not consciously directed.
initiate uterine contractility and, with certain ascribed characteristics of the woman, decrease placental progesterone production\(^{77}\) which, partly by permitting prostaglandin synthesis,\(^{78}\) further encourages uterine irritability and the cervical changes leading to preterm labour (Bragonier et al., 1984). A final group of preterm births results from obstetric intervention. When the indication for delivering electively preterm is flimsy, perinatal death or morbidity may be iatrogenic. However, when continuing the pregnancy endangers the woman's life, for instance because of fulminating pre-eclampsia,\(^{79}\) perinatal complications may be unavoidable.

The risks associated with post-term births have probably been exaggerated, a lengthened gestation (42 weeks or beyond) itself usually presenting few problems. Complications such as birth trauma suffered by the large baby after a long gestation most often reflect some other condition in the woman such as toxaemia or diabetic states.\(^{80}\) Only when such a disorder already presents is pregnancy not usually permitted to proceed past term, since placental incompetence can then induce the postmaturity syndrome.

2.6.2 MATERNAL DISEASE

Some maternal diseases, for example placenta praevia, are peculiar to pregnancy, whilst others, such as the cardiac diseases, can exist, of course, before the pregnancy, and both affect, and be affected by, the pregnancy. The most widespread complications of either group are the hypertensive disorders of pregnancy (see section 5.3.1), which may be

\(^{77}\) Progesterone is an important hormone for the maintenance of pregnancy.
\(^{78}\) Prostaglandins are hormone-like substances, one effect of which is to cause uterine contractions.
\(^{79}\) Pre-eclampsia or toxaemia is characterized by high blood pressure, the swelling of certain body parts, and protein in the urine.
\(^{80}\) Diabetic states are characterized by a deficiency of the hormone insulin.
unrelated to the present pregnancy. The next most prevalent, serious complications are *abruptio placentae* and spontaneous premature rupture of the membranes (see section 5.3.3).

The last complication can create an interesting dilemma for doctors. Spontaneous premature labour usually quickly follows premature rupture of the membranes, even with tocolysis, but the question arises of how actively to seek prolongation of the latent period between membrane rupture and delivery: inhibition of labour may be necessary to allow foetal lung maturity to occur following the administration of steroids, yet this protocol increases the risk of chorioamnionitis. Also relevant are ethical considerations raised in section 2.4.3 concerning the aggressiveness of treatments.

### 2.7 CHROMOSOMAL AND GENETIC FACTORS

Perinatal deaths are often potentially preventable given the preponderance of environmental aetiologies. However, the possibilities for prevention are limited for the chromosomal and single gene aberrations that cause perinatal deaths mainly by producing recognizable birth defects and usually symmetrical foetal growth retardation. Some precautionary steps may be possible as, for example, through the completion of childbearing before the advanced maternal ages that are known risk factors for such defects as Downs syndrome; but, in any event, most babies with this syndrome 'are born to younger women because of the composition of the childbearing population' (Donnai, 1986: 15).

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81 Tocolytic therapy uses betamimetics to try to inhibit the progress of premature labour in order to improve the perinatal outcome.

82 When the growth retardation is symmetrical, the head-to-abdomen ratio is normal throughout, except for both variables being smaller than expected for the gestational age.
More promising is the larger aetiological contribution of environmental factors to many of the lethal anomalies of complex genetic aetiology such as the neural tube defect, anencephaly. Anomalies especially of the central nervous system have been associated with the pregnancy health factors discussed in sections 2.8 to 2.8.5; with maternal disease such as diabetes (Mills, 1982); with reproductive history factors; and with low social class (Baird, 1980; Janerich and Polednak, 1983). Environmental factors can also influence the genetic expression of multifactorial disorders. Diet, for example, can prevent the expression of disease in individuals with phenylketonuria (Janerich and Polednak, 1983).

2.8 PREGNANCY HEALTH

Pregnancy health factors or stressors describe the behaviour and psychological state of the pregnant woman. Those to be presently discussed are psychological stress, physical activity, physical agents, chemical hazards, and nutrition. These stressors can affect perinatal outcome, most commonly by stimulating the autonomic nervous system to release catecholamines. This can have two effects which probably vary not only with the character of the insult but with the status and responsiveness of the foetus (Reece et al., 1986).

The first effect is constriction of the uterine vessels with a concomitant reduction in the flow and composition of blood in the intervillous space - that is, in what is available for transfer to the foetus. In any foetus, a moderate reduction in uterine and placental blood flow of long duration can cause foetal growth retardation in

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83 Phenylketonuria is a rare metabolic disease characterized by the presence in the urine of phenylketones.
84 Other consequences include teratogenicity.
85 However, Clapp (1979) reported that an epinephrine-induced decrease in the rate of uterine blood flow in ten pregnant ewes did not concomitantly decrease uterine oxygen intake.
utero. A reduction in blood flow of greater magnitude but shorter duration can lead to severe asphyxia, producing foetal death or brain injury (Adamsons and Myers, 1979). The second effect of catecholamine secretion is to increase uterine irritability, which indirectly decreases placental function and can initiate a cascade of changes leading to preterm labour (Bragonier et al., 1984).

Through both sets of mechanisms, pregnancy health factors can affect perinatal outcome, for example, by promoting physiological immaturity of the foetus. Because these factors can be controlled more easily than most other risk factors, understanding their effects is especially important from the standpoint of primary prevention. At both the individual and societal levels, opportunities for change are provided by an increased commitment to a healthy lifestyle irrespective of the material standard of living.

2.8.1 PSYCHOLOGICAL STATUS

Many women suffer profound changes in their emotions at some time during pregnancy. These changes induce psychoneuroendocrinal responses86 that can affect the foetus via the placenta. Psychological stress has long been known to cause the sympathetic nervous system to redistribute blood flow away from the abdominal viscera87 to the heart, brain and voluntary muscles. Now, animal experimental studies, such as by Myers (1979), confirm that the uterus and its supplying blood vessels are also highly reactive to emotional stress. Sympathetic nervous system stimulation, secondary to psychological stress, can lead to neurotransmitter release and the discharge of catecholamines, causing the uterine vessels to constrict or, possibly, uterine

86 Psychoneuroendocrinal responses are hormonal and nervous system responses to psychological stress.
87 The abdominal viscera are the internal organs of the abdomen.
contractions to begin. Perfusion\textsuperscript{88} of the intervillous space is thus reduced as is oxygen delivery to the foetus, while the risk of preterm labour is increased (Adamsons and Myers, 1975; Myers, 1979; Bragonier et al., 1984). Psychological stress may result from life events, these being the occurrence of particular events in a person's social environment (see section 7.1) which find expression in state or trait anxieties.

**State anxiety:** State anxiety, resulting from acute or sudden stress, is anxiety as a transitory emotional state which can cause marked asphyxia of short duration with persistent foetal bradycardia. This can lead in turn to foetal death or less commonly to brain injury. High levels of state anxiety consistently contribute to obstetric complications. The relation holds for life stresses before and especially during pregnancy. Disagreement exists, however, over the period during which state anxieties are suffered by women with obstetric complications \textit{vis-à-vis} those with problem-free pregnancies. Obstetric complications associated with state anxiety include increases both in foetal heart rates and body movements; toxaemia; prematurity and light-weight babies; and intense pain during labour (Zichella and Pancheri, 1979; Wolkind, 1981).

**Trait anxiety:** Trait anxiety is chronic stress typically produced by relatively enduring personality characteristics. In these circumstances the reduction in uterine and placental blood flow is usually moderate but of long duration (Adamsons and Myers, 1975). The main effect is growth retardation of the foetus, although other obstetric complications associated with trait anxiety are long labours, intense labour pains, and increased neonatal health difficulties (Erickson, 1976; Falorni et al., 1977; Lubin et al.,

\textsuperscript{88} Perfusion here means the flow of maternal blood through the intervillous space.
1977). Some studies reveal no differences in trait anxiety between patients experiencing normal and abnormal perinatal outcomes (Edwards and Jones, 1970; Gorusch and Key, 1974; Spielberger and Jacobs, 1979). But in patients with a recognizable psychiatric disorder, the incidence of obstetric complications is rarely questioned; and the more serious the disorder, the greater these complications are (Mednick et al., 1971; Cohler et al., 1975; Zax et al., 1977).

2.8.2 PHYSICAL ACTIVITY

Four variables in the conceptual model describe the level of physical activity of the pregnant woman: exercise, work, coitus, and rest and sleep. These activities are important, mainly because at least moderate physical activity and a concomitant increase in body temperature divert blood from the splanchnic area, reducing uterine circulation and increasing uterine contractility.

Exercise: Mild to moderate exercise\textsuperscript{90} is encouraged in many women. Despite some uncertainty it probably does not seriously diminish uterine blood flow (Gorski, 1985), affect the foetal heart rate (Carpenter et al., 1988), increase uterine activity (Zahn and Raabe, 1984; Veille, 1985), or reduce birthweights (Colling et al., 1983; Hauth et al., 1982). Appropriate exercising may have psychological benefits. It has been claimed that exercise improves digestion, stimulates the lungs, promotes sleep and (although refuted, for example, by Colling et al. (1983)), provides for an easier labour and delivery (Cedeno et al., 1980).

\textsuperscript{89} The splanchnic area refers to the abdomen and its internal organs.
\textsuperscript{90} This describes a maternal heart rate of 140 to 160 beats per minute (approximately 70 to 80 per cent of the maximal heart rate) in the average pregnant woman. Strenuous activities are less than 15 minutes in duration.
Even women accustomed to sedentary lifestyles may initiate during pregnancy some, especially non-weight bearing, activities such as cycling, necessarily at low frequencies and intensities. A few sports, for example, water skiing, are specifically contraindicated during pregnancy (Lumley, 1986), but generally, the safety of exercise is relative to normal types and levels of activity (Paisley and Mellion, 1988 and see section 7.2.2). Certain guidelines for exercise during pregnancy should perhaps be applied in these terms. Such recommendations\(^{91}\) include the prohibition of activities involving ballistic movements (Gauthier, 1986) and a supine position (Paisley and Mellion, 1988).

Of course, physical activity may be contraindicated by pregnancy complications like diabetes.\(^{92}\) Even in reasonably active women,\(^{93}\) vigorous or protracted exercise may cause foetal hypoxia, secondary to decreased uterine blood flow and prolonged foetal bradycardia or tachycardia (Carpenter et al., 1988). Severe or chronic elevations in core temperature should be avoided (Gorski, 1985). According to Smith et al. (1988), teratogenic effects are induced by a threshold elevation of core temperature of 2.5° C. irrespective of the basal temperature. German (1985) reports that this threshold temperature depends on the duration of the exercise.

**Maternal Work:** Consistent with these findings is the consensus that arduous work, usually defined for operational purposes by participation in the paid labour force, contributes to low

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\(^{91}\) One set was developed in 1985 by the American College of Obstetricians and Gynecologists.

\(^{92}\) Controlled diabetes is a relative contraindication to exercise during pregnancy, which means that this condition may benefit from an exercise program that is individualized and medically supervised. Uncontrolled diabetes is a general contraindication to exercise during pregnancy.

\(^{93}\) Anecdotal evidence, without supporting scientific studies, indicates that high-performance athletes who become pregnant have much higher levels of exercise tolerance than the average pregnant woman.
birthweights and, for example, preterm births (Saurel-Cubizolles and Kaminski, 1986). Working conditions hazardous for the foetus include the woman standing continuously, lifting heavy objects, working long hours and using public transport involving lengthy commuting times (Papiernik and Kaminski, 1974; Papiernik, 1984; and see section 7.2.1). These and other variables were brought together by Mamelle and Laumon (1984) in a 'hardness of work' index - comprising posture, work on machines, physical load, mental load and the environment - which in the same study correlated positively with preterm births.

Contemporaneously, most surveys in developed countries (for example, Gofin, 1979; Murphy et al., 1984) have shown pregnancy outcomes to be favourable for working women. However, there may be some selection in that the more physically fit may participate in the workforce. Also associated with increased female participation in the workforce have been improvements in the social and economic statuses of women. These gains have reduced the housework women perform (Daniels, 1979), and contributed to the decreasing proportions of women undertaking the most physically arduous jobs. Moreover, within these latter occupations, working conditions have improved. Social legislation has brought earlier and more widespread maternity leave of longer duration than previously (Saurel-Cubizolles and Kaminski, 1986; Glezer, 1988).

In sum, work itself does not seem to be a risk factor for perinatal death. What is important in assessing risks are such factors as the type and conditions of work, the employment interruption date and the characteristics of the individual woman.

Coitus: Obstetric risks associated with physical activity are exemplified further by coitus. Because coitus is usually perceived as a form of moderate physical activity, no restrictions are placed on the routine patient. However, coitus may be unwise, particularly in
the first and last trimesters, if the pregnancy is unstable or if there has been a bad obstetric history. For instance, previous miscarriages or bleeding in early pregnancy can make coitus inadvisable.

In addition, in women with vascular disease, coitus, like exercise, can diminish intervillous space perfusion, and retard growth in utero (Haesslein, 1981a). Coitus has also been implicated through two mechanisms in the aetiology of preterm birth. First, coitus carries pathogenic bacteria\(^{94}\) to the urinary tract, or to the cervical os\(^{95}\) and thus to the amniotic membranes. Rupture of the membranes is a possible sequel (see section 5.3.3), but according to Naeye (1979), amniotic fluid infection in the presence of intact foetal membranes is the most important foetal condition contributing to death among immature infants. The second mechanism is the single or combinative action of coitus, orgasm and cervical or seminal prostaglandins in stimulating uterine contractions (Haesslein, 1981b).

Sexual contact can also lead to the transmission of infectious diseases including, very rarely, the potentially fatal human immunodeficiency virus, for which the incidence of foetal contamination is approximately 40 per cent (Henrion, 1988). Acute venereal diseases can impair growth and cause preterm birth and newborn morbidity.

**Rest and Sleep:** Bed rest and sleep are especially important for pregnant women, particularly when these women are exposed to more than everyday physical activity or to conditions, such as pre-eclampsia, that reduce intervillous space perfusion. Rest decreases maternal anxiety and, by increasing uterine blood flow when the woman lies on

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94 Pathogenic means causing disease.
95 The cervical os is the opening of the cervix into the vagina.
her side, possibly helps to increase birthweight and improve the birth outcome.

It has become customary to admit women with multiple pregnancy, for example, to hospital for bed rest. However, this last practice is controversial. Some researchers, including Hartikainen-Sorri and Jouppila (1984), suggest that in multiple pregnancy, prophylactic hospital bed rest in the third trimester promotes rather than averts the onset of preterm labour. One reason for this may be that hospitalization is stressful.

2.8.3 PHYSICAL ENVIRONMENT

Factors in the physical environment affecting perinatal survival may include ionizing radiation, ambient noise, vibrations, altitude, temperature and air pollution. Exposure to radiation from man-made sources, that is X-rays, poses potential dangers to offspring. In the perinatal period they include genetic mutations\textsuperscript{96} that, depending on the number of X-rays and their strength, type and timing during pregnancy, can cause congenital abnormalities.

Uncertain risks are associated with emissions of both ionizing and non-ionizing radiation from video display terminals. Arbitrary methods of establishing radiation standards, improper functioning of 'fail safe' mechanisms, power surges and the cumulative effect of even low levels of radiation exposure are adduced of the potential dangers of the machines (Chernier, 1982). Consideration of the thermal effect of micro-wave radiation is inadequate to reach any conclusion (World Health Organization, 1983).

\textsuperscript{96} Genetic mutations bring a change in a gene from parent to offspring.
The role of noise in causing pregnancy complications is probably mediated through psychological stress. Although noise is poorly studied for teratogenic effects, an increased incidence of birth defects has been associated with residence near an airport landing (Jones and Tauscher, 1978). Industrial, high power ultrasound can destroy living cells and organisms, but no deleterious effects have been observed to follow the use of diagnostic ultrasound during pregnancy. Local vibration from hand-held tools is cited as causing autonomic effects (Futasuka et al., 1980, in Chamberlain 1984a) and augmenting the risk of preterm delivery (Mammelle et al., 1984).

Pregnancies at very high altitudes, where oxygen tension is low, carry increased perinatal risks. For women raised at low altitudes, successful pregnancies at high altitudes seem to require a period of adaptation, and even then there is often a reduction in mean birthweights (Gruenwald, 1975). For example, the mean birthweight in Lake County, Colorado (3 000 metres above sea level) is nearly 300 grams below that for the population of Denver (Adamsons and Myers, 1975).

Climatic factors affecting pregnancy include temperature, which, for example, can influence diet and the type and tolerance of physical activities, and high levels of air pollution. The rate of ventilation during pregnancy increases from seven to about ten litres per minute at term, so that pregnant women may inhale large quantities of toxic air contaminants that have teratogenic effects (Brunt and Hricko, 1980). After birth, inhaled chemicals such as lead can contaminate breast milk and adversely affect the infant.
2.8.4 CHEMICAL FACTORS

Maternal pregnancy health requires the control of exposure to
therapeutic and recreational, non-therapeutic drugs, and to chemical
hazards produced by the workplace. This is because of the
pharmacological and/or toxic effects of these agents, and the woman's
autonomic reactivity to them.

Therapeutic Drugs: According to Bleyer et al. (1970), in their study
from the United States, private maternity patients reported in a day
to day diary taking an average of 8.7 drugs during the last trimester.
An average of 80 per cent of these drugs were taken without medical
supervision or knowledge. A different picture emerges from a more
recent and larger scale, prospective study by Rubin et al. (1986).
These authors found that in Scotland, a mere six per cent of women
ingested drugs during the first trimester, a further two-thirds of
women using no drugs at any stage of their pregnancies. Only about
nine per cent of pregnant women self-administered drugs, usually
non-narcotic analgesics. These results indicate a lower usage of all
groups of drugs than was reported by Bleyer's study and by the last
preceding survey of pregnancy drug-use in the United Kingdom by Forfar
and Nelson (1973).

For most drugs, the consequences for the human foetus are unknown.
This is because animal research may be irrelevant to humans, and
because a substantial number of cases involving the human foetus must
be assessed before a small increase in defects attributable to a
particular drug can be detected (Lo Bue, 1981; Berglund et al., 1985).
The thalidomide97 malformations, phocomelia98 or amelia,99 are salient

97 Until the thalidomide (Distavel) revelations in 1961, this drug was
prescribed during the fourth to eighth week of pregnancy as a sedative or as
an antiemetic for morning sickness.
98 Phocomelia is a congenital absence of the upper arm(s) and/or upper leg(s).
99 Amelia is the total congenital absence of the arms or legs.
examples of the ultimate horrors to which therapeutic drugs can give rise. When a woman views an experimental intervention as the only way to save the baby, the ethics of appropriate informed consent are pertinent.

In addition to the chemical properties of a drug, its effects on the foetus depend on the timing and duration of exposure, the drug concentration, and personal factors such as maternal age and body-weight. The dangers of drug-induced abnormalities, for instance, are greatest between the third and eighth gestational weeks when the organs and skeleton are being formed. The dangers decrease progressively thereafter. Adverse effects may also result from the abuse of therapeutic drugs, for example the benzodiazepines such as Serepax, or from failure to take prescribed medications.

Non-Therapeutic drugs: Non-therapeutic drugs are classified here not according to their generalized pharmacological effects as, for example, depressants or stimulants, but with respect to their social acceptability and the resultant type and prevalence of their use in developed countries. Two groups of drug ingestion are discussed. The first includes tobacco smoking and, although a less normative behaviour, marijuana use, together with alcohol intake. The second group refers to the use or abuse of cocaine and the opiate, heroin, which are more strongly deviant social behaviours.

Tobacco: Smoking tobacco, or more specifically cigarettes, is the most widespread, known chemical hazard to which pregnant women are exposed: between one-quarter and one-third of women in various developed countries have been reported to smoke cigarettes during pregnancy (Prager et al., 1984; Rubin et al., 1986; Behrens et al., 1987). The

100 Cocaine, or 'coke' (benzoyl methylecgonine), is a central nervous system stimulant, whereas heroin (diacetylmorphine) is a central nervous system depressant. Both drugs have mood-altering effects.
most common effect of maternal smoking is a reduction in birthweight. The babies of women who smoke are on average 40 to 430 grams smaller than the babies of non-smoking women, the most commonly observed mean reduction in birthweight being about 200 grams (Abel, 1983, Werler et al., 1985). The large range of average differences reflects, for example, whether confounding factors are controlled for, as well as the dosage - the more a woman smokes, the lower her baby's birthweight - and the period of exposure. The birthweights of babies born to smokers who do not smoke during pregnancy, or who stop smoking after the first half of their pregnancies, resemble those of non-smokers more than those of smokers (Abel, 1983).

According to Milstein (1981), the babies of women who smoke are often small but not small for gestational age. However, most often the birthweight reductions are explained by intrauterine growth retardation, especially when smoking occurs after the sixteenth week (Brar and Rutherford, 1988). Usually, smoking does not affect the length of gestation, or at most by only a few days (Abel, 1983).

Various mechanisms have been hypothesized to explain the retardation in foetal growth and reduction in birthweights among maternal smokers. Most persuasive are three additive mechanisms through which smoking per se, rather than any physiological characteristic of the smoker herself ('Constitutionality hypothesis') reduces foetal oxygen delivery. Smoking, first, stimulates sympathetic activity, which reduces blood circulation in the intervillous space (Quigley et al., 1979; Trease and Evans, 1983). Secondly, smoking increases carboxyhaemoglobin\textsuperscript{101} and thiocyanate levels\textsuperscript{102} in maternal and foetal blood (Bottoms et al., 1982; Visnjevac and Mikov, 1986). The third

\textsuperscript{101} Carboxyhaemoglobin is formed when carbon monoxide combines with haemoglobin, a pigment in the red blood cells. This reduces the oxygen carrying capacity of the blood by up to 12 per cent (Abel, 1983).

\textsuperscript{102} Thiocyanate is a salt of thiocyanic, or sulfocyanic, acid.
mechanism is altered placental function and morphology, which could also account for the increased occurrence of antepartum haemorrhage in smokers (Van der Veen and Fox, 1982; Van der Velde et al., 1983; Werler et al., 1985).

It is unlikely that smoking retards foetal growth by affecting maternal food consumption and foetal nutrient availability. In contrast to the effects of maternal undernutrition, smoking during pregnancy reduces birthweight rather than length. As Davies et al. (1976) explain, to achieve the reduction in birthweights associated with heavy smoking by women, maternal diets would need to be nearly as inadequate as those observed during the Dutch famine of 1944-45 (see section 2.8.5).

Maternal smoking has also been linked to congenital anomalies through an increased incidence of chromosomal exchange-type aberrations. However, the evidence for this from both human and animal studies is conflicting. An apparent benefit of maternal smoking is a reduced incidence of the respiratory distress syndrome in infants, possibly because smoking is believed to accelerate foetal pulmonary maturation (Curet et al., 1983).

The only certainty is the decrease in birthweight associated with the woman smoking in the second half of her pregnancy. Considering the size of the birthweight reduction produced by smoking, which yields a high mean birthweight for low birthweight babies (Goldstein, 1972), likely relations between smoking and perinatal death emerge. Women who are already in high-risk groups for perinatal death increase this risk by smoking during pregnancy. However, smoking does not

103 The same effect has been noted for maternal alcohol ingestion (Ioffe and Chernick, 1987) and heroin (Flandermeyer, 1987). However, the association is not unequivocal. For example, Zervoudakis et al. (1980) found a higher incidence of respiratory distress syndrome in the infants of women who were treated with ethanol to arrest preterm labour.
substantially increase the risk of perinatal death for women who are otherwise low-risk candidates for this outcome (Andrews and McGarry, 1972; Werler et al., 1985). Also, paradoxically, among low birthweight babies maternal smoking is associated with a lower risk of perinatal mortality than is pregnant women not smoking (Abel, 1983 and see section 7.3).

Marijuana: Research from North America conservatively estimates, on the basis of self-reporting, that at least 10 per cent of women use marijuana during pregnancy (Fried et al., 1983; Hatch and Bracken, 1986). Despite this high prevalence of marijuana usage, the effects of the drug on pregnancy outcome remain inconclusive.

Australian evidence indicates a strong, statistically significant association between using marijuana more than once a week and preterm labour and its sequelae - for example, low birthweight and perinatal death (Gibson et al., 1983). Yet Fried et al. (1984) observe a small, but statistically significant, reduction in gestation length (of 1.1 weeks) only among marijuana users of six or more joints per week. This was after controlling for nicotine and other potential confounding factors, similar adjustments yielding a very small reduction in birthweight. Hatch and Bracken (1986) likewise found that marijuana, only when used at least two to three times per month and exclusively by white women, yields increased relative risks of being born preterm, small for gestational age and of low birthweight. Hingson et al. (1982) report that marijuana use lowers birthweight, but not gestation length, in a dose-response relation.

Marijuana ingestion has been further observed to produce complications of the labour and delivery. Two papers by Greenland et al. (1982, 1983) report the same qualitative pattern of elevated rates of dysfunctional labour, precipitate labour and meconium staining.
However, observed differences are smaller in the second study of women enrolled in a home-birth centre than in the earlier study of a hospital delivery population. Fried et al. (1983) also find marijuana use to be linked to an increased incidence of precipitate labour, although not of meconium staining. Their study did not report statistically significant differences between the marijuana users and their matched controls with respect to presentation at birth and Apgar scores.

Nor did this study observe any association between in utero exposure to marijuana and major physical abnormalities, but two minor anomalies did uniquely characterize the offspring of heavy marijuana users. The anomalies were an unusual amount of skin covering the nasal portion of the eye and an atypically wide separation of the eyes. Linn et al. (1983) note that major malformations more often affected the babies of marijuana users, but logistic regression did not show the relation to be statistically significant. Hingson et al. (1982) found the infants of marijuana users to experience a fivefold increase in features compatible with the foetal alcohol syndrome, and the plausibility of developmental anomalies is supported by substantial research in animals (for example, Abel, 1980, 1981) describing numerous teratogenic effects, especially at high doses.

**Alcohol:** Heavy alcohol use affects perinatal outcome adversely, the babies of alcoholic women, for example, having a 30 to 50 per cent chance of being born with the foetal alcohol syndrome (Saunders, 1985). Even among women who are heavy drinkers, but not alcoholics,

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104 The foetal alcohol syndrome affects many infants born to women who are alcoholics or who drink alcohol excessively. The syndrome is characterized by symmetrically retarded foetal growth, central nervous system dysfunction and facial anomalies.

105 This usually refers to a chronic, average daily consumption of about 85 ml of absolute alcohol. However, also important may be binge drinking at critical periods during the first trimester (Hadi et al., 1987).
this syndrome, or complications including foetal growth retardation and congenital abnormalities, are sometimes also reported (Rosett et al., 1983; Little et al., 1986; Ernhardt et al., 1987).

Studies reach little agreement about the effects of light and moderate drinking on the developing foetus, most investigations seeking evidence of increases in retarded growth in utero and, thus, reduced birthweights. The unknown risks of these sequelae may reflect differences in studies' abilities to quantify alcohol ingestion (for example, according to the type of beverage) and to adjust for confounding factors; and the period(s) during, and before recognition of, pregnancy when the alcohol consumption is being studied. Without considering individual studies according to these criteria, some results relating consumption levels to birthweight are summarized.

Olsen et al. (1983) found that five to nine drinks per week reduced mean birthweight by 40 grams. For Mills et al. (1984), the daily consumption of less than one drink and one to two drinks in the first trimester yielded mean birthweight reductions of 14 and 83 grams respectively. This is not inconsistent with Little's (1977) finding that one drink per day in the month preceding pregnancy brought a mean decrease in birthweight of 91 grams. However, Little et al. (1986) noted that this same average daily consumption in the week before pregnancy accompanied a mean birthweight reduction of 225 grams. A comparable decrease in birthweights for male, but not female, infants was associated with one drink per day between seven and fifteen weeks and during the week prior to the first prenatal visit. Olsen et al. (1983) found more than ten drinks per week to lower mean birthweights by 150 grams, while for Mills et al. (1984), a reduction of 165 grams required between three and five drinks on average each day in the first trimester.
**Cocaine and Heroin:** Multiple problems hinder the collection of reliable information on the perinatal effects of cocaine and the opioids such as heroin. Users may habituate the ingestion of several drugs; live in an unstable social, economic and emotional environment; and have a history of drug abuse manifest frequently in associated medical complications. For example, amenorrhoea\(^{106}\) among heroin addicts can partly explain their typically late antenatal attendance. Confronted by these difficulties, research has attempted to unravel the effects on pregnancy outcome of using illicit drugs such as cocaine and heroin.

In recent years, cocaine has been the major substance of abuse in the United States (Keith et al., 1986), and this period has likewise seen the use of this drug increase in Australia (Tudehope, 1989). Cocaine affects perinatal outcome mainly by causing vasoconstriction of the uterine vessels which reduces both the blood flow and oxygen delivery (also the amount of cocaine transfer) to the foetus (Woods et al., 1987). In turn cocaine ingestion during pregnancy has been linked to a higher incidence than in matched controls of low birthweight newborn resulting from preterm birth (Keith et al., 1989) and intrauterine growth retardation (MacGregor et al., 1987). Chasnoff et al. (1989) found these symptoms only to attend sustained cocaine abuse throughout pregnancy.

The abrupt elevation in blood pressure, secondary to vasoconstriction of the placental vessels, may explain the positive association, reported by many studies (for example, Acker et al., 1983; Chasnoff et al., 1987; Keith et al., 1989), between cocaine use and abruptio placentae. Yet Chouteau et al. (1988), for example, found no such association in their retrospective, multivariate study of several

\(^{106}\) Amenorrhoea is the absence of menstruation.
hundred women at a large urban hospital in the United States. A correlation is sometimes noted between maternal abuse of cocaine and developmental abnormalities. These abnormalities include defects of the skeletal system, as in skull-ossification defects (Bingol et al., 1987), and of the cardiovascular system (Little et al., 1989) similar to those found in the offspring of laboratory animals given cocaine during pregnancy. In neonates there is no predictable sequence of physical withdrawal.

In Australia, cocaine use is less widespread than that of heroin. As with cocaine, the ingestion of heroin significantly reduces placental blood flow and has been found cross-sectionally to attend increased incidences of premature rupture of the membranes and growth retardation in utero, as well as, to a lesser extent, abruptio placentae and toxaemia (Keith et al., 1986; Flandermeyer, 1987).

Neither heroin nor methadone is believed to be teratogenic (Bingol et al., 1987; Caviston, 1987) but passive addiction by neonates to heroin or methadone may be evidenced in signs of acute opioid withdrawal.107

Occupational Hazards: In addition to the cognizant ingestion of drugs, exposure to various chemicals, especially in work-related activities, is potentially dangerous to the foetus. Especially great is the risk of spontaneous abortion, but perinatal complications can also result. For the sake of brevity, discussion focuses only on the potential reproductive effects of prolonged exposure to lead and anaesthetic gases.

Lead has a long history as an abortifacient, yet maternal exposure also increases the risks of both stillbirth and neonatal death.

Occupational groups affected include typographers, the makers and

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107 A narcotic withdrawal syndrome has been identified to include hyperflexia tremors, irritability, excessive high-pitched crying and disturbed sleep. Other symptoms involve the gastrointestinal tract.
users of paint and pesticides, farmers, ceramic and pottery makers and petrol attendants (Chenier, 1982). Health care workers may experience prolonged exposure to anaesthetic gases such as halothane and nitrous oxide. The exposure of female workers brings increased foetal wastage and increased congenital anomalies (Cohen et al., 1974; Knill-Jones et al., 1975; Infante and Tsongas, 1985).

2.8.5 NUTRITION

The human foetus is well protected against the effects of a poor maternal diet (Schneider and Dancis, 1975; Hytten, 1979), although inadequate nutrition can impair the delivery of oxygen and nutrients to the foetus. The degree to which this contributes to pregnancy complications depends on the nature, timing and duration of the nutritional deficiency; the nutritional and reproductive histories of the pregnant woman; her age, physical activity and lifestyle; and the presence of concomitant illness.

In terms of these factors, nutritional deficiency can increase the risk of general medical disorders and complications specific to pregnancy. The major general disorder associated with a poor diet is infectious disease. Inadequate nutrition impairs the maintenance of the body's cellular integrity and undermines its immune competence, predisposing the individual to infections she or he is unable to handle (Gray, 1983).

Specific obstetric complications can result from nutritional deficiency. Iron and folate deficiency in the pregnant woman can cause anaemia, producing in extreme cases smaller foetuses (Adamsons and Myers, 1975). However, the importance of preanaemic folate

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108 Anaemia is defined as a reduced number of red blood cells or a lack of haemoglobin in them.
deficiency in causing pregnancy complications is controversial. Zinc depletion has been associated with congenital malformations (Sandstead, 1985) and deficiencies of both this mineral and magnesium plus thiamine and other B vitamins appear to contribute to foetal growth retardation and pregnancy hypertension (Patrick et al., 1982; Wynn and Wynn, 1988). Strong evidence for restricting or increasing sodium intake is lacking (Pitkin, 1976), and an inadequate intake of vitamin D can cause foetal hypoglycaemia which may be fatal (de Swiet, 1986).

In today's developed societies, birthweight reductions resulting from dietary inadequacy are clearly much less than those associated with near starvation during the hunger winter in Northwest Holland from 1944 to 1945. Owing to growth retardation in utero, this experience brought mean reductions in birthweight of about 300 grams, the length of babies being much less affected (Stein et al., 1975; Hytten, 1979). The German siege of Leningrad in 1942 brought larger reductions in birthweight, averaging about 500 grams, but conditions in the Russian situation were more complex than simple dietary inadequacy; the extreme cold exacerbated a nutritional deprivation that was more severe and prolonged than in the Dutch famine (Gruenwald, 1975; Schneider and Dancis, 1975; Pitkin, 1976).

Also supporting the notion of limited dietary impacts on birthweight are the observed effects of prenatal dietary supplementation in developed populations. When, in Northwest Holland, nutritional deficiency affected the first half of pregnancy, deficits were recovered by resuming a normal diet in late pregnancy. Yet only modest increases in birthweight - between 40 and 70 grams - were achieved by trials involving the supplementing of diets of low-income, but not overtly malnourished populations in Montreal (Rush et al.,
1976) and New York (Rush et al., 1980). Indeed, in both studies the supplemented women with a previous low birthweight baby recorded lower birthweights than did the offspring of untreated controls with the same history.

2.9 PATERNAL FACTORS

Ascribed and acquired characteristics of the father can influence the risks of perinatal death. With rising paternal age comes increased incidence of certain genetic defects such as Marfan’s syndrome,\textsuperscript{109} while the extra chromosome in Down’s syndrome (Trisomy 21) can derive from the father. Paternal emotions and behaviours can also influence perinatal well-being. They can alter the maternal lifestyle or directly affect the foetus as, for example, through passive smoking, which has teratogenic potential (Grab et al., 1988) and may contribute to a reduction in birthweight (Martin and Bracken, 1986), or through occupational exposure to certain chemicals.

For example, exposure by male workers to anaesthetic gases such as halothane and nitrous oxide is associated with increased risks of preterm delivery (Chenier, 1982) and birth defects, especially of the central nervous and musculo-skeletal systems (Tomlin, 1979). These defects, like those suspected among the offspring of male workers exposed to vinyl chloride monomer\textsuperscript{110} (Infante, 1976), may be the result of chromosomal anomalies produced by paternal exposure (Armstrong and Stanley, 1984). In the case of vinyl chloride monomer, however, the evidence for mutagenic or teratogenic effects is equivocal. For example, Edmonds et al. (1978) found no relationship between birth defects and parental occupation in Kanawha County, West Virginia,

\textsuperscript{109} Marfan’s syndrome is characterized by excessive height, abnormally long, slender fingers, and congenital defects of the heart and eyes.

\textsuperscript{110} Vinyl chloride monomer (VCM) is processed into the plastic, polyvinyl chloride (PVC).
where there is a polyvinyl plant, and Thieriault et al. (1983) were unable to substantiate an association between vinyl chloride monomer and birth defects in the exposed community of Shawinigan, Quebec.

A related issue is whether the risk of birth defects is increased by the father’s military service in Vietnam, possible mechanisms including not only chemical exposure, for example to the defoliant Agent Orange, but infections and the emotional and behavioural consequences of serving in Vietnam (Armstrong and Stanley, 1984). An individually matched case-control study by Donovan et al. (1984) did not find an excess of congenital anomalies in general, diagnosed at or shortly after birth, in the offspring of Vietnam veteran fathers.

PART B: BACKGROUND VARIABLES

Three groups of background variables influence perinatal outcome indirectly, their effects always operating through the proximate variables. Two of the groups, family social factors and the family medical history, operate at the micro-level. Exogenously-defined, family social factors include sociobiological factors, sociocultural factors, socioeconomic factors and geographic factors. All these relate to both parents, but in this review only maternal characteristics are considered because of the paucity of literature pertaining to men. Endogenous factors describing the family medical history are both parents’ families’ medical histories and the woman’s own general and obstetrical histories. Each set of micro-level variables is contextualized by macro-level background factors. However, these latter effects are integrated with the discussion of factors at the individual level which now follows.
2.10 FAMILY MEDICAL HISTORY

This section first briefly describes how risk factors for perinatal death may present in the general medical history of the pregnant woman and in her and her partner's families' medical histories. Attention is secondly given to variables that specifically relate to the maternal obstetrical history.

2.10.1 FAMILY AND MATERNAL GENERAL MEDICAL HISTORIES

Both parents' families' medical histories and the pregnant woman's own general medical history facilitate an assessment of perinatal risks, especially when, as with the primigravida, there is no obstetric record. Possible risk factors, for example, include genetic disease or multiple pregnancy in the family histories of either parent. A maternal history of prior surgery, medications, blood transfusions and allergies may also be relevant as could be the presence of certain medical conditions, hypertension for one. The pregnancy may be complicated by such a disorder, the disorder may be aggravated by the pregnancy, or both outcomes may eventuate.

2.10.2 OBSTETRICAL HISTORY

The obstetric or reproductive history of the expectant woman is discussed with reference to three interrelated variables. These are parity and, in the multigravida, the preceding interpregnancy interval and earlier pregnancy outcomes (see also section 8.1). This shift in focus between parity and gravidity reflects merely the emphasis in the literature.

Parity: Cross-sectional studies, for example, by Selvin and Garfinkel (1976), have usually found the relation between perinatal death and
parity to be J-shaped. Perinatal mortality falls after the first birth, before rising with the fourth birth and at subsequent parities. Failure to replicate this pattern is most often associated with neonates. For example, Forbes and Pickering (1985) found the risks of neonatal and perinatal death, but not of stillbirth, to decline with increasing parity. Da Vanzo et al. (1983) suggest that the mortality risks of high birth orders emerge later in infancy.

Where high parities do characterize perinatal death, they may not be inherently more risky than low parities since the women who present for high-order births are typically already at increased risk for perinatal death: high parity births are most commonly to women of advanced maternal age who may also have at least one prior unsuccessful pregnancy outcome; successful reproducers may withdraw from childbearing earlier. Moreover, according to Bakketeig et al., (1984), the cross-sectional approach is biased by pooling across sibships.

These last researchers, when examining the parity-perinatal outcome nexus, have attempted to circumvent the dynamic nature of populations by comparing mortality rates for defined cohorts of women at different birth orders. However, this longitudinal approach produces its own distortions (Yudkin and Baras, 1983). The results of Bakketeig and Hoffman (1979), which show declining perinatal mortality with increasing parity, are themselves an artefact of keeping sibship size fixed - so long as previous pregnancy outcomes influence reproductive decisions (Mantel, 1979; Kiely et al., 1986).

Both the cross-sectional and longitudinal approaches, therefore, have limitations, although wider support seems to attend the former approach. Its practitioners have demonstrated a growing acceptance that measuring the effect of parity per se requires adjusting for such
factors as maternal age and, in particular, different reproductive histories.

Yudkin and Baras (1983) show that the risks of an adverse outcome in the current pregnancy are increased fourfold by one or more previous poor outcomes. With adjustment for this factor, and for short pregnancy intervals, parity is not a significant risk factor. Consistent with this conclusion is the finding by Kiely et al. (1986) that high parity is strongly associated only with intrapartum death, placental abruption\textsuperscript{111} providing a possible intervening variable, whilst a strong age-parity interaction occurs only for neonatal death.

**Pregnancy Intervals:** Rarely reported is any effect of interpregnancy intervals on stillbirths. These deaths have, on occasion, been linked to long intervals of varying length (James, 1968; Erickson and Bjerkedal, 1978), but short birth intervals appear to have little effect on stillbirth rates (Hobcraft et al., 1983, 1985). By contrast, short pregnancy intervals are often noted to increase the risk of neonatal death (Fedrick and Adelstein, 1973; Spiers and Wang, 1976; Fortney and Higgins, 1984).

This elevated mortality risk of neonates has been postulated to be mediated through a reduction in birthweight. Empirical evidence for this mechanism is lacking in developed populations, but Jelliffe's (1966) hypothesis of a 'maternal depletion syndrome', according to which repeated, short interpregnancy intervals deny the woman full physical and nutritional recovery, may apply to subgroups such as multiparous adolescents (Cramer, 1987). Plausible at least is that it is the 'high-risk women of low socioeconomic status [that occupy] the short spacing categories because of their failure to control their fertility' (Miller, 1989: 483).

\textsuperscript{111} Placental abruption is a translation of *abruptio placentae*.
Although Fortney and Higgins (1984) found the risk of delivering a low birthweight baby to be increased by birth intervals of nine to twelve months, the authors had no information on prematurity, and by definition, preterm births have shorter birth to birth intervals (Eastman, 1944). Live birth to conception intervals of fewer than nine months were observed by Brody and Bracken (1987) to increase the risk of a low birthweight delivery. However, in this cohort study of Connecticut births, adjustment was not made for the birthweight of the immediately prior sib, and the finding was based on very small numbers and thus unstable relative risk estimates (Klebanoff, 1988).

Prospective studies by Erickson and Bjerkedal (1978) and Klebanoff (1988) conclude that there is no causal association between short interpregnancy intervals and low birthweights. There is a tendency for low birthweight babies to be followed by shorter interpregnancy intervals than are non-low birthweight babies. This is because low birthweight babies face increased risks of neonatal death, and, in this event, women have increased propensities to repeat pregnancies in a short time to compensate for, or replace, losses. Correlates of high risk include sociobiological factors such as maternal age, socioeconomic status, and the reproductive history (Klebanoff, 1988).

**Previous Pregnancy Experiences:** The complications and outcomes of previous pregnancies can affect the most recent pregnancy. First, a prior, unsuccessful pregnancy is often closely followed by another pregnancy. This is because there is a tendency toward 'replacement', and the resumption of menstruation and ovulation is accelerated when lactation either does not take place, because of a stillbirth, or terminates prematurely with a neonatal death. Secondly, history tends to repeat itself. For example, complications such as preterm birth (Keirse et al., 1978; Kaltreider and Kohl, 1980; Hoffman and
Bakketeig, 1984) have an increased risk of recurrence in the subsequent pregnancy.

A further risk factor is the cumulative effect of induced abortions, performed especially before the early 1970s, on subsequent pregnancies. To allow curettage or evacuation, most first trimester terminations then first required forcible dilation of the cervix under general anaesthesia. Particularly with repeated terminations and infection, this could affect the structural integrity of the cervix (Obel, 1979a, 1979b), and so produce cervical incompetence. Future risks of preterm birth and perinatal death were thus increased, especially for nulliparae (Bragonier et al., 1984). In recent years, these dangers have been minimized by the 'earlier interruption of pregnancy, paracervical block anaesthesia, the use of small flexible cannulae and suction, and the use of prostaglandins or laminaria for cervical ripening' (Bragonier et al., 1984: 74). Complications can still follow the use of other methods, such as hysterotomy, after 12 weeks' gestation. For example, there is the danger of uterine rupture in a subsequent pregnancy.

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112 Curettage involves scraping tissue from the internal surface of the uterus.
113 Evacuation uses suction to remove the unwanted products of conception.
114 An incompetent cervix is one unable to hold the foetus in the uterus because it dilates prematurely.
115 The paracervical block requires infiltration of a local anaesthetic immediately lateral to the cervix to block sensation from the uterus toward the end of the first stage of labour.
116 Cannulae are small hollow tubes inserted into the uterus to achieve evacuation (see Footnote 113).
117 Laminaria tents are rod-shaped structures which, having been prepared from the dried stems of the seaweed 'laminaria', are inserted into the cervix where, by absorbing water and so expanding, they cause the cervix to dilate.
118 Hysterotomy, which is an incision into the uterus extending into the uterine cavity, is in essence a caesarean section performed before the foetus is viable.
2.11 FAMILY SOCIAL FACTORS

Four sets of social factors - sociobiological factors, sociocultural factors, socioeconomic factors and geographic factors - operate at the family level to influence perinatal survival. The potential aetiological effects of these factors are discussed in sections 2.11.1 to 2.11.4 mainly with reference to the woman.

2.11.1 SOCIOBIOLOGICAL FACTORS

Attention is given to two sets of sociobiological factors in the expectant woman. They are her pre-pregnancy height and weight, and her age.

Pre-Pregnancy Height and Weight: Short maternal stature,\textsuperscript{119} which may reflect genetic factors, inadequacies of nutrition and growth in early life, or both factors, is associated with low birthweight infants and, because maternal height is a guide to pelvic size, with labour complications and stillbirths (Keay and Morgan, 1978, Buckfield et al., 1983). Maternal prepregnancy weight, which is frequently a function of height, further influences the risk of perinatal complications.

In the underweight woman\textsuperscript{120} the likelihood of lightweight babies is substantially increased (Petros-Barvazian and Behar, 1978), while the overweight woman\textsuperscript{121} faces an elevated risk of general disorders and obstetric complications specific to pregnancy. The former problems include hypertension, diabetes and cardiovascular disorders.

Complications due to pregnancy include pre-eclampsia, difficulties in

\textsuperscript{119} Short maternal stature refers to a maternal height below 152 centimetres.

\textsuperscript{120} The underweight woman is one whose prepregnant weight is 10 per cent or more below the ideal weight for her height and age.

\textsuperscript{121} Moderately overweight and very overweight women enter pregnancy weighing, respectively, at least 20 and 35 per cent above the ideal weight for their height and age.
the diagnosis and management of malpresentations, and prolonged labour if the foetus is large. Also increased is the likelihood of caesarean delivery, and the risk of lactation failure is greatest in obese women. However, in the absence of antenatal complications, obesity introduces no increase in perinatal mortality (Garbaciak et al., 1985).

**Maternal Age:** Increased risks of perinatal death are associated cross-sectionally with women below age 20 years and, especially, aged 35 years and over. For adolescents, except at very young ages, apparent age effects may represent the social, economic and emotional contexts of pregnancies.

**Mature Women:** Women over 35 years of age carry a substantially increased risk for stillbirths (Grimes and Gross, 1981; Forman et al., 1984), the risk increasing monotonically from the late twenties to the early forties (Kiely et al., 1986). It becomes twofold for women in their mid to late thirties, and threefold to fourfold for women in their early forties (Hansen, 1986), although risks vary among different socioeconomic groups and countries. The association between maternal age and neonatal mortality is less pronounced, being greatest for the very oldest maternal age groups (Hansen, 1986).

Stillbirths to mature women tend to be antepartum, but not intrapartum, foetal deaths associated with three groups of congenital anomalies: congenital syndromes affecting multiple systems; anomalies of the heart and circulation; and neural tube defects (Kiely et al., 1986). Hansen (1986) explains these anomalies among mature pregnant women in terms of increased chromosomal abnormalities, especially trisomies 13, 18 and 21 and sex chromosome aneuploidies.122

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122 Sex chromosome aneuploidies arise when the chromosome number of a cell is not an exact multiple of the normal number.
Biological explanations for these late foetal deaths include reduced foetal oxygenation associated with impaired uterine blood flow (Naeye, 1983); increased hypertension and toxaemia (Grimes and Gross, 1981); and diabetes mellitus (O'Sullivan et al., 1973). These problems are consistent with high incidences among mature women of lightweight babies - both preterm and small for gestational age (Kajanoja and Widholm, 1978; Forman et al., 1984), and babies weighing more than 4 000 grams (Grimes and Gross, 1981; Kirz et al., 1985). In addition caesarean section rates increase markedly with advancing maternal age (Blum, 1979; Grimes and Gross, 1981; Kujansuu et al., 1981; Kirz et al., 1985). The contemporary significance of these complications and of the increased risk for stillbirths among mature women arises, in Australia, from the increased proportion of births to women aged 35-39 years (see section 1.4.1).

**Adolescent Women:** Women under 20 years of age face an increased risk for neonatal death, recording higher incidences of low birthweight and preterm infants than do women aged less than 35 years (Blum and Goldhagen, 1981). However, the effect of adolescence itself is unclear, except possibly for women below age 15 years, who are characterized by relative physiological immaturity (Tyrer, 1978). The uncertainty arises because adolescent pregnancies are often associated with an adverse psychosocial environment which is not conducive to good maternal health habits and early antenatal care, the effects of which may be relatively stronger than those of young age (Elster, 1984; Leppert et al., 1986).

Controlling for antenatal care usually shows that adolescence per se does not predispose to infants of low birthweight and high mortality risk (Horon et al., 1983; Zukerman et al., 1983). Indeed the young woman receiving early antenatal care is often a low-risk patient
(Elster, 1984; Davidson and Fukushima, 1985; Clark et al., 1986). On the other hand, some studies, such as by Leppert et al. (1986), still find young women to be at increased risk for obstetric and neonatal complications even with antenatal care and other factors controlled. Less disagreement surrounds the high risks for adolescents experiencing a second or subsequent pregnancy, especially one characterized by an inter-pregnancy interval of less than six months (Spiers and Wang, 1975; Eisner et al., 1979).

2.11.2 SOCIOCULTURAL FACTORS

Four variables are taken to represent sociocultural influences on perinatal death: ethnicity and race; education; marital status; and household type. These risk factors, including the first one, are postulated to describe mainly acquired characteristics of parents that permit intervention and change.

**Ethnicity and Race:** Ethnicity is defined mainly by cultural traits ascribed by way of common ancestry and historical experience, but continually influenced by intergroup contact and relations (Buetow, 1983). Typically, the concept of race more strongly emphasizes distinctive biological features of groups and individuals, yet this definition is not unequivocal. For example, although genetic traits may predispose to ill-health, as with thalassaemia among Mediterranean and Southeast Asian peoples, and Tay-Sachs Disease among Ashkenazi and Moroccan Jews, Cooper (1986: 101) argues that 'no discrete "package" of gene differences has ever been described between two races'. This absence of a consistent difference in many traits reduces the race concept almost always to different social and cultural environments. Of course, this does not obviate the need to determine just when genetic differences apply.
Across racial and ethnic groups, predominant cultural traits that have implications for perinatal survival are manifest, for example, in nutrition, language, and group perceptions of health and morbidity. These perceptions, which are also influenced by intergroup relations, affect the evaluation of symptoms, use of mainstream health services and compliance with different treatment regimes (Harwood, 1981). Since it is often also unrealistic to separate ethnic factors from socioeconomic position, Gordon (1964) devised the 'ethclass' concept. This conflates the historical and social forces of class in the individual's ethnic group. Risk factors for perinatal death, such as nutritional intake, can then be conceived as deriving from this interaction insofar as it defines the locus of subsocietal participation. In section 8.2.1, a case study is presented of the risk factors affecting Aboriginal people in New South Wales, whilst in section 8.2.2, the maternal region of birth is investigated as a risk factor for perinatal death.

**Education:** The effect on perinatal survival of, especially, maternal education remains uncertain (Da Vanzo et al., 1983). A negative relation between maternal education and foetal and neonatal mortality was found by Hobcraft et al. (1984), but in the 1982 study of Paneth et al., maternal education lowered mortality only at birthweights less than 1 500 grams. Shoham-Yakubovich and Barell (1988) report an inverse relation between maternal education and proportions of low birthweight infants. However, using mortality ratios standardized for maternal age and parity, the only significant effect of maternal education on low birthweight-specific neonatal mortality rates was a survival advantage at the lowest educational level. Excess mortality of normal birthweight neonates was detected in the highest level of maternal education.
On substantive grounds, an inverse relation between maternal education and perinatal death is the most plausible. This is irrespective of whether education affects perinatal outcome merely as a proxy for other social or economic correlates, such as material well-being, or, as with child survival, also in its own right (Ruzicka, 1985). The latter possibility could exert downward pressure on perinatal mortality in several ways.

For one, education enhances knowledge, and presumably the practice, of prophylactic behaviours: McDonald and Coburn (1988) found poorly educated women, especially without a high school education, to be the least likely to receive adequate antenatal care. Education further promotes the recognition of illness and sources of effective treatment, whilst engendering increased confidence, respect for, and an ability to deal effectively with bureaucratic authority (Cleland, 1989).

**Marital Status:** Never married and formerly married women often have reduced economic and social supports, and diminished satisfaction with their personal and emotional lives (McDonald, 1985). Henrique et al. (1986) found both non-married groups, when compared with married women, less likely to commence antenatal care before the third trimester or to attend antenatal classes, and more inclined to smoke cigarettes. Formerly married women were especially likely to be heavy smokers and to have a history of bleeding, while never-married women were the more prone to anaemia. In a second study by the same authors (Golding et al., 1986), the two non-married groups delivered lower birthweight babies than did the married group after adjustment for maternal age, parity and smoking history. Reduced birthweights were associated mainly with preterm birth, and helped to explain increased
perinatal mortality among non-married, especially formerly married, women.

However, most perinatal research on the aetiological contribution of marital status has focused on never-married women (for example, Elster, 1984). This is presumably because conceptions have been less common to formerly married women, despite recent increases in marital breakdowns, than to never-married women who conceive and deliver ex-nuptially. Both never-married and formerly married women are included in the analysis in section 8.2.3.

The following discussion considers ex-nuptial births, and so, without distinguishing between the never-married and formerly married, excludes the women who married or remarried between conception and confinement. Yet, in Australia since 1971, the proportion of women marrying after a non-marital pregnancy has declined markedly, particularly at ages less than 25 years (Choi and Ruzicka, 1987). Proportions of ex-nuptial births have continued to rise, in Australia from 13.2 to 16.8 per cent of total live births from 1981 to 1986 (Australian Bureau of Statistics, 1987a), both because nuptial fertility has declined and because the rate and number of ex-nuptial births have increased (Khoo and McDonald, 1988).

From 1981 to 1986 in Australia, the largest proportionate increases in ex-nuptial confinements were to women aged over 24 years, ex-nuptial confinements to women aged 20-24 years continuing, nevertheless, to account for the largest share, approximately one-third, of total ex-nuptial confinements. During the same period, the proportion of all ex-nuptial confinements occurring to adolescent women fell by 7.5 percentage points to 25.1 per cent in 1986 (Australian Bureau of Statistics, 1987a). However, ex-nuptial births became a larger proportion of total births to adolescents (see section 1.4.1).
Assessment of how these changes might have influenced perinatal death requires an understanding of the changing context of ex-nuptiality per se. Specifically, ex-nuptial childbearing has become less characteristic of mainly single women than of women in consensual unions (Khoo and McDonald, 1988). De facto couples might generally have become of lower socioeconomic status, frequently valuing marriage and the family, but choosing not to marry at least until they feel more financially secure (Hugo, 1986; Khoo and McDonald, 1988 and see section 1.4.1).

Ex-nuptial births to adolescent women cause particular concern for perinatal well-being. Single pregnant women, especially adolescents — who are probably the most likely to be genuinely single (that is, not cohabiting) — have often still been forced to contend with an adverse psychosocial environment. It is true that there has been a liberalization of societal attitudes towards cohabitation and childbearing outside marriage (Hugo, 1986), as evidenced in Australia, for example, by the Supporting Parent’s Benefit which has been available to single women since July 1973. But the difficulties described below, even though they are probably less common today than they once were, still deserve reporting.

The unmarried, adolescent girl, fearing parental condemnation or rejection by her partner, may attempt to conceal her pregnancy which decreases the likelihood of early and regular antenatal care. Family may indeed over-react to the pregnancy if, or when, it is revealed, or the pregnant woman may have no family or partner to turn to. Meanwhile, the community, whilst having grown more tolerant, continues to promulgate prejudiced, even punitive, attitudes which may be instrumentalized even by health care advisers toward illegitimacy, single parenthood and de facto unions.
Especially when support from family and society are lacking, conflicts may arise over whether to marry precipitately, induce abortion, keep the baby or, rarely in Australia these days, surrender it for adoption. These decisions can be complicated by difficulties of finance and accommodation, and by the woman's probable immaturity which may be associated with a lack of responsibility and inadequate coping ability. All these problems, which produce psychological stress (see section 2.8.1), are likely to increase the risks of an adverse perinatal outcome.

Further, given that, in Australia, induced abortion has become more readily available since the early 1970s, it is important to ask who the adolescents are that have continued to give birth ex-nuptially. Some have been in de facto unions that are perceived to be stable. However, more generally, it may be that single and cohabiting adolescent women have come mainly from disadvantaged groups that are less able or inclined to use abortion facilities (G. Carmichael, pers. comm., 1989). This hypothesis has yet to be tested.

**Household Type:** The nuclear family and household type, comprising a head, spouse and dependent children, has long prevailed in developed countries, usually providing a stable, beneficial environment for pregnancy and a newborn baby. However, in recent years alternative living arrangements have grown in popularity and probably increased net perinatal risks.

In recent years, ex-nuptial births have increasingly occurred to women in consensual unions who, by inference, live in nuclear household situations. The same household type may apply especially to adolescent women who continue living with their parents. However, some of these women have established single-person or single-parent households, which typically are burdened by economic difficulties and
often the stresses of residential relocation, (re)entry into the labour force, and alterations in household status (Bane and Weiss, 1980).

The shift to increased self-responsibility can increase this distress, especially in the presence of young dependent children who create extra work in the home during pregnancy and, after the delivery, compete with the baby for the mother's attention and other household resources. On the other hand, non-coresident family members may be psychologically and interactively present for significant periods, and the new households may face less stress by escaping conflict and developing new affective structures (Thompson and Gongla, 1983).

2.11.3 **SOCIOECONOMIC FACTORS**

Socioeconomic position is often defined for ease of measurement, but not interpretation, using either income as a proxy or some amalgam of factors which yields an index of social class. In both cases, socioeconomic position is usually found to vary inversely with the level of perinatal mortality, particularly when the income range is broad (Gortmaker, 1979; and see section 8.3).

There are two main explanations for this negative association, which hinge on two concepts: patients' demand for care, which is what they want for themselves and their babies, and patients' need for care, which is what medical opinion, instrumentalized through professional suppliers, determines as proper. Patients' demand for care is weakened by limited education and knowledge manifest in individual behavioural risk factors, such as tobacco smoking, psychological stress, low relative weight and less education, and in distinctive neighbourhood environments (Salonen, 1982, Haan et al., 1986).
The result is that what patients want is diminished in terms of what they expect, can afford and thus have access to, and these constraints are translated into a reduced consumption of health care items such as health insurance (Flegg, 1979; and see section 8.3). Poorer patients also have less control over the type of care regimes determined appropriate by their medical advisors, although according to Poston (1980), the mother’s demand for care is in any event usually quite irrelevant.

What matters most are assessments of patient needs by medical advisors. This is shown by an Australian study (Skuja et al., 1982) of factors governing consumer demand for obstetric services. Expectant women wanted control over the birth event and opposed most forms of medical intervention. Yet these wishes were frequently ignored by doctors who, questioning women’s abilities to make informed decisions about obstetric management, encouraged patients to accept virtually on trust the health care plan prescribed for them.

Women should be involved in decisions about their own care. But it can be potentially dangerous for patients at risk of complications to ignore the advice of their doctors. Notwithstanding this, professional assessments of patients’ needs internalize a social bias. According to Poston (1980), these needs are influenced not only by physiological requirements but by patients’ middle class beliefs and superior resources. Such symbols of success ensure that health care advisors afford these women optimal use of available health services and existing medical technologies. As shown in sections 2.12 to 2.12.3, intrapartum technologies are sometimes used inappropriately, so that, ironically, high status women may not always receive the best health care during parturition. That they enjoy more successful
perinatal outcomes than poorer patients may reflect their superior pregnancy health and antenatal care.

Of course, middle class women may reject the health needs prescribed for them because, for example, of their negative feelings toward the pregnancy or infant, or toward hospitals or medical personnel and procedures. However, these situations are for the most part exceptional: rich and poor people continue to receive different 'expert' definitions of their respective health needs which militate against poor people in health care delivery and use, and hence increase perinatal risks.

Finally, socioeconomic factors at the individual level should be noted especially to exist within the larger institutional structures that define the political economy of any region. In terms of implications for perinatal survival, prospects depend on economic growth equitably shared by the total population. However, in countries including notably the United States, the life circumstances of some families approach economic, social and political marginality. Their poverty can only really be understood in terms of the specific social arrangements that perpetuate class-based structural inequalities, for example, in health standards and care, education, and employment conditions and prospects.

2.11.4 GEOGRAPHICAL FACTORS

Place of residence can be defined in various ways, such as according to function or to demographic criteria such as the size, density and heterogeneity of populations. Overall, these characteristics can be summarized in typologies like rural-urban, in terms of which mortality differentials can be conveniently studied.
Demographic and epidemiologic transition theories respectively suggest that these differentials converge in response to a modernization that permeates the entire country, and to changes in the types of disorders producing death (Notestein, 1945; Omran, 1971). However, the extent of persistence in urban-rural differences in perinatal survival remains unclear. While most studies (for example, Aitken and Neville, 1978; Farmer et al., 1984) suggest that rural areas have higher infant mortality than urban areas, Clifford and Brannon (1985) find rural areas most conducive to survival during the neonatal period.

The effect of rural-urban residence on foetal death is uncertain, although, in the aggregate, pregnancy outcomes are certainly disadvantaged in remote areas of Australia (National Health and Medical Research Council, 1983b). The effects of urban living, for example, can be seen to increase and decrease perinatal survival chances.

Urban settings are characterized by technological progress and augmented social consumption. This can have salutary impacts since, by implication, urban populations are the most amenable to change, in part because high population densities permit the rapid diffusion and assimilation of new ideas. Urban areas also have the threshold numbers and densities to sustain high order goods and services as well as those of low order over large trade areas. The markets served by the few highest-order places dominate access to, and use of, almost all services, including those of the best quality (Carter, 1977).

This is exemplified by health care. The price of accessibility to the most sophisticated care, as found in major hospitals, is usually residence in the main cities or their tributary areas (Harvey, 1976). Although the bias is mitigated in a regionalized and, therefore, highly accessible health care system (in which patients are matched
with facilities appropriate to their needs), regionalization has its problems. For instance, transfers of babies may not always take place when needed because of problems of supply and demand (see sections 6.1 to 6.2).

On the other hand, it can be argued that urbanism, while providing effective treatment of medical conditions, is conducive to complications developing in the first place. It is true that the environment of rural areas has changed, having come to adopt many urban-oriented life style habits including nutrition, and work, smoking and drinking behaviours. However, the urban lifestyle probably still militates most severely against the primary prevention of illness, while perhaps more often ensuring survival by permitting superior treatment regimes. Examples of negative externalities in urban areas are increased pollution and the prevalence of increased psychological stress and mental illness.

One explanation for the last problem is that urbanism as a way of life is depersonalized and anomic, which can result in poorly defined social roles, the decay of traditional value systems and weakened family and kinship ties (Wirth, 1938), to an extent not experienced in rural locations. During pregnancy and the postnatal period, women may lack support from primary social groups, being confronted instead with impersonal procedures and institutions. In addition the intense stimuli of urban environments can generate a 'psychic overload' (Milgram, 1970), producing anxiety, loneliness and nervous strain with an increased incidence of mental illness.

2.12 CONCLUSION

The contents of this discussion yield the conceptual framework presented in section 2.0. Thus the framework, which suggests how
proximate and background factors affect perinatal death (and its stated causes), preceded and gave structure to the literature review from which it had in fact been produced. According to the framework there are five main groups of proximate factors: the physiological maturity of the conceptus; maternal and infant health care; the family lifestyle; obstetrical factors; and chromosomal and genetic factors. The first three of these factors were suggested to reflect exogenous forces, and to be amenable therefore to intervention and primary prevention. All five proximate factors influence perinatal death directly or as proxies for the background factors which comprise family social and medical history factors. These background factors always affect perinatal death indirectly, and operate with other micro-level proximate factors in the context of macro-level institutional factors.

Many of these risk factors affect a small proportion of the maternity population and, because perinatal death is usually associated with multiple risk factors, they tend not to occur in isolation. A fictional example, abstracted from the review of literature, should help to clarify the extent and nature of interrelationships between these risk factors. A 40 year old woman of gravidity 7, parity 3, works as a machine operator and smokes cigarettes heavily. Her potential risk factors in pregnancy would include her maternal age, high gravidity, previous poor reproductive history and, presumably, her low education and low income. The woman's occupation and added housework might represent risk factors for preterm birth, while her smoking could retard foetal growth. One could speculate further about factors like stress and the adequacy of her diet and attendance at antepartum care. Let us assume that our imaginary patient enters labour at, say, 32 weeks. Her doctor uses internal foetal heart rate monitoring which leads to caesarean section for foetal distress,
foetal blood sampling having not being performed. The preterm baby is born alive of low birthweight, to receive intensive care that is discontinued care after two days because of poor outcome chances. The baby dies and the stated cause of death is the respiratory distress syndrome.

This simple example gives a sense of the rare tapestry of multiple risk factors that can interweave to affect a single pregnant woman. The conceptual framework gave structure to a detailed review of how each of these factors operates directly or more often indirectly to influence perinatal mortality. Moreover, pathways were identified, both well-tracked and relatively unexplored, that require empirical study, and they form the subject matter of subsequent chapters.
CHAPTER 3
OPERATIONAL FRAMEWORK

3.0 INTRODUCTION

This chapter presents sixteen hypotheses, extracted from the conceptual model and literature review, before describing the case-control method used, among other things, to test these hypotheses. Most of the discussion focuses on the collection of two sets of primary data. The first set contains existing data collected at the New South Wales State level and includes birth and perinatal death registrations, and the State Health Department's Maternal and Perinatal Statistical Collection. The second data set is based on original information derived from patient records and interviews with patients of six large maternity hospitals. Each data set provides information relating to its constituent data sources and an expanded database produced through record linkage of these sources. A discussion of the methods of data analysis employed in the investigation is presented last.

3.1 HYPOTHESES

The conceptual framework and underlying research literature give rise to a general hypothesis that in turn gives structure to sixteen more specific hypotheses concerning the causes of perinatal deaths in New South Wales in 1985-86. The hypotheses internalize the concept of proximate and background origins for these deaths, and each hypothesis requires testing.
The broad hypothesis is that perinatal death can result from five groups of proximate determinants, which are
a. the physiological maturity of the conceptus,
b. obstetrical factors,
c. the health care received by women and infants,
d. maternal pregnancy health,
e. chromosomal and genetic factors.¹

Specific hypotheses associated with each group include the following:

**Newborn's Physiological Maturity**
1. that among low birthweight babies, intrauterine growth retardation is associated with a lower risk of perinatal death than is preterm birth;
2. that among low birthweight babies, intrauterine growth retardation tends to precede preterm birth, especially as gestation increases; and
3. that among low birthweight babies, intrauterine growth retardation and preterm birth are associated with a higher risk of perinatal death when they occur together than when either complication occurs alone;

**Obstetrical Factors**
4. that Apgar scores at one minute are better associated with perinatal mortality risk than are Apgar scores at five minutes;
5. that low neonatal ponderal indices are a useful predictor of neonatal death;
6. that perinatal death is less frequent among multiple than singleton births at the same gestational ages before term;

¹ Hypotheses relating to genetic and chromosomal factors are not tested.
7. that two characteristics of male births predispose to perinatal death: an increased incidence
   a. of preterm birth, and
   b. of hypertensive disorders of pregnancy, *placenta praevia* and preterm rupture of the membranes;

*Health Care Factors*

8. that the babies at highest risk of perinatal death are born in the hospitals most equipped to care for them;
9. that a positive relation exists between perinatal outcome and the quantity of antenatal care received;
10. that before term, induction yields a higher risk of perinatal death than does caesarean section;
11. that the incidence of caesarean section can be reduced for
   a. dystocia in nulliparous women, and
   b. preterm and multiple breech babies;

*Pregnancy Health Factors*

12. that the risk of perinatal death is increased for the pregnant woman who
   a. has a high level of psychological stress;
   b. has adverse work conditions in the labour force or in the home;
   c. starts a new type of exercise during the pregnancy; or continues to consume a non-therapeutic drug at a high level;

*Background Factors*

13. that perinatal mortality is elevated most at the highest gravidities, specifically when these gravidities are composed of
   a. more poor than successful reproductive outcomes, in particular when
   b. the immediately preceding pregnancy failed;
14. that Aboriginal women experience increased risks of perinatal death at all birthweights;
15. that women born in Australia enjoy a perinatal survival advantage over immigrant women born in developing regions;
16. that never-married women are at greater risk of perinatal death than are formerly married or currently married women.

Each of these hypotheses is classified into one or two of three groups. The first group, being mutually exclusive from the second, contains aetiologically new hypotheses, no or little previous research having been conducted thereon. The second group of hypotheses is a collection of findings documented from elsewhere of which further testing is needed because of different results, for example, in some geographical settings. The third group of hypotheses has important monitoring implications.

The broad hypothesis about proximate determinants is considered original because it deals with a new conceptual model. The sixteen more specific hypotheses are grouped as follows. Those invoking some key sense of originality are hypotheses 4, 6, 7b, 11a, the last part of 12b, 14 and 15 and the part of 16 relating to formerly married women. The hypotheses based more strongly on findings from elsewhere are 1, 2, 3, 5, 7a, 8, 9, 10, 11b, 12a, the first part of 12b, and 12c, 13 and the remainder of 16. The health care factors, including especially hypotheses 8 and 9, have strong monitoring implications.

Of final note is that the testing of these hypotheses will permit assessment of the severity of the perinatal mortality risks associated with individual factors or different levels thereof. However, a comparison of risks between factors is sought only in section 8.4 (see section 3.8).
The study area for testing these hypotheses is New South Wales which, in recent decades, has been one of the States least successful in reducing its perinatal mortality. This lack of success by New South Wales, combined with its housing the largest population of any Australian State, has seen it continue to record the most perinatal deaths in Australia (see section 1.3).

During the study period, New South Wales had clearly defined structures for administering health (see Figures 3.0 and 3.1). Twelve health regions incorporated clusters of local government areas which, for most of the period, emphasized service-based or institutionally-based health service provision. However, in September 1986 incipient area health services were formed and co-ordinated within the regions, this change signifying a movement toward defined area populations. Regional offices retained autonomy in making decisions affecting local health service provision, but area health boards, and boards of individual public hospitals not included under the 1986 Area Health Services legislation, became responsible for administering community health services.

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2 Victoria and the Australian Capital Territory (A.C.T) were also to have been included in the study area. However, poor support from Victorian record holders made the inclusion of this State unrealistic, and lack of time made it necessary to exclude a cooperative A.C.T.

3 In September 1988, five Department of Health Regions (the Sydney Metropolitan, Hunter and Illawarra regions) with their 23 Area Health Services were disbanded and replaced by ten new Area Health Services with expanded geographic area and service delivery responsibilities. Extant regions are the Central Western, New England, North Coast, South-eastern, South-western regions and Orana and Far Western regions.
Figure 3.0
Health Regions of New South Wales, Including the Australian Capital Territory, by Local Government Area, 1985-86

See Figure 3.1.
Figure 3.1
Health Regions of Metropolitan Sydney by Local Government Area, 1985-86
KEY TO FIGURES 3.0 AND 3.1

Health regions of New South Wales as defined by Local Government Areas.

Metropolitan Sydney
1. Southern Metropolitan Region
   1. Ashfield M. 10. Marrickville M.
   2. Botany M. 11. Randwick M.
   3. Burwood M. 12. Rockdale M.
   5. Concord M. 14. Sutherland S.
   6. Drummoyne M. 15. Sydney C.
   8. Kogarah M. 17. Woollahra M.
   9. Leichhardt M.

2. Northern Metropolitan Region
   1. Gosford C. 7. Mosman M.
   2. Hornsby S. 8. North Sydney M.
   3. Hunter's Hill M. 9. Ryde M.
   5. Lane Cove M. 11. Willoughby M.

3. Western Metropolitan Region
   1. Auburn M. 8. Fairfield C.
   2. Bankstown C. 9. Hawkesbury S.
   5. Blue Mountains C. 12. Parramatta C.

Country
4. Central Western Region
   1. Bathurst C. 8. Lachlan S.
   2. Bland S. 9. Lithgow, Greater
   3. Blayney S. 10. Oberon S.

5. Far West Region
   1. Broken Hill C. 3. Unincorporated Area
   2. Central Darling S.

6. Hunter Region
   1. Cessnock, Greater C. 8. Murrurundi S.
   2. Dungog S. 9. Muswellbrook S.
   5. Lake Macquarie M. 12. Scone S.
   7. Merriwa S.

7. Illawarra Region
   1. Kiama M. 4. Wingecarribee S.
   2. Shellharbour M. 5. Wollongong C.
   3. Shoalhaven M.
<table>
<thead>
<tr>
<th>Region</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>New England Region</strong></td>
<td></td>
</tr>
<tr>
<td>1. Armidale C.</td>
<td>11. Narrabri S.</td>
</tr>
<tr>
<td>2. Barraba S.</td>
<td>12. Nundle S.</td>
</tr>
<tr>
<td>5. Glen Innes M.</td>
<td>15. Severn S.</td>
</tr>
<tr>
<td>7. Guyra S.</td>
<td>17. Tenterfield S.</td>
</tr>
<tr>
<td>8. Inverell S.</td>
<td>18. Uralla S.</td>
</tr>
<tr>
<td><strong>North Coast Region</strong></td>
<td></td>
</tr>
<tr>
<td>2. Bellingen S.</td>
<td>11. Kyogle S.</td>
</tr>
<tr>
<td>7. Grafton C.</td>
<td>16. Richmond River</td>
</tr>
<tr>
<td>8. Taree, Greater C.</td>
<td>17. Tweed S.</td>
</tr>
<tr>
<td><strong>Orana Region</strong></td>
<td></td>
</tr>
<tr>
<td>1. Bogan S.</td>
<td>8. Dubbo C.</td>
</tr>
<tr>
<td>2. Bourke S.</td>
<td>9. Gilgandra S.</td>
</tr>
<tr>
<td>5. Coolah S.</td>
<td>12. Walgett S.</td>
</tr>
<tr>
<td><strong>South Eastern Region</strong></td>
<td></td>
</tr>
<tr>
<td>1. Bega Valley S.</td>
<td>9. Harden S.</td>
</tr>
<tr>
<td>2. Bombala S.</td>
<td>10. Mulwaree S.</td>
</tr>
<tr>
<td>5. Crookwell S.</td>
<td>13. Tallaganda S.</td>
</tr>
<tr>
<td>7. Goulburn C.</td>
<td>15. Yass S.</td>
</tr>
<tr>
<td><strong>South West Region</strong></td>
<td></td>
</tr>
<tr>
<td>2. Balranald S.</td>
<td>17. Leeton S.</td>
</tr>
<tr>
<td>5. Conargo S.</td>
<td>20. Murrumbidgee S.</td>
</tr>
<tr>
<td>7. Cootamundra S.</td>
<td>22. Temora S.</td>
</tr>
<tr>
<td>8. Corowa S.</td>
<td>23. Tumbarumba S.</td>
</tr>
<tr>
<td>10. Deniliquen M.</td>
<td>25. Urana S.</td>
</tr>
<tr>
<td>15. Jerilderie S.</td>
<td>30. Windouran S.</td>
</tr>
</tbody>
</table>

**A. The Australian Capital Territory**
3.3 DATA COLLECTION AND PREPARATION

The case-control method is used to test the stated hypotheses for the study area in 1985-1986. This required that the researcher first develop empirical referents for the potential risk factors described in the conceptual framework, and then collect data on the derived operational variables for a case or study group, and a control or comparison group.

Within the temporal and geographic boundaries set above, the case group is defined by incident occurrences of perinatal death, together with the women who experienced these losses. The control group is defined by new occurrences of infants surviving the perinatal period, and by the mothers of these survivors. Perinatal events are defined in accordance with national statistical definitions used in Australia (see section 1.1).

Clinical and social data were collected for every newly-occurring case in the study area in 1985 and 1986. These data were also collected for stratified samples of controls for 1985 and 1986, including all very low birthweight survivors, and random selections of additional control members after group matching for birthweights greater than or equal to 1 500 grams and for maternal age.
Data for the case group were collected from 1985-87 birth and perinatal death registrations. For both the case and the control groups, data were collected from three sources: the 1986 Maternal and Perinatal Statistical Collection; 1985 and 1986 patient maternity records at six public hospitals performing 2 000 or more deliveries annually; and interviews with patients of these hospitals in 1986.

The result is two sets of data, each of which contains information collected from separate sources, and an expanded database that combines this information through record linkage on the basis of common identifiers for individual records. The first data set contains 1986 registration and Health Department data collected for the State of New South Wales. The second data set comprises information collected from six public hospitals. These data are summaries of clinical records, and interviews with former patients. A detailed discussion of each separate data source and of procedural and ethical issues associated with their collection, and subsequent preparation and analysis, forms the body of this chapter.

3.4 REGISTRATION DATA

Registrations are legal records, theoretically of all vital events in a defined territory,\(^4\) and an important — indeed sometimes the only — source of certain statistical information. For example, the Medical Certificate of Cause of Perinatal Death, commonly referred to as the death certificate,\(^5\) is relied upon not only to permit registration of perinatal death, but to furnish details for preparing statistics of

\(^4\) Registrations of births and deaths refer primarily to the State or Territory in which the events occur. Births or deaths outside Australia may be registered in New South Wales, if they have not been registered elsewhere, under the conditions listed in Sections 16 and 22 of the Registration of Births, Deaths and Marriages Act, 1973.

\(^5\) Strictly speaking, the death certificate is issued by the Registrar-General after he or she is satisfied with the details supplied by the medical practitioner.
causes of death and ancillary information, for example, about the
timing of death.

Permission was sought, therefore, to obtain from the Australian Bureau
of Statistics, listings of perinatal deaths registered between 1985
and 1987, which could be linked with corresponding birth
registrations, and subsequently with the Health Department’s Maternal
and Perinatal Collection. These requests were approved in accordance
with the researcher’s compliance with accepted ethical principles.

The listings originally received from the Australian Bureau of
Statistics are assumed to be virtually complete. They include 1985
and 1986 perinatal deaths registered from 1985 up to, and including,
the June quarter of 1987. These data were modified in three ways:
the data were made to accord with the definitions used to compile
Australian perinatal statistics; the deaths not occurring in 1985 or
1986 were excluded; and the remaining deaths were organized according
to their year of occurrence rather than their year of registration.

Although analysis of risk factors for perinatal death is confined to
1986 data (see section 3.5), 1985 and 1986 registrations are used to
describe the timing and stated cause(s) of perinatal death. For this
latter purpose, 1985 and 1986 data were aggregated because differences
between the perinatal deaths in these years are negligible in the
amended registration data. For example, 44.1 (n=416) and 44.6 (387)

6 This is not true of published perinatal death statistics for New South
Wales, the percentages of neonatal deaths in 1985 and 1986 being 44.0 (n=433)
and 51.4 per cent (n=510) respectively. According to the Australian Bureau of
Statistics (1985), the number of neonatal deaths, but not the number of
stillbirths, is likely to have been affected by abnormal delays in the
processing of registrations in the New South Wales Registry of Births, Deaths
and Marriages in 1984. The magnitude of the effect is not known.
per cent of 1985 and 1986 registered perinatal deaths were neonatal
deaths.\textsuperscript{7}

National statistical definitions differ from the legal ones used for
registration purposes, and from the statistical definitions used in
New South Wales. To be registered in New South Wales, the stillborn
child must be of at least twenty gestational weeks or weigh at least
400 grams at delivery, whilst a neonatal death relates to all liveborn
infants who die within 28 days of birth. These legal criteria are
also used by this State when publishing its own perinatal statistics.
However, national statistics are prepared using, for all perinatal
deaths, a minimum birthweight criterion of 500 grams or, only if the
birthweight is unknown, the corresponding gestational age or body
length (see section 1.1).

In addition, a stillbirth is defined by statute in New South Wales
when breathing does not take place, irrespective of when cessation of
the heart beat occurs. Yet for State and national statistical
reporting purposes, live births and neonatal deaths are indicated if
the heart beats after the delivery even if the infant does not
breathe; that is, there is some evidence of life. Applying the last
difference resulted in the reclassification of 40 of the 1985 and 37
of the 1986 registered stillbirths as statistical live births ending
in neonatal death.

For neonates, successful matching of the amended registrations took
place on 78.2 per cent (n=628) of the 803 live births ending in
neonatal death in 1985-86. For stillbirths, it was possible to match

\textsuperscript{7} In this study, n refers to the numerator rather than to the denominator;
any deviation from this rule should be clear from the position of the
parenthesized n value. (See section 3.8).
82.0 per cent (n=393) of the 479 stillbirths occurring only in 1986.\(^8\) Unsuccessful linkages are best explained by parents' failure to complete a Form of Information of Birth, especially for stillbirths, on which birth registrations are based. However, human error could also account for missed linkages and for spurious ones.

### 3.4.1 QUALITY OF REGISTRATION DATA

Registration data are qualified by two potential classes of error, those of coverage and those of content, although registrations in fact provide an excellent source of population data. With respect to coverage, the main error is omission: if vital events are not to escape registration, particulars sufficient to warrant registration must be furnished to, or otherwise be established by, registrars.

For the Medical Certificate of Cause of Perinatal Death, such registration was able invariably to take place because the signing of this document by a medical practitioner and its furnishing to the registrar were strictly enforced. The same control was not exercised over the birth registrations of perinatal deaths, producing consequent errors of coverage. This is because the onus of notification here fell on the bereaved parents. Although notification of live births and stillbirths was compulsory, registrars were reluctant to exhort grieving parents to provide a Form of Information of Birth when this had not been furnished. Thus, up to one-fifth of the births ending in a perinatal death in 1985-86 were never registered.\(^9\)

---

\(^8\) Although this percentage of successful matches was higher than for 1985-86 neonatal deaths, a similar percentage of birth and perinatal death registrations, 82.9 per cent (n=321), was achieved for 1986 neonatal deaths alone.

\(^9\) As described above, linkage of perinatal death and birth registrations was possible in only about 80 per cent of the cases.
To help resolve this problem, registrars collected for neonatal deaths a Form of Information of Death prepared by the funeral parlours. Yet the data given on this form were less complete, for example with regard to paternal details, than those provided when notifying a birth. A Form of Information of Death was sometimes also furnished for stillbirths, even though this was not the approved form. The practice was condoned because, as Forms of Information of Birth were often not completed for stillbirths, it was better to collect some information than none at all.

Content errors could have resulted from three groups of causes. The first is inadequate knowledge or the exercise of poor care or judgement by informants when reporting details. Presumably, such errors most often occurred when informants reported for someone else. For example, inaccurate or incomplete data could have resulted if the medical practitioner, who completed the Death Certificate, stated maternal characteristics, such as whether the mother was Aboriginal, of which he or she was uncertain. Similarly, on the births form one parent might have reported erroneous details for the other parent. For example, the father of a child might have incorrectly reported the mother's place of birth.

Content errors could also have arisen when informants self-reported, as with the medical practitioner's statement of the cause(s) of death on the death certificate. Error would have resulted if the cause of death was not stated correctly or if it was not stated clearly enough for proper classification by Australian Bureau of Statistics officers.

10 The approved form is the form approved by the Principal Registrar for provision of the 1975 Amendment of the Birth, Deaths and Marriages Act, 1973. The approved form here, is the Form of Information of Birth.
11 That is, according to the W.H.O. International Statistical Classification of Diseases, Injuries and Causes of Death, Ninth Revision, 1977-78.
In fact four checks were used to ensure the accurate and ethical reporting of causes of death, but these checks sometimes brought their own problems. The first check was the option of postmortem examination. Among other things, this helped to determine the cause of death, but autopsy findings,\(^\text{12}\) which may contravene clinical diagnoses (see section 4.8.2), were often still to be received at the time of registration. Secondly, the perinatal death could be reported to a coroner (see sections 4.2 and 4.7), and if a non-attending practitioner\(^\text{13}\) completed the death certificate, this had to occur. Thirdly, the Australian Bureau of Statistics could refer any death certificate back to the certifying doctor for additional information. Too often, no replies were received to queries (see section 4.8.1), although of the queries made, most related only to the fourth digit of the cause of death category; that is, a general description of the aetiology usually already existed. The final check was that there could be no cremation until the medical certificate had been received and passed by an independent medical panel.

The second, possible cause of content errors was the purposive misrepresentation of facts. Examples may include upgrading the father’s occupation or claiming native birth when the person was foreign-born. The third explanation for incomplete and inaccurate reporting is memory failure. Parents were encouraged to furnish the birth form within one month following the birth or stillbirth. However, particulars were sometimes furnished after this period expired, and as the time interval since the vital event increased, so did the potential for recall errors.

\(^\text{12}\) The terms postmortem and autopsy are used interchangeably. Both terms, and in some other works also that of the necropsy, refer to examination and dissection of the body after death.

\(^\text{13}\) A non-attending practitioner is one who either did not attend the mother of a stillborn child during her confinement, or was not in attendance during the last illness of a liveborn child.
3.5 THE MATERNAL AND PERINATAL COLLECTION

In New South Wales, public obstetric hospitals since 1981, and private obstetric hospitals since 1983, have been required by the Health Department to complete and furnish a Perinatal Statistics Form for all live births, and all stillbirths of at least 400 grams birthweight or at least 20 gestational weeks.¹⁴ The completed forms have contributed data about women and individual babies¹⁵ to the Department’s Maternal and Perinatal Statistical Collection. The Collection experienced early difficulties in achieving acceptable response rates, but by 1986, 99.0 per cent of hospitals were furnishing reasonably complete and accurate returns. This recently enabled the Department, in conjunction with the Australian Bureau of Statistics, to produce for the first time a half yearly report on New South Wales perinatal statistics (New South Wales Department of Health, 1988).

The 1986 Collection offers researchers and policy makers a valuable data source. By covering virtually all obstetric hospitals in a State where 99.5 per cent of confinements are hospital-based (New South Wales Department of Health, 1988), the Collection provides numerically large, population-based data devoid of sampling biases. These data are based on statistical rather than legal definitions of live birth and perinatal death. The Collection provides a medical summary of information that must in theory agree with that in the hospital patient records; and which extends the limited range of variables covered by registrations.

Moreover, an acceptable quality of data should be guaranteed by the method of collecting information. Forms are completed by a midwife or

¹⁴ If the delivery precedes admission to hospital, such that a form has not been completed, the hospital first admitting the woman is responsible for completing the form.
¹⁵ With multiple births, a separate form is completed for each baby.
medical practitioner who attended the delivery, and at the processing stage automatic checks or edits are used against most missing and invalid data. Such problems might result from incorrect recording or from mispunching or omissions when entering data.

A further advantage of the Collection is its inclusion of all live births, which enables researchers to select and study a control group. Collection data do not reveal whether infants survived the full perinatal period, but a reasonable proxy for this outcome is the babies who were discharged. Although some infants subsequently died, for example after being transferred to another hospital, they were comparatively few in number and should be identifiable through linking the Collection data with perinatal death registrations (see page 157). Also, there exists no better method of selecting the control group at the State level.

For all these reasons and in light of the research plan described in section 3.4, permission was sought to use data from the 1986 Maternal and Perinatal Collection in a form that would permit record linkage with the amended registration data. Permission for this being given, data suitable for the researcher's needs were provided for each of the 81,887 births which occurred in New South Wales in 1986. The 1985 Collection was not used because it was deficient in coverage and completeness, and lacked final editing checks.

From the Collection, 122 births were immediately excluded because either the birthweight was less than 500 grams or, if the birthweight was unknown, the gestational age was less than 22 weeks. Selections followed of a separate group of cases and controls from the remaining 81,765 births. This second case group, the first having been identified from perinatal death registrations, was selected for use in the identification of risk factors for perinatal death (chapters 5 to
This is because the Collection data relate to a larger number of variables than the registration data, and, given this fact, because it becomes desirable to use this same group of cases for all the explanatory analyses.

To obtain, as far as possible, a complete ascertainment of cases, three stages were executed. The first stage involved selecting the 686 perinatal deaths reported by the hospital of birth that furnished the Collection form; they included 445 stillbirths and 241 neonatal deaths. Then, these deaths were matched on six variables\textsuperscript{16} with 866 death and birth registrations to yield 505 (73.7 per cent) successful linkages; the linkages that failed are attributed to errors more often at the recording stage than during matching. In the third stage, the infants who were transferred postnatally were matched with the death registrations for which a successful match had not been found.\textsuperscript{17} This was to identify neonates who died after being transferred and who were not, therefore, reported as deaths by the Collection.\textsuperscript{18} Record linkage was achieved for 45 infants having this characteristic,\textsuperscript{19} and hence there were 731 perinatal deaths identified from the Collection, including 19 babies (11 stillbirths and 8 neonatal deaths) with missing birthweights but gestational ages of at least 22 weeks, plus 712 other perinatal deaths (see Table 3.0) comprising 434 stillbirths and 278 neonatal deaths.\textsuperscript{20}

\textsuperscript{16} These variables, common to each data source, were the baby's date of birth, date of death, birthweight and sex, and its mother's age and place of residence as indicated by the Australian Standard Geographical Classification.

\textsuperscript{17} Excluding the variable, date of death, which did not apply, the same matching variables as described in Footnote 16 were used.

\textsuperscript{18} It was not feasible to estimate the prevalence of neonatal death among the 79 008 discharged infants who were, at birth, of at least 500 grams birthweight or, when the birthweight was unknown, of at least 22 gestational weeks.

\textsuperscript{19} More of these infants should have been discovered, and it is unclear why this did not happen.

\textsuperscript{20} The matching of Collection and registration data also yielded a data set of 550, approximately three-quarters, of the identified perinatal deaths.
An important issue is the representativeness of these 731 perinatal deaths of the population of registration data. This matter is addressed in Table 3.1 which compares the prevalence of variables common to both data sets and in the (up to) 135 (55.3 per cent) missing cases. This table permits the same analyses for the women aged 20 to 34 years, with respect to whom, for reasons to be presently explained, the explanatory analysis in chapters 5 to 8 is largely undertaken.

Table 3.1 indicates that neonatal deaths constitute three-quarters (n=101) of the perinatal deaths recorded in the registration data but not in the merged Collection-registration data. Thus, neonatal deaths account for only 39.1 per cent (n=286) of these latter deaths compared with 44.6 per cent (n=387) in the registration data,21 this difference, and that between the sample proportions at maternal ages 20 to 34 years, being statistically significant at the 0.01 level.

Table 3.1: Distribution of Birthweight by Maternal Age for the Case Group Selected from the Maternal and Perinatal Collection, 1986

<table>
<thead>
<tr>
<th>Birthweight (grams)</th>
<th>Maternal Age (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Under 20</td>
</tr>
<tr>
<td></td>
<td>No.</td>
</tr>
<tr>
<td>500-1,499</td>
<td>21</td>
</tr>
<tr>
<td>1,500-2,499</td>
<td>9</td>
</tr>
<tr>
<td>2,500-3,999</td>
<td>15</td>
</tr>
<tr>
<td>4,000+</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>24</td>
</tr>
<tr>
<td>Grand Total</td>
<td>45</td>
</tr>
</tbody>
</table>

(Source. Maternal and Perinatal Collection, 1986)

21 The latter percentage approximates the reported Australian Bureau of Statistics percentage of 43.6 per cent (1,227/2,812) for 1986.
Table 3.1: Number and Percentage of Perinatal Deaths with Selected Characteristics by Data Source, 1986

<table>
<thead>
<tr>
<th>Variable</th>
<th>Perinatal Death Registrations (By year of occurrence) (n=866)</th>
<th>Merged M &amp; PC and Regs. (n=731)</th>
<th>Missing records (n=135)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td><strong>All maternal ages</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foetus or infant</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neonatal death</td>
<td>387</td>
<td>44.6</td>
<td>286</td>
</tr>
<tr>
<td>Low birthweight</td>
<td>586</td>
<td>68.7</td>
<td>470</td>
</tr>
<tr>
<td>Preterm birth</td>
<td>569</td>
<td>65.7</td>
<td>458</td>
</tr>
<tr>
<td>Male</td>
<td>461</td>
<td>53.2</td>
<td>386</td>
</tr>
<tr>
<td>Multiple birth</td>
<td>81</td>
<td>9.4</td>
<td>70</td>
</tr>
<tr>
<td><strong>Mother</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 or fewer antenatal visits</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aboriginal</td>
<td>30</td>
<td>3.9</td>
<td>30</td>
</tr>
<tr>
<td>Non-metropolitan residence</td>
<td>23</td>
<td>2.7</td>
<td>16</td>
</tr>
<tr>
<td>Non-Australian birthplace</td>
<td>319</td>
<td>36.8</td>
<td>301</td>
</tr>
<tr>
<td><strong>Maternal ages 20-34 years</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foetus or infant</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neonatal death</td>
<td>310</td>
<td>44.8</td>
<td>237</td>
</tr>
<tr>
<td>Low birthweight</td>
<td>472</td>
<td>69.2</td>
<td>385</td>
</tr>
<tr>
<td>Preterm birth</td>
<td>458</td>
<td>66.2</td>
<td>376</td>
</tr>
<tr>
<td>Male</td>
<td>366</td>
<td>52.9</td>
<td>310</td>
</tr>
<tr>
<td>Multiple birth</td>
<td>71</td>
<td>10.3</td>
<td>61</td>
</tr>
<tr>
<td><strong>Mother</strong></td>
<td></td>
<td></td>
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<tr>
<td>2 or fewer antenatal visits</td>
<td></td>
<td></td>
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<tr>
<td>Aboriginal</td>
<td>21</td>
<td>3.4</td>
<td>20</td>
</tr>
<tr>
<td>Non-metropolitan residence</td>
<td>17</td>
<td>2.5</td>
<td>12</td>
</tr>
<tr>
<td>Non-Australian birthplace</td>
<td>257</td>
<td>37.1</td>
<td>244</td>
</tr>
<tr>
<td><strong>Maternal age 45 years</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Footnotes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>a The numerator in the calculation of percentages is the total group size minus the number of missing values which varies, of course, with each variable.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b The statistical test used to assess the difference between two sample proportions is detailed in Mendenhall (1983), Introduction to Probability and Statistics, Duxbury Press, Boston: 316-317.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| * The difference between the two proportions is statistically significant at the 0.05 level using a one-tailed test.  
| ** The difference between the two proportions is statistically significant at the 0.01 level using a one-tailed test. |

Percentages of low birthweights and non-metropolitan places of residence are smaller and larger respectively in the amended Collection data than in the writer’s registration data. The differences are statistically significant at the 0.01 and 0.05 levels respectively for all the women, but not for the women aged 20 to 34 years. In both maternal groups, low birthweights and, in particular, preterm births, constitute very high proportions of the records missing from the Collection data. Only at ages less than 20 years and more than 34 years do almost all the missing records pertain to a non-Australian birthplace. Similar prevalences in the two data sets are found for male and multiple births, and, in the woman, for fewer than two antenatal visits and for Aboriginality.

A sample group of controls was selected (see Table 3.2) because the population data provided by the Health Department were unnecessarily large for the purpose of the research. There were 81 080 infants eligible for selection on the basis of two criteria: the infants were discharged or transferred\(^\text{22}\) from the hospital furnishing the Collection form, and at birth the infant weighed at least 500 grams or, when the birthweight was unknown, the infant was of at least 22 weeks’ gestation.

The selection of the sample involved three stages. First, through stratified sampling, every woman was selected who delivered a surviving infant between 500 and 1 499 grams. However, about one-fifth of these 454 babies are estimated to have died, in fact, during the perinatal period. Specifically, at very low birthweights, 90 registered deaths were not included in the deaths selected from the Collection data, that is 90 (58.4 per cent) of the total (all known

\(^{22}\) As 46 transfers (2.1 per cent) were known to have died, a search was made for these same infants among the selected sample of controls; 14 such infants had been selected and they were deleted from the control group accordingly.
birthweights) of 154 (866 minus 712) missing deaths, and the level of misclassification is 19.8 per cent (90/454). For the women aged 20 to 34 years, the figure is 19.2 per cent (71/369).

Secondly, when the birthweight exceeded 1 499 grams, random sampling was used to frequency match controls with the cases already identified from the Collection. This matching was done on categories of the woman's age and the baby's birthweight at an approximate 5:1 ratio of controls to cases. In the third stage, fourteen transferred infants who had been selected as controls were removed from the control group because they were found to have died. They were not replaced since all but two of these infants weighed less than 1 500 grams at birth, and every surviving infant in this birthweight category had already been selected.

Table 3.2: Distribution of Birthweight by Maternal Age for the Control Group Selected from the Maternal and Perinatal Collection, 1986

<table>
<thead>
<tr>
<th>Birthweight (Grams)</th>
<th>Under 20</th>
<th>20-34</th>
<th>Over 34</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>500-1 499</td>
<td>31</td>
<td>6.8</td>
<td>369</td>
<td>81.3</td>
</tr>
<tr>
<td>1 500-2 499</td>
<td>45</td>
<td>2.3</td>
<td>585</td>
<td>30.5</td>
</tr>
<tr>
<td>3 500-3 999</td>
<td>75</td>
<td>3.9</td>
<td>895</td>
<td>46.7</td>
</tr>
<tr>
<td>4 000+</td>
<td>0</td>
<td>0.0</td>
<td>75</td>
<td>3.9</td>
</tr>
<tr>
<td>Total</td>
<td>120</td>
<td>6.3</td>
<td>1555</td>
<td>81.2</td>
</tr>
</tbody>
</table>

(Source. Maternal and Perinatal Collection, 1986)
The use of sampling complicates estimation of the extent of any misclassification of perinatal deaths at birthweights 1 500 grams and over. However, following the same procedure as above and adjusting for the sampling fraction in each birthweight interval, crude estimates of the misclassification are well below 1 per cent in each birthweight category.

The rationale for, and implications for analysis produced by, these selections of the case and control groups is now stated. The discussion relates to the database derived from the Maternal and Perinatal Collection but also to the data similarly selected from hospital sources (see section 3.6). In each collection the control group was chosen according to the birthweight and maternal age distributions of the case group because, first, birthweight and maternal age are risk factors for perinatal death, and secondly, because the intention is to control for their effects in order to ascertain the aetiological contributions of other study exposures.

The more important potential confounder is birthweight. That low birthweight in particular is a key determinant of perinatal death was indicated in section 2.3.1, and can be verified for New South Wales from Tables 3.3 and 3.4.

Table 3.3: Distribution of Birthweight by Maternal Age for the Population of New South Wales, 1986

<table>
<thead>
<tr>
<th>Birthweight (grams)</th>
<th>Maternal Age (years)</th>
<th>&lt;20</th>
<th>20-34</th>
<th>&gt;34</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>500-1 499</td>
<td>21</td>
<td>46.7</td>
<td>31</td>
<td>0.7</td>
<td>286</td>
</tr>
<tr>
<td>1 500-2 499</td>
<td>9</td>
<td>20.0</td>
<td>117</td>
<td>20.2</td>
<td>2966</td>
</tr>
<tr>
<td>2 500-3 999</td>
<td>15</td>
<td>33.3</td>
<td>4032</td>
<td>86.0</td>
<td>179</td>
</tr>
<tr>
<td>4 000+</td>
<td>0</td>
<td>7.7</td>
<td>360</td>
<td>2.6</td>
<td>7770</td>
</tr>
<tr>
<td>Total</td>
<td>45</td>
<td>100.0</td>
<td>4697</td>
<td>100.0</td>
<td>579</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
(Source: Maternal and Perinatal Collection, 1986)
Table 3.3 shows that two-thirds of perinatal deaths, but just 5 per cent of perinatal survivors, occur at low birthweights, the differential of twelve times increasing to 77 times at very low birthweights. Irrespective of survival, low birthweights are most prevalent in numerical terms when women are aged 20 to 34 years since this is the maternal age group at which most births occur.

Adolescents record high proportions of perinatal deaths and survivors at low birthweights, whilst also at low birthweights the percentages of perinatal deaths for mature-aged women, and perinatal survivors for women aged 20 to 34 years, are low compared with those in the other maternal age groups.

Table 3.4: Age-Birthweight-Specific Perinatal Death Rates for New South Wales, 1986

<table>
<thead>
<tr>
<th>Birthweight (grams)</th>
<th>Maternal Age (years)</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt; 20</td>
<td>20-34</td>
<td>&gt;34</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>500-1 499</td>
<td>.404 (.271, .537)</td>
<td>.421 (.382, .459)</td>
<td>.426 (.326, .525)</td>
<td>.420 (.386, .455)</td>
<td></td>
</tr>
<tr>
<td>1 500-2 499</td>
<td>.033 (.012, .054)</td>
<td>.038 (.031, .045)</td>
<td>.040 (.020, .060)</td>
<td>.038 (.032, .044)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>.092 (.061, .123)</td>
<td>.104 (.094, .114)</td>
<td>.118 (.089, .148)</td>
<td>.105 (.096, .114)</td>
<td></td>
</tr>
<tr>
<td>2 500-3 999</td>
<td>.004 (.002, .006)</td>
<td>.003 (.003, .004)</td>
<td>.005 (.003, .007)</td>
<td>.003 (.003, .004)</td>
<td></td>
</tr>
<tr>
<td>4 000+</td>
<td>.002 (.001, .003)</td>
<td>.004 (.000, .009)</td>
<td>.002 (.001, .003)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>.010 (.007, .013)</td>
<td>.008 (.007, .009)</td>
<td>.013 (.010, .016)</td>
<td>.009 (.008, .010)</td>
<td></td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a The parenthesized values are the lower and upper confidence limits at the 0.95 level
(Source: Maternal and Perinatal Collection, 1986)

These exposures in the groups of deaths and survivors are reconciled in Table 3.4 into single age-birthweight specific perinatal death rates, that for all very low birthweight babies being 0.420 per 1 000. This rate is consistent with Tudehope’s and Thearle’s (1989) results showing survival by 26, 55 and 87 per cent of infants weighing less than 750, 750 to 999 and 1 000 to 1 499 grams respectively at Brisbane’s Mater Mothers’ Hospital from 1977 to 1982. These authors
reported the survival of 95 and 98 per cent of infants weighing 1,500 to 1,999 and 2,000 to 2,499 grams respectively, whereas in Table 3.4, the percentage surviving is 96.2 per cent.

The overall rate of perinatal death at low birthweights, without consideration of maternal age, is 0.105 per 1,000, reaching 0.118 per 1,000 among mature aged women (for whom it may be as high as 0.148 per 1,000 or as low as 0.089 per 1,000) and a nadir of 0.092 per 1,000 among adolescent women. Collapsing over birthweight, the highest perinatal death rate of 0.13 per 1,000 is recorded by women aged 35 years and over, followed by 0.10 per 1,000 for women under aged 20 years and finally 0.08 per 1,000 for women aged 20 to 34 years. This supports the knowledge that women at adolescent and mature ages are at increased risk of perinatal death.

Accepting that low birthweight is an important cause of perinatal death, what should now benefit from investigation is why some low birthweight babies survive when others do not, adjusting for factors including maternal age. The samples of controls from the Maternal and Perinatal Collection and hospital sources (see section 3.6) facilitate the study of this problem by permitting retrospective comparison of differences between the case and groups in factors affecting perinatal outcomes in different birthweight and age strata.

The main problems with using birthweight in this manner are that birthweight is only one index of risk and its measurement occurs after exposures, such as antenatal care, that occur during pregnancy. However, foetal weight can be estimated in utero, most simply by the symphyseal-fundal height, and more accurately, when ultrasound is available, by measurement of the biparietal diameter and abdominal circumference (New South Wales Department of Health, 1989d). Measurement of the infant’s weight at birth is straightforward when
accurate scales and standardized procedures are used (Lancaster, 1989b).

Another constraint is that the sampling design does not facilitate examination of the complete sample of control members with its skewed birthweight and age distributions, low birthweights and adolescent and mature women being over-represented. The sample is intentionally more amenable to the examination of individual birthweight and age strata. Yet it would be cumbersome and impracticable for basic, tabular analyses to display four categories of birthweight and three of age even before entry of the study factor(s) of interest. The solution adopted is that, while small numbers of logistic regressions incorporate all three age groups, basic methods of analysis examine each birthweight stratum only for the highly prevalent group of women aged 20 to 34 years for whom age per se is not a risk factor (see sections 3.8 to 3.8.2).

3.6 HOSPITAL SOURCES

The 1986 Maternal and Perinatal Collection has, despite its usefulness, two main limitations: because the Collection is a summary of medical facts, albeit a primary one, some items are not asked for or, secondly, they are poorly reported by the midwives or medical practitioners. Omitted items include clinical details such as a maternal haemoglobin value for estimating nutritional status, and most seriously, behavioural information about the expectant mother’s environment. The latter omission is important because lifestyle

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23 Age is a nuisance factor adjusted early out of the regressions.
24 Collapsing over the age stratification in the absence of differing factor effects across the age strata would lead to inconsistency in reporting tables, and such an assessment is also not possible when cell sizes are made too small by the inclusion of a large number of factors.
information is often needed to understand why aetiological conditions develop in the first place.

Other variables are asked about, but they are poorly reported on the Collection form because the information needed is frequently missing from the hospital patient records. This applies especially to antenatal details, such as the timing of the first antenatal visit, because the private practice antenatal record card or even a copy of this card is not usually included in the hospital patient record.

It was decided, therefore, to make two requests of the fifteen largest obstetric hospitals in New South Wales, which were the eight teaching hospitals and seven non-teaching hospitals annually performing 2000 or more deliveries.25 The requests were

(1) that the researcher be permitted to use for statistical purposes the clinical information contained in 1985 maternity records; and

(2) that the informed consent of selected 1986 women be obtained to extract statistical data from their maternity records and to conduct an interview in their homes.

Having determined on the need to contact selected hospitals, the next stage was to gain the support of the New South Wales Department of Health’s Maternal and Perinatal Steering Committee. A negative response from the committee would save future time wasting, and it seemed prudent to prevent hospitals asking the Department for a directive before the researcher had first presented his research protocol to that body. Committee support would add credibility to the project, and because some of Sydney’s leading obstetricians compose

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25 The same requests were made of three hospitals in both Victoria and the A.C.T.
the committee, its approval was expected not only to speak for the Health Department, but directly to strengthen the protocol in individual hospitals. In any case, the committee’s considerations could probably not have been avoided since its approval was also needed, on the basis of the protocol, for the researcher to use the Maternal and Perinatal Statistical Collection. 26

Approval by the committee was deferred until the protocol had been independently assessed by a nominated biostatistician, and when this assessment recommended that the study proceed, the committee agreed that the researcher should approach the hospitals, and have access to the Maternal and Perinatal Collection.

Securing hospitals’ approval to conduct the research became, and was always expected to be, a time-consuming and difficult task. The hospitals and, where they existed, their area health boards, to which the protocol might be referred, required the full research protocol and often a summary of this document in their own formats. For the study to proceed, these materials had to be approved by institutional ethics committees and, sometimes, by separate research committees. One or more of these committees might consider the project more than once and ask to meet the researcher, and this could occur either before or after hospital departments such as obstetrics had considered the protocol. At any of these stages, the project could be delayed or rejected.

This machinery was used to assess the protocol’s defensibility on ethical and research grounds, yet also important is how its working parts made their determinations. To assert that approval depended on

26 Access to the Maternal and Perinatal Collection also required the permission of the Information and Data Services Committee of the New South Wales Department of Health. Moreover, permission to undertake the investigation itself had earlier followed the approval of the Ethics and Human Experimentation Committee of the Australian National University.
the quality of the protocol per se would be to ignore other influences. For example, seeking the cooperation of multiple institutions invoked a domino effect: the largest hospitals, in particular, were influenced by the responses of other hospitals, and coordinating the reporting of these responses across hospitals required considerable care.

In this context, therefore, the researcher, and not merely the written protocol, had to be demonstrated to be credible. This required that an effective communication base be established with a large number of hospitals. Indeed this was especially important given the unusual circumstances of the researcher: he was unknown, a single scholar working outside all the hospitals, lacking in medical training and asking to undertake sensitive research involving hospital patients and their personal records. Not surprisingly, persuading the hospitals to consider the researcher and his protocol seriously, at first proved difficult: eight hospitals claimed that they never received the copy of the protocol first sent to them, and two hospitals claimed that they lost the protocol twice.27

Fortunately, with persistence, relations became easier and, as noted above, once approval was received from one or two hospitals, other units appeared more willing to express their support. Eventually the research protocol was approved by six New South Wales hospitals.28 They are not named owing to the writer's commitment to preserve their anonymity. However, some general comments are made about the characteristics of the hospitals and their catchment populations.

27 Explanation may also be found in the inefficiencies of hospital bureaucracies, as exemplified by staff changeovers without replacements always being fully briefed by their predecessors, and in possible mishandling by the Australian postal service.

28 The protocol was also approved by the three public hospitals in the A.C.T., and it was rejected by the three Victorian hospitals contacted. Lack of time finally prevented the researcher from including the former hospitals in the data collection.
The six participating hospitals were of varying sizes above the minimum threshold of 2,000 annual deliveries, and they extended over five health regions. Three of the hospitals, and one of the two located in the same health region, were in Sydney, and three hospitals were also equipped to provide intensive (level 3) neonatal care (see section 6.1). These last units were major referral centres for urban and rural women at high risk of perinatal complications. Two hospitals, although one in particular, served a large proportion of women from non-English speaking backgrounds. These women tended to be of low socioeconomic status, living in neighbourhoods that either were densely populated and close to the local business district or in peripheral localities with low population densities. Conspicuous among the four remaining catchment populations were, for two hospitals, financially secure or at least upwardly mobile women, and, for the remaining hospitals, populations whose socioeconomic status was heterogenous.

A broad spectrum of women was thus served by the participating hospitals, such that prima facie evidence suggests that collectively these units were probably not unlike other large hospitals. Nevertheless it is helpful to assess statistically whether the hospitals that approved the protocol were representative, first of all the hospitals contacted and secondly, of all the hospitals in New South Wales. The answers to these questions are important because for statistical inference to be justifiable, samples must be representative or typical of the populations whence they come. To provide answers, levels of selection bias were measured using chi-square tests of significance, in which representativeness was arbitrarily defined when significance levels reached 0.05.

Table 3.5 relates to selection bias resulting from only some of the largest hospitals approving the protocol. The results show no
statistically significant differences in the birthweight, maternal age and gravidity distributions of the case group observed at participating hospitals vis-à-vis the case group found at all large maternity hospitals in New South Wales. The findings are less pleasing when infants surviving the perinatal period are considered.

Table 3.5: Representativeness of Participating Hospitals of New South Wales Hospitals Performing 2 000 or More Deliveries Annually, 1986

<table>
<thead>
<tr>
<th>Variable</th>
<th>Chi-Square Statistic</th>
<th>Degrees of Freedom</th>
<th>Significance Level (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case Group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birthweight</td>
<td>5.89</td>
<td>3</td>
<td>0.12</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>1.02</td>
<td>2</td>
<td>0.59</td>
</tr>
<tr>
<td>Gravidity</td>
<td>2.98</td>
<td>5</td>
<td>0.70</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Control Group</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Birthweight</td>
<td>11.85</td>
<td>3</td>
<td>0.01</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>13.72</td>
<td>2</td>
<td>0.00</td>
</tr>
<tr>
<td>Gravidity</td>
<td>5.97</td>
<td>5</td>
<td>0.31</td>
</tr>
</tbody>
</table>

(Source. Maternal and Perinatal Collection, 1986)

Only on gravidity was it probable that the sample of controls found at participating hospitals was not randomly derived. For the distributions of birthweight and maternal age, the likelihoods of no statistically significant differences were 0.008 and 0.001 respectively. This is because the participating hospitals had more expectant mothers aged 35 years and over, and more babies of very low birthweight than would be expected given the maternal age and birthweight distributions for perinatal survivors at all large maternity hospitals.

Table 3.6 shows the extent of selection bias attributable to the participating hospitals not being representative of all hospitals in the State. For the case group selected from the participating hospitals, there was an acceptably high probability of selecting the
same sample, or one less like the population, on maternal age and gravidity. However, there was almost nil probability ($p = 0.002$) that a sample with the same birthweight distribution, or one worse, would be selected again from this population: the participating hospitals had more babies dying at birthweights less than 1500 grams, and fewer babies dying at birthweights 2500 to 3999 grams than would be expected from the birthweight distributions of all hospitals.

However, with regard to birthweight, the bias was an intentional effect of the sampling design. Low birthweight infants were intentionally over-sampled by contacting only hospitals performing at least 2000 deliveries annually in order to permit at the analysis stage statistical control of confounding effects due to physiological immaturity.

Table 3.6: Representativeness of Participating Hospitals of all New South Wales Hospitals, 1986

<table>
<thead>
<tr>
<th>Variable</th>
<th>Chi-Square Statistic</th>
<th>Degrees of Freedom</th>
<th>Significance Level ($p$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case Group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birthweight</td>
<td>14.99</td>
<td>3</td>
<td>0.00</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>1.44</td>
<td>2</td>
<td>0.49</td>
</tr>
<tr>
<td>Gravidity</td>
<td>2.22</td>
<td>5</td>
<td>0.82</td>
</tr>
<tr>
<td>Control Group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birthweight</td>
<td>143.60</td>
<td>3</td>
<td>0.00</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>68.29</td>
<td>2</td>
<td>0.00</td>
</tr>
<tr>
<td>Gravidity</td>
<td>5.66</td>
<td>5</td>
<td>0.34</td>
</tr>
</tbody>
</table>

(Source. Maternal and Perinatal Collection, 1986)

Selection biases pertaining to the control group were of more serious concern. Only on gravidity was the control group likely to have been representative of perinatal survivors at all hospitals. The variable least representative was birthweight owing to twice as many very low birthweight babies surviving at the participating hospitals; other
birthweight categories show small proportionate differences, but large group sizes produced a very high chi-square statistic. The low significance level found for maternal age is accounted for by the latter problem and by an excess of women aged 35 years and over.

3.6.1 SELECTION OF HOSPITAL PATIENTS

In each participating hospital, 'suitable' subjects were selected for inclusion in the study. Usually using hospital registers of births and deaths, case and control groups were selected separately for 1985 and 1986 in a similar manner to that described in section 3.5, and thus permitting the same method of analysis. Specifically, all cases and very low birthweight survivors were selected, and additional control members were randomly selected after group matching at a 1:1 ratio on the maternal age and birthweight distributions of hospital cases.

For 1985, these procedures yielded 201 cases and 246 controls for whom patient record summaries were completed. These data extractions were made without patients' consent to spare these women, from whom an interview was not sought, unnecessary anxiety. Explaining the research and obtaining consent was considered likely to cause the women more anxiety than the research itself ever would have done (National Health and Medical Research Council, 1985a: 14); and this argument was accepted by each participating hospital.

Similar totals of 1986 women were selected: 214 cases and 242 controls. However, to be included in the project these women were asked to confirm their selection on a confidential basis by giving written informed consent to one or both of two requests: to the researcher extracting information for statistical purposes from their hospital patient records, and to an interview in their home.
Each woman received a letter from her hospital (see Appendix 1 for one example). This covered a second, enclosed letter from the researcher which, without containing the woman's name or address (see Appendix 2), explained the project more fully. Forms of consent to participate in the project (see Appendix 3) and a freepost envelope were also provided. The consent forms contained the general purpose, methods, risks and expected returns of the study. Subjects were asked to consent to the two requests stated above, and the women were told that they could withdraw from the project at any time.

Only after signed consent to an interview was received, either in the researcher's post office box or by the hospital, did the researcher make personal contact with patients. This contact usually involved a telephone call to thank the respondent for agreeing to help; to offer to clarify any points about the project; to confirm the woman's willingness still to participate; and to arrange a convenient time for an interview.

Table 3.7 shows response rates, and interviews plus record extractions completed for the selected case and control groups. Of the case women contacted, 77 (36.0 per cent) consented to an interview and to the use of their patient records. However, both collections were made of only 72 women: an interview was completed for three women whose records could not be traced, and two interviews were prevented by difficulties in arranging an interview. A further nine women consented just to the use of their records, so that 40.2 per cent (n=86) of women who experienced a perinatal death showed some willingness to aid the research. For the women who consented to both requests, the mean response rate across the six participating hospitals was 39.9 per cent, but the sample standard deviation was 12.2 per cent owing to response rates ranging from 52.6 per cent to 23.2 per cent.
Table 3.7: Consent to an Interview and to Patient Record Extractions by Selected Case and Control Groups, 1986

<table>
<thead>
<tr>
<th>Consent to:</th>
<th>Interview and use of records</th>
<th>Use of records only</th>
<th>Total response</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td><strong>Case group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number contacted, n=214</td>
<td>77</td>
<td>36.0</td>
<td>9</td>
</tr>
<tr>
<td>Completed:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interview and record summary</td>
<td>72</td>
<td>93.5</td>
<td>-</td>
</tr>
<tr>
<td>Interview only</td>
<td>3</td>
<td>3.9</td>
<td>-</td>
</tr>
<tr>
<td>Record summary only</td>
<td>1</td>
<td>1.3</td>
<td>6</td>
</tr>
<tr>
<td>No collection</td>
<td>1</td>
<td>1.3</td>
<td>3</td>
</tr>
<tr>
<td>Total interviews</td>
<td>75</td>
<td>97.4</td>
<td>-</td>
</tr>
<tr>
<td>Total record summaries</td>
<td>73</td>
<td>94.8</td>
<td>6</td>
</tr>
<tr>
<td><strong>Control group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number contacted, n=242</td>
<td>68</td>
<td>28.1</td>
<td>6</td>
</tr>
<tr>
<td>Completed:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interview and record summary</td>
<td>62</td>
<td>91.2</td>
<td>-</td>
</tr>
<tr>
<td>Interview only</td>
<td>1</td>
<td>1.5</td>
<td>-</td>
</tr>
<tr>
<td>Record summary only</td>
<td>5</td>
<td>7.4</td>
<td>4</td>
</tr>
<tr>
<td>No collection</td>
<td>0</td>
<td>0.0</td>
<td>0</td>
</tr>
<tr>
<td>Total interviews</td>
<td>63</td>
<td>92.6</td>
<td>-</td>
</tr>
<tr>
<td>Total record summaries</td>
<td>67</td>
<td>98.5</td>
<td>4</td>
</tr>
</tbody>
</table>

(Source. Interview data, 1986; Hospital patients' records, 1985-86)
As Table 3.7 also shows, comparatively poor response rates were achieved for the control group: 68 of these women (28.1 per cent) consented to both requests, with interviews and record extractions being completed for 62 women. Of the remaining six women, only one was interviewed owing to difficulties in re-establishing contact with patients at one large hospital. Including the women who consented only to use of their records, just 30.6 per cent (n=74) of the control group were prepared to participate in the project. Across the six hospitals, the mean response rate for consenting to both requests was 31.1 per cent with a sample standard deviation of 7.6 per cent; the range extended from a high of 42.1 per cent to 20.0 per cent.

3.6.2 EXTRACTION OF DATA FROM MATERNITY RECORDS

The information sought from maternity records is shown in Appendix 4. The main problem in extracting these data from records, as also found by Cartwright et al. (1987), was determining with certainty that particular events did not take place. Not only did identifying events require familiarity with the layout of notes, but entries were written in different hands. Certain factors of interest were not reported, as was noted earlier with respect to antenatal details for private and transferred patients.29

Understanding the medical jargon associated with the factors of interest was not a major problem, but inconsistencies appeared, for instance, in assessments of the gestational age at birth. Still, more worrying than identifying events wrongly was the possibility of overlooking them, and for this reason, plus omissions on the records, data extractions might have underestimated the frequency with which complications occurred and procedures were carried out.

29 Sometimes this information could be furnished by the 1986 women who consented to an interview.
Unfortunately, although in retrospect predictably, the variables least well reported on the Maternal and Perinatal Collection Form were also those exhibiting the greatest errors of coverage in the medical records. Notwithstanding this, the records furnished useful information, for example, on prescribed medications not elsewhere collected, and the data extracted from the 1985 records supplemented the Maternal and Perinatal Collection data limited to 1986.

Collecting the data first-hand and the ready availability of medical advice contributed to the researcher's understanding of the data being collected.

Viewing the records also afforded the opportunity to check the validity of reporting on the Collection form, especially for variables against which edits could not be made. Recording errors were few, but they sometimes resulted from medical records staff, who dispatched the forms to the Health Department, wrongly entering details upon finding omissions or apparent errors. These forms should have been returned to the midwife or medical practitioner who signed them, for rechecking and if necessary, correction. But in some cases this seems not to have happened, in one hospital because of a perceived lack of cooperation from the maternity ward. Most errors were presumably identified through editing by the Health Department.

3.7 INTERVIEWS

Interviews were used to collect original data on the pregnancy environments of small groups of 1986 case and control women. These data promise potentially valuable insights, but their method of collection makes them especially liable to biases resulting from characteristics of the interview task, and interviewer and respondent characteristics (Bradburn and Sudman, 1979).
Dijkstra and Van der Zouwen (1982) distinguish between structural task characteristics and characteristics of the questions asked. Structural task characteristics describe the method of administering the interview schedule, so as, for example, to place respondents at ease and to create respondent motivation. These characteristics are particularly important because they condition the response effects of question variables (see section 3.7.2) and interviewer and respondent variables (see section 3.7.3). In practice, the most important structural task variables were found to be the development of a strong rapport and a professional manner. These ends are exemplified by the interview setting and the method of asking questions, in terms of which the following comments derive mainly from the experiences of the writer, since he conducted most of the interviews, whilst according with those of an assistant interviewer (see section 3.7.3).

The interviews, based on the schedule presented in Appendix 5, were held in each woman's home. Every effort was taken to avoid an audience since the presence of other persons can bias answers. However, the interviewer did not object when, in a small number of instances, the woman wanted her husband present; and these interviews appeared no less candid than those where the women spoke alone. Seating was found that let the respondent and the interviewer be comfortable, usually placing the interviewer next to rather than across from the respondent.

Although this seating arrangement let respondents read the interview schedule and possibly be influenced by knowing which questions followed, it permitted the interviewer to involve the women in the interview process as active, informed and strongly motivated participants. With the matrix reconstructions, the women could see how the charts were built up and visually cross-check them for their
internal validity. Also, the seating arrangement was set by the practice of first showing women the information extracted from their hospital patient records. Most women found the record summary interesting, and they appeared reassured to see how the data had been coded for analysis in aggregated form.

The method of asking questions was situationally directed toward achieving respondent ease, interest and motivation. To these ends, the set questions for completing the matrices (sections A to D) were not rigidly adhered to, but used instead to structure a less formal, conversational approach to asking questions. This provided, especially in contingency situations, for more efficient construction of the matrices, and although perhaps it caused stimulus conditions to vary slightly across the interviews, it permitted a sensitivity of questioning that must have helped to elicit full and open responses.

The effort was made, moreover, to adhere strictly to certain rules when asking questions. The interviewer influenced respondents' answers as little as possible by acting non-directively and in accordance with the question objectives. That is, as far as possible, nothing in the interviewer's words or manner implied approval, disapproval or surprise either at the questions themselves or at respondents' answers. The interviewer tried not to offer unrelated information likely to distract respondents from answering questions or to imply or suggest particular responses. At the same time, the interviewer sought always to be warm, sensitive and non-judgemental.

3.7.2 QUESTION CHARACTERISTICS

The level of recall bias\(^{30}\) depended partly on structural task characteristics, but also on the extent to which 'formal' characteristics, but also on the extent to which 'formal'

---

\(^{30}\) Recall bias is defined as the differential recall of prior exposures to potential risk factors between the case and the control groups.
characteristics of questions resolved three potential problems. First, subjects inevitably forgot specific events that occurred more than eighteen months previously. Although salient events were the least likely to be forgotten, seemingly inconspicuous events were also recalled, not only by the case women but also by the women whose surviving infants had faced the risk of perinatal morbidity or death; this had prompted these women to review their pregnancies with care. A second problem associated with memory was likely to have been telescoping, which means that frequent events were brought forward in time.

Thirdly, some women, presumably especially in the case group, might not have recalled behaviours such as heavy smoking about which they felt guilty or embarrassed. Yet the women who consented to an interview were, because they had consented to help, unlikely to withhold information or give intentionally false answers. In practice, this was shown by respondents' candour in relating events that may not be 'socially acceptable'. However, problems such as 'effort after meaning' are described in section 7.1.

To reduce all three sets of difficulties, a novel approach was taken when designing the interview instrument. The first part of the schedule (Sections A to D) was made to comprise four pregnancy and postpartum history matrices. These were constructed to interest respondents and to increase motivation, while helping to stimulate, order and crosscheck the recall of events by directly relating them to each other. The interviewer and respondent became able to reconstruct together a concise, consistent and probably valid, chronological record of events.

In the matrices, the attempt was made to use non-biased phraseology in wording questions; items were factual in content, relatively simple
and devoid of judgemental language. The most threatening questions were placed last because rapport was then most likely to be well-established (DeLamater, 1982).

Recall bias was probably not serious in the second part of the schedule (section E) which contained short-answer questions about the woman's antenatal life; these closed or structured questions were the least personal. Again, biases relating to the wording of questions and to response categories were avoided as far as possible. Threatening topics were placed toward the end of sets of related items, although probably only small response effects are associated with the location of questions (Molenaar, 1982).

A third and final part of the schedule (section F) was originally intended to introduce open-ended questions encouraging the case women to discuss their feelings and ideas about the pregnancy and perinatal death. However, in practice, certain questions tended to generate the same responses and a small number of women found the questions distressing. For these reasons and because these data were the least important to the study - important less for themselves than for their abilities to contextualize the meaning and significance of the structured antenatal information - the researcher decided to drop the questions in their existing format.

Instead, in relaxed conversation outside the formal interview, it was a simple matter to ask questions that were situationally relevant such as why a respondent felt she had gone into preterm labour. It thus remained possible to move beyond undirectional, extractive questioning to collect the informant's data based on her priorities rather than the researcher's. In these terms, the analysis of perinatal outcomes still acquired a broad, holistic perspective.
It is worth noting that other steps had earlier been taken to help ensure that informants would not be distressed by the interview. The women contacted had all lost their babies at least eighteen months previously so that they were likely to have entered the reorientation phase of their bereavement. The initial contact encouraging participation came from the hospital to preserve subjects' anonymity and to minimize the interference to their lives. Formal arrangements were made for women to receive follow-up professional counselling if they considered this necessary, although this help was never asked for.

Because of these precautions, and the researcher's willingness to adjust the interview schedule in the manner described, very few women appeared to find the interview distressing. Indeed, this confirms the finding of other studies that contact and interviews are well received (Nichol et al., 1986; Tudehope et al., 1986). The interview allows these women to seek information and to review the pregnancy, delivery and subsequent events (Rowe et al., 1978; Clyman et al., 1980); these instrumentalities facilitate the catharsis of unresolved feelings of guilt and helplessness (Raphael, 1984). Moreover, the women seemed to feel good about doing something that may help prevent the same tragedy recurring, either to themselves or to other women.

3.7.3 INTERVIEWER AND RESPONDENT CHARACTERISTICS

Bias can also result from ascribed characteristics of the interviewer. However, in this study the extent of that bias cannot be measured quantitatively because the researcher, then 27 years old and never-married, conducted most of the interviews, that is, 86.7 per cent (n=65) of the interviews with the case group, and 98.4 per cent (n=62) of the interviews with the control group. The remaining interviews were conducted, after suitable training, by a married
mother who had personally experienced a neonatal death and who was the current secretary of the Stillbirth and Neonatal Death Association in Sydney. This woman was completing an undergraduate degree in psychology and possessed a wonderfully warm and compassionate personality. All these qualifications produced an ideal person to help conduct the interviews, and no significant bias is attributable either to her performance or to that of the researcher.

With regard to respondent characteristics, selection bias depends on the extent to which the women who consented to an interview differed from the women who did not consent to an interview. Direct measurement of this bias cannot take place because failure to consent to an interview, *ipso facto*, prevented the extraction of details pertaining to these women. However, the 1985 women, for whom data were collected from hospital records, can be treated as surrogates for the contacted groups of 1986 women. This was done in an attempt to measure the representativeness of the interviewed groups as earlier defined by $p > 0.05$.

The results, displayed in Table 3.8, show that based on the distributions of maternal age and gravidity, the case sample may be typical of the population contacted. However, with the given rejection level, this is not true for birthweight: on this variable, there were no statistically significant differences between the groups in less than 0.01 per cent of subjects. The problem is that more women who lost a small baby, particularly one weighing 1 500 to 2 499 grams at birth, consented to an interview than would be expected on the basis of the birthweight distribution of all cases; and of course the reverse holds for babies of higher birthweight. Acceptably high probabilities were found of selecting a control group whose distributions on all three variables were at best the same.
These biases resulted from factors encouraging non-participation in the study. They are likely to have included difficulties in adjusting to the loss, and hence an unwillingness to be reminded of what happened or to suffer further distress. Other deterrents to participation could have been changes in the place of permanent residence which prevented women from receiving the correspondence from their hospital; language problems, making it difficult to understand this correspondence or producing actual or perceived obstacles in fulfilling its requirements; and barriers of physical distance, also causing doubts about the feasibility of an interview. Other women might have intended to sign and return the consent forms, but have forgotten to do so, perhaps until it appeared too late, while some control women in particular might have lacked sufficient interest and motivation to agree to help.

**Table 3.8:** Representativeness of Interviewed Women of all Women Asked for an Interview, 1986

<table>
<thead>
<tr>
<th>Variable</th>
<th>Chi-Square Statistic</th>
<th>Degrees of Freedom</th>
<th>Significance Level</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Case Group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birthweight</td>
<td>8.95</td>
<td>2</td>
<td>0.01</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>3.72</td>
<td>2</td>
<td>0.16</td>
</tr>
<tr>
<td>Gravidity</td>
<td>0.52</td>
<td>3</td>
<td>0.92</td>
</tr>
<tr>
<td><strong>Control Group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birthweight</td>
<td>3.17</td>
<td>2</td>
<td>0.20</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>0.12</td>
<td>1</td>
<td>0.73</td>
</tr>
<tr>
<td>Gravidity</td>
<td>2.12</td>
<td>3</td>
<td>0.55</td>
</tr>
</tbody>
</table>

(Source. Interview data, 1986)

3.8 METHODS OF DATA ANALYSIS

Previous sections have focused on what, and how, data were collected and subsequently prepared in order, for example, to permit analyses capable of testing hypotheses. The present discussion considers more
directly how these data are analysed. As implied in section 3.5, the
data analyses are exploratory, involving simple statistical methods
associated with two- and higher-order contingency tables. This is
because the groups, especially of perinatal deaths, are small in size,
representing a time period of one or at most two years, and because it
is important to attain familiarity with one's data before proceeding
to more complex analyses. Stratifying on two or more variables, which
is one method of eliminating biased comparisons due to confounding,
does in any case permit a basic form of multivariate analysis not
dependent on the validity of the assumptions underlying parametric
models.

As described more fully below, stratification is usually attended by
summary estimates of odds ratios 'adjusted' for the effects of the
variables used in the stratification(s). Further adjustment for
confounding is achieved by limiting tables to specified sub-groups
such as women aged 20 to 34 years (see section 3.5), which improves
the presentation of results. Of course, this latter approach loses
some information, and as variables of interest increase in number, the
analysis of multiple cross-classifications becomes less feasible. A
further disadvantage of these tables is their comparative inability
vis-à-vis multivariate methods to explore interactive effects. Thus,
logistic regression models are briefly developed once apparently key
risk factors, and the reasonableness of assumptions underlying these
models, have been assessed on the basis of the earlier simpler
analyses. First though to the basics: two concepts are fundamental to
measuring the risk of perinatal death: the prevalence, meaning the
frequency of occurrence, of death, survival or both events, and the
relative risk of death, each concept being associated with exposure to
known or suspected risk factors.
3.8.1 ESTIMATES OF PREVALENCE

Calculated rates of period prevalence relate events or attributes of persons during specified periods to the total populations during these periods. For some, but not all, exposures, measures of prevalence may also be interpreted to reflect incidence, which is the number of new events during a specified period. Antenatal care exemplifies an exposure for which rates of incidence are not shown from the data collected because such care might not have originated after the beginning of the period, say 1986, but instead be carried over from 1985. It should also be noted that the numerator is indicated by the \( n \) values associated with each percentage included in the text.

The measurement of prevalence is undertaken almost exclusively for women aged 20 to 34 years, and mainly in individual birthweight strata; that is, the denominator is the number of women or infants in any particular birthweight stratum. When the denominator is some other total number of births, derived through aggregation of the different birthweights shown in the table, proportions are calculated after weighting the birthweight distributions of (a) the control group (separately for singleton births and all surviving neonates) drawn from the Maternal and Perinatal Collection, and (b) the case and control groups selected from hospital sources (see Table 3.9). Weights are estimated as the inverse of the proportion of the selected number of infants out of the population total, or best estimate of this total, for the relevant survival status.

The case group drawn from the Collection data is not weighted to the birthweight distribution of the perinatal death registrations because this would lead to overcounting of the perinatal deaths misclassified, at least at very low birthweights, in the control group. In any event, because both the underenumeration of the case group and its contribution to the total population is small, so too is the bias.
associated with estimating the prevalence of exposures of interest in the combined case and weighted control groups.

With the data collected from interviews and patient records, it is not a problem to weight the case group to the birthweight distribution of the deaths identified from the Maternal and Perinatal Collection. No deaths are misclassified as survivors in these sample data and although some deaths are included in the population of surviving infants that forms the denominator used to calculate weights - with the result that deaths enter the weighted control group - these deaths were found through record linkage to be different from 95 per cent (n=58) of the 61 1986 deaths covered in the interviews and patient records. Thus, double counting does not occur here. However, it should be noted that the 1985 sampling distributions are weighted to resemble the 1986 population in the absence of the requisite 1985 population data.

Table 3.9: Weightings of Birthweight Strata in Between-Group Analyses of Exposure by Women Aged 20 to 34 Years, according to Data Source, 1985-86

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Singletons</td>
<td>All plural</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>500-1 499</td>
<td>1.00</td>
<td>1.00</td>
<td>2.20</td>
<td>2.91</td>
<td>7.66</td>
</tr>
<tr>
<td>1 500-2 499</td>
<td>5.18</td>
<td>5.04</td>
<td>2.39</td>
<td>41.49</td>
<td>7.31</td>
</tr>
<tr>
<td>2 500-3 999</td>
<td>65.19</td>
<td>65.32</td>
<td>2.67</td>
<td>869.57</td>
<td>22.37</td>
</tr>
<tr>
<td>4 000+</td>
<td>103.52</td>
<td>103.63</td>
<td>2.14</td>
<td>3846.15</td>
<td>7.50</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
(Source. Hospital Patients' Records, 1985-86; Interviews, 1986; Maternal and Perinatal Collection, 1986)

First, though, qualifications affecting the weighting procedure are noted. The weighting is crude and rather messy, especially as it pertains to the interview and patient record data for which small cell sizes yield very high and potentially unreliable weights at the larger
birthweights. Additional caveats are that none of the weights accommodates missing values in the numerator and, at each birthweight, the weights are assumed to apply to each level of the factor being studied. The weighted estimates, particularly those calculated for the interview and patient record data, must, therefore, be interpreted with considerable caution, which is not to deny their usefulness as broad indicators of prevalence. Still, the weighting is avoided whenever possible, so that, for example, low birthweight babies are treated as a single group when, as with the interview data (see chapter 7), small numbers prevent evaluation of separate birthweight strata below 2 500 grams.

Because of the stratified sampling, combining all low birthweight babies yields a group with lower birthweights than in the populations from which they came. Yet the resultant group is not a highly special one. Low birthweight babies are a well-defined group and aetiological factors should be similar in the joined birthweight strata. The meaning of results, in terms of the identification of aetiological processes associated with low birthweight, should still be useful, and representativeness may be assumed for a population other than that from which the samples were drawn.

3.8.2 ESTIMATES OF RELATIVE RISK

The relative risk of death is the risk of death associated with exposure to a risk factor relative, or compared, to the risk of death associated with non-exposure to the same risk factor. Using the notation in Table 3.10, the relative risk of death is \( \frac{af}{ec} \). In most case-control studies, including this one, the relative risk of an event cannot be exactly determined,\(^{31}\) but for rare occurrences\(^ {32} \) such

\[ \text{In Table 3.10, the marginal totals } e \text{ and } f \text{ are arithmetically derived, and may not reflect the frequency of exposure in the total population; the sampling fractions are unknown.} \]
as perinatal death, estimation of the odds ratio (θ) closely approximates the relative risk. Using Table 3.10, the odds ratio is the odds of death in exposed individuals relative to unexposed individuals, the odds being the ratio p/q where p is the probability of an event occurring, and θ=1−p. Put most simply, the odds ratio is written

\[ \theta = \frac{ad}{bc} \]

where if the odds ratio differs from unity, the exposure being studied is estimated to be associated with the risk of death. If θ > 1, the association is said to be positive; if θ < 1, the association is negative.

This interpretation likewise applies to a summary odds ratio estimated in chapters 5 to 8 from a series of 2 * 2 tables. This Mantel-Haenszel estimate (θ_{mh}), which adjusts for the confounding effects of the stratification variable(s), is calculated as

\[ \theta_{mh} = \frac{\sum_{i=1}^{k} (a_i d_i / i)}{\sum_{i=1}^{k} (b_i c_i / i)}. \]

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Death</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
<th>Odds of death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>Yes</td>
<td>a</td>
<td>b</td>
<td>e</td>
<td>a/b</td>
</tr>
<tr>
<td>No</td>
<td></td>
<td>c</td>
<td>d</td>
<td>f</td>
<td>c/d</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>g</td>
<td>h</td>
<td>i</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(a + c)</td>
<td>(b + d)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Odds of exposure: a/c b/d

Table 3.10: Relationship between Death and Exposure

Table 3.11 shows the method of presenting odds ratios used in this study. The empirical contents of the table are discussed below, but

32 See Chapter 1, footnote 3.
first some of its distinctive methodological features are explored. The table has two main features. To begin with, it assesses the individual and joint effects on perinatal death of two variables: the timing of the first visit to a hospital antenatal clinic in 1986, and the birthweight of the baby. The table secondly, after adjustment for birthweight, expresses the effect on perinatal death of when the first visit occurs. Hence the role of birthweight is assessed in two different ways: as an exposure and as a variable to be controlled for.

To express birthweight as an exposure the odds ratios in this study relate the odds of death for different combinations of cases and controls to a specified subgroup which becomes a standard of reference. Because the odds ratios are not independent, tests for heterogeneity do not apply and comparison is facilitated. The reference group chosen is usually that hypothesized to present the least risk for perinatal death, although this is not so in Table 3.11.

An alternative approach would make the odds ratios sub-group specific. In Table 3.11 the reference group would then change for each birthweight category when comparing the odds of perinatal death between early and late attenders. The main focus of the odds ratios would be on early versus late attenders. This approach is probably easier to understand than the one used but by failing to give equal weight to birthweight it suffers one important constraint: variation by birthweight in the baseline rates makes a direct comparison of the odds ratios not very informative (Schlesselman, 1982).

The second characteristic of, for example, Table 3.11, namely adjustment for the effect of birthweight, is achieved in two ways. First, odds ratios associated respectively with early versus late attenders can be compared within (as well as across) birthweight strata. If the stratum of interest is, say, very low birthweights,
the odds ratios associated with early and late attendance estimate the risk occurring at very low birthweights compared to that occurring at normal birthweights. It is this relation rather than very low birthweights per se that is kept constant.

The second method of adjusting for birthweight uses the Mantel-Haenszel estimate. When the exposure is a factor with more than two levels, a reference group facilitates comparison in the same manner as described above. The Mantel-Haenszel estimate is more clearly a summary of the individual odds ratios when they are not linked to a reference group, as in the alternative approach. But it is helpful nevertheless to show the two perspectives of birthweight in one table.

A further characteristic of Table 3.11 and most subsequent tables is that approximate tests of statistical significance are performed on all odds ratios. For 2*2 tables, the null hypothesis of no association, $H_0: \theta=1$, is tested using the Yates continuity corrected chi-square test. Fisher's exact results are taken when at least one expected cell value is less than 5. For Mantel-Haenszel estimates of the odds ratio, approximate chi-square tests are performed with a 1/2 correction for continuity.

Against this background and looking specifically at Table 3.11, measures of proportions, both unweighted and weighted, and estimates of relative risk can now be discussed. In terms of prevalence, early clinic attendance is related inversely to birthweight: attendance is less common before than after eight weeks in each birthweight stratum, but this is least so for the mothers of very low birthweight babies (20.4 per cent, n=22; that is 22/108 since, as noted above, in this study n refers to the numerator). At birthweights 1 500 to 2 499 grams and 2 500 to 3 999 grams, early attendance characterizes 15.4 per cent (n=19) and 12.9 per cent (n=21) of women respectively.
Weighting is necessary to estimate that 15 per cent (n=1 628) of all women (irrespective of the baby's birthweight) visited a hospital clinic during the first eight weeks of pregnancy.

The Mantel-Haenszel estimate of the odds ratio suggests that visiting a clinic early reduces the risk of perinatal death to 0.47 that of the reference group which received care from the ninth week onwards. Other odds ratios, unadjusted for birthweight, are relative to the reference group of normal birthweight infants of late clinic attenders. Thus, \( \theta = 2.03 \), for example, is the estimated relative odds of death for a very low birthweight baby whose mother attended care early -

\[
\theta = \frac{(6 \times 119)}{(22 \times 16)} = 2.03
\]

all the estimates in fact being higher when care begins after the eighth week, although they decline in both periods with increasing birthweight.

**Table 3.11: Relation of Perinatal Death to Week of First Antenatal Visit by Women Aged 20 to 34 Years Attending a Hospital Clinic, according to Birthweight, 1985-86**

<table>
<thead>
<tr>
<th>First Antenatal Visit (weeks)</th>
<th>Birthweight (grams)</th>
<th>P.D.</th>
<th>Ctl.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-1 499</td>
<td>1 500-2 499</td>
<td>2 500-3 999</td>
</tr>
<tr>
<td></td>
<td>P.D. Ctl. ( \theta )</td>
<td>P.D. Ctl. ( \theta )</td>
<td>P.D. Ctl. ( \theta )</td>
</tr>
<tr>
<td>1-8</td>
<td>6. 16 2.03</td>
<td>1 18 0.30</td>
<td>1 20 0.27</td>
</tr>
<tr>
<td>&gt;8\textsuperscript{a}</td>
<td>27 59 2.47</td>
<td>19 85 1.12</td>
<td>22 119 1.00\textsuperscript{b}</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
\textsuperscript{a} Reference group for Mantel-Haenszel estimated odds ratios
\textsuperscript{b} Reference group for other odds ratios
(Source. Hospital patients' records, 1985-86)

Interaction effects cannot be efficiently identified from simple crosstabulations. Tests for synergy can reveal additive models of mortality risk but in this study the search for interaction is left to
the fitting of linear logistic (logit) models, the final model being reported. It should be noted that only the logistic models effectively compare the severity of risks associated with different factors or covariates. For the contingency tables to do this, the estimated relative risks would need both to control for more variables and be used (with the amount of exposure thereto) to calculate attributable risks. These last measures would give the relative risks a policy focus that is outside the scope of this project, and hence they are not calculated.

The fitted logistic models permit analyses of the individual and joint effects of multiple predictor variables on a dichotomous dependent variable, the probability (p) of perinatal death vis-à-vis perinatal survival. Briefly, because variations in proportions (between 0 and 1) are of interest and must be described by parameter estimates, proportions are transformed on a logistic scale. This is done so that, first, models do not predict impossible values less than 0 or greater than 1, and secondly, large changes near the middle of the range, 0.5, are made no more important than small changes near 0 and 1. The logistic model specifies the effects of variables, \( x_1, x_2 \ldots x_p \), on the log odds (logit) of death in the following way:

\[
\text{logit}(p) = \log_e(p/(1-p)) = (\beta_0 + \beta_1 x_1 + \ldots + \beta_p x_p) = \alpha \quad \text{say}
\]

such that \( p = \frac{e^\alpha}{1 + e^\alpha} \)

Using the GLIM (General Linear Interactive Modelling) system, logistic parameters (\( \beta_i \)) are estimated via the maximum likelihood approach which, given the binomial distribution of the dependent variable, leads to model fitting by iterative weighted least squares. The goodness of fit of each model is shown by the scaled deviance, an appropriate function of the weighted residuals.
Models are fitted in this study according to three criteria. First, the building of models is governed by the conceptual underpinnings of the thesis, so that the entries of successive variables are sensitive to causal paths. Secondly, models are comparatively simple and parsimonious, high-order interaction terms being omitted. Thirdly, the models aspire to fit the data well. This is attempted using a forward stepwise algorithm by which variables are entered in accordance with their comparative abilities to reduce the scaled deviance (equivalent to the log likelihood ratio) with minimal loss of degrees of freedom. The statistical significance of the deviance change is also examined since (asymptotically) this change follows an approximate chi-square sampling distribution (Glim Working Party, 1986).

3.9 CONCLUSION

Progress in the primary prevention of perinatal death depends on researchers having access to environmental and nonenvironmental data. Yet no one data source collects such data and they are not readily available even from multiple sources: data on some variables may not exist, and strict ethical procedures govern perinatal research; approval may be given of the sound protocol, but necessarily after heavy demands have been made on the time and other resources of both researcher and record holders. To help reduce these difficulties, existing data collections by hospitals and the Health Department need to be expanded to include information on maternal, or indeed family, lifestyle practices during pregnancy. This will reduce the need to collect original data, and give researchers a choice of data sources they presently lack. Nevertheless, for this researcher, the time spent producing a primary database was invaluable; the experience, with all its difficulties, was intellectually rewarding and it gave human meaning to a statistical analysis of perinatal death.
4.0 INTRODUCTION

Perinatal death was defined in sections 1.1. and 3.4., but a more comprehensive, descriptive characterization of this phenomenon, as observed in the study area, is reserved for this chapter. The timing of perinatal death is first briefly considered. Using a detailed typology, perinatal death is disaggregated within two overlapping frameworks: a simple stillbirth-neonatal death dichotomy and a transcendent tripartite classification of antepartum, intrapartum and postpartum deaths - within which further, temporal refinements are made. Because of its simplicity, the dichotomous approach is retained when secondly, the discussion moves on to summarize the stated causes of death. They include the main aetiological conditions in the foetus or infant and the mother, as well as the underlying cause of death. Finally, discussion focuses on factors including the type of certification and options available both to the certifier and to the registrar in determining these causes. In contrast to published statistics, all events are reported according to their year of occurrence rather than year of registration. The desired result of the chapter is a consideration of the timing and stated causes of the object of this study: perinatal death in New South Wales during 1985 and 1986.

4.1 TIMING OF PERINATAL DEATH

In New South Wales in 1985 and 1986, there were 1808 perinatal deaths to foetuses or infants of at least 500 grams birthweight, with an
associated perinatal death rate of 10.7 per 1 000 total births. This is seen in Table 4.0 to reflect a predominance of stillbirths. They account for 55.6 per cent of perinatal deaths, the percentage having increased in recent years owing to advances in newborn intensive care producing proportionately fewer neonatal deaths.

However, refinement of the stillbirth-neonatal death dichotomy into antepartum, intrapartum and postnatal deaths reveals postnatal deaths as the most prevalent. Whereas they account for almost all neonatal deaths, one-third of stillbirths result from intrapartum foetal deaths, these latter deaths occurring mainly during labour and before delivery. The risk for postnatal death decreases with infant age, almost 80 per cent (n=577) taking place before the end of the first week. Likewise, only a small proportion of deaths in utero (10.4 per cent, n=105) occurs more than six days before the onset of labour.

4.2 CERTIFICATION OF DEATH

When a perinatal death is thought to have occurred, the medical practitioner called to the body must first confirm that death has taken place. Then, the attending practitioner must, and the non-attending practitioner may, either sign a Medical Certificate of Cause of Perinatal Death or sign a notice of intention to issue this certificate, if he or she is in a position to do so; that is, if the nature of the death does not oblige the medical practitioner to refer the death to a coroner (see section 4.7).

On the medical certificate, the certifier must state the causes(s) of death and the underlying cause of death. From the five sections providing these details, officers of the Australian Bureau of Statistics summarily produce two pieces of information. The first relates to the main disease or condition in the foetus or infant,
Table 4.0: Timing of Stillbirths, Neonatal Deaths and Perinatal Deaths, 1985-86

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>% of Stillbirths or Neonatal Deaths</th>
<th>% of Perinatal Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stillbirths</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Antepartum Foetal Deaths</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No breathing at birth, and heart beat ceased before labour commenced:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Under 1 Hour</td>
<td>7</td>
<td>0.7</td>
<td>0.4</td>
</tr>
<tr>
<td>1-23 Hours</td>
<td>130</td>
<td>12.9</td>
<td>7.2</td>
</tr>
<tr>
<td>1-6 Days</td>
<td>366</td>
<td>36.4</td>
<td>20.2</td>
</tr>
<tr>
<td>7-27 Days</td>
<td>105</td>
<td>10.4</td>
<td>5.8</td>
</tr>
<tr>
<td>More than 27 days</td>
<td>10</td>
<td>1.0</td>
<td>0.6</td>
</tr>
<tr>
<td>Missing</td>
<td>54</td>
<td>5.4</td>
<td>3.0</td>
</tr>
<tr>
<td>Total antepartum deaths</td>
<td>672</td>
<td>66.9</td>
<td>37.2</td>
</tr>
<tr>
<td><strong>Intrapartum Foetal Deaths</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No breathing at birth, and heart beat ceased during labour but before delivery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not known whether before or during labour</td>
<td>266</td>
<td>26.5</td>
<td>14.7</td>
</tr>
<tr>
<td>Not known whether before or during delivery</td>
<td>45</td>
<td>4.5</td>
<td>2.5</td>
</tr>
<tr>
<td>Missing</td>
<td>20</td>
<td>2.1</td>
<td>1.1</td>
</tr>
<tr>
<td>Total intrapartum foetal deaths</td>
<td>332</td>
<td>33.0</td>
<td>18.4</td>
</tr>
<tr>
<td>Total stillbirths</td>
<td>1005</td>
<td>100.0</td>
<td>55.6</td>
</tr>
<tr>
<td><strong>Neonatal Deaths</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Intrapartum Infant Deaths</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No breathing at birth, but heart beat ceased after delivery:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Under 1 hour</td>
<td>62</td>
<td>7.7</td>
<td>3.4</td>
</tr>
<tr>
<td>1-23 hours</td>
<td>12</td>
<td>1.5</td>
<td>0.7</td>
</tr>
<tr>
<td>Missing</td>
<td>1</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Total intrapartum infant deaths</td>
<td>75</td>
<td>9.3</td>
<td>4.1</td>
</tr>
<tr>
<td>Total intrapartum deaths</td>
<td>407</td>
<td>-</td>
<td>22.5</td>
</tr>
<tr>
<td><strong>Postnatal Deaths</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breathing at birth, and heart beat ceased after delivery:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Under 1 hour</td>
<td>85</td>
<td>10.6</td>
<td>4.7</td>
</tr>
<tr>
<td>1-23 hours</td>
<td>266</td>
<td>33.1</td>
<td>14.7</td>
</tr>
<tr>
<td>1-6 days</td>
<td>226</td>
<td>28.1</td>
<td>12.5</td>
</tr>
<tr>
<td>7-27 days</td>
<td>150</td>
<td>18.7</td>
<td>8.3</td>
</tr>
<tr>
<td>Missing</td>
<td>1</td>
<td>0.1</td>
<td>0.1</td>
</tr>
<tr>
<td>Total postnatal deaths</td>
<td>728</td>
<td>90.7</td>
<td>40.3</td>
</tr>
<tr>
<td>Total neonatal deaths</td>
<td>803</td>
<td>100.0</td>
<td>44.4</td>
</tr>
<tr>
<td>Total perinatal deaths</td>
<td>1808</td>
<td>-</td>
<td>100.0</td>
</tr>
</tbody>
</table>

(Source: Perinatal death registrations, 1985-87)
which is the pathological condition making in the certifier’s opinion the greatest contribution to the foetal or infant death. The second piece of information is the main maternal disease or condition affecting the foetus or infant, which is the disease or condition in the mother that in the certifier’s opinion most adversely affected the foetus or infant. A further section provides for entry of the underlying cause of death, which is the single disease or condition that the certifier believes initiated the train of events leading to death. Since 1979, the Australian Bureau of Statistics has classified these three causes of death in accordance with the Ninth Revision (1975) of the *International Statistical Classification of Diseases, Injuries and Causes of Death*. Succeeding sections describe the prevalence of these stated causes of perinatal death, before detailing the manner in which these causes were arrived at, and hence factors qualifying their interpretation.

### 4.3 MAIN DISEASE OR CONDITION IN THE FOETUS OR INFANT

Table 4.1 classifies perinatal deaths by the main disease or condition in the foetus or infant. In this context the most frequently stated cause of perinatal death is *hypoxia, birth asphyxia and other respiratory conditions*, which accounts for 39.0 per cent (n=705) of perinatal deaths, and occurs especially among stillbirths, 43.3 per cent (n=437) being affected. Also conspicuous is *foetal death from hypoxia before labour or at an unspecified time*; this characterizes one-third of stillbirths or 76.2 per cent (n=333) of stillbirths attributed to hypoxia, and 18.4 per cent (n=333) of perinatal deaths. Almost all other stillbirths are classified as *foetal deaths from hypoxia during labour*. 
Table 4.1: Certified Cause of Stillbirths, Neonatal Deaths and Perinatal Deaths. Main Disease or Condition in the Foetus or Infant in New South Wales, 1985-86

<table>
<thead>
<tr>
<th>Main Disease or Condition in the Foetus or Infant</th>
<th>Stillbirths</th>
<th>Neonatal Deaths</th>
<th>Perinatal Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slow Foetal Growth, Foetal Malnutrition and Immaturity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slow foetal growth and foetal malnutrition</td>
<td>29 2.9</td>
<td>4 0.5</td>
<td>33 1.8</td>
</tr>
<tr>
<td>Extreme immaturity</td>
<td>58 5.8</td>
<td>70 8.7</td>
<td>128 7.1</td>
</tr>
<tr>
<td>Other preterm infants</td>
<td>6 0.6</td>
<td>0 0.0</td>
<td>6 0.3</td>
</tr>
<tr>
<td>Birth Trauma</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birth injury to brain, spine or spinal cord</td>
<td>2 0.2</td>
<td>16 2.0</td>
<td>18 0.9</td>
</tr>
<tr>
<td>Other birth injury</td>
<td>1 0.1</td>
<td>16 2.0</td>
<td>17 0.9</td>
</tr>
<tr>
<td>Hypoxia, Birth Asphyxia and Other Respiratory Conditions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foetal death from asphyxia or hypoxia before onset of labour or at unspecified time</td>
<td>333 33.1</td>
<td>0 0.0</td>
<td>333 18.4</td>
</tr>
<tr>
<td>Foetal death from asphyxia or hypoxia during labour</td>
<td>91 9.0</td>
<td>0 0.0</td>
<td>91 5.0</td>
</tr>
<tr>
<td>Foetal distress or birth asphyxia in liveborn infant.</td>
<td>0 0.0</td>
<td>41 5.1</td>
<td>41 2.3</td>
</tr>
<tr>
<td>Respiratory distress syndrome</td>
<td>0 0.0</td>
<td>76 9.5</td>
<td>76 4.2</td>
</tr>
<tr>
<td>Congenital pneumonia</td>
<td>6 0.6</td>
<td>14 1.7</td>
<td>20 1.1</td>
</tr>
<tr>
<td>Massive aspiration syndrome</td>
<td>5 0.5</td>
<td>29 3.6</td>
<td>34 1.9</td>
</tr>
<tr>
<td>Interstitial emphysema and related conditions</td>
<td>0 0.0</td>
<td>12 1.5</td>
<td>12 0.7</td>
</tr>
<tr>
<td>Pulmonary haemorrhage</td>
<td>2 0.2</td>
<td>6 0.7</td>
<td>8 0.4</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>0 0.0</td>
<td>68 8.5</td>
<td>68 3.8</td>
</tr>
<tr>
<td>Other respiratory conditions of foetus and newborn</td>
<td>0 0.0</td>
<td>22 2.7</td>
<td>22 1.2</td>
</tr>
<tr>
<td>Foetal and Neonatal Haemorrhage</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foetal blood loss</td>
<td>4 0.4</td>
<td>1 0.1</td>
<td>5 0.3</td>
</tr>
<tr>
<td>Intraventricular haemorrhage</td>
<td>2 0.2</td>
<td>65 8.1</td>
<td>67 3.7</td>
</tr>
<tr>
<td>Subarachnoid haemorrhage</td>
<td>2 0.2</td>
<td>1 0.1</td>
<td>3 0.2</td>
</tr>
<tr>
<td>Haemolytic Disease of Foetus or Newborn</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Haemolytic disease due to Rh isoimmunization</td>
<td>14 1.4</td>
<td>5 0.6</td>
<td>19 1.1</td>
</tr>
<tr>
<td>Haemolytic disease due to other and unspecified isoimmunization</td>
<td>6 0.6</td>
<td>0 0.0</td>
<td>6 0.3</td>
</tr>
<tr>
<td>Haemolytic disease due to other and unspecified isoimmunization</td>
<td>8 0.8</td>
<td>5 0.6</td>
<td>13 0.7</td>
</tr>
</tbody>
</table>

Continued overleaf
Table 4.1. Continued:

<table>
<thead>
<tr>
<th>Main Disease or Condition in the Foetus or Infant</th>
<th>Still-births</th>
<th>Neonatal Deaths</th>
<th>Perinatal Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Other Conditions Originating in the Perinatal Period</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infections specific to the perinatal period</td>
<td>306</td>
<td>30.4</td>
<td>50</td>
</tr>
<tr>
<td>Endocrine and metabolic disturbances specific to the foetus and newborn</td>
<td>17</td>
<td>1.7</td>
<td>19</td>
</tr>
<tr>
<td>Haematological disorders of the foetus and newborn</td>
<td>6</td>
<td>0.6</td>
<td>1</td>
</tr>
<tr>
<td>Perinatal disorders of the digestive tract</td>
<td>1</td>
<td>0.1</td>
<td>5</td>
</tr>
<tr>
<td>Hydrops fetalis not due to isoimmunization</td>
<td>0</td>
<td>0.0</td>
<td>15</td>
</tr>
<tr>
<td>Other conditions involving the integument and temperature regulation of the foetus and newborn</td>
<td>0</td>
<td>0.0</td>
<td>8</td>
</tr>
<tr>
<td>Other and ill-defined conditions originating in the perinatal period</td>
<td>269</td>
<td>26.6</td>
<td>2</td>
</tr>
<tr>
<td>Congenital Anomalies</td>
<td>137</td>
<td>13.6</td>
<td>280</td>
</tr>
<tr>
<td>Anencephalus and similar anomalies</td>
<td>41</td>
<td>4.1</td>
<td>23</td>
</tr>
<tr>
<td>Spina bifida</td>
<td>6</td>
<td>0.6</td>
<td>18</td>
</tr>
<tr>
<td>Congenital hydrocephalus</td>
<td>23</td>
<td>2.3</td>
<td>12</td>
</tr>
<tr>
<td>Other deformities of central nervous system</td>
<td>8</td>
<td>0.8</td>
<td>6</td>
</tr>
<tr>
<td>Congenital anomalies of heart and circulatory system</td>
<td>9</td>
<td>0.9</td>
<td>69</td>
</tr>
<tr>
<td>Congenital anomalies of respiratory system</td>
<td>0</td>
<td>0.0</td>
<td>38</td>
</tr>
<tr>
<td>Congenital anomalies of digestive system</td>
<td>2</td>
<td>0.2</td>
<td>7</td>
</tr>
<tr>
<td>Congenital anomalies of genital organs and urinary system</td>
<td>8</td>
<td>0.8</td>
<td>12</td>
</tr>
<tr>
<td>Congenital anomalies of musculoskeletal system and limbs</td>
<td>4</td>
<td>0.4</td>
<td>39</td>
</tr>
<tr>
<td>Chromosomal anomalies</td>
<td>15</td>
<td>1.5</td>
<td>34</td>
</tr>
<tr>
<td>Other and unspecified congenital anomalies</td>
<td>21</td>
<td>2.1</td>
<td>22</td>
</tr>
<tr>
<td>Infectious and Parasitic Diseases</td>
<td>3</td>
<td>0.3</td>
<td>1</td>
</tr>
<tr>
<td>All Other Causes</td>
<td>5</td>
<td>0.5</td>
<td>42</td>
</tr>
<tr>
<td>Other specified conditions</td>
<td>5</td>
<td>0.5</td>
<td>24</td>
</tr>
<tr>
<td>Symptoms, signs and ill-defined conditions</td>
<td>0</td>
<td>0.0</td>
<td>18</td>
</tr>
<tr>
<td>Total</td>
<td>1005</td>
<td>100.0</td>
<td>803</td>
</tr>
</tbody>
</table>

(Source. Perinatal death registrations, 1985-87)
Among liveborn infants, birth asphyxia accounts for fewer deaths (33.4 per cent, n=268), but its incidence remains high, particularly for two conditions. The first is the respiratory distress syndrome. This accounts for 4.2 per cent (n=76) of perinatal deaths, but 9.5 per cent (n=76) of neonatal deaths and 28.4 per cent of the neonatal deaths attributed to asphyxia. Almost as important as a cause of neonatal asphyxia is atelectasis: 3.8 per cent of perinatal deaths and 8.5 per cent (n=68) of neonatal deaths are attributed to this condition; as many as 83.8 per cent of the neonatal deaths resulting from atelectasis occur before the first completed day.

The second most frequently stated main cause of death in the foetus or infant is congenital anomalies: 23.1 per cent of perinatal deaths result from this cause. However, slightly over two-thirds (n=280) of these deaths are found among liveborn infants, congenital anomalies accounting for only 13.6 per cent (n=137) of stillbirths. Congenital anomalies of the heart and circulatory system stand out as causing 8.6 per cent (n=69) of neonatal deaths, and one-quarter of the neonatal deaths attributable to congenital anomalies; the same malformations explain 4.3 per cent (n=78) of perinatal deaths. Other salient anomalies are those of the musculoskeletal system and limbs, which account for 4.9 per cent (n=39) of neonatal deaths, and congenital anomalies of the respiratory system; in the certifier’s opinion, this was the main cause of 4.7 per cent (n=38) of neonatal deaths.

Chromosomal anomalies, especially Patau’s syndrome and Edward’s

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1 The respiratory distress syndrome and atelectasis are defined in chapter 2, footnote 43.
2 Clinical characteristics of Patau’s syndrome, or trisomy 13-15, include cerebral and cardiac defects, ocular anomalies, malformed ears and cleft lip and palate.
syndrome,\(^3\) is the main condition in the foetus or infant causing 4.2 per cent (n=34) of neonatal deaths.

The most important congenital anomalies causing stillbirths are the neural tube defects, *anencephalus and similar anomalies*, which are found in 4.1 per cent (n=41) of deaths, and less frequently, *congenital hydrocephalus*. Yet having twice the frequency of congenital anomalies as causes of stillbirth, are other and ill-defined conditions originating in the perinatal period. The latter conditions account for 26.6 per cent (n=269) of stillbirths and 15.0 per cent (n=271) of perinatal deaths.

Of continued importance as a main cause of 8.1 per cent (n=65) of neonatal deaths is *intraventricular haemorrhage*;\(^4\) and more important than this as causes of all perinatal deaths is *slow foetal growth*, *foetal malnutrition and immaturity*. These conditions are certified as the main aetiological condition in the foetus or infant in 9.2 per cent (n=167) of perinatal deaths, almost identical proportions occurring among stillbirths and neonatal deaths. Few perinatal deaths are attributed to *birth trauma, infectious and parasitic diseases or all other causes*.

4.4 **MAIN DISEASE OR CONDITION IN THE MOTHER AFFECTING THE FOETUS OR INFANT**

Three categories are conspicuous by the frequency with which they are reported as the main condition in the mother contributing to perinatal death. As Table 4.2 shows, the two most important of these categories in fact predominate among stillbirths or neonatal deaths, but not

---

\(^3\) Edward's syndrome, or trisomy 18, is marked by more delicate facial features including low set, malformed ears and smallness of the jaw and mouth, as well as overlapping fingers, cardiac and central nervous defects and low birthweight.

\(^4\) Intraventricular haemorrhage is a cerebral haemorrhage, which begins on the lateral ventricle of the brain, in infants below 34 weeks' gestation.
Table 4.2: Stated Cause of Stillbirths, Neonatal Deaths and Perinatal Deaths. Main Maternal Disease or Condition Affecting the Foetus or Infant in New South Wales, 1985-86

<table>
<thead>
<tr>
<th>Main Maternal Disease or Condition Affecting the Foetus or Infant</th>
<th>Stillbirths</th>
<th>Neonatal Deaths</th>
<th>Perinatal Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal Conditions that may be Unrelated to the Present Pregnancy</td>
<td>110</td>
<td>10.9</td>
<td>54</td>
</tr>
<tr>
<td>Maternal hypertensive disorders</td>
<td>65</td>
<td>6.5</td>
<td>31</td>
</tr>
<tr>
<td>Maternal renal and urinary tract diseases</td>
<td>2</td>
<td>0.2</td>
<td>1</td>
</tr>
<tr>
<td>Maternal infections</td>
<td>5</td>
<td>0.5</td>
<td>3</td>
</tr>
<tr>
<td>Maternal injury</td>
<td>5</td>
<td>0.5</td>
<td>0</td>
</tr>
<tr>
<td>Other maternal conditions</td>
<td>33</td>
<td>3.3</td>
<td>19</td>
</tr>
<tr>
<td>Maternal Complications of Pregnancy</td>
<td>147</td>
<td>14.6</td>
<td>350</td>
</tr>
<tr>
<td>Incompetent cervix</td>
<td>16</td>
<td>1.6</td>
<td>19</td>
</tr>
<tr>
<td>Premature rupture of membranes</td>
<td>36</td>
<td>3.6</td>
<td>54</td>
</tr>
<tr>
<td>Oligohydramnios</td>
<td>4</td>
<td>0.4</td>
<td>8</td>
</tr>
<tr>
<td>Polyhydramnios</td>
<td>26</td>
<td>2.5</td>
<td>18</td>
</tr>
<tr>
<td>Multiple pregnancy</td>
<td>15</td>
<td>1.5</td>
<td>15</td>
</tr>
<tr>
<td>Other maternal complications of pregnancy</td>
<td>50</td>
<td>5.0</td>
<td>236</td>
</tr>
<tr>
<td>Complications of Placenta, Cord and Membranes</td>
<td>450</td>
<td>44.8</td>
<td>81</td>
</tr>
<tr>
<td>Placenta praevia</td>
<td>15</td>
<td>1.5</td>
<td>12</td>
</tr>
<tr>
<td>Other placental separation and haemorrhage</td>
<td>166</td>
<td>16.5</td>
<td>35</td>
</tr>
<tr>
<td>Other and unspecified morphological and functional abnormalities of placenta</td>
<td>108</td>
<td>10.7</td>
<td>7</td>
</tr>
<tr>
<td>Placental transfusion syndromes</td>
<td>6</td>
<td>0.6</td>
<td>0</td>
</tr>
<tr>
<td>Prolapsed cord</td>
<td>13</td>
<td>1.3</td>
<td>7</td>
</tr>
<tr>
<td>Other compression of umbilical cord</td>
<td>96</td>
<td>9.6</td>
<td>4</td>
</tr>
<tr>
<td>Chorioamnionitis</td>
<td>26</td>
<td>2.6</td>
<td>0</td>
</tr>
<tr>
<td>Other complications of placenta, cord and membranes</td>
<td>20</td>
<td>2.0</td>
<td>16</td>
</tr>
<tr>
<td>Other Complications of Labour and Delivery</td>
<td>29</td>
<td>2.9</td>
<td>138</td>
</tr>
<tr>
<td>Breech delivery and extraction</td>
<td>13</td>
<td>1.3</td>
<td>19</td>
</tr>
<tr>
<td>Other malpresentation, malposition and disproportion of labour and delivery</td>
<td>3</td>
<td>0.3</td>
<td>7</td>
</tr>
<tr>
<td>Delivery by caesarean</td>
<td>2</td>
<td>0.2</td>
<td>80</td>
</tr>
<tr>
<td>Other complications of labour and delivery</td>
<td>11</td>
<td>1.1</td>
<td>32</td>
</tr>
<tr>
<td>No maternal condition reported</td>
<td>269</td>
<td>26.8</td>
<td>180</td>
</tr>
<tr>
<td>Total</td>
<td>1005</td>
<td>100.0</td>
<td>803</td>
</tr>
</tbody>
</table>

(Source. Perinatal death registrations, 1985-87)
both. Most widespread among stillbirths is complications of the placenta, cord and membranes. This accounts for 44.8 per cent (n=450) of stillbirths or 29.4 per cent of perinatal deaths attributed to this cause. Of these complications, other placental separation and haemorrhage, including especially abruptio placentae, is most salient.

Neonatal deaths are associated mainly with maternal complications of pregnancy, this being the main maternal condition in 43.6 per cent (n=350) of neonatal deaths, and 27.5 per cent (n=497) of perinatal deaths. No single pregnancy complication, possibly excepting premature rupture of the membranes in 6.7 per cent (n=54) of neonatal deaths, is conspicuous by its frequency. The third, prominent category is no maternal condition reported, which characterizes almost one-quarter (n=449) of perinatal deaths, and 26.8 per cent of stillbirths (n=269).

Having secondary aetiological importance for perinatal death are two residual groups of causes, the first of which is other complications of labour and delivery. While this also provides negligible explanatory power for stillbirths, the cause is important in 17.2 per cent (n=138) of neonatal deaths, among which caesarean delivery is stated as the main maternal cause in 10 per cent (n=80) of cases.

Conversely, maternal conditions that may be unrelated to the present pregnancy is most frequently reported among stillbirths. It is the main maternal condition affecting the foetus or infant in 9.1 per cent (n=164) of perinatal deaths, although 62.3 per cent (n=93) of deaths attributed to this cause resulted more specifically from the maternal hypertensive disorders.
4.5 CONCURRENCE OF MATERNAL WITH FOETAL AND INFANT CAUSES OF PERINATAL DEATH

Table 4.3 crosstabulates the main maternal condition affecting the foetus or infant with selected main causes of death in the foetus or infant. *Complications of placenta, cord and membranes* is the most important maternal condition producing physiological immaturity and asphyxia or anoxia; the latter respiratory complications occurred especially before or during labour, and hence mainly among stillborn infants. This set of maternal conditions is also frequently reported when the main cause of death in the foetus or infant is other and ill-defined conditions originating in the perinatal period.

The main maternal complications during pregnancy are associated with at least three-quarters of two aetiological conditions found mainly in the neonate: the *respiratory distress syndrome* and *atelectasis*. In addition *extreme immaturity*, *infections specific to the perinatal period*, and certain malformations such as *anencephalus and similar anomalies* arise when complications of pregnancy are the leading maternal conditions affecting the foetus or infant.

The locus of no maternal condition reported is conspicuously the congenital anomalies, especially of the musculoskeletal system and limbs, and of the heart and circulatory system. Simple arithmetic shows that the reporting of no maternal condition is more prevalent when congenital anomalies resulted in stillbirth (53.4 per cent, n=62) than in neonatal death (39.3 per cent, n=110). No maternal condition is also strongly associated with the *massive aspiration syndrome*, the coincidence of these factors being 44.1 per cent (n=15) of perinatal deaths, although the aetiology mostly affects neonates (see Table 4.1). Less salient but nonetheless significant is the absence of a reportable maternal condition in approximately one-fifth of the
Table 4.3 continued:
Main Disease or Condition in the Foetus or Infant

<table>
<thead>
<tr>
<th>No maternal Condition Reported</th>
<th>Maternal complications unrelated to present pregnancy</th>
<th>Maternal complications of pregnancy</th>
<th>Complications of placenta, cord, and membranes</th>
<th>Other complications of labour and delivery</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Infections specific to the perinatal period</td>
<td>8</td>
<td>22.2</td>
<td>3</td>
<td>8.3</td>
<td>16</td>
</tr>
<tr>
<td>Other and ill-defined conditions originating in the perinatal period</td>
<td>78</td>
<td>28.8</td>
<td>31</td>
<td>11.4</td>
<td>26</td>
</tr>
<tr>
<td>Anencephalus and similar anomalies</td>
<td>26</td>
<td>40.6</td>
<td>2</td>
<td>3.1</td>
<td>24</td>
</tr>
<tr>
<td>Congenital hydrocephalus</td>
<td>13</td>
<td>37.1</td>
<td>2</td>
<td>5.7</td>
<td>8</td>
</tr>
<tr>
<td>Congenital anomalies of heart and circulatory system</td>
<td>41</td>
<td>52.6</td>
<td>6</td>
<td>7.7</td>
<td>11</td>
</tr>
<tr>
<td>Congenital anomalies of respiratory system</td>
<td>12</td>
<td>31.6</td>
<td>1</td>
<td>2.6</td>
<td>15</td>
</tr>
<tr>
<td>Congenital anomalies of musculoskeletal system and limbs</td>
<td>23</td>
<td>53.5</td>
<td>2</td>
<td>4.7</td>
<td>10</td>
</tr>
<tr>
<td>Chromosomal anomalies</td>
<td>16</td>
<td>32.7</td>
<td>3</td>
<td>6.1</td>
<td>14</td>
</tr>
<tr>
<td>Other and unspecified congenital anomalies</td>
<td>13</td>
<td>30.2</td>
<td>2</td>
<td>4.7</td>
<td>16</td>
</tr>
</tbody>
</table>

(Source: Perinatal Death Registrations, 1985-87)
Table 4.3: Crosstabulation of Selected Maternal, and Foetal and Infant Causes of Perinatal Death, in New South Wales, 1985-86

<table>
<thead>
<tr>
<th>Main Disease or Condition in the Foetus or Infant</th>
<th>No maternal condition reported</th>
<th>Maternal complications unrelated to present pregnancy</th>
<th>Maternal complications of pregnancy</th>
<th>Complications of placenta, cord, and membranes</th>
<th>Other complications of labour and delivery</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foetal growth retardation</td>
<td>6 20.7</td>
<td>5 17.2</td>
<td>2 6.9</td>
<td>16 55.2</td>
<td>0 0.0</td>
<td>29 100.0</td>
</tr>
<tr>
<td>Extreme immaturity</td>
<td>0 0.0</td>
<td>4 6.9</td>
<td>28 48.3</td>
<td>25 43.1</td>
<td>1 1.7</td>
<td>58 100.0</td>
</tr>
<tr>
<td>Foetal death from asphyxia or anoxia before the onset of labour or at an unspecified time</td>
<td>76 22.8</td>
<td>41 12.3</td>
<td>15 4.5</td>
<td>201 60.4</td>
<td>0 0.0</td>
<td>333 100.0</td>
</tr>
<tr>
<td>Foetal death from asphyxia or anoxia during labour</td>
<td>9 9.9</td>
<td>7 7.7</td>
<td>23 25.3</td>
<td>40 44.0</td>
<td>12 13.2</td>
<td>91 101.1</td>
</tr>
<tr>
<td>Foetal distress or birth asphyxia in liveborn infants</td>
<td>3 7.3</td>
<td>6 14.6</td>
<td>11 26.8</td>
<td>15 36.6</td>
<td>6 14.6</td>
<td>41 99.9</td>
</tr>
<tr>
<td>Respiratory distress syndrome</td>
<td>2 2.6</td>
<td>6 7.9</td>
<td>57 75.0</td>
<td>8 10.5</td>
<td>3 3.9</td>
<td>76 99.9</td>
</tr>
<tr>
<td>Massive aspiration syndrome</td>
<td>15 44.1</td>
<td>4 11.8</td>
<td>0 0.0</td>
<td>3 8.8</td>
<td>12 35.3</td>
<td>34 100.0</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>0 0.0</td>
<td>5 7.4</td>
<td>54 79.4</td>
<td>8 11.8</td>
<td>1 1.5</td>
<td>68 101.1</td>
</tr>
<tr>
<td>Intraventricular haemorrhage</td>
<td>2 3.0</td>
<td>3 4.5</td>
<td>42 62.7</td>
<td>9 13.4</td>
<td>11 16.4</td>
<td>67 100.0</td>
</tr>
</tbody>
</table>

Continued overleaf
perinatal deaths attributed mainly to two causes: foetal growth retardation and foetal death from asphyxia or anoxia before the onset of labour or at an unspecified time.

These sets of maternal causes of perinatal death have been shown to cluster among specific aetiologies in the foetus or infant. However, maternal complications unrelated to the present pregnancy and other complications of labour and delivery do not share this characteristic, even though the latter set of complications is reported in 35.3 per cent (n=12) of perinatal deaths attributed mainly to the massive aspiration syndrome.

4.6 UNDERLYING CAUSE OF DEATH

Another way of bringing together the main aetiological conditions in the mother and her foetus or infant is to examine the frequency of the underlying causes of death. This distribution is shown in Table 4.4 in which two groups of causes most often initiate the train of events leading, in the certifier's opinion, to death. The first group, complications of placenta, cord and membranes, is noted in 29.9 per cent (n=541) of perinatal deaths, although this reflects the prevalence of these complications (83.9 per cent, n=454) among stillbirths vis-à-vis neonatal deaths; indeed these complications characterize 45.0 per cent of all stillbirths.

Congenital anomalies is the second group. This is the underlying cause of 23.5 per cent (n=425) of perinatal deaths, owing to the dominance of such malformations among neonatal deaths: 67.8 per cent (n=288) of congenital anomalies occur among neonatal deaths, and these anomalies account for over one-third of all deaths to neonates. Thus what emerges from examining the main and underlying causes of death is
<table>
<thead>
<tr>
<th>Underlying Cause in the Foetus or Infant</th>
<th>Stillbirths</th>
<th>Neonatal Deaths</th>
<th>Perinatal Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Maternal Conditions that may be Unrelated to the Present Pregnancy</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal hypertensive disorders</td>
<td>62</td>
<td>22</td>
<td>84</td>
</tr>
<tr>
<td>Other maternal conditions</td>
<td>35</td>
<td>9</td>
<td>44</td>
</tr>
<tr>
<td><strong>Maternal Complications of Pregnancy</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Incompetent cervix</td>
<td>11</td>
<td>31</td>
<td>42</td>
</tr>
<tr>
<td>Premature rupture of membranes</td>
<td>26</td>
<td>42</td>
<td>68</td>
</tr>
<tr>
<td>Multiple pregnancy</td>
<td>21</td>
<td>21</td>
<td>42</td>
</tr>
<tr>
<td>Other maternal complications of pregnancy</td>
<td>20</td>
<td>25</td>
<td>45</td>
</tr>
<tr>
<td><strong>Complications of Placenta, Cord and Membranes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placenta praevia</td>
<td>14</td>
<td>13</td>
<td>27</td>
</tr>
<tr>
<td>Other placental separation and haemorrhage</td>
<td>160</td>
<td>39</td>
<td>199</td>
</tr>
<tr>
<td>Other and unspecified morphological and functional abnormalities of placenta</td>
<td>109</td>
<td>8</td>
<td>117</td>
</tr>
<tr>
<td>Prolapsed cord</td>
<td>13</td>
<td>9</td>
<td>22</td>
</tr>
<tr>
<td>Other compression of umbilical cord</td>
<td>99</td>
<td>4</td>
<td>103</td>
</tr>
<tr>
<td>Other complications of placenta, cord and membranes</td>
<td>59</td>
<td>14</td>
<td>73</td>
</tr>
<tr>
<td><strong>Other Complications of Labour and Delivery</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slow Foetal Growth, Foetal Malnutrition and Immaturity</td>
<td>6</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Birth Trauma</td>
<td>1</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>Hypoxia, Birth Asphyxia and Other Respiratory Conditions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Foetal death from asphyxia or hypoxia before onset of labour or at unspecified time</td>
<td>78</td>
<td>0</td>
<td>78</td>
</tr>
<tr>
<td>Foetal death from asphyxia or hypoxia during labour</td>
<td>9</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Respiratory distress syndrome</td>
<td>0</td>
<td>26</td>
<td>26</td>
</tr>
<tr>
<td>Congenital pneumonia</td>
<td>0</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Massive aspiration syndrome</td>
<td>0</td>
<td>26</td>
<td>26</td>
</tr>
<tr>
<td>Atelectasis</td>
<td>0</td>
<td>18</td>
<td>18</td>
</tr>
<tr>
<td>Other respiratory conditions of foetus and newborn</td>
<td>6</td>
<td>18</td>
<td>24</td>
</tr>
</tbody>
</table>

Continued overleaf
<table>
<thead>
<tr>
<th>Underlying Cause in the Foetus or Infant</th>
<th>Still-births</th>
<th>Neonatal Deaths</th>
<th>Perinatal Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Foetal and Neonatal Haemorrhage</td>
<td>6</td>
<td>0.6</td>
<td>66</td>
</tr>
<tr>
<td>Intraventricular haemorrhage</td>
<td>2</td>
<td>0.2</td>
<td>65</td>
</tr>
<tr>
<td>Other foetal and neonatal haemorrhage</td>
<td>4</td>
<td>0.4</td>
<td>1</td>
</tr>
<tr>
<td>Haemolytic Disease of Foetus or Newborn</td>
<td>12</td>
<td>1.2</td>
<td>8</td>
</tr>
<tr>
<td>Haemolytic disease due to Rh isoimmunization</td>
<td>6</td>
<td>0.6</td>
<td>2</td>
</tr>
<tr>
<td>Haemolytic disease due to other and unspecified isoimmunization</td>
<td>6</td>
<td>0.6</td>
<td>6</td>
</tr>
<tr>
<td>Other Conditions Originating in the Perinatal Period</td>
<td>101</td>
<td>10.1</td>
<td>47</td>
</tr>
<tr>
<td>Infections specific to the perinatal period</td>
<td>7</td>
<td>0.7</td>
<td>18</td>
</tr>
<tr>
<td>Perinatal disorders of the digestive tract</td>
<td>0</td>
<td>0.0</td>
<td>14</td>
</tr>
<tr>
<td>Other and ill-defined conditions</td>
<td>94</td>
<td>9.4</td>
<td>15</td>
</tr>
<tr>
<td>Congenital Anomalies</td>
<td>137</td>
<td>13.6</td>
<td>288</td>
</tr>
<tr>
<td>Anencephalus and similar anomalies</td>
<td>40</td>
<td>4.0</td>
<td>23</td>
</tr>
<tr>
<td>Spina bifida</td>
<td>6</td>
<td>0.6</td>
<td>19</td>
</tr>
<tr>
<td>Congenital hydrocephalus</td>
<td>23</td>
<td>2.3</td>
<td>11</td>
</tr>
<tr>
<td>Other deformities of central nervous system</td>
<td>8</td>
<td>0.8</td>
<td>6</td>
</tr>
<tr>
<td>Congenital anomalies of heart and circulatory system</td>
<td>9</td>
<td>0.9</td>
<td>71</td>
</tr>
<tr>
<td>Congenital anomalies of respiratory system</td>
<td>0</td>
<td>0.0</td>
<td>32</td>
</tr>
<tr>
<td>Congenital anomalies of genital and urinary system</td>
<td>8</td>
<td>0.8</td>
<td>10</td>
</tr>
<tr>
<td>Congenital anomalies of musculoskeletal system and limbs</td>
<td>4</td>
<td>0.4</td>
<td>48</td>
</tr>
<tr>
<td>Chromosomal anomalies</td>
<td>16</td>
<td>1.6</td>
<td>35</td>
</tr>
<tr>
<td>Other and unspecified congenital anomalies</td>
<td>23</td>
<td>2.3</td>
<td>33</td>
</tr>
<tr>
<td>All other Causes</td>
<td>9</td>
<td>0.9</td>
<td>41</td>
</tr>
<tr>
<td>Total</td>
<td>1005</td>
<td>100.0</td>
<td>803</td>
</tr>
</tbody>
</table>

(Source. Perinatal Death Registrations, 1985-87)
their temporal specificity: the importance of particular causes of death depends on when death occurs.

4.7 TYPE OF CERTIFICATION

Medical practitioners were usually able to eliminate on medical evidence, doubts or suspicions surrounding the cause(s) of death. Consequently, 97.3 per cent (n=1760) of perinatal deaths were certified by a medical practitioner rather than by a coroner, this proportion being higher for stillbirths and lower for neonatal deaths (see Table 4.5).

It is not surprising that coroners rarely became responsible for certification. Only, for example, when the death occurred in unusual or suspicious circumstances, or when the death was sudden⁵ and of unknown cause(s), was the medical practitioner required to report the death to the coroner.⁶

Table 4.5: Coronial and Non-Coronial Certifications, 1985-86

<table>
<thead>
<tr>
<th>Certification</th>
<th>Still Births</th>
<th>Neonatal Deaths</th>
<th>Perinatal Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
<td>No.</td>
</tr>
<tr>
<td>Non coronial</td>
<td>1001</td>
<td>99.6</td>
<td>759</td>
</tr>
<tr>
<td>Coronial</td>
<td>4</td>
<td>0.4</td>
<td>44</td>
</tr>
<tr>
<td>Total</td>
<td>1005</td>
<td>100.0</td>
<td>803</td>
</tr>
</tbody>
</table>

(Source. Perinatal death registrations, 1985-87)

⁵ For example, a postmortem examination is mandated by law if a baby dies within 24 hours of a surgical operation.

⁶ The conditions under which death is reportable to a coroner are described under section 13(3) of the New South Wales Coroners' Act (1980).
The prevalence of non-coronial certifications may also reflect medical practitioners' knowledge of patients' obstetric and paediatric histories. It cannot be argued, however, that the conduct of postmortem examinations helped to circumvent coronial certification by eliminating uncertainty over the cause(s) of death: the signing of the death certificate or of a notice of intention to sign this certificate had to precede any autopsy.7

Neonatal deaths were much more likely than stillbirths to be reported to a coroner because any death known to be a stillbirth is not reportable to this official. Only the death of a legally liveborn infant, one who shows evidence of breathing at birth, can come under a coroner's jurisdiction. In four cases, in which there was uncertainty over whether the death was a stillbirth or a neonatal death, the case was reported to the coroner for performance of a postmortem examination. Once the autopsy findings revealed the baby to be stillborn, the coroner no longer had a legal interest in the case and an order authorizing disposal of the body was signed by the coroner's delegate.

4.8 DEFICIENCIES IN CAUSE OF PERINATAL DEATH REGISTRATIONS

Deficiencies in cause of perinatal death registrations might have resulted from two sets of factors, each of which is discussed in turn. These factors relate to unanswered queries by the Registrar, and to aspects of the reporting and conduct of postmortem examinations.

7 The terms autopsy, necropsy and postmortem examination all refer to dissection and examination of a body after death.
4.8.1 UNANSWERED QUERIES

The possibility of errors of content in the certification of cause(s) of perinatal death was introduced in section 3.4.1. Now, other specific problems require exposition, the first relating to queries made by the registrar when there was inadequate reporting of the cause(s) of death, usually the underlying cause of death.

Table 4.6 shows that almost one-quarter of certifications prompted such a query, but that no reply was received in 51 (2.8 per cent) cases. This may not be seriously problematic since the figures are small, yet the queries were sufficiently important to warrant making, and cause-of-death amendments were made on 54.6 per cent (n=194) of the 355 certificates for which queries were answered.

Table 4.6: Query on the Underlying Cause of Death or Main Maternal Condition Affecting the Foetus or Infant, 1985-86

<table>
<thead>
<tr>
<th></th>
<th>Still-Births</th>
<th>Neonatal deaths</th>
<th>Perinatal deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No</td>
<td>%</td>
<td>No</td>
</tr>
<tr>
<td>No query</td>
<td>792</td>
<td>78.8</td>
<td>610</td>
</tr>
<tr>
<td>Query sent but no reply</td>
<td>23</td>
<td>2.3</td>
<td>28</td>
</tr>
<tr>
<td>Query and cause of death unchanged</td>
<td>103</td>
<td>10.2</td>
<td>58</td>
</tr>
<tr>
<td>Query and cause of death amended</td>
<td>87</td>
<td>8.7</td>
<td>107</td>
</tr>
<tr>
<td>Total</td>
<td>1005</td>
<td>100.0</td>
<td>803</td>
</tr>
</tbody>
</table>

(Source, Perinatal death registrations, 1985-87)

If this amendment rate can be extrapolated to the queries receiving no reply, an estimated 28 cases have at least one cause of death stated 'incorrectly'. Indeed this probably understates the true figure.
because while neonatal queries were the least likely to receive replies, amendments, resulting from queries, occurred most often among neonatal deaths: 64.8 per cent (n=107) of replies to queries led to amendments of the (usually underlying) cause of neonatal death.

4.8.2 THE PERINATAL AUTOPSY: ITS REPORTING AND CONDUCT

The remaining difficulties relate to reporting of the perinatal autopsy as required by question 25 of the death certificate. First, there were delays in receiving autopsy findings. Results confirming the cause(s) of perinatal death were received in only 19.6 per cent (n=355) of all cases. Autopsy findings were still to be received in 40.0 per cent (n=723) of cases (see Table 4.7). That death certificates were being dispatched before autopsy evidence became available, constitutes misuse of an important potential role of the autopsy: to facilitate the production of accurate statistics that can aid patient care.

Table 4.7: Performance of Perinatal Autopsy by Type of Certification, 1985-86

<table>
<thead>
<tr>
<th>Type of certification</th>
<th>Non-coronal</th>
<th>Coronal</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
</tr>
<tr>
<td>Cause of death confirmed by autopsy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>328 18.6</td>
<td>27 57.4</td>
<td>355 19.6</td>
</tr>
<tr>
<td>Autopsy information may be available later</td>
<td>706 40.1</td>
<td>17 36.2</td>
<td>723 40.0</td>
</tr>
<tr>
<td>Autopsy not held</td>
<td>578 32.8</td>
<td>0 0.0</td>
<td>578 32.0</td>
</tr>
<tr>
<td>None of the above</td>
<td>148 8.4</td>
<td>4 6.4</td>
<td>152 8.4</td>
</tr>
<tr>
<td>Total</td>
<td>1760 100.0</td>
<td>48 100.0</td>
<td>1808 100.0</td>
</tr>
</tbody>
</table>

(Source. Perinatal death registrations, 1985-87)
At a more fundamental level, serious problems affected the validity of the data collected. The problems relate to poor wording of item 25 and the quality of the postmortem examinations being performed. Thus secondly, there is uncertainty about what is intended by the response statement 'the cause of death has been confirmed by autopsy'. Does 'the cause of death' refer to the main cause in the foetus or infant, to the main condition in the mother affecting the foetus or infant, to the underlying cause, or to some combination of these causes?

The most likely answer is the main condition in the foetus or infant. This is because pathologists may not receive the placenta along with the dead baby, and even if the main aetiological condition in the mother can be identified through autopsy, it is unclear how the item would accommodate multiple causes: for example, what would happen if the autopsy rejected the clinically observed main condition in the mother, but accepted the clinical diagnosis in the foetus or infant?

The intention of the response statement is thus probably as noted above, but this should be stated explicitly or at least referred to in the registrar's booklet for helping medical practitioners complete the death certificate (Australian Bureau of Statistics, 1984). This does not occur because the item is unimportant for registration purposes.

The third difficulty with item 25 also relates to confusion about the frequency with which autopsies were performed. The difficulty results from two causes, the first being the ambiguous wording of the response category 'autopsy information may be available later'. This may mean an autopsy has been held but autopsy results are not yet available, or alternatively, an autopsy may be held in which case autopsy findings will later become available. The latter possibility would arise if parents were still deciding at the time the death certificate was signed, whether to consent to an autopsy. Given this ambiguity and no
guidelines for interpreting the statement, likely differences across hospitals would detract from the value of information collected.

The second cause of uncertainty about the frequency of perinatal autopsies concerns the *reason* some certifiers failed to identify an autopsy category on the registration form. This might have occurred when the pathological findings of postmortem examinations did not confirm clinical diagnoses since direct provision is not made for such occurrences. The cause of death statement takes autopsy evidence into account, but question 25 (which relates specifically to the perinatal autopsy) makes no provision for this evidence when it disagrees with clinical information obtained during life; and this could explain why 8.4 per cent (n=152) of certifiers did not answer this question: they could not answer it. Although this failure to assess the correspondence between clinical and pathological diagnoses is important *ipso facto*, for now it is emphasized for revealing additional deaths on which a postmortem examination might have been performed. In this latter context different estimates become available of the frequency with which perinatal autopsies were performed.

Table 4.7 shows that an autopsy was unequivocally held for only 19.6 per cent (n=355) of perinatal deaths. An autopsy might have been held in 59.6 per cent of cases (n=1078), if deaths for which 'autopsy information may be available later' in fact involved an autopsy; or at most 68.0 per cent (n=1230) of deaths if failure to identify any of the provided categories resulted from the omission noted above. What is clear is that in only about one-third of deaths was a perinatal autopsy certainly not conducted, and it is hard to conceive of this proportion rising much in the period following certification; it accords with the larger than normal rate of 75 per cent for perinatal
autopsies to have been expected from a large maternity hospital in Sydney (pers. comm. H. Jeffery, 1989).

Also clear from the table is the more frequent conduct of postmortem examinations in coronial certifications. Coroners routinely ordered a postmortem examination, with no deaths probably being excluded from this investigation, for two important reasons: the exceptional nature of these deaths, the causes of which were likely to have been unknown, and statutes facilitating universal, legal conduct of the perinatal autopsy. For example, section 48(1) of the New South Wales Coroners' Act, 1980 gives authority to coroners to direct postmortem examinations, immune from challenge by relatives or other involved persons, if the results of such examinations may render the holding of an inquest unnecessary.

The fourth, main problem with the data describing postmortem examinations concerns the validity of postmortem results per se. The potential problem is that in New South Wales a postmortem examination may be performed by any legally qualified medical practitioner, not necessarily a qualified pathologist, and not necessarily a paediatric pathologist or one who frequently conducts perinatal autopsies and is interested in doing them. The last qualifications are most important if one accepts that the skill of the person conducting an autopsy bears a direct relation to the value of evidence derived from it (Plueckhahn, 1983). Yet it is not known how often suitable, rather than merely legally qualified, specialists performed or supervised perinatal autopsies. Such occurrences may help to explain some

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8 Coronial protection was previously given under common law, only with the holding of an inquest. In non-coronial certifications, the consent of relatives is required before an autopsy can be legally performed (See, for example, sections 28(2) and 29(2) of the New South Wales Human Tissue Act (1983)).
failures to make additional or supplementary diagnoses through postmortem examination.

As a result of all these problems, epidemiological records are left incomplete and inaccurate, and the efficacy of the perinatal autopsy becomes difficult to assess. Although efficacy is not the only reason for performing the examination - for example, the parents or coroner may request it - the perinatal autopsy may reasonably be expected in a large proportion of cases to add clinically important information particularly about the disease process and cause of death. Whether this occurs is obscure at the aggregate level in New South Wales because autopsy results are incomplete at the time of certification; are not put into precisely defined and mutually exhaustive categories; and are not directly and accurately related to clinical diagnoses.

Consequently, considerations of efficacy prompt a look at overseas research. In England, Porter and Keeling (1987) detected in 36 per cent of 150 stillbirths and in 44 per cent of 66 neonatal deaths, autopsy results that differed in important respects from the clinical information obtained during life. In a study by Meier et al. (1986), the autopsy was the sole means of establishing the cause of death and the need for genetic counselling in 26 and 22 per cent respectively of 139 perinatal deaths. In each investigation, a large proportion of the autopsy results provided important additional information.

Yet these studies also showed that in the majority of examinations, the cause(s) of death could be accurately determined from investigations performed during life, and that the perinatal autopsy confirmed what was already known. Depending on the objectives of the autopsy, this may suggest the need for careful selection of the deaths for which an autopsy is to be conducted. With stillbirths, in particular, establishing a cause of death in the foetus is especially
difficult. If it is severely macerated, identification can usually be made of developmental abnormalities but not of conditions such as intrauterine infections.

This likelihood of discovering new and clinically important information should perhaps be the determinant of whether parents are actively encouraged to consent to a perinatal autopsy. Contemporaneously, medical practitioners need to appreciate that even if the cause(s) of death can be determined, this knowledge may not necessarily relieve parents of their grief and existential guilt (Barr, 1988). On the other hand, guilty feelings may be compounded by not having the opportunity to understand why the baby died, so that an autopsy will not augment this guilt by leaving a negative unknown. At best, parents must be fully and honestly informed about the methods and likely benefits of the examination. Since no-one would dispute that the issue of perinatal autopsy is extremely complex, remedying the lacunae on the death certificate must occur before proper assessment of the practice of perinatal autopsies can take place.

4.10 CONCLUSION

The concept of perinatal death is essentially classificatory, a convenient way of understanding losses around the period of birth. However, this chapter's discussion of distinctive, particularistic characteristics of perinatal deaths, including their timing and stated causes, is a necessary precursor to the efforts of chapters 5 to 8 to determine risk factors for perinatal death per se.

In this chapter, perinatal death in New South Wales during 1985-1986 was found to reflect a slightly larger proportion of stillbirths than neonatal deaths, and to occur more often antepartum, or especially postnatally, than during labour and delivery. Certain groups of
causes of death were most prevalent. The main cause of perinatal death, and more commonly stillbirth, reported in the foetus or infant was hypoxia, birth asphyxia and other respiratory conditions. This was followed by congenital anomalies, especially among neonatal deaths. The main maternal disease or condition contributing to perinatal death, and most conspicuously to stillbirth, was complications of the placenta, cord and membranes. Maternal complications of pregnancy was nearly as important because of its prevalence among neonatal deaths, while no maternal condition was reported in almost one-quarter of perinatal deaths. The most frequently reported underlying cause of perinatal death was complications of the placenta, cord and membranes, especially among stillbirths. Almost all certifications reporting these causes were non-coronal, and most certifications did not accurately report the results of postmortem examinations, valid data being needed on this procedure to ascertain its usefulness in some circumstances.
5.0 INTRODUCTION

The physiological maturity of the conceptus and, as defined in section 2.6, obstetrical factors, are two of the most proximate and important determinants of perinatal outcome. Physiological immaturity, its most common empirical referent being low birthweight, is probably the key determinant (see section 2.3.1) as exemplified for New South Wales in section 3.5. Accordingly, initial inquiries in this chapter focus mainly on whether different causes of low birthweight, namely preterm birth and intrauterine growth retardation, aid the explanation of why some low birthweight babies survive whilst others do not. Less attention is given to babies that are post-term, about one-fifth of whom may suffer from the postmaturity syndrome (Haesslein, 1981), or that are large for gestational age; small numbers of infants at high birthweights permit limited consideration of this group. Subsequently examined is the relation of five obstetrical factors to perinatal death. The effects of the sex and plurality of the foetus are separately considered before these variables provide a common theme for exploring the aetiological effects of three sets of obstetric complications in the mother: the hypertensive disorders of pregnancy, placenta praevia and premature rupture of the membranes.

5.1 PHYSIOLOGICAL MATURITY OF THE FOETUS AND INFANT

The main index of perinatal risk used in this study is low birthweight. Despite its limitations (see section 3.5), the index is
simple, is easily measured and summarizes many maternal and foetal complications that are too rarely reported to be consistently incorporated into basic methods of analysis. Two variables as ubiquitous as birthweight are gestational age and birthweight for gestational age.

Commonalities between all three variables, and small numbers especially in the case group, explain the decision usually not to adjust for gestational age as well as for birthweight when other variables are added. Ideally, of course, this should be done because, despite their similarities, the two measures of physiological maturity are different: birthweight best describes growth, whereas gestational age more directly indicates development of the baby's organ or physiological systems.

However, the following analysis can elucidate the relations between perinatal death and the stated measures of physiological maturity. This is done by observing how the aetiological effects mainly of low birthweight vary across categories of gestational age, especially preterm birth, and of the appropriateness of intrauterine growth for this gestational age, emphasizing the infant who is growth retarded in utero. These two sets of variables are considered separately and then together.

5.1.1 GESTATIONAL AGE AND GROWTH RETARDATION IN UTERO

In developed countries preterm birth is the most frequent cause of low birthweight (see section 2.6.1). The association of preterm birth with perinatal death reflects the causation not merely of low birthweight, however, but of developmental abnormalities which can

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1 Section 3.5 elaborates on this failure to control for the confounding effects of variables such as gestational age.
make adaptation to extrauterine life difficult. Preterm birth may thus be hazardous even when the birthweight is normal, although the greatest danger attends the coincidence of both characteristics. Post-term birth, while not usually problematic, can produce complications.

Less widespread than preterm birth, but an important cause of perinatal death, is growth retardation in utero. Its effects, like those of preterm birth, vary with the timing of the 'aetiologic insult'. Yet compared with preterm birth, retarded intrauterine growth brings higher risks during the labour and delivery, and lower risks during the neonatal period (Chiswick, 1978; and see section 2.6.1). Large for gestational age infants experience increased perinatal risks of, for example, hypoglycaemia\(^2\) and hypocalcaemia\(^3\).

Before empirically assessing these complications' aetiological contributions, three methodological problems are discussed. First, the accurate estimation of gestational age is difficult. This information is in question for 20 to 50 per cent of women in reported series owing to uncertain menstrual data (Beischer, 1983),\(^4\) which are themselves associated with low birthweight and low socioeconomic and sociodemographic statuses, plus late booking for antenatal care (Buekens et al., 1984).

Even when the date of the last menstrual period is known, estimation of gestational age can be undermined by menstrual irregularities, the misinterpretation of pregnancy as post-pill amenorrhoea, and slight bleeding in early pregnancy (Lancaster, 1989b). Before 20, and

\(^2\) Hypoglycaemia is an abnormally low blood sugar.

\(^3\) Hypocalcaemia is a low level of calcium in the blood.

\(^4\) However, from the reported date of the last normal menstrual period, Kliewer and Stanley (1989) were unable to calculate the gestational age of only 2.6 per cent of singleton live births in Western Australia during 1980-86.
especially between 8 and 12 weeks' gestation, ultrasound measurement can permit an accurate clinical assessment of gestational age from foetal size. However, after the 20th week of pregnancy, estimates of gestational age become increasingly inaccurate, so that the gestational age may remain uncertain especially in women who commence antenatal care late (New South Wales Department of Health, 1989d).

Secondly, a probably minor problem is the confounding effect of lung maturation therapy. The foetus that receives high doses of glucocorticoids between 28 and 34 weeks' gestation, and possibly earlier (for 24 to 48 hours), may be at lower risk of respiratory distress syndrome and hence perinatal mortality than a foetus of more advanced gestation that does not receive such treatment.

Thirdly, relating foetal growth to gestational age is problematic. Foetal growth retardation (that is, smallness for gestational age) is rarely symptomatic before 28 to 30 gestational weeks. Although serial ultrasonic examination can show foetal growth retardation well ahead of clinical signs, the condition is not usually diagnosed antenatally and can be precisely identified only at birth (Beischer, 1983; New South Wales Department of Health, 1989d). Concomitantly, the ability of intrauterine growth charts to assess appropriateness of foetal growth depends on the degree of similarity between the group to which the charts are being applied and the population from which the charts were developed (see page 223 and Table 5.0).

With these caveats, examination is made of the separate contributions to perinatal death of gestational age per se (see Table 5.1) and of the appropriateness of foetal growth for different gestational ages

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5 Glucocorticoids may be contraindicated in the woman with pre-eclampsia with proteinuria, rupture of the membranes or cervical suture because of the increased risk of perinatal death (New South Wales Department of Health, 1989d).
(see Table 5.2), and then of the joint aetiological contributions of these variables (see Table 5.3). The last two assessments involved using fourteen charts which show gestation-specific percentile birthweights. These charts were prepared by the Commonwealth Department of Health (1985) from a Western Australian population of singleton, Caucasian live births occurring between 1979 and 1982. Two charts, one for each sex, describe the period from 22 to 31 gestational weeks. Twelve charts represent the remaining births categorized by sex, maternal height (tercile) and parity (0, >1). Births affected by pathological influences on growth, such as diabetes and smoking, could not be excluded in preparing the charts.

In the present study, estimates after 31 weeks' gestation show the mean effect of terciles of height on growth estimates by sex and gestational age. All estimates, so that they relate directly to the charts, are for singleton births, of known sex, after at least 22 weeks' gestation; to complement this presentation in Table 5.0, the analysis of gestational age per se (Table 5.1) is conducted in like terms. Although multiple births are not considered, they could be related indirectly to the charts, and this is done for stillbirths.

Before examining these tables, it is pertinent, as indicated above, that the appropriateness of foetal growth is most accurately assessed for the population on which the intrauterine growth charts are based, and then for other groups with maternal and foetal or infant characteristics similar to those of this source population. The bias should be minimal in the analysis below because, with reference to singleton births, certain similarities between the populations of New South Wales and Western Australia are clear from the selected variables shown in Table 5.0.
Nulliparity and caesarean section yield near-identical proportions, and there are high levels of induction in both States, although especially in Western Australia. The two sociodemographic profiles are alike, the percentage of Aboriginal women being smaller for New South Wales, probably owing in part to under-reporting of Aboriginal women in this State (see section 8.2.1). New South Wales also recorded proportionately fewer women with pre-eclampsia, the most common obstetric complication in both populations, but in each region, more than two-thirds of the women experienced no obstetric complication; New South Wales was safer apparently in this respect. Each State showed low levels of newborn physiological immaturity and perinatal death. Thus, despite almost 3.4 times fewer singleton births in Western Australia (N=23 290) than in New South Wales, there is a reasonable equivalence between the populations (Health Department of Western Australia, 1988; Maternal and Perinatal Collection, 1986).

Table 5.0: Sociodemographic and Obstetric Characteristics of New South Wales and Western Australian Singleton Births, a 1986

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>New South Wales</th>
<th>Western Australia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td></td>
<td>No.</td>
<td>No.</td>
</tr>
<tr>
<td><strong>Mother</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sociodemographic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age 20-34 years</td>
<td>85.9</td>
<td>86.2</td>
</tr>
<tr>
<td>Married/de facto</td>
<td>84.8</td>
<td>88.3</td>
</tr>
<tr>
<td>Aboriginal</td>
<td>1.6</td>
<td>5.3</td>
</tr>
<tr>
<td><strong>Obstetric</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nulliparous</td>
<td>38.9</td>
<td>38.9</td>
</tr>
<tr>
<td>Pre-eclampsia</td>
<td>4.7</td>
<td>7.5</td>
</tr>
<tr>
<td>No obstetric complication</td>
<td>76.8</td>
<td>69.4</td>
</tr>
<tr>
<td>Induction</td>
<td>20.8</td>
<td>26.9</td>
</tr>
<tr>
<td>Caesarean section</td>
<td>15.5</td>
<td>15.4</td>
</tr>
<tr>
<td><strong>Foetus or infant</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low birthweight</td>
<td>4.6</td>
<td>4.9</td>
</tr>
<tr>
<td>Preterm</td>
<td>4.9</td>
<td>7.0</td>
</tr>
<tr>
<td>Perinatal deaths</td>
<td>8.3</td>
<td>10.5</td>
</tr>
</tbody>
</table>

a Excludes births below 500 grams birthweight

(Source. Health Department of Western Australia, 1988; Maternal and Perinatal Collection of New South Wales, 1986)
Returning to the analysis of physiological maturity per se, comparison of Tables 5.1 and 5.2 indicates that, at low birthweights, preterm birth is more prevalent than foetal growth retardation irrespective of the perinatal outcome, although especially in the case group.\(^6\)

Retarded growth in utero is more prevalent in the control group. Hence, preterm birth characterizes 78.3 per cent (n=799) and 90.8 per cent (n=276) of all low birthweight and non-surviving low birthweight newborn respectively, the corresponding figures for growth retarded infants being 61.2 per cent (n=624) and 53.9 per cent (n=164).

Preterm birth especially, but also intrauterine growth retardation, is less strongly associated with larger babies. At birthweights 2 500 to 3 999 grams, 5.4 per cent (n=53) of infants are born before term and 11.5 per cent (n=113) are growth retarded. With non-survival, the differential disappears. With survival, the occurrence of preterm birth is almost halved to 2.8 per cent (n=23), while the incidence of intrauterine growth retardation is little affected at 10.2 per cent (n=83). In the weighted sample of all infants, birth before term, with an incidence of 5.3 per cent (n=3 369), is the less common of the two conditions by a factor of 2.2. However, preterm birth (63.7 per cent, n=307) is more prevalent than is retarded growth (40.2 per cent, n=194) in the case group.

Separate schedules of the perinatal mortality risks associated with being born preterm or small for gestational age are more clearly represented by the estimated odds ratios in the two tables than by the derived proportions. The odds ratios, after adjustment for

\(^6\) Analyses of the proportions calculated from subsequent tables relate to all births, usually without regard to survivorship since this is implicit in the estimated odds ratios; although proportions in the case group can usefully express the results of the odds ratios. Here, because estimates of relative risk are not directly comparable across Tables 5.1 and 5.2, measures of prevalence are widely described for all births and also for the case and control groups separately.
Table 5.1: Relation of Perinatal Death to Gestational Age according to Birthweight of Singleton Infants of Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Gestational Age (completed weeks)</th>
<th>Birthweight (grams)</th>
<th>500-1 499</th>
<th>1 500-2 499</th>
<th>2 500-3 999</th>
<th>4 000+</th>
<th>θ&lt;sub&gt;mh&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P.D. Ctl. 0</td>
<td>P.D. Ctl. 0</td>
<td>P.D. Ctl. 0</td>
<td>P.D. Ctl. 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22-27</td>
<td>129</td>
<td>56</td>
<td>14.04**</td>
<td>6.10</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>28-32</td>
<td>61</td>
<td>174</td>
<td>2.14***</td>
<td>41</td>
<td>3.42***</td>
<td>1</td>
</tr>
<tr>
<td>33-36</td>
<td>17</td>
<td>44</td>
<td>2.35**</td>
<td>45</td>
<td>207</td>
<td>1.33</td>
</tr>
<tr>
<td>37-41&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3</td>
<td>10</td>
<td>1.83</td>
<td>24</td>
<td>182</td>
<td>0.80</td>
</tr>
<tr>
<td>&gt;41</td>
<td>0</td>
<td>0</td>
<td>-</td>
<td>1</td>
<td>6.10</td>
<td>9</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
<sup>a</sup> Reference group for Mantel-Haenszel estimated odds ratios
<sup>b</sup> Reference group for other odds ratios
** Statistically significant at the 0.01 level
*** Statistically significant at the 0.005 level
(Source. Maternal and Perinatal Collection, 1986)

Table 5.2: Relation of Perinatal Death to Appropriateness of Birthweight for Gestational Age according to Birthweight of Singleton Infants of Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Appropriateness of Birthweight for Gestational Age</th>
<th>Birthweight (grams)</th>
<th>500-1 499</th>
<th>1 500-2 499</th>
<th>2 500-3 999</th>
<th>4 000+</th>
<th>θ&lt;sub&gt;mh&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P.D. Ctl. 0</td>
<td>P.D. Ctl. 0</td>
<td>P.D. Ctl. 0</td>
<td>P.D. Ctl. 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small for Gestational Age</td>
<td>98</td>
<td>159</td>
<td>3.37***</td>
<td>66</td>
<td>301</td>
<td>1.20</td>
</tr>
<tr>
<td>Appropriate for Gestational Age&lt;sup&gt;a&lt;/sup&gt;</td>
<td>95</td>
<td>112</td>
<td>4.64***</td>
<td>18</td>
<td>122</td>
<td>0.81</td>
</tr>
<tr>
<td>Large for Gestational Age</td>
<td>17</td>
<td>13</td>
<td>7.15***</td>
<td>10</td>
<td>9</td>
<td>6.07***</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
<sup>a</sup> Reference group for Mantel-Haenszel estimated odds ratios
<sup>b</sup> Reference group for other odds ratios
*** Statistically significant at the 0.005 level
(Source. Maternal and Perinatal Collection, 1986)
birthweight, are greatest before 28 weeks ($\theta_{mh}=7.67$) relative to the reference group of term babies. Estimates of the relative risk of perinatal death continue to fall with increasing gestational age until 42 weeks, the Mantel-Haenszel odds ratio for post-term birth ($\theta_{mh}=2.33$) being lower than for any comparable estimate of risk before term. Overall, being born preterm increases the estimated relative risk of death almost threefold ($\theta_{mh}=3.05$).

The crude odds ratios are likewise predictably highest at 22 to 27 weeks' gestation when they decline with increasing (low) birthweights. However, at other, successive gestational ages before term, the highest odds ratios attend the largest birthweight at which babies of these gestational ages are born. Thus, at 33 to 36 weeks' gestation, the highest odds ratio is for infants with birthweights 2 500 to 3 999 grams ($\theta=7.42$), taking term infants of normal birthweight as the reference group. Curiously, this odds ratio exceeds all estimates of relative risk at the earlier gestational ages of 28 to 32 gestational weeks.

Table 5.2 estimates that after adjustment for birthweight, newborn who are large for gestational age face the greatest risk of death ($\theta_{mh}=1.17$) relative to babies whose birthweight is appropriate for gestational age. Without adjustment, this risk is restricted to small numbers of infants of low birthweight; estimates are relative to newborn whose birthweights of 2 500 to 3 999 are appropriate for gestational age.

Intrauterine growth retardation is most hazardous at very low birthweights and more so at normal birthweights than at birthweights 1 500 to 2 499 grams; fortunately growth retardation is less common in

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7 Birthweights that are appropriate for gestational age lie between the tenth and ninetieth percentiles. The terms small for gestational age and large for gestational age are defined in the glossary and in chapter 1, footnote 51.
the heavier babies. Irrespective of the appropriateness of foetal growth for gestational age, the foetus of very low birthweight is most at risk of dying during the perinatal period.

The next step is to explore how survival chances reflect relations between gestational age and the appropriateness of birthweight for gestational age. Using Table 5.3, this is done, first, by relating each level of the appropriateness of growth for gestational age to differences in gestational age and birthweight. The discussion mainly articulates the effect of preterm birth (plus other gestational ages) on the birthweight of newborn who are small for gestational age.

Secondly, perinatal risks for different gestational age and birthweight combinations are compared across levels of appropriateness of birthweight for gestational age.

Table 5.3: Relation of Perinatal Death to Gestational Age according to Birthweight and Appropriateness of Birthweight for Gestational Age of Singleton Infants of Determinate Sex among Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Appropriateness of Birthweight for Gestational Age</th>
<th>Gestational Age (completed weeks)</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-1499</td>
<td>1500-2499</td>
</tr>
<tr>
<td>Small for Gestational Age</td>
<td>P.D. Ctl. 0</td>
<td>P.D. Ctl. 0</td>
</tr>
<tr>
<td>22-27</td>
<td>36 12 8.68***</td>
<td>0 0 -</td>
</tr>
<tr>
<td>28-32</td>
<td>42 93 1.31</td>
<td>1 5 0.58</td>
</tr>
<tr>
<td>33-36</td>
<td>40 113 1.02</td>
<td>0 1 -</td>
</tr>
<tr>
<td>37-41</td>
<td>24 182 0.38***</td>
<td>28 81b 1.00</td>
</tr>
<tr>
<td>&gt;41</td>
<td>0 0 -</td>
<td>1 1 2.89</td>
</tr>
<tr>
<td>Appropriate for Gestational Age</td>
<td>P.D. Ctl. 0</td>
<td>P.D. Ctl. 0</td>
</tr>
<tr>
<td>22-27</td>
<td>77 35 6.36***</td>
<td>0 0 -</td>
</tr>
<tr>
<td>28-32</td>
<td>18 77 0.48</td>
<td>13 29 1.30</td>
</tr>
<tr>
<td>33-36</td>
<td>0 0 -</td>
<td>5 93 0.16***</td>
</tr>
<tr>
<td>37-41</td>
<td>0 0 -</td>
<td>0 0 -</td>
</tr>
<tr>
<td>&gt;41</td>
<td>0 0 -</td>
<td>0 0 -</td>
</tr>
<tr>
<td>Large for Gestational Age</td>
<td>P.D. Ctl. 0</td>
<td>P.D. Ctl. 0</td>
</tr>
<tr>
<td>22-27</td>
<td>16 9 5.14***</td>
<td>1 1 2.89</td>
</tr>
<tr>
<td>28-32</td>
<td>1 4 0.72</td>
<td>9 7 3.72*</td>
</tr>
<tr>
<td>33-36</td>
<td>0 0 -</td>
<td>0 1 -</td>
</tr>
<tr>
<td>37-41</td>
<td>0 0 -</td>
<td>0 0 -</td>
</tr>
<tr>
<td>&gt;41</td>
<td>0 0 -</td>
<td>0 0 -</td>
</tr>
</tbody>
</table>

P.D.: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
* Statistically significant at the 0.025 level
** Statistically significant at the 0.01 level
*** Statistically significant at the 0.005 level
(Source. Maternal and Perinatal Collection, 1986)
Of the low birthweight babies that are estimated to be growth retarded in utero (n=624), almost two-thirds (64.6 per cent, n=403) are also preterm. Their estimated relative risk of death, after adjustment for birthweight, is 2.6 times higher (p < 0.005) than for the reference group of term but growth retarded infants. The result is that 83.4 per cent (n=136) of the growth retarded and low birthweight babies that died are also preterm, whilst at very low birthweights, the percentage is 96.9 per cent (n=95).

More detailed examination of the effect of gestational age on the survival of growth retarded babies is made taking each birthweight category in turn. The most common gestational ages for which newborns of very low birthweight are small is 28 to 32 weeks, but the risk of mortality to growth retarded and very low birthweight babies is greatest before 28 gestational weeks (θ=8.68) relative to infants that are small for gestational age and of normal birthweights.

Notwithstanding this, even for the case group, growth retardation is most prevalent among babies born between 28 and 32 weeks: 42.8 per cent (n=42) of very low birthweight non-survivors are then growth retarded, compared with 36.7 per cent (n=36) before 28 weeks.

For babies with birthweights between 1 500 and 2 499 grams, growth retardation is shown to be most hazardous (on a sample of two) after 41 weeks but, before term, from 33 to 36 weeks' gestation (θ=1.02). The latter statistic is the more meaningful since 41.7 per cent (n=153) of the growth retarded infants weighing 1 500 to 2 499 grams at birth are born immediately before term when their estimated relative risk of death is 2.7 times higher than at term (θ=0.38).

During the latter period, retardation in utero is even more prevalent. Term accounts for 56.1 per cent (n=206) of growth retarded babies in
the specified birthweight interval, growth retardation then being most likely asymmetrical owing to its probable later onset.

Presumably, this causation even better explains growth retardation at birthweights 2500 to 3999 grams, with uteroplacental insufficiency resulting less commonly from post-dates than from complications of pregnancy including late third trimester pre-eclampsia. The low prevalence of mortality after 41 weeks is fortunate because it yields an odds ratio (θ=5.79), admittedly on small numbers, which is almost six times higher than that found at term—the locus of the reference group.

Among babies appropriate for gestational age, 95.8 per cent (n=45897) reach term after weighting (see sections 3.8.1 and 3.8.2). Odds ratios, which presumably best indicate the effect of gestational age, are largest for very low birthweight babies (θ=6.36), but a surprisingly high odds ratio of 3.91 is associated with newborn of normal birthweight and of gestational ages 33 to 36 weeks. Among babies large for gestational age, very low birthweights before 28 weeks are predictably most hazardous. In the weighted sample, just 5.3 per cent (n=421) of large for gestational age infants are not born after 36 weeks' gestation.

A second approach relates perinatal death to the appropriateness of birthweights for gestational age at different birthweights and gestational durations. Of most interest are preterm babies of low birthweight, half (n=403) of which are estimated to be growth retarded; the same proportion is found for newborn of very low birthweight. Taking survivorship into account affects only the latter babies among whom the coincidence of preterm birth falls by 4.8 percentage points.
Further analysis reveals that among all preterm babies of low birthweight, advancing gestational age increases the proportion of growth retarded infants. This is clearest for babies of very low birthweight. The percentage of growth-retarded newborn increases from 25.9 per cent (n=48) before 28 weeks to 57.4 per cent (n=135) at 28 to 32 weeks' gestation to all babies (n=61) between 33 and 36 weeks' gestation. After 27 weeks, the same trend is found for babies weighing 1 500 to 2 499 grams at birth.

Coupled with these exposures are two patterns of estimated relative risks associated with birthweights. With growth retardation, estimated odds ratios in each gestational group before term decline with increasing birthweight. After 36 weeks' gestation, they assume a J-shaped pattern. Among babies that are appropriate or large for gestational age, a direct association occurs after 27 weeks' gestation between birthweights and odds ratios.

Necessarily all low birthweight infants from 37 weeks onwards are growth retarded. However, only 12.1 per cent (n=112) of babies of normal birthweight and born after 36 weeks are estimated as small for gestational age; although for the case group this percentage rises to 22.2 (n=30). Almost all post-term newborn are appropriate for gestational age, while just about one quarter of term babies (n=19) with birthweights of at least 4 000 grams are appropriate for dates (θ=0.58).

Finally, at low birthweights and making adjustment for birthweight, infants who are preterm and appropriate for gestational age yield $\text{OR}_{\text{mh}}=0.95$ relative to infants who are preterm and small for gestational age. Yet the risk is smaller only at birthweights 1 500 to 2 499 grams. Taking the same reference group, the odds ratio is $\text{OR}_{\text{mh}}=0.39$ for the babies who are small for gestational age and born at term, which
suggests that growth retardation by itself is a lesser threat to perinatal survival than is preterm birth or are both complications together. Notwithstanding this, being born at 33 to 36 weeks' gestation and appropriate for gestational age is apparently very safe ($\theta=0.16$).

5.1.2 PONDERAL INDICES

The problem with measuring physiological maturity by birthweight and gestational age is that these variables ignore the body size and length of the baby. To quantify thinness or obesity in infants, and to predict neonatal outcome, some investigators, such as Patterson and Pouliot (1987), have used the neonatal ponderal index. This is the product of the birthweight in grams, and a constant of 100, divided by the crown-heel length cubed.

A normal ponderal index, estimated by the writer to increase from about 1.75 to 2.75 with increasing gestational age in standard populations, is found when the weight and length are of similar size, for example, in newborn that are symmetrically small (low birthweight and short length). A low or high index is produced when the birthweight or the body-length is large or small for the gestational age.

A baby that is asymmetrically small, for example, with perhaps a low birthweight but normal body-length, has a low ponderal index.

Additional advantages of the ponderal index are that it is not affected by sex or race. Potential disadvantages include any error induced by cubing the length (Brar and Rutherford, 1988).

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8 The in utero ponderal index has also been used, for example by Yagel et al. (1987) to estimate foetal growth and outcome.
Terciles of neonatal ponderal indices, based on data collected from patient records, are reported in Table 5.4. Hospitals’ failure to record the crown-heel length for surviving babies, particularly when of very low birthweight, explains the greater prevalence of neonatal death vis-à-vis survival in each tercile. Low indices are confined almost entirely to very low birthweights and characterize 18.3 per cent (n=16) of the smallest liveborn babies. At birthweights 1 500 to 2 499 grams, 90.2 per cent (n=55) of the infants have normal ponderal indices, the largest proportion of high indices at any birthweight being one-quarter at birthweights 2 500 to 3 999 grams.

Table 5.4: Relation of Neonatal Death to Ponderal Indices of Physiological Maturity according to Birthweight of Infants of Women Aged 20 to 34 Years, 1985-86

<table>
<thead>
<tr>
<th>Ponderal Indices</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-1 499</td>
</tr>
<tr>
<td>P.D. Ctl. 0</td>
<td></td>
</tr>
<tr>
<td>0.98-1.74</td>
<td>12</td>
</tr>
<tr>
<td>1.75-2.75a</td>
<td>46</td>
</tr>
<tr>
<td>2.76-6.56</td>
<td>6</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
(Source. Hospital patient records, 1985-86)

For very low birthweight infants with low ponderal indices, the estimated risk of neonatal death is 2.57 relative to the babies whose birthweight and ponderal index are both normal. At birthweights below 1 500 grams, elevated odds ratios are also associated with other ponderal indices, high indices, for example, yielding the same odds ratio of 2.57. Measurable effects of low indices are restricted to the very smallest neonates, but high indices are hazardous at all birthweights except 2 500 to 3 999 grams, and attest to the apparently
very low sensitivity of the indices at low birthweights (see sections 2.5 and 5.1.3); that is, the indices appear to have minimal ability to predict neonatal death in small babies. The safety of high indices at normal birthweights (0=0.50) explains the Mantel-Haenszel estimate of 0.87, taking the reference group as normal ponderal indices.

5.1.3 APGAR SCORING

Apgar scores\(^9\) permit simple assessment of the condition of infants immediately after birth and of their mortality risk. However, there is uncertainty about whether there is any difference in the risk of perinatal death associated with Apgar scores taken at one and five minutes. According to Goetzman (1981), the one-minute Apgar score is the more useful predictor of survival, but Nelson and Ellenberg (1981) report that neonatal death correlates much better with low Apgar scores (0 to 3) at five minutes than at one minute.

In fact, low scores at one and five minutes both correlate highly with perinatal mortality (Mulligan et al., 1980), which is not to deny the importance of knowing whether higher mortality risks accompany Apgar scores - that may, or may not, be low - at one minute or at five minutes. This question is now addressed by comparing one-minute and five-minute Apgar scores with neonatal death, when scores exceed 0 at one or five minutes (see Table 5.5). Definitions of low scores differ between studies, complicating the assessment of scores' predictive abilities at different times. In this investigation scores of 0 to 2 describe severe depression, scores of 0 being sufficiently rare not to warrant separate consideration. Apgar scores of 4 to 6 indicate moderate depression, while those of 7 to 10 describe infants in excellent condition.

\(^9\) See section 2.3.2.
The lowest scores reported in Table 5.5 are most important. After weighting, they constitute 1.9 per cent and 0.2 per cent of Apgar scores at one and five minutes respectively, remaining comparatively rare even at low and very low birthweights. For example, 19.0 per cent (n=29) of 500 to 1 499 gram babies have low Apgar scores at one minute, this percentage being reduced at five minutes to 11.6 per cent (n=19) and at both times by higher birthweights. In the weighted sample, low birthweight babies are estimated to account for one-quarter of low scores at one minute, but 84.0 per cent (n=95) of these scores at five minutes. This matters because low scores are associated with a substantially increased neonatal mortality risk especially for the very smallest babies.

Table 5.5: Relation of Neonatal Death to Apgar Scores at One and Five Minutes according to Birthweight of Infants of Women Aged 20 to 34 Years, 1985-86

<table>
<thead>
<tr>
<th>Minute</th>
<th>Apgar Score</th>
<th>Birthweight (grams)</th>
<th>500-1 499 P.D. Ctl. o</th>
<th>1 500-2 499 P.D. Ctl. o</th>
<th>2 500-3 999 P.D. Ctl. o</th>
<th>4 000+ P.D. Ctl. o</th>
<th>O_{mh}</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0-2</td>
<td>28</td>
<td>1 184.80***</td>
<td>9</td>
<td>5</td>
<td>11.88***</td>
<td>1 0</td>
</tr>
<tr>
<td></td>
<td>3-6</td>
<td>24</td>
<td>51 3.11***</td>
<td>6</td>
<td>11</td>
<td>3.60</td>
<td>6 10</td>
</tr>
<tr>
<td></td>
<td>7-10</td>
<td>7</td>
<td>42 1.10</td>
<td>4</td>
<td>44</td>
<td>0.60</td>
<td>8 48</td>
</tr>
<tr>
<td>5</td>
<td>0-2</td>
<td>16</td>
<td>3 35.20***</td>
<td>4</td>
<td>1</td>
<td>26.40***</td>
<td>6 0</td>
</tr>
<tr>
<td></td>
<td>3-6</td>
<td>22</td>
<td>17 8.54***</td>
<td>7</td>
<td>2</td>
<td>23.10***</td>
<td>6 0</td>
</tr>
<tr>
<td></td>
<td>7-10a</td>
<td>19</td>
<td>87 1.44</td>
<td>9</td>
<td>61</td>
<td>0.97</td>
<td>10 66</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control

a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
** Statistically significant at the 0.01 level
*** Statistically significant at the 0.005 level
(Source: Hospital patients' records, 1985-86)

This is shown by estimates of the relative risk of neonatal death expressed relative to the reference group of newborn with normal birthweights and Apgar scores of 7 to 10 at five minutes. For infants of very low birthweight, the estimated relative risk of neonatal death associated with low one-minute Apgar scores is 184.80. This is five and a quarter times higher than at five minutes (0=35.2). Death
results for 91.7 per cent (n=44) of very low birthweight babies with low Apgar scores at one or five minutes.

This increased risk associated with low scores at one minute is not found for newborn weighing 1 500 to 2 499 grams. The odds ratio associated with low scores at five minutes (θ=26.40) is more than twice that recorded at one minute (θ=11.88). Exposure to scores of 0 to 2 is increased for 1 500 to 2 499 gram infants vis-à-vis very low birthweight infants by a factor of two between one and five minutes. The much increased risk of perinatal death for the tiniest infants best explains the birthweight-adjusted Mantel-Haenszel estimates for low scores of 38.78 at one minute and 24.98 at five minutes; these estimates are relative to five minute Apgar scores greater than six. The former estimates accommodate the odds ratio of 52.80 for neonates of normal birthweight.

Apgar scores of 3 to 6, indicating moderate depression, also yield estimates of increased mortality risk at low birthweights, and so give some evidence of the low sensitivity of the scores (see section 2.3.2) at least at five minutes. However, although larger risks attend scores of 3 to 6 at five minutes, these scores are then less common than at one minute, which most affects birthweights below 1 500 grams. The result is similar proportions of deaths. Of specific interest is that one-minute scores for lightweight infants are conspicuously alike (θ=3.11 and 3.60) by comparison with elevated estimates at five minutes for the corresponding birthweights; these estimates are 8.54 for very low birthweight newborn and 26.40 for the smaller number of affected infants with birthweights 1 500 to 2 499 grams. Adjustment for birthweight yields a Mantel-Haenszel estimate at five minutes (θ_{mh}=7.58), 2.9 times larger than the one minute estimate of 2.65.
Infants with high Apgar Scores (7-10), especially at one but also at five minutes, have the best survival chances; the one-minute, Mantel-Haenszel estimated odds ratio is 0.89. Unadjusted odds ratios are predictably higher at very low and at high birthweights.

In sum, whether Apgar scores at one minute or at five minutes better predict perinatal mortality depends on the size of these scores. Neonatal mortality risks are more strongly increased by low Apgar scores (0 to 2) at one minute than at five minutes, but by higher scores (3 to 10) at five minutes than at one minute.

5.2 OBSTETRICAL FACTORS

Contributing either directly to perinatal outcome, or more often by affecting the physiological maturity of the foetus, are obstetrical factors. Five such factors are discussed here. The plurality and sex of the foetus are separately explored, the latter variable being related mainly to the propensities of boys for preterm birth. Particularly the sex of the foetus then provides the theme for examining three other conditions in the mother - hypertensive disorders of pregnancy, pre-eclampsia and premature rupture of the membranes. Hypotheses are tested that these complications are more prevalent when the foetus is a boy than a girl. The focus on differentials by sex is associated only with singleton births in order to avoid multiple births of different sexes.

5.2.1 PLURALITY

The literature is replete with documentation of the increased risks for perinatal death, and for its antecedent complications including especially prematurity, associated with multiple birth (see section 2.6.1). However, rarely reported is that perinatal death is less
frequent among twins delivered preterm, and at least at birthweights 1 500 to 2 499 grams, than among singletons of equivalent gestational ages and birthweights. Further, the mortality of singletons, but not of twins, declines after 37 weeks' gestation (Rydhstrom, 1985; Fabre et al., 1988). The result is that after adjustment for birthweight, for example, the excess in the foetal and perinatal mortality rates of twins is markedly decreased, while the difference in the early neonatal mortality rates of singletons and twins disappears (Fabre et al., 1988).

Replication of these relations is sought in Table 5.6 using the dichotomy of singleton and multiple pluralities. The table is observed, first, to show that multiple births are more strongly associated in the weighted sample with low birthweights (52.1 per cent, n=713) and, to a lesser extent, with preterm gestational ages (34.2 per cent, n=477) than are singleton births for which the corresponding percentages are 4.4 per cent (n=2 776) and 5.3 per cent (n=3 347) respectively.

Even at low birthweights, the ratio of singleton birth to multiple birth is more than four times, the lowest ratio of 2.6 describing the small number of term babies with birthweights 500 to 1 499 grams. With this qualification, plurality differentials by birthweight are greatest after 32 weeks and then especially at birthweights 2 500 to 3 999 grams. Thus, among multiple births, low birthweights persist even at term, while normal birthweights at any gestation are uncommon.

Nevertheless, differences in plurality by birthweight and gestational age suggest greater relative maturity of the organ systems in multiple births. Although very low birthweights bring no increased propensity for preterm birth at either plurality, singletons weighing 1 500 to 3 999 grams at birth are more likely than are multiples to be born
before 37 gestational weeks. This indicates that, at these latter birthweights, the survival chances associated with multiple births may be better than for singleton births.

This possibility is further strengthened by the pattern of relative risks associated with different pluralities. Taking the reference group as singleton term births, estimated Mantel-Haenszel birthweight-adjusted odds ratios are consistently lower for multiple than for singleton births, suspiciously even at term. After 41 weeks, the pattern is not measurable because no multiple births take place.

<table>
<thead>
<tr>
<th>Gestational Age (completed weeks)</th>
<th>&lt;28</th>
<th>28-32</th>
<th>33-36</th>
<th>37-41</th>
<th>&gt;41</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plurality</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiple</td>
<td>29</td>
<td>10</td>
<td>3</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Singleton</td>
<td>132</td>
<td>65</td>
<td>17</td>
<td>17</td>
<td>0</td>
</tr>
<tr>
<td>Birthweight (grams)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>500-1 499</td>
<td>P.D. Ctl. 0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1 500-2 499</td>
<td>P.D. Ctl. 0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2 500-3 999</td>
<td>P.D. Ctl. 0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4 000+</td>
<td>P.D. Ctl. 0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
- Reference group for Mantel-Haenszel estimated odds ratios
- Reference group for other odds ratios

** Statistically significant at the 0.01 level
*** Statistically significant at the 0.005 level
(Source: Maternal and Perinatal Collection, 1986)

The survival advantage of multiple infants is most salient between 28 and 32 weeks' gestation when multiple births are also safer than term singleton births, and more consistently at birthweights 1 500 to 2 499 grams. When the reference group is taken to be singleton term infants with normal birthweights, estimated relative risks of perinatal death...
are higher for multiple than singleton infants only at very low
birthweights before 28 weeks and at 33 to 36 weeks' gestation, and
also at birthweights 2 500 to 3 999 among 33 to 36 week old infants.

These findings are broadly consistent with the evidence, summarized
above, that at birthweights 1 500 to 2 499 grams and before term, the
multiple newborn is at lower risk of perinatal death than is the
singleton newborn of equivalent birthweight and gestational age.

Curiously, the table shows persistence of this survival advantage for
multiples at term (θ=0.63) and, after controlling for gestational age,
at birthweights 2 500 to 3 999 grams (θ_mh=0.48). However, these last
two results are clearly contaminated, presumably by small cell
numbers. Limited data for post-term births, which are only for
singleton(s), yield estimated relative risks commensurate with those at
33 to 36 weeks' gestation.

In sum, odds ratios follow the expected pattern of lower perinatal
mortality risks for multiple than for singleton babies of equivalent
gestational ages before term, especially at birthweights 1 500 to
2 499 grams. One explanation is the greater physiological maturity of
multiple newborn. Singletons at birthweights 1 500 to 3 999 grams
were found more likely to be born before term.

The corollary is the hypothesis that multiple infants are more likely
than are singleton infants of equivalent (especially, low)
birthweights to be growth retarded in utero, in which event more
advanced gestations, which may escape gestational age categorizations,
and thus increased physiological maturity, would attend these
birthweights. It may also be that twin pregnancies are identified
early and receive a very high standard of health care compared with
singleton pregnancies of similar gestations.
5.2.2 SEX OF THE FOETUS AND INFANT

Female babies are usually slightly less frequent than male babies, and experience reduced risks of perinatal death, the differential, at a cross-sectional level, being clearest for neonatal deaths but applying also to stillbirths (Australian Bureau of Statistics, 1987; and see section 2.6.1). Research priorities include elucidating the aetiological contribution of the sex of the foetus by adjusting for potential confounding factors and investigating its constituent features, for example, with regard to how the sex of the foetus influences its physiological maturity. This knowledge could supplement other obstetrical information in planning the optimal time for delivery of the high risk foetus.

Factors that may confound the relation between perinatal death and the sex of the foetus or infant are biological and sociodemographic determinants of the sex ratio at birth. These have been comprehensively reviewed by Chahnazarian (1989) who describes how biological factors affect the sex ratio at conception, apparently through physiological characteristics of both the sperm and the mother's reproductive tract, and the chances of survival of embryos in response, for example, to sex selective causes of death. Sociodemographic effects on the sex ratio at birth are small, but are higher in white than in black populations. These effects appear to be inversely associated with parity and paternal age, or, without adjustment for these variables, with the age of the mother. Increases in socioeconomic status possibly increase the likelihood of sons.

10 The sex ratio at birth typically varies between 104 and 107 boys per 100 girls. The usual figure for white populations is 105 or 106, whilst for black populations it is consistently lower, ranging from 102 to 104 (Chahnazarian, 1989).
In the following discussion, the sociodemographic effects of race, maternal age and plurality are accounted for, to some extent, when analysing the relation of perinatal outcome to the sex of the foetus or infant. Comparable adjustments are not possible for the biological variables, in part because they are less clearly defined - which prompts the analyses in sections 5.3.1 to 5.3.3.

For now, the necessary and possible statistical adjustments permit investigation of the aetiological effect of the sex of the foetus in the broader context of the joint effect on perinatal outcome of the foetus’s sex and physiological maturity. Specifically, an understanding of the survival advantage of females, despite their lower mean birthweights after about 24 weeks’ gestation, is sought by relating birthweight to gestational age.

Hall and Carr-Hill (1982) found that boys deliver spontaneously at earlier gestational ages than girls do. If this is regarded as a truism, birthweight equivalence by sex conceals lower levels of maturity in the organ or physiologic systems of boys and hence, despite overall higher birthweights, reduced abilities by boys to survive extrauterine life. To test these still uncertain ideas, Table 5.7 considers relations between the sex of the foetus, and birthweight and gestational age.

The table suggests that gestational age helps to explain a mortality differential favouring girls, the sex ratio for non-Aboriginal, singleton births being 1.10 (32 752/29 920) after weighting. There is a higher percentage of low birthweight girls (5.2 per cent, n=1 542) than low birthweight boys (3.7 per cent, n=1 207), but, including all birthweights, more similar proportions of both sexes are born preterm in the weighted sample: preterm birth characterizes 5.7 per cent
(n=1 693) of girls and 5.0 per cent (n=1 632) of boys. This suggests that proportionately more boys of low birthweight are also preterm.

In fact, although at very low birthweights there is no sex differential in preterm birth, higher proportions of preterm boys are found at birthweights 1 500 to 2 499 grams in particular, and to a lesser extent at birthweights 2 500 to 3 999 grams. At birthweights 1 500 to 2 499 grams, 68.4 per cent (n=154) of the boys, yet only 54.5 per cent (n=157) of the girls, are preterm. At normal birthweights, 6.3 per cent of the boys (n=32) and 4.3 per cent (n=20) of the girls are preterm. Both birthweight groups together account for 85.7 per cent (n=2 851) of preterm births and 88.1 per cent (n=55 229) of all births.

Table 5.7: Relation of Perinatal Death to Sex of Singleton Infants of Non-Aboriginal Women Aged 20 to 34 Years, according to Birthweight and Gestational Age, 1986

<table>
<thead>
<tr>
<th>Gestational Age (completed weeks)</th>
<th>Sex</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>500-1 499</td>
</tr>
<tr>
<td>&lt;28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>65</td>
<td>24</td>
</tr>
<tr>
<td>Female</td>
<td>61</td>
<td>31</td>
</tr>
<tr>
<td>28-32</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>30</td>
<td>79</td>
</tr>
<tr>
<td>Female</td>
<td>30</td>
<td>94</td>
</tr>
<tr>
<td>33-36</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>10</td>
<td>21</td>
</tr>
<tr>
<td>Female</td>
<td>6</td>
<td>22</td>
</tr>
<tr>
<td>37-41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Female</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>&gt;41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Female</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
** Statistically significant at the 0.01 level
 *** Statistically significant at the 0.005 level
(Source: Maternal and Perinatal Collection, 1986)

Sex ratios of births before and after term help to explain these birthweight-specific propensities for male preterm births. Among
newborn weighing 1,500 to 2,499 grams, the greater association of boys than girls with preterm delivery results from fewer non-preterm boys in the denominator of total births; the sex ratio after 36 weeks' gestation is 0.54 (71/131) compared with 0.98 (154/157) before term. Moreover, this shortage of boys at term is associated mainly with the control group.

At birthweights 2,500 to 3,999 grams, numerator effects are the more important: the sex ratios from and before 36 weeks' gestation are 1.08 (478/441) and 1.6 (32/20) respectively. The latter sex ratio is based almost completely on births at 33 to 36 gestational weeks, the surplus of boys being characteristic only of the case group. Note also that both sex ratios exceed 1.0, whereas neither did at low birthweights.

These findings of greater exposure by boys than girls to preterm birth, in proportionate terms and at normal birthweights also in absolute terms, suggest greater relative immaturity of boys' physiological systems and thus overall higher estimates of the relative risks of male perinatal death at birthweights 1,500 to 3,999 grams. Specifically, at birthweights 1,500 to 2,499 grams, fewer boys than girls are exposed to the reduced risks associated with term birth compared with preterm birth, although term births, as implied above, attend higher male than female mortality. At birthweights 2,500 to 3,999 grams, more boys than girls are exposed to the increased mortality risks associated with preterm birth (actually just 33 to 36 weeks) and, as it happens, births at term.

In this context, odds ratios for boys and girls are estimated as follows: making the reference group girls born at term, birthweight-adjusted estimated odds ratios associated with preterm birth are 5.09 for boys and 3.56 for girls (both p < 0.005 level). These estimates reflect higher odds ratios for boys than girls,
especially before 28 gestational weeks and from 33 to 36 weeks' gestation by a factor in both periods of about 1.5. Indeed while estimates decline consistently for girls until 41 weeks, the trend for boys is again interrupted (see section 5.2.1) by increased risks from 33 to 36 weeks' gestation. Term is likewise more hazardous for boys, which explains why, taking all girls as the reference group, Mantel-Haenszel odds ratios for boys are about 30 per cent higher than for girls irrespective of whether adjustment is made for gestational age.

Odds ratios unadjusted for birthweight show differences generally favouring girls, except especially at very low birthweights at term; the estimated relative risk of death for girls (0=2.82) is then 1.6 times that for boys. Survival prospects also militate against girls with birthweights 1 500 to 2 499 grams before term. This is pertinent since, as stated above, higher proportions of boys than girls are born before term at this birthweight in particular. However the most hazardous combination for boys, by a factor of approximately 3, is birthweights 1 500 to 2 499 grams at term, which could help to explain why the sex ratio still favours girls.

5.3 OBSTETRIC COMPLICATIONS

The following discussion explores the relation to perinatal death of three obstetric complications in the mother: hypertensive disorders of pregnancy, which may exist before the pregnancy (see section 2.6.2), and placenta praevia and premature rupture of the membranes which are both peculiar to the pregnancy itself. The main focus, which develops the discussion of section 5.2, is on whether these complications are more prevalent among, and also more hazardous for, boys than girls.
Briefly considered is the effect of parity on differences in perinatal mortality associated with the stated obstetric complications.

5.3.1. HYPERTENSIVE DISORDERS OF PREGNANCY

The hypertensive disorders of pregnancy are an important set of maternal conditions affecting the foetus or infant (see sections 4.4 and 4.5), being associated universally with a high rate of perinatal death and increased maternal risk (World Health Organization, 1987). Maternal hypertensive disorders promote intrauterine growth retardation or preterm birth secondary to uteroplacental insufficiency and other disturbances of circulatory adaptation, for example, to pregnancy (Lin et al., 1982; World Health Organization, 1987).

Mediated by these complications, mortality risks increase with rising diastolic pressure\textsuperscript{11} and, according to most studies,\textsuperscript{12} for example, Fedrick and Adelstein (1978) and MacGillivray (1980), especially in the presence of proteinuria.\textsuperscript{13}

These inherent pathological effects on perinatal survival are influenced in turn by the mother’s general state of health and by therapeutic but potentially toxic measures to control blood pressure and arrest seizure tendency. In fact such interventions are of uncertain benefit in disorders characterized by mild or moderate hypertension, conditions which can also be difficult to define (Lubbe, 1987; World Health Organization, 1987).

Before summarizing current evidence of predisposing factors for hypertensive disorders of pregnancy, the discussion centres on two such disorders analysed in this study using data from the

\textsuperscript{11} The diastolic blood pressure is the lower and more important pressure of blood in the arteries when the ventricles (the two lower chambers of the heart) relax and refill.

\textsuperscript{12} An exception is the study by Wildschut et al. (1983).

\textsuperscript{13} Proteinuria is defined by at least 300 mg of protein per litre in either a clean-catch, mid-stream specimen of urine, or a 24 hour collection of urine.
population-based Maternal and Perinatal Collection. The disorders are unclassified hypertension and pre-eclampsia.

The former disorder refers to what is described by the New South Wales Health Department as essential hypertension,\(^{14}\) where this indicates a blood pressure of 140/90 mmHg\(^ {15}\) on the second antenatal visit before 20 weeks' gestation. However, the term 'unclassified hypertension' is preferred here for these data, since, as the Health Department itself noted in a much earlier report, then under the title of the Health Commission of New South Wales, 'for the patient who has hypertension documented before the onset of pregnancy, or who is observed to be hypertensive early in pregnancy, it is not possible during pregnancy to exclude with precision underlying renal disease' (Health Commission of New South Wales, 1980: 24). Data collected from hospitals, therefore, most likely include women in whom the underlying pathology of pre-existing or early onset hypertension is unknown. Accordingly, the term essential hypertension is replaced with unclassified hypertension.

This problem of ascribing idiopathic hypertension\(^ {16}\) is compounded by at least two problems in differentiating between normotensive\(^ {17}\) and hypertensive patients. The first problem is that because blood pressure normally falls during the first half of pregnancy (Redman and Jefferies, 1988), hypertension can be difficult to diagnose in women that fail to attend early for antenatal care; this problem may be greatest in primigravidae who, by definition, have no pregnancy histories to refer to.

---

\(^{14}\) Essential hypertension is abnormally high blood pressure in an individual who is otherwise healthy.

\(^{15}\) The higher level of 140 mmHg (millimetres of mercury) is the systolic blood pressure, which is the maximum pressure when the ventricles contract. The lower level is the diastolic blood pressure defined in footnote 13.

\(^{16}\) That is, hypertension of unknown cause.

\(^{17}\) The blood pressure is 'normal'. During the first half of pregnancy, the diastolic blood pressure should be below 75 mmHg.
The second problem relates to what may be varying levels of adherence by hospital doctors to the Health Department’s definition of hypertension. Some doctors may not diagnose hypertension by only a diastolic blood pressure of 90 mmHg. They may use different levels of diastolic pressure, ranging perhaps from 80 mmHg to 95 mmHg, or use a rise in blood pressure to define, rather than merely to indicate, hypertension in pregnancy. Such diagnostic differences would probably lead to misreporting in terms of Health Department requirements, this being observed for other interventions such as augmentation (New South Wales Department of Health, 1989c: 37) and, by the researcher during fieldwork, for elective and emergency caesarean sections.

The latter difficulties of defining hypertension may well be greatest for the second disorder to be discussed, pre-eclampsia, as the Health Department provides no definitional guidelines for this condition. This problem is potentially important because gestational hypertension, along with significant proteinuria, is the key diagnostic criterion of pre-eclampsia. Of course, pre-eclampsia, like unclassified hypertension, is a descriptive term, and when hypertension and proteinuria arise from 20 to 36 weeks of pregnancy, the underlying pathology is usually unknown. Pre-eclampsia that is purely idiopathic tends to appear between 37 and 40 weeks of gestation (Ihle et al., 1987).

A small number of factors are known to influence the development of these, and other, hypertensive disorders of pregnancy. Risk factors

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18 This latter option is to be discouraged. The amount of proteinuria correlates with the level, and not the magnitude of the rise, of diastolic blood pressure.

19 Oedema is now often considered premonitory rather than evidential of hypertensive disorders of pregnancy. Assessment of oedema is necessarily subjective, and oedema can occur in normal pregnancy or be absent from hypertensive disorders of pregnancy (World Health Organization, 1987; Redman and Jefferies, 1988).
include adolescence and especially maternal ages 35 years and over; primigravidity; and multiple births (MacGillivray, 1983). Literature reviewed by the World Health Organization (1987) also showed familial, genetic and racial and ethnic factors to be associated with hypertensive disorders of pregnancy. The same study found inconsistent evidence that these conditions are influenced by maternal-foetal blood groups, consanguinity, the sex of the foetus, behavioural and socioeconomic factors such as diet, and the physical environment.

Of particular interest here is whether the male foetus is at increased risk of the two stated hypertensive disorders. Earlier attempts to answer this question are confined mainly to the first half of this century. Chesley (1978) reports four studies, dating to 1903, three of which suggest an association between eclampsia and an excess of male foetuses. More recent research by Toivanen and Hirvonen (1970) indicates a positive correlation between the secondary sex ratio and hypertensive disorders of pregnancy. Yet MacGillivray (1983) finds no preponderance of singleton male issue from pre-eclamptic pregnancies in North-east Scotland.

Chesley (1978) claims more girls are born to older primigravidae and multiparae. The next step may be to test for an effect of foetal sex in categories of maternal age and either parity or gravidity. Investigating only singleton births to women aged 20 to 34 years, therefore, Table 5.8 considers changes in perinatal outcome in categories of sex and gravidity. Gravidity is used instead of parity because an abortion, especially a late spontaneous abortion, may give some protection to a future pregnancy (Beck, 1985; Campbell et al., 1985). The discussion focuses on sex as the variable of main
interest, although the table also permits gravidity to assume this role.

Caution is required when interpreting this table, the results for unclassified hypertension being affected possibly by sex differentials in wastage before the perinatal period and by especially small numbers. The predominant sex in the table varies with the type of hypertensive disorder and therein with gravidity. After weighting, unclassified hypertension is associated with a sex ratio of 1.76 (51/28), boys being more common when the mother is multigravid; the sex ratio for multigravidae is 2.5. Women with pre-eclampsia have fewer male than female births, the sex ratio being 0.68 (164/242).

**Table 5.8: Relation of Perinatal Death of Low Birthweight Singleton Infants to Hypertensive Disorders of Pregnancy according to Sex of the Infant and Gravidity of Non-Aboriginal Women Aged 20 to 34 Years, 1986**

<table>
<thead>
<tr>
<th>Gravidity</th>
<th>Sex</th>
<th>Hypertensive Disorder</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>500-1 499</td>
</tr>
<tr>
<td>Primigravid</td>
<td>Male</td>
<td>Unclassified hypertension</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>Pre-eclampsia</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>Unclassified hypertension</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>Pre-eclampsia</td>
<td>1</td>
</tr>
<tr>
<td>Multigravid</td>
<td>Male</td>
<td>Unclassified hypertension</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Male</td>
<td>Pre-eclampsia</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>Unclassified hypertension</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>Pre-eclampsia</td>
<td>3</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
* Statistically significant at the 0.025 level
(Source. Maternal and Perinatal Collection, 1986)
The preponderance of daughters is most conspicuous among primigravidae (64.9 per cent, n=148), who account for 56.2 per cent of all births, and at birthweights 1 500 to 2 499 grams. The latter infants, who constitute four-fifths (n=323) of the low birthweight babies in the weighted sample, yield a sex ratio that is 15 percentage points lower than that of 0.80 at very low birthweights.

Higher male perinatal mortality, as measured by Mantel-Haenszel birthweight-adjusted estimates, is associated with both obstetric complications. In absolute terms, the estimated relative risk of losing a son is greater for women with unclassified hypertension than for women with pre-eclampsia. Multigravidae, especially with unclassified hypertension, more often experience the perinatal death of a boy than do women having their first pregnancies. This last result differs from findings by MacGillivray (1961, 1983) that hypertensive disorders are typically less severe after the first pregnancy, when they are also less common.

However, unclassified hypertension, but not pre-eclampsia, is more hazardous for girls whose mothers are primigravidae than girls born to multigravidae. Also, only when the mothers of daughters are primigravidae is unclassified hypertension estimated to produce higher perinatal mortality for girls, 10 times so, than is pre-eclampsia. The largest sex differential in perinatal mortality risks is observed for primigravid women with pre-eclampsia. For these women the Mantel-Haenszel odds ratio is 3.8 times higher for boys ($\theta_{mh}=0.73$) than for girls ($\theta_{mh}=0.19$), yet both risk estimates are seen to be low.

The reference group for unadjusted odds ratios is girls weighing 1 500 to 2 499 grams with multigravid, pre-eclamptic mothers. Relative to this standard, the risks of perinatal death attending each complication tend to be higher for boys than girls and for the
offspring of multigravidae and women with unclassified hypertension. The most stable relative risk estimates are for pre-eclamptic women. In this group, the most hazardous combination is a very low birthweight boy born to a multigravid woman (θ=2.00), while least at risk of perinatal death are the daughters of primigravidae, especially at very low birthweights (θ=0.17); 96 per cent (n=27) of these babies survive the perinatal period.

If the gravidity stratification is collapsed, and the reference group is taken to comprise the daughters of pre-eclamptic mothers, most at risk after adjustment for birthweight are boys born to women with unclassified hypertension (θ_{mh}=3.52) or, then, pre-eclampsia (θ_{mh}=2.24). Girls born to women with unclassified hypertension record the Mantel-Haenszel odds ratio of 2.00. Further adjustment for gravidity reduces these estimates to 2.39, 1.96 and 1.59 respectively.

Finally, gravidity is made the study factor of interest, and pre-eclamptic women with more than one pregnancy become the reference group. Adjustment for birthweight and sex yields estimated relative risks of perinatal death for a first pregnancy of 0.34 when the mother has pre-eclampsia, and 0.97 when she has unclassified hypertension. When this latter complication afflicts multigravidae with unclassified hypertension, θ_{mh}=1.23.

Tentative conclusions from these analyses reject the hypothesis that more boys than girls are born to women with pre-eclampsia. Weak support for the hypothesis derives from sparse data for unclassified hypertension, which emerges as the more hazardous condition. As expected, boys record higher perinatal mortality than girls, but contrary to existing knowledge, especially when mothers are multigravidae.
5.3.2 **PLACENTA PRAEVIA**

As with hypertensive disorders of pregnancy, definitional problems confound comparative research into aetiologies for placental abruption. Some reports, such as Jones (1982), do not distinguish between different types of abruption, pooling them instead under one label: antepartum haemorrhage. Moreover, cases of placenta praevia, which are considered exclusively here, may themselves be of different types. As noted by Fribourg (1982: 850), 'some are total, some are partial, some are marginal and some low-lying placetas are classified as placenta praevia'. Distinctions between these types are seldom made or specified even though they have quite different management needs and outcomes. The same problem characterizes data from the Maternal and Perinatal Collection on which the analysis below is made.

The aetiology of low placental implantation is uncertain, although some risk factors have been identified. One is previous caesarean section (Singh et al., 1981, Clark et al., 1985; McShane et al., 1985), yet increased rates of this operation have not been cited as raising concurrent levels of placenta praevia. The mechanism of the disorder is presumably secondary to scarring, this also accounting for correlations between placenta praevia and uterine curettage (Rose and Chapman, 1986). Of consequent interest is an association between this disorder and induced abortion in some studies (e.g. Barrett et al., 1981) but not others (for example, Grimes and Techman, 1984; Rose and Chapman, 1986). This may reflect a preference for suction over curettage in performing early elective terminations. Other correlates

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20 While recognition of commonalities is useful, evidence pertaining to different placental disorders may not be generalizable as suspected risk factors.

21 One theory, referred to by Rose and Chapman (1986), proposes that damage to the endometrium or myometrium could alter the site of implantation.
of *placenta praevia* are increasing maternal age, multiparity and multiple pregnancy (Lo Bue, 1981, Chamberlain, 1984b).

As with hypertensive disorders of pregnancy, evidence is mixed for an effect on *placenta praevia* attributable to the sex of the foetus. MacGillivray et al. (1986) report a positive association between the secondary sex ratio and *placenta praevia*. They conclude that fertilization late in the menstrual cycle predisposes to this disorder by increasing the ratio of males to females at conception. However, Mills et al. (1987) could not replicate the association between *placenta praevia* and the sex ratio at birth, which also, by inference, provides evidence against the MacGillivray et al. (1986) hypothesis. This uncertainty prompts the analysis in Table 5.9 of the aetiological influence of the sex of the foetus in women with *placenta praevia* at different parities.

### Table 5.9: Relation of Perinatal Death of Low Birthweight Singleton Infants to *Placenta Praevia* according to Sex of the Infant and Parity of Non-Aboriginal Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Parity</th>
<th>Sex</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-1 499</td>
<td>1 500-2 499</td>
</tr>
<tr>
<td></td>
<td>P.D. Ctl. θ</td>
<td>P.D. Ctl. θ</td>
</tr>
<tr>
<td>Multipara</td>
<td>Male</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>8</td>
</tr>
<tr>
<td>Primipara</td>
<td>Male</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Female&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control  
<sup>a</sup> Reference group for Mantel-Haenszel estimated odds ratios  
<sup>b</sup> Reference group for other odds ratios  
(Source. Maternal and Perinatal Collection, 1986)
Based on very small numbers, the table suggests that rather than a surplus of boys, placenta praevia is associated with a sex ratio of 0.91 (61/67) that is considerably lower than that of 1.10 reported in Table 5.7. However, the preponderance of girls occurs mainly among multiparous women and especially at very low birthweights when the sex ratio is 0.55 (16/29).

Taking girls born to primiparae as the reference group and adjusting for birthweight, boys have lower estimated relative risks of perinatal death from placenta praevia at both parities, especially when mothers are multiparae ($\theta_{mh}=0.86$). Among multiparous women, the relative odds of a perinatal death are 1.67 times higher for girls ($\theta_{mh}=1.44$) than boys.

Crude odds ratios reveal that in women with placenta praevia, the risk of perinatal death is greater for girls than for boys, except when primiparae deliver babies with very low birthweights; the estimated relative risk of perinatal death is then 1.14 times higher for boys ($\theta=1.00$) than for girls ($\theta=0.88$), the reference group being first-born girls with birthweights 1,500 to 2,499 grams. The relative risk of perinatal death is otherwise estimated to be consistently higher for girls than boys at birthweights 1,500 to 2,499 grams and when the mother is multiparous. When these two characteristics coincide, the odds ratio for girls ($\theta=1.25$) is estimated to be 2.5 times higher than for boys ($\theta=0.50$). Only among girls born to primiparae does an increase in birthweight not reduce the risk of perinatal death, this attenuation in risk being especially great for boys.

Adjustment for parity and birthweight yields a Mantel-Haenszel estimate for boys, relative to girls, of 0.73. Collapsing the sex stratification produces $\theta_{mh}=1.32$ for the issue of multiparae which is little different to the estimated odds ratio for these infants after
adjustment for sex and birthweight ($\theta_{mh}=1.24$). The main finding is not to confirm the hypothesis of a preponderance of boys born to mothers with placenta praevia: more girls than boys are born to these women and experience higher perinatal mortality, but this result is not statistically significant at the 0.025 level.

5.3.3. PREMATURE RUPTURE OF THE MEMBRANES

Reported incidences of premature rupture of the membranes range from 2 to 40 per cent (Bada and Alojipan, 1977; Vintzileos et al., 1985) owing to inadequate definition of the condition (Evaldson et al., 1980), or at least to carelessness in the reporting of inquiries, including the denominator in rates. These considerations are explored.

Premature rupture of the membranes is commonly defined as spontaneous rupture of the chorioamniotic membranes before the onset of labour. Yet investigators may focus on all spontaneous membrane ruptures or only on those occurring before or after term. The causes and potential maternal and perinatal complications of membrane ruptures may be different at various gestational ages.

The condition is especially hazardous before term, producing the dual problems of avoiding infection and minimizing the risks of prematurity. Since prematurity is the more important cause of perinatal death (Eggers et al., 1979), expectant management is most common (Fisk, 1988). However, implicit in this teleological prescription is that even conservative regimes are often ineffective. When membrane rupture leads to preterm labour, tocolysis appears able to prolong pregnancy by only one or two days (Garite, 1985).

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22 The aim is to prolong pregnancy in order to deliver a baby that can survive normally. When very early gestational ages promise little hope of neonatal survival, maternal needs are pre-eminent; intervention for foetal indications is not at issue.
Perinatal death is thus especially likely to result from early spontaneous rupture of the membranes mediated by preterm labour and delivery (Flood and Naeye, 1984). With membrane rupture at or very near term, the main risks — infection to the mother and baby — are not usually serious. Yet if labour does not start spontaneously within a short time and if, as particularly in nulliparous women, the cervix is unripe, induction may fail causing foetal distress and leading to an increased rate of caesarean section (Rydstrom et al., 1986).

Various issues associated with gestational duration may thus be addressed in a study of premature rupture of the membranes. Since the most worrisome outcome is usually spontaneous membrane rupture before term, Drife (1982) advocates that premature rupture of the membranes be renamed preterm rupture of the membranes. The present writer suggests using the root term, spontaneous rupture of the membranes, indicated by the logical fallacy of premature rupture of the membranes at term, to which the qualifier of either preterm or premature can be appended when the membranes rupture before term. Without this root term, it is important to implement Drife’s proposal.

The problem in sum, therefore, is that attaching different meanings to the same term, without this being clearly stated, can produce complications. These are exemplified by discrepancies in the measurement of incidence or prevalence: the denominator may include all pregnancies or only preterm births. If research is to be meaningful and comparison possible, standard definitions and concepts must be produced and uniformly applied.

Of course, timing differences in the manifestation of illness affect the study not only of membrane ruptures but of other complications including those discussed in sections 5.3.1 and 5.3.2. Ideally, gestational age effects would have been accommodated in these sections.
(see section 5.1). At least this is done in Table 5.10 where intrinsically it is even more important, for preterm rupture of the membranes. For the present, discussion focuses on the broader term used in the literature.

Isolated cases of premature rupture of the membranes result from cervical incompetence, hydramnios or trauma (Naeye and Peters, 1980). More often, spontaneous rupture of the membranes before term is unexplained. Theories about its genesis concentrate on physical and chemical characteristics of the membranes.

Skinner et al. (1981) reported an abnormally low collagen content in membranes rupturing spontaneously before term. Yet mixed evidence for this mechanism suggests localized rather than generalized weakness in the membranes (Naeye, 1982). Damage could result from enzymatic depolymerization of collagen fibres, which may be secondary to cervicovaginal and chorioamniotic infection (Naeye and Peters, 1980) and to 'collagenase-like' enzymes in ejaculate (Naeye, 1982) in the presence of female orgasm (Lavery and Miller, 1981). Naeye and Peters (1980) found infections more severe when coitus occurred in the month before delivery.

A positive association between coitus and premature rupture of the membranes has not, however, been consistently shown. One reason may be the different time frames employed. Catalysts for membrane rupture could be the occurrence of coitus between the last clinic visit and delivery (Flood and Naeye, 1984) or the concurrence of chorioamnionitis (Naeye, 1982).

---

23 Collagen, a protein, is the main constituent of white fibrous connective tissue. Although relatively inelastic, it has a high tensile strength.

24 This is the initiated or accelerated breakdown of polymers, substances formed by the linkage of many smaller molecules (monomers).
Evaldson et al. (1980) found heavy smokers over-represented among cases of spontaneous membrane rupture before 36 weeks' gestation. Yet in the study by Naeye (1982), cigarette smoking contributed to premature rupture of the membranes only in pregnancies that continued to term. Additional, possibly predisposing factors include, first, the prior occurrence of premature rupture of foetal membranes and of genital or cervical surgery, and secondly, advanced maternal age and increasing parity (Evaldson et al., 1980; Flood and Naeye, 1982). The influence of parity requires elucidation. It may reflect damage to the cervix, or the influence of age which could progressively weaken the foetal membranes.

A suspected risk factor, rarely cited, is the sex of the foetus. As noted in 5.2.2, Hall and Carr-Hill (1982) report that in Aberdeen, from 1973 to 1979, boys delivered spontaneously at earlier gestational ages than girls did. This observation was confirmed by studies in Cape Town (MacGillivray and Davey, 1985) and Japan (Seki and Kato, 1987) which also found premature rupture of the membranes to occur most often in pregnancies with male foetuses. It was suggested by MacGillivray and Davey (1985) that membrane rupture results from infection of the amniotic fluid and membranes to which pregnancies with male foetuses are predisposed because of boys' slower immunologic development. The study by Seki and Kato, but not by MacGillivray and Davey, also found a preponderance of boys in preterm labour beginning with contractions. Unfortunately, it is not possible to look at many biological influences here, even though they are relevant.

With this qualification, Table 5.10 does not support the hypothesis of a preponderance of boys born to women whose membranes spontaneously rupture before term. To the contrary, there are slightly more girls than boys, yielding a sex ratio of 0.87 (249/287), which is lower than
the sex ratio of 0.96 (1 632/1 693) for all singleton preterm births to non-Aboriginal women (see Table 5.7). However, this low sex ratio is in fact characteristic only of the newborn of multiparous women, 0.79, and of infants with birthweights 1 500 to 2 999 grams (0.85). Only at very low birthweights do primiparae deliver more boys (52.8 per cent, n=28) than girls. At birthweights 1 500 to 2 499 grams, there is no difference by parity in the ratio of boys to girls.

Table 5.10: Relation of Perinatal Death of Low Birthweight Singleton Infants to Preterm Rupture of the Membranes according to Sex of the Infant and Parity of Non-Aboriginal Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Parity</th>
<th>Sex</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>500-1 499</td>
</tr>
<tr>
<td></td>
<td></td>
<td>P.D.</td>
</tr>
<tr>
<td>Multipara</td>
<td>Male</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>19</td>
</tr>
<tr>
<td>Primipara</td>
<td>Malea</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>Female</td>
<td>8</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control

a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios

** Statistically significant at the 0.05 level
(Source. 1986 Maternal and Perinatal Collection)

These distributions are the result, of course, of higher estimated relative risks of death for girls than boys, particularly at birthweights less than 1 500 grams and when the mother is multiparous. The coincidence of both characteristics yields the highest odds ratio of θ=4.83 for girls, taking the reference group as 1 500 to 2 499 grams boys25 born to primiparae. Except when primiparae deliver at birthweights 1 500 to 2 499 grams, boys record lower estimated odds ratios than do girls, this being most salient for 1 500 to 2 499 gram

25 Boys, and not girls, born to primiparae compose the reference category because of a sampling zero in the latter group.
boys born to multiparae: \( \theta = 0.28 \), which is four times lower than the comparable odds ratio for girls. Moreover, this lowest odds ratio is more than eight times smaller than the odds ratio for very low birthweight boys born to multiparae.

The highest Mantel-Haenszel odds ratio, relative to boys born to primiparae, is thus \( \theta_{mh}=2.10 \) for girls with multiparous mothers. Collapsing over the parity stratification yields an estimate of \( \theta_{mh}=1.55 \) for girls compared to boys, whilst adjustment for parity \( (\theta_{mh}=1.52) \) has little effect on this estimated relative risk of death for girls. Likewise, the increased risk for issue of multiparae, without adjustment for sex \( (\theta_{mh}=1.66) \) changes only to \( \theta_{mh}=1.62 \) once adjustment is made.

5.4 CONCLUSION

This chapter began by assessing the aetiological contributions of gestational age and the appropriateness of birthweight for gestational age to low birthweight babies in particular. Preterm gestational age was more prevalent and hazardous than in utero growth retardation in low birthweight babies. However, newborn small for gestational age experienced high risks, especially at very low birthweights and before 28 weeks' gestation. Between 28 and 36 weeks, these risks were lower than in the fewer babies large for dates. Preterm birth was often associated with growth retardation, increasingly so at advancing gestations, and to a greater extent newborn small for gestational age were preterm. Both complications were largely confined to low birthweight infants, among whom these complications were more hazardous when they occurred together than, after adjustment for birthweight, when either complication occurred alone. However, the
added risk of growth retardation in the preterm baby was apparent only at very low birthweights.

Neonatal ponderal indices were found to have low sensitivity, a problem that less seriously affected Apgar scores especially at one minute. Neonatal mortality risks were increased fivefold by low Apgar scores (0 to 2) at one minute compared with five minutes. However, higher scores (3 to 10) were better able to predict neonatal death at five minutes than at one minute.

The discussion of obstetrical risk factors focused, first, on the plurality and sex of infants. At low birthweights, multiple births were found to be safer than singleton births at equivalent gestational durations before term - especially from 28 to 36 weeks. As for the sex of newborn, girls were more likely than boys to be of low birthweight, but not to be preterm. Specifically, higher proportions of boys weighing 1 500 to 2 499 grams, yet also of normal birthweights, were preterm. This helped to account for higher estimates of boys' relative risks of perinatal death by affecting the exposure of each sex to the mortality schedules associated with different gestational age and birthweight combinations. An interesting finding was that at very low birthweights, boys did not seem to be more prone to preterm birth.

Both plurality and sex, but particularly sex, provided foci for exploring three sets of obstetrical complications in the mother: hypertensive disorders of pregnancy, placenta praevia and preterm rupture of the membranes. Of most interest was whether a high sex ratio contributed to increased incidences of these complications, the aetiological roles of either parity or gravidity being given secondary importance. Analyses suggested that rather than a preponderance of boys, as documented by a minority of studies, more girls attended
these maternal conditions, excluding, based on very small numbers, unclassified hypertension. Girls also experienced higher perinatal mortality when the maternal complication was *placenta praevia* or preterm membrane rupture.
CHAPTER 6
PROXIMATE DETERMINANTS OF PERINATAL DEATH:
HEALTH CARE FACTORS

6.0 INTRODUCTION

This chapter estimates the contribution to perinatal death of two sets of health care factors. The first set relates geographically to different places of birth and to the antenatal and postnatal movements that define, in particular, the effectiveness of perinatal referral in New South Wales. The second set of factors focuses more temporally on the different amounts and types of health care received during the pregnancy and confinement, comparing mainly the safety of caesarean section with alternative methods of managing the abnormal labour and delivery. Both discussions consider for the most part neonatal rather than all perinatal outcomes.

6.1 PUBLIC HOSPITAL BIRTH

The safety of hospital confinements is investigated with respect to the differential risks associated with birth at public hospitals in New South Wales. These hospitals operate within a regionalized perinatal health care system that implies, first, size and role delineation, or relative areas of responsibility, in the amount and level (or complexity) of maternity and neonatal care provided, and secondly, a coordinated and cooperative networking of hospitals that matches patients with hospitals appropriate to their needs.

In New South Wales, guidelines for assigning hospitals to levels of obstetric and neonatal care have, until recently, been based on
numbers of beds or births per annum.¹ It was the 1989 Task Force Review of maternity services in New South Wales that pointed out that these criteria are, in themselves, inadequate. The classification of each unit's level of care should instead be based on the availability of specialist staff and the role of the unit in servicing a discrete geographic region (New South Wales Department of Health, 1989a).

Accordingly, the Task Force produced revised guidelines for delineating the levels of obstetric and neonatal care provided by individual obstetric and special units. Six levels of obstetric care and four of neonatal care were described, but the positions of individual hospitals on these scales have yet to be established. Application of the new schedules, that of neonatal care being the more pertinent to this research, is thus confounded.

However, individual hospitals have been designated levels of a classification of neonatal care, produced by the Department of Neonatology of Sydney's Royal Alexandra Hospital for Children, that retains the holistic focus of the Task Force Guidelines. The resulting statistics, which were kindly made available to the writer, identify five levels of neonatal care and assign these to individual hospitals in three ways.

The five levels are described in ascending order of the complexity of care provided. Level 0 hospitals have no booked maternity admissions and any births are 'emergencies'. These units, which might once have had obstetric services, can provide basic care if needed. Level 1 hospitals service uncomplicated deliveries and normal neonates, and management aims at stabilization when any transfer of the expectant mother or newborn becomes necessary; hospitals coded 1- are deficient

¹ These guidelines are found in the 1978 and 1983 Maternal and Perinatal Committee reports, the Hosplan Guidelines (1984) and the Health 2000 Hospital Role Delineation Guidelines
in some area. Level 2 hospitals are subdivided into two groups. Level 2a hospitals provide level 1 services while managing low-risk pregnancies and healthy infants over 35 weeks gestation. Level 2b hospitals provide this care and attend to many neonatal problems. A special care nursery exists for sick neonates and there is a separate observation nursery for infants who cannot be looked after in rooming-in situations. These hospitals can manage neonates in need of oxygen therapy, cardio-respiratory monitoring, transcutaneous oxygen monitoring and parenteral fluid therapy. Short-term ventilatory support can be given before any transfer. Level 3 hospitals usually provide these functions, and have an intensive care nursery that can tend the most critically ill. Infants admitted to a level 3 unit are those requiring medium- to long-term assisted ventilation, prolonged parenteral nutrition and sometimes neonatal surgery.

Using this classification, each hospital received three measures of its level of neonatal care in a survey undertaken in 1988-1989. The first measure was how each hospital's staff perceived and reported its own ability to provide neonatal care. The second measure was an objective assessment, made by the researcher who led the study, according to set criteria of equipment and staffing. The third measure was a functional one based on how the newborn emergency transport service perceived each unit's provision of neonatal care at the time of retrieval. The measure was the result of first-hand

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2 The monitoring involves piercing the skin.
3 The fluid is administered intravenously.
4 The two methods of assisted ventilation used are continuous distending airway pressure, usually continuous positive airway pressure administered via a face mask, nasal prongs or an endotracheal tube; and positive pressure ventilation by means of a mechanical ventilator (Tudehope and Thearle, 1988).
5 The two Children's Hospitals, out of the seven Level 3 neonatal units in New South Wales, do not provide maternity care, or level 1 or level 2 neonatal services, but these hospitals perform neonatal surgery, which is not provided by the other Level 3 hospitals.
evaluations of whether or not assistance was being given, or being
given effectively, at the time of transfer.

As an assessment of the actual abilities of hospitals to care for
infants, this last measure is the best of the three. It yields
Figures 6.0 and 6.1 which show neonatal care to be localized in
eastern New South Wales, and especially in Sydney which, for example,
contains six of the seven hospitals providing level 3 neonatal care.
However, the maps do exaggerate the geographic bias at low levels of
care because hospitals performing fewer than 200 deliveries per annum
are the most likely not to have been functionally assessed. Rather
than lose these hospitals from Table 6.0, the greater part of which is
also based on a functional role delineation, their level of care is
determined by the second, objective assessment based on hospital
facilities, and is presented at the base of the table.

The results of the table are first described. Estimates are made of
the 'exposures' of live born infants of different birthweights to the
abilities of hospitals to provide neonatal care and to associated
risks of neonatal mortality. Then, two issues are explored: the
safety of small maternity units and the appropriateness of
cross-referral patterns between units with different roles in the
regionalized perinatal health care system. The caveat is noted that
while the classification of hospitals is based on 1988-89 data, the
information on perinatal outcome is derived from the 1986 Maternal and
Perinatal Collection.

Births at level 0 hospitals are, by definition, non-routine. Also
comparatively rare, according to Table 6.0, are births at hospitals
providing level 1 care, whether this level is defined functionally or
in terms of facilities. Only 2.4 per cent (n=11) of very low
birthweight infants, but as many as 9.5 per cent (n=53) of 1 500 to
Figure 6.0
Levels of Neonatal Care Provided by Hospitals in New South Wales, 1988-89

Levels of neonatal care assessed by the Neonatal Emergency Transport Service
(Source: Royal Alexandra Hospital for Children, Unpublished Data, 1989)

Figure 6.1
Levels of Neonatal Care Provided by Hospitals in the Sydney Metropolitan Health Regions, 1988-89

Levels of neonatal care assessed by the Neonatal Emergency Transport Service
(Source: Royal Alexandra Hospital for Children, Unpublished Data, 1989)
2.499 gram infants are shown to be born at either type of level 1 unit. The latter proportion approaches the 12.5 per cent (n=100) of normal birthweight babies born at level 1 units. Level 1 units are estimated to account for 8.2 per cent (n=4903) of births in the weighted sample.

The birthweight distributions characterizing the hospitals providing level 2a and level 2b neonatal care are similar. Very low birthweight infants appear slightly more prevalent at level 2b hospitals, the reverse holding for other 1500 to 2499 gram babies. Yet the important point is that a little less than one-third of all very low birthweight babies are born at level 2 hospitals. Since level 3 care is necessary for these babies (New South Wales Department of Health, 1989d), many of them should presumably have been transferred, when possible, in utero. Including all the hospitals for which levels of care are functionally defined, level 2 units account for 55.6 per cent (n=302) and 66.5 per cent (n=497) of infants weighing 1500 to 2499 grams.

### Table 6.0: Relation of Neonatal Death to Level of Neonatal Care at the Public Hospital of Birth of Infants of Women Aged 20 to 34 Years according to Birthweight, 1986

<table>
<thead>
<tr>
<th>Level of Neonatal Care</th>
<th>Birthweight (grams)</th>
<th>P.D. Ctl.</th>
<th>0</th>
<th>P.D. Ctl.</th>
<th>0</th>
<th>P.D. Ctl.</th>
<th>0</th>
<th>P.D. Ctl.</th>
<th>0</th>
<th>P.D. Ctl.</th>
<th>0</th>
<th>θ_{mh}</th>
</tr>
</thead>
<tbody>
<tr>
<td>500-1499</td>
<td></td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
<td>0</td>
<td>17</td>
<td>0</td>
<td>1</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1500-2499</td>
<td></td>
<td>3</td>
<td>4</td>
<td>5.58*</td>
<td>3</td>
<td>37</td>
<td>0.60</td>
<td>8</td>
<td>64</td>
<td>0.93</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>2500-3999</td>
<td></td>
<td>15</td>
<td>44</td>
<td>2.54*</td>
<td>6</td>
<td>156</td>
<td>0.29**</td>
<td>15</td>
<td>241</td>
<td>0.46*</td>
<td>3</td>
<td>22</td>
</tr>
<tr>
<td>4000+</td>
<td></td>
<td>21</td>
<td>54</td>
<td>2.89***</td>
<td>8</td>
<td>132</td>
<td>0.45</td>
<td>16</td>
<td>225</td>
<td>0.53</td>
<td>0</td>
<td>23</td>
</tr>
<tr>
<td>Hospitales performing fewer than 200 deliveries per annum</td>
<td>68</td>
<td>242</td>
<td>2.09***</td>
<td>18</td>
<td>182</td>
<td>0.74</td>
<td>25</td>
<td>186</td>
<td>1.00b</td>
<td>0</td>
<td>9</td>
<td>1.00c</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control

- Level as perceived by Neonatal Emergency Transport Service (functional assessment).
- Reference group for individual odds ratios
- Reference group for Mantel-Haenszel estimated odds ratios
- Level as rated in terms of facilities (objective assessment)

* *** Statistically significant at the 0.005 level

(Source. Maternal and Perinatal Collection, 1986)
grams and 2,500 to 3,999 grams at birth. The salient characteristic of level 3 hospitals is their estimated delivery of exactly half (n=510) of all low birthweight babies including 68.1 per cent (n=310) and just 36.0 per cent (n=200) of babies at birthweights 500 to 1,499 and 1,500 to 2,499 grams respectively. After weighting, almost one-quarter (n=14,353) of all births occurring to women aged 20 to 34 years are estimated to take place in level 3 hospitals.

Using only functional levels of care, odds ratios are elevated at level 1 hospitals compared with level 2 hospitals, but including also the hospitals classified level 1 on objective criteria, the risk differential disappears except at very low birthweights. The implication is that level 1 hospitals are comparatively safe except for the very smallest babies, which were noted to be comparatively few, and except perhaps when level 1 units perform 200 or more deliveries per year,6 which describes 95 per cent (n=21) of the hospitals classified level 1 on functional criteria. The safety of 1,500 to 2,499 gram babies serves as a reminder that birthweight is only one measure of risk and may not in itself justify transfer, except in the circumstance of very low birthweights.

After adjustment for birthweight, infants born at level 2 hospitals, whether level 2a or level 2b, have lower estimated relative odds of perinatal death than do infants belonging to the reference group of level 3 hospital births. The Mantel-Haenszel estimates for levels 2a and 2b hospitals are $\theta_{mh}=0.68$ and 0.83 respectively, yielding an apparent increase in risk from level 2a to level 3 hospitals. This is to be expected since the risk statuses of maternal and newborn populations rise directly with the level of care received.

6 Of these hospitals, three-quarters (n=16) performed 200 to 399 deliveries per annum.
However, only at birthweights over 1 499 grams do level 2 hospitals yield lower estimated relative odds of death than do level 3 hospitals. At very low birthweights, the relative risks of perinatal death are higher at level 2a ($\Theta=2.54$) and especially at level 2b ($\Theta=2.89$) units than at level 3 hospitals ($\Theta=2.09$), the reference group being normal birthweight newborn at level 3 hospitals. This again implies that birthweight, excluding very low birthweights, is only one measure of risk and that, overall, infants at level 2 hospitals are of lower risk than those of equivalent birthweights at level 3 hospitals. Added support is given to the belief that very low birthweight infants should be transferred in utero to level 3 units.

These results present two issues deserving of discussion: the safety of level 1 units, and, to be considered more thoroughly in section 6.2, the difficulties of antenatal transfers of, for example, very low birthweight neonates from level 2 units when these babies may have better survival chances in level 3 units. These issues are discussed in turn.

### 6.1.1 SMALL MATERNITY UNITS

The overall safety of level 1 units, as indicated by their management of only low-risk deliveries, implies effective risk assessment and compliance with strict referral protocols. This conclusion was reached also, for example, by Lumley et al. (1987) in their study of small maternity units in Victoria, and by the 1989 Task Force review in New South Wales (New South Wales Department of Health, 1989a). The Task Force rejected the conclusion of the 1983 Maternal and Perinatal Committee (New South Wales Department of Health, 1983) that non-viable units were those performing fewer than 80 deliveries per annum,\(^7\)

\(^7\) That is, except in geographically isolated areas or in the presence of unfavourable environmental factors.
arguing instead that the 'number of births should not be the sole
criterion for determining unit viability unless contiguous units are
underutilised in which case amalgamation may need to be considered' (New South Wales Department of Health, 1989a: 97).

The 1989 report recommended that the viability of small, country
maternity units should instead be determined by staff availability,
the degree of geographic isolation, and the changing demand of the
catchment population. The report also cited evidence that small units
are not 'obstetrically undesirable and uneconomic to maintain' (New
South Wales Department of Health, 1983: 8), that they are popular with
families and are an integral part of the fabric of rural communities,
and that their closure leads to cost escalations for consumers plus
the difficulties and hazards of increased travel.

However, the 1989 report indicated no support for reopening any of the
35 (47.9 per cent, n=73) small, country maternity units closed between
1983 and 1987 following the recommendation of the 1983 report to
effect that action,8 despite the 1989 report's contesting of the
criteria on which that recommendation was made. One reason for this
unwillingness to undo the past is presumably the paucity of
appropriately qualified staff prepared to work in small hospitals.
Yet the cost of providing financial incentives to work in rural areas
could be recouped by the lower cost per confinement of birth in a
small hospital than in a large hospital (New South Wales Department of
Health, 1989a).

A second reason could be difficulties associated with country
transport to a referral hospital. This problem is explored in the
context of the second issue to be discussed, which centres on the

8 Although, owing to lack of patronage, small maternity units had in fact
been slowly closing for more than a decade.
appropriateness of referral patterns especially for very low birthweight babies from level 2 to level 3 hospitals.

6.2 ANTENATAL TRANSFER

The analysis in section 6.1 estimated that in 1986 almost one-third of very low birthweight babies were born at level 2 hospitals, and that at least some of these infants might have had improved survival chances if they had been born at level 3 units. The problem was not overlooked by the 1989 Task Force (New South Wales Department of Health, 1989a) which noted the low rate of in utero referral in New South Wales compared with other States, and estimated on six months data for 1986 that 90 per cent (n=154) of infants weighing 750 to 1500 grams and delivered in units without level 3 neonatal care would have required such care. The problem invites examination of the working success of mechanisms for antenatal cross-referral in particular between level 2 and level 3 units.

There are several constraints to antenatal transfer. First, the risk factor(s) that necessitate transfer must be identified, and identified early enough for transfer to be undertaken. On the one hand, the risk factors themselves are well-known, and the infant at risk of respiratory failure can usually be identified using modern diagnostic procedures such as ultrasound.9 However, particularly in patients who default antenatal care or who attend late, there may be limited opportunities during pregnancy to observe these risk factors and to assess the need for intensive neonatal care.

9 In fact, as discussed later, these tools tend to overpredict the need for ventilatory support.
The gestational duration may be uncertain, and preterm labour can be difficult to diagnose\textsuperscript{10} or be diagnosed late. According to the 1989 Task Force, up to 40 per cent of babies at risk cannot be detected before labour begins (New South Department of Health, 1989c). Even then, active labour may not be able to be arrested, even though this is essential for transport to be undertaken; attempts to inhibit labour pharmacologically (usually with a salbutamol infusion) may themselves be ineffective or such treatment may be contraindicated by factors including maternal shock.

There may also be insufficient time to transfer patients, or to transfer them directly to the appropriate level of care, over large physical distances or from areas made inaccessible by poor roads and weather conditions.\textsuperscript{11} The impact of geographic obstacles can be seen from Table 6.1, which relates neonatal outcomes for very low birthweight newborn at level 3 hospitals to movement by pregnant women living in different health regions. Movement is defined when a woman delivers her baby in a level 3 hospital but resides in a local government area that does not contain a level 3 hospital or, if the area has no maternity hospital, is closer to a lower level unit. Non-movement is defined when a woman lives in a level 3 designated area. Although movement thus encompasses movements by choice as well as antenatal transfers, the analysis should be relevant to such transfers.

Residency in these regions implies different degrees of physical accessibility to level 3 neonatal care. Few women move from remote regions such as the Orana and Far West regions (2.0 per cent, n=5) and movement from these regions is also comparatively safe (\(\theta=0.71\))

\textsuperscript{10} Between 30 and 50 per cent of women who appear to be in labour before term are not in labour at all (New South Wales Department of Health, 1989d).

\textsuperscript{11} Unfortunately, there are no data enumerating the women not transferred for these reasons.
relative to non-movement from the southern metropolitan health region.
Conversely, most movement (77.2 per cent, n=190) originates in the
metropolitan health regions, particularly Sydney’s western
metropolitan health region,\(^{12}\) containing level 3 units. In these
regions, movement is safer than non-movement except for residents of
the northern metropolitan health region. It is unclear whether
residence in metropolitan regions improves perinatal outcomes compared
with residence in country regions.

Table 6.1: Relation of Neonatal Death of Very Low Birthweight
Infants in a Level 3 Hospital to Maternal Health Region of
Usual Residence according to Antenatal Movement from this Region,
1986

<table>
<thead>
<tr>
<th>Health Region of Usual Residence</th>
<th>Antenatal Movement from:</th>
<th>No Antenatal movement from:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P.D. Ctl. θ</td>
<td>P.D. Ctl. θ</td>
</tr>
<tr>
<td>Metropolitan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Southern Metropolitan</td>
<td>4 13 0.88</td>
<td>7 20 1.00b</td>
</tr>
<tr>
<td>Northern Metropolitan</td>
<td>13 26 1.43</td>
<td>1 16 0.18</td>
</tr>
<tr>
<td>Western Metropolitan</td>
<td>23 82 0.80</td>
<td>2 6 0.95</td>
</tr>
<tr>
<td>Country</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Central Western</td>
<td>3 8 1.07</td>
<td></td>
</tr>
<tr>
<td>Far West</td>
<td>0 0 -</td>
<td></td>
</tr>
<tr>
<td>Hunter</td>
<td>2 27 0.21</td>
<td>2 8 0.71</td>
</tr>
<tr>
<td>Illawarra</td>
<td>4 6 1.90</td>
<td></td>
</tr>
<tr>
<td>New England</td>
<td>0 7 -</td>
<td></td>
</tr>
<tr>
<td>North Coast</td>
<td>4 15 0.76</td>
<td></td>
</tr>
<tr>
<td>Orana</td>
<td>1 4 0.71</td>
<td></td>
</tr>
<tr>
<td>South Eastern</td>
<td>0 0 -</td>
<td></td>
</tr>
<tr>
<td>South West</td>
<td>0 4 -</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>54 192 1.00</td>
<td>12 50</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for individual odds ratios
(Source: Maternal and Perinatal Collection, 1986)

\(^{12}\) This health region covers 72 per cent of the total Sydney region and, as of
1986, housed almost one-quarter of the population of New South Wales (New
South Wales Department of Health, 1989a).
When the risk is detected and there is sufficient time to undertake a transfer, whether or not that movement takes place depends secondly on whether the advantages of movement are perceived to outweigh the disadvantages by both the doctor and the patient. The consensus is that on medical criteria, antenatal movements are safe and can improve perinatal outcomes. However, medical practitioners may advise against antenatal transfer for a variety of reasons.

Particularly when long distances are involved, there may be financial and emotional costs to the pregnant woman (which also helps to explain Table 6.1). In probably rare circumstances, the affordability of short-term movement may be restricted for low-income families, specially among certain groups such as Aboriginals and in such regions as the Central West and New England. The psychological impact of antenatal transfer is perhaps the more widespread consideration.

Antenatal movement can be stressful and disrupt the stability of family life. However, the level of stress and any advice to transfer would reflect personal and situational factors including patients’ emotional, social and economic environments. Indeed some characteristics of transfer could help to relieve stress. These include being in the same hospital as one’s sick baby during its first, hazardous days of life, proximity being especially desirable if the baby should need prolonged treatment or should die, but being important anyway for the development of normal mother-child relations (Blake et al., 1979). In these situations, there is a need for the largest maternity units to provide accommodation for the mother’s partner or some other support person.

From the doctor’s perspective, antenatal transfer disrupts the continuity of care. Controversially, it is possibly also perceived by
some specialists at level 2 hospitals to subordinate their abilities
to the perinatal services of the largest hospitals around which the
system is built (Paneth, 1982). The result is that some specialists
may treat the patient whom they, but not their hospitals, are equipped
to manage. A problem with this explanation for failure to transfer
patients is that obstetric consultants from the largest units are
often actively involved in the provision of maternity care at smaller
hospitals.

A further consideration is accessibility to, and acceptance of, a
large second-tier hospital vis-à-vis a level 3 hospital. Upward
mobility from the Illawarra Region, as represented in Table 6.1, seems
low given its closeness to Sydney, probably because of competition
from the Illawarra’s Wollongong Hospital; four-fifths of the
population of the Illawarra live within 50 kilometres of Wollongong
City. The lack of movement from the South Eastern Region may likewise
be explained by proximity to large maternity units in Canberra and
even Victoria.

Yet the major constraint to transfer has not really been a lack of
demand for this service but, thirdly, the inability of newborn
intensive care facilities at tertiary centres to accommodate many more
sick neonates. Owing to the lack of an available level 3 neonatal
ventilator cot, level 6 obstetric units\(^{13}\) have had to redirect requests
for in utero transfer to other perinatal units. On 30 days during
1987, a Level 3 ventilator cot was not available at any neonatal
intensive care unit in New South Wales, and on 60 days none was
available in a Level 6 obstetric unit (New South Wales Department of

\(^{13}\) Level 6 obstetric units are supra-regional units caring for the highest-
risk obstetric patients. Because antenatal transfers are referred to
obstetric units rather than neonatal units, appropriate use is seldom made of
a computerized Neonatal Bed-State Network which daily updates the availability
of Level 3 ventilator cots (New South Wales Department of Health, 1989a).
Health, 1989a). However, ideally, high-risk infants should be born in an obstetric unit where intensive care is immediately available, this being fundamental to a perinatal referral system, as distinct, for example, from the system in West Germany (Obladen, 1988).14 The estimated shortfall as of 1988 in New South Wales was 16 funded Level 3 ventilator cots at neonatal intensive care units,15 and the problem is not merely a lack of beds.

There are also shortages of suitably qualified staff, inadequate resources for equipment repair and replacement, and problems with back-transport to referring units (New South Wales Department of Health, 1989a). All these problems have resulted especially from the increased survival of very low birthweight babies who consume high technology care for longer periods and at greater financial cost than previously, as well as from increases in antenatal transfers (Bryce and Stanley, 1985). Yet the potential for these transfers has nevertheless been stifled by augmentation of the normal demand for, and utilization of, maternity beds at large tertiary centres such as Sydney’s King George V (New South Wales Department of Health, 1989a).

Women’s reasons for choosing particular maternity units were investigated by a telephone survey, commissioned by the 1989 Task Force, of 724 maternity patients at seven major obstetric units. The survey found that for private patients, the hospital affiliation patterns of doctors were particularly important in determining the place of birth. Most private patients chose their doctor before their hospital, and this usually involved a doctor-hospital combination.

14 There, sophisticated neonatal transport systems and special care units, identified with paediatric vis-d-vis neonatal care, instrumentalize aggressive therapeutic approaches. Regionalization programs for in utero transfer and perinatal centres for high-risk deliveries have not evolved (Obladen, 1988).
15 There were 44 funded Level 3 ventilator cots at the seven neonatal intensive care units in New South Wales, but the estimated bed requirement is 60 such cots (New South Wales Department of Health, 1989a).
For the 41 per cent of women who were given some choice of hospital by their doctor, this choice was a limited one. On the other hand, more than a quarter of the private patients actively chose their hospital before their doctor, and public patients, of course, choose the hospital clinic they would attend.

Patients' reasons for selecting a particular hospital varied less with differences between the women themselves than with perceived differences between the hospitals, relating mainly to the quality of nursing care; closeness; facilities and technology; and then, the recommendation of the doctor. Non-local units were chosen mainly by private patients. Positive criteria for selecting particular units were more important than reasons for bypassing the local hospital, the most important of which was the doctor's choice.

Level 3 neonatal services are thus overstretched in part because (or from a different perspective, despite the fact that) private patients select specialists frequently associated with these hospitals, and to a lesser extent because private and public patients actively and positively choose these hospitals. Both mechanisms have the effect of circumventing, for these women, the transfer problems identified, whilst at the same time exacerbating these difficulties for other women.

The potential problems outlined are demonstrated empirically. The local government area in which each woman usually resides was assigned the rating of the highest level maternity hospital in this area or, when no hospital existed therein, closest to this area. The place of expected delivery, on this basis of residential status, was then compared with the actual hospital of birth. The results in Table 6.2 relate neonatal outcome of very low birthweight babies to these actual and expected places of birth.
The table suggests that little more than one-fifth (n=68) of births at hospitals providing level 3 neonatal care are to women living in level 3 designated areas; and only 10 per cent (n=33) of newborn at these hospitals are from level 1 areas. Residents of level 2a areas occupy the most level 3 beds (42.3 per cent, n=131), but approximately half and three-quarters of the births at level 2b and level 2a hospitals respectively are to women living in areas served by these hospitals.

Estimated relative odds of perinatal death are generally lower at level 3 units irrespective of the expected hospital of birth, although especially when this was a level 1 hospital. Adjustment for the expected hospital of birth yields a suspiciously low estimate of the relative risk of perinatal death at level 2a hospitals.

Table 6.2: Relation of Neonatal Death of Very Low Birthweight Infants to Actual Place of Birth according to Expected Place of Birth for Women aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Actual Hospital of Birth (Level of Care)</th>
<th>Expected Hospital of Birth (Level of care)</th>
<th>P.D.</th>
<th>Ctl.</th>
<th>P.D.</th>
<th>Ctl.</th>
<th>P.D.</th>
<th>Ctl.</th>
<th>P.D.</th>
<th>Ctl.</th>
<th>θ_{mh}</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1</td>
<td>4</td>
<td>0</td>
<td>2.20</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1.00</td>
</tr>
<tr>
<td>2a</td>
<td>0</td>
<td>6</td>
<td>0</td>
<td>13</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1.03</td>
</tr>
<tr>
<td>2b</td>
<td>8</td>
<td>8</td>
<td>3.86*</td>
<td>3</td>
<td>9</td>
<td>1.29</td>
<td>9</td>
<td>30</td>
<td>1.16</td>
<td>1.63</td>
</tr>
<tr>
<td>3a</td>
<td>7</td>
<td>26</td>
<td>1.04</td>
<td>35</td>
<td>96</td>
<td>1.41</td>
<td>11</td>
<td>66</td>
<td>0.64</td>
<td>1.00</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control

a Reference level for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
* Statistically significant at the 0.05 level
*** Statistically significant at the 0.005 level
(Source. Maternal and Perinatal Collection, 1986)

The Task Force, recognizing the problem of increased demand for confinements at level 3 hospitals, recommended improving the childbirth services and physical facilities of intermediate-sized hospitals, and promoting these units to the public as well as to
doctors. In fact, it may become necessary not only to cross-accredit doctors within areas, as the Task Force suggests, but to restrict their visiting rights at the largest units. The Task Force recommends decentralizing some specialist facilities to western Sydney growth areas. However, as important as this recommendation is, there is perhaps a larger solution, neglected by the Task Force, to the problem of increased demand for level 3 care: this is a thorough, qualitative reassessment of which patients should be transferred antenatally.

Chiswick (1982) reports that only about one-fifth of the babies transferred antenatally to his hospital, the St. Mary's Hospital in Manchester, require definitive ventilatory assistance. This means that valuable resources, especially nursing time, are taken up by the remaining 80 per cent of babies, leading potentially to refusals of neonates who require medium to long term ventilatory care. The lack of more selective antenatal transfers in New South Wales, as perhaps in Britain, is partly the result of difficulties in readily obtaining expert perinatal management advice and of diagnostic tools that lack sharpness in identifying impending asphyxia.

The answer, taken up from Chiswick (1982), may be to reduce the need for antenatal (and postnatal) transfers by improving the standard of essential neonatal care at level 2 maternity hospitals, while recognizing that centralization is needed of the most sophisticated care regimes. Intermediate-sized units would continue to transfer pregnant women and newborn babies for indications such as impending respiratory failure. But a more selective policy of antenatal transfer would see level 2 hospitals retain premature babies in whom respiratory failure is not imminent. These hospitals would provide

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16 This had led to a State-wide protocol for a co-ordinated telephone advisory service called the Perinatal Advice/Access Line (P.A.L.) (New South Wales Department of Health, 1989a).
the larger babies, whose stay is short and who are less likely to need prolonged ventilatory support, with level 3 neonatal intensive care (Chiswick, 1982).

Obstacles to this approach will continue to be inadequate funding in New South Wales, and the problem that low-risk status is a retrospective diagnosis. Yet, at least in theory, decentralizing neonatal care provision would allocate resources more equitably and probably more efficiently and cheaply than does the present system. This is not to suggest that these alternative ideas have not already been instituted in some places, but rather that there may be room for improvement, and that there is a need to address the increase in demand for antenatal transfer and not merely the stretched capacities of the largest neonatal units to accommodate critically ill babies.

Such, at least, is indicated by Table 6.3 which shows, from survey data, the distribution by birthweight of in utero transfers from non-level 3 hospitals to three hospitals providing level 3 neonatal care in 1985 and 1986. Eighteen Level 3 ventilator cots were available at these units in 1987, yet, for example, at birthweights 1 500 to 2 499 grams, ten babies (one-third of the infants at these birthweights) had been transferred in utero. Many of these babies might not have required prolonged mechanical ventilation and some of them might have been able to be cared for effectively at the hospital of origin if its facilities were improved.

The table shows that at birthweights 2 500 to 3 999 grams, only 12.2 per cent (n=5) of the infants born at these tertiary centres had been transferred in utero, compared with 61.8 per cent (n=55) at very low birthweights. Yet since low and normal birthweight babies are over- and under-sampled respectively in Table 6.3, transferring even a small proportion of the latter and most prevalent babies, would produce a
large number to manage. About four per cent of neonates \((n=1367)\) in the weighted sample are estimated to have been transferred in utero from units not providing level 3 neonatal care to hospitals providing this care.

Also of interest is the confirmed safety of antenatal transfer, despite the high-risk status of these transferred babies. After adjustment for birthweight, Table 6.3 gives estimated relative odds of perinatal death following antenatal transfer, relative to no transfer, of 0.56. At very low birthweights the survival advantage of transfer \((\theta=2.12)\) compared with no transfer \((\theta=8.86)\) is 4.2. The high estimated relative risk associated with the transfer of infants 2500 to 3999 grams is almost certainly an artefact of the small number of transfers. The main finding, which supports existing evidence, is that some very low birthweight newborn might have improved survival chances if transferred in utero to hospitals providing level 3 neonatal care - given existing services at lower level units.

Table 6.3: Relation of Neonatal Death at Level 3 Hospitals to Antenatal Transfer from Level 0, 1 or 2 Hospitals by Women Aged 20 to 34 Years according to Birthweight, 1985-86

| Antenatal Transfer | Birthweight (grams) |  |  |  |
|--------------------|---------------------|---|---|---|---|
|                    | 500-1499            | 1500-2499 | 2500-3999 | 4000+ |
|                    | P.D. Ctl. \(\theta\) | P.D. Ctl. \(\theta\) | P.D. Ctl. \(\theta\) | P.D. Ctl. \(\theta\) | \(\theta_{mh}\) |
| Yes                | 14 | 41 | 2.12 | 2 | 8 | 1.55 | 4 | 1 | 24.80** | 0 | 0 | 0.56 |
| No\(^a\)           | 20 | 14 | 8.86*** | 6 | 14 | 2.66 | 5 | 31 | 1.00\(^b\) | 1 | 1 | 6.20 | 1.00 |

P.D: Perinatal Death; Ctl: Control

\(^a\) Reference group for Mantel-Haenszel estimated odds ratios

\(^b\) Reference group for other odds ratios

** Statistically significant at the 0.01 level

*** Statistically significant at the 0.005 level

(Source: Hospital patients' records, 1985-86)
6.3 POSTNATAL TRANSFER

Antenatal transfer is not always possible, for the types of reasons suggested and because symptoms, for example of cardiac disease or intestinal atresia,\(^{17}\) may not be apparent until the baby is born. However, lives are saved by the postnatal transfer of neonates, which is a key mechanism influencing where sick newborn receive health care. Sims et al. (1982), for example, report neonatal survival rates of babies with respiratory failure that were improved by half following admission, compared with non-admission, to a neonatal intensive care unit.

Comparison of the risks of transfer before with after delivery is complicated by substantive factors and, in this study, by constraints of the data. The former considerations include differences between the groups undergoing the transfer. Whereas the foetus transferred \textit{in utero} can die as a stillbirth, or be potentially at high-risk of an adverse perinatal outcome, postnatal transfer involves only neonates who are always very sick. On the other hand, neonates have already demonstrated some survival ability \textit{vis-à-vis} still born infants and may be seen to constitute a more potentially salvageable group (Chiswick, 1986). Differential risks associated with antenatal versus postnatal transfer are thus likely to reflect characteristics of the baby being transferred as much as the type or timing of the transfers \textit{per se}; and any comparison should be made in these terms. Studies documenting improvements in survival when babies are transferred antenatally have little value in relation to postnatal transfers (Chiswick, 1982; and see section 2.2.1).

\(^{17}\) This refers to a congenital absence or abnormal narrowing of an intestinal body opening.
This is notwithstanding the increased aetiological risk of, for example, intraventricular haemorrhage associated with postnatal transfer, and the intellectual attractiveness of the notion that intrauterine carriage is the safest method of transport. For these and other reasons including the ability of the referring hospital to care for the sick neonate until the retrieval team arrives, and the desire not to separate the mother and newborn baby (Blake et al., 1979), neonatal transfers have declined, for example in Rhode Island, in concert with increased transfers antenatally (Cowett and Coustan, 1986).

The second set of difficulties in comparing the risks of postnatal and antenatal transfer is that of identifying the babies who died after postnatal transfer. As data exist for only a small number of these infants (see section 3.5), caution is exercised in interpreting the findings of Table 6.4. However, to reduce the bias as much as possible, postnatal transfers to level 3 units have been compared for all hospitals of origin, rather than exclusively for level 1 and level 2 public hospitals.

Table 6.4: Relation of Neonatal Death to Postnatal Transfer to a Level 3 Public Hospital for Women Aged 20 to 34 Years according to Birthweight, 1986

<table>
<thead>
<tr>
<th>Postnatal Transfer</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-1 499</td>
</tr>
<tr>
<td></td>
<td>P.D. Ctl. 0</td>
</tr>
<tr>
<td>Yes</td>
<td>6  68 0.46</td>
</tr>
<tr>
<td>No(^a)</td>
<td>262 301 4.57***</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
\(^a\) Reference group for odds ratios relative to no postnatal transfer
\(^b\) Reference group for other odds ratios
*** Statistically significant at the 0.005 level
(Source: Maternal and Perinatal Collection, 1986)
The table is studied in its own right, and then compared to Table 6.3 within the limits just described. Table 6.4 suggests that postnatal transfer compared with no transfer is most prevalent (11.6 per cent, n=74) at very low birthweights when such movement is also especially safe (θ=0.46) relative to non-movement (θ=4.57); the reference group is non-transferred infants of normal birthweight. Failure to transfer is also hazardous at birthweights 1 500 to 2 499 grams, but at birthweights 2 500 to 3 999 grams postnatal transfer appears to be unsafe.

This is interesting because, in the literature, the birthweight-specific neonatal transfer rates show no clear trend. Cowett and Coustan (1986) report, for Providence, Rhode Island, an increase in the proportion of transferred neonates weighing at least 2 500 grams. However, the opposite is documented by Floyd et al. (1989) for Georgia, in the United States, between 1974 and 1982. From Table 6.4, after adjusting for birthweight, and making the reference group to include neonates not transferred after birth, the relative risk of death is estimated to fall with postnatal transfer (θ_{nh}=0.41).

Comparison is undertaken of the risks of babies transferred antenatally vis-à-vis postnatally. Antenatal transfer to a level 3 hospital was found in Table 6.3 usually to decrease the estimated relative odds of neonatal death, also, like postnatal transfer, at low and especially at very low birthweights, but not at normal birthweights. Overall, postnatal transfer is shown to be 'safer', not necessarily because of the safer timing of the transfer but as a result of the characteristics of the babies being moved. The result implies that if high-risk infants can survive birth, their risks of dying with postnatal transfer to a level 3 unit are lower than for infants transferred there in utero from a non-level 3 unit.
6.4 ANTENATAL CARE: ASPECTS OF QUANTITY

Frequently reported is the value of early, regular and qualitatively appropriate antenatal care for improving perinatal risks. For example, the 1989 Task Force states that 'the cornerstone of good obstetrics is a high standard of antenatal care' (New South Wales Department of Health, 1989c). Owing to a paucity of data, no assessment in the present study is made of the effects of different types of health care, but testing is undertaken of an hypothesized positive relation between perinatal survival and the amount of antenatal care as measured by the number of antenatal visits recorded. A potential problem with the analysis is that any one antenatal visit may not have the same value for different patients or indeed for the same patient, even though such equality must be assumed to exist.

From Table 6.5, just 1.1 per cent (n=754) of the weighted sample did not make two or more antenatal visits, which is below the State Department of Health's half-yearly estimate for 1986 of 1.6 per cent (New South Wales Department of Health, 1988a). This lack of attendance is greatest for the mothers of low (4.1 per cent, n=51) and especially very low birthweight (5.1 per cent, n=29) babies, and reaches its nadir for the women delivering normal birthweight babies (1.0 per cent, n=11). Without two or more antenatal visits, the weighted incidence of low birthweights is increased approximately threefold to 16.4 per cent (n=3 407).

Nevertheless, taking two or more antenatal visits as the reference group and making adjustment for birthweight, the relative odds of perinatal death associated with fewer than two antenatal visits is just 1.09. This lack of attendance is estimated to increase the relative risk of perinatal death most for infants weighing 2 500 to 3 999 grams. The odds ratio (θ=1.93) for these infants, based on
their small numbers, is almost two times that of newborn at the same birthweights whose mothers made at least two antenatal attendances.

Table 6.5: Relation of Perinatal Death to Two or More Antenatal Visits by Women Aged 20 to 34 Years according to Birthweight, 1986

<table>
<thead>
<tr>
<th>Two or More Visits</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-1 499</td>
</tr>
<tr>
<td>P.D. Ctl. θ</td>
<td>P.D. Ctl. θ</td>
</tr>
<tr>
<td>No</td>
<td>11</td>
</tr>
<tr>
<td>Yes</td>
<td>230</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratio
b Reference group for other odds ratios
*** Statistically significant at the 0.005 level
(Source: Maternal and Perinatal Collection, 1986)

In fact, fewer than two antenatal visits is shown to be more hazardous than is two or more visits at all birthweights of 1 500 grams and over (this minimum quantity of care reducing the estimated relative odds of death to that of the reference group) but the increases in risk are not statistically significant at the 0.05 level. By contrast, at very low birthweights, making at least two antenatal visits is not beneficial and \( p < 0.005 \). However, it is reassuring that other age groups have outcomes for very low birthweight babies that improve with two or more antenatal visits (see Table 6.6).

This finding is clearest for adolescent women whose estimated relative risk of perinatal death falls from 1.37 to 0.82, the reference group being antenatal care receivers aged 20 to 34 years. Similarly, among women aged 35 years or more, the relative risk of perinatal death falls from 1.37 to 0.99. Because 20 to 34 year-old women account for 81.7 per cent of the very smallest babies, 0.99 is the age-adjusted Mantel-Haenszel estimate of the odds ratio associated with inadequate antenatal care.
Using sample data extracted from patient maternity records, an examination is made of the effect of the numbers of antenatal visits made by women aged 20 to 34 years during 1985-1986 (see Table 6.7). The table shows that 6.2 per cent (n=8) of the women having very low birthweight babies made fewer than two antenatal visits, but that higher birthweights almost never followed this lack of attendance. While this result may reflect a sampling bias, it is worth recalling the observation of the Ministerial Task Force in New South Wales that attendance rates differ widely by individual hospital, with 'in one part of Western Sydney for example at least 30 per cent of women ... [making] fewer than two antenatal visits' (New South Wales Department of Health, 1989a: 156).

Table 6.6: Relation of Perinatal Death of Very Low Birthweight Infants to Two or More Antenatal Visits according to Maternal Age, 1986

<table>
<thead>
<tr>
<th>Two or More Visits</th>
<th>Age of the Mother (completed years)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15-19</td>
</tr>
<tr>
<td></td>
<td>P.D. Ctl. θ</td>
</tr>
<tr>
<td>No</td>
<td>3 3 1.37</td>
</tr>
<tr>
<td>Yes(^a)</td>
<td>15 25 0.82</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratio
b Other odds ratios
(Source. Maternal and Perinatal Collection, 1986)

Factors noted by the Task Force to contribute to poor attendances include congestion and continuity problems in hospital antenatal clinics, and a failure to meet the individual needs of known high-risk groups, such as Aboriginal women, in the delivery of antenatal care. To help raise the number and quality of antenatal visits, the Task Force supported, among other things, increased shared
care arrangements; community-based rather than institution-based antenatal care; and greater recognition of the special requirements of identified high-risk groups. The report emphasized the needs of the pregnant woman rather than the convenience of her care giver.

Further noted from Table 6.7 is that more than three-quarters (n=169) of the mothers of low birthweight babies made 2 to 9 antenatal visits, only 17.1 per cent (n=37) of these women making 10 or more visits. By contrast, this largest number of visits was undertaken by 56.7 per cent (n=55) of the mothers of normal birthweight babies.

Table 6.7: Relation of Perinatal Death to Antenatal Visits By Women Aged 20 to 34 Years according to Birthweight, 1985-86

<table>
<thead>
<tr>
<th></th>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-1</td>
<td></td>
<td>6</td>
<td>2</td>
<td>3.88</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0.82</td>
</tr>
<tr>
<td>2-5</td>
<td></td>
<td>25</td>
<td>28</td>
<td>1.15</td>
<td>5</td>
<td>13</td>
<td>0.50</td>
<td>7</td>
<td>3</td>
<td>3.01</td>
<td>0</td>
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<td>6-9</td>
<td></td>
<td>30</td>
<td>29</td>
<td>1.34</td>
<td>14</td>
<td>25</td>
<td>0.72</td>
<td>15</td>
<td>16</td>
<td>1.21</td>
<td>2</td>
</tr>
<tr>
<td>10+</td>
<td></td>
<td>7</td>
<td>3</td>
<td>3.01</td>
<td>16</td>
<td>11</td>
<td>1.88</td>
<td>24</td>
<td>31</td>
<td>1.00b</td>
<td>2</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control

a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios

(Source. Hospital patients' records, 1985-86)

The main finding associated with the estimated odds ratios attending different numbers of antenatal visits is that at low birthweights, perinatal death is more likely to follow 10 or more antenatal visits than fewer visits, this being translated into the Mantel-Haenszel estimates. Two possible explanations of this result are given. First, the mothers of low birthweight babies that receive the largest

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18 Specifically, midwives and general practitioners could provide more antenatal care for women at low or moderate risk of complications in consultation with specialist obstetricians.
amounts of antenatal care may do so because they are identified as at greater risk of perinatal complications than the other women who deliver at low birthweights. Although high risks of perinatal death occur despite the large quantity of care received, these risks might have been even greater if fewer visits had been made. The second, more polemical perspective is that antenatal care, whatever its quantity, does not always effectively monitor the wellbeing and risk status of the pregnant woman. Therefore, such care, irrespective of its informational value, may not prevent some complications developing and, although treatment may be initiated early, some cases of perinatal death (see section 2.5).

Yet Table 6.7 also shows that most dangerous at any individual birthweight is fewer than two antenatal visits (θ=3.88) for newborn weighing less than 1 500 grams, this estimate being relative to 10 or more visits by mothers of infants weighing 2 500 to 3 999 grams. Also hazardous is fewer than six antenatal visits at normal birthweights, the odds ratio for 2 to 5 visits being θ=3.01 Fewer than ten attendances at antenatal care is shown least to affect the perinatal survival of babies weighing 1 500 to 2 499 grams at birth. The main result is that at low birthweights, a large quantity of antenatal care, although it may reduce perinatal mortality, cannot do so sufficiently to yield low estimated relative odds of perinatal death.

6.5 INTRAPARTUM CARE

Sections 6.5 to 6.8 are concerned mainly with the comparative risks, in different circumstances, of caesarean section vis-à-vis induction, augmentation and vaginal breech delivery. A key problem is whether the risk schedules vary because of differences in the methods themselves, or more because of the characteristics of the women and
babies exposed to the methods. To deal with this difficulty, the following approach is taken.

Two assumptions are made. First, caesarean section is almost always possible should it be considered necessary (see section 6.6). Secondly, vaginal delivery is usually undertaken for lower risk women than is a caesarean section (see section 2.4.3). This is notwithstanding that when it becomes apparent before the birth that a baby cannot be saved, for example because of lethal congenital anomalies, delivery will almost always be vaginal because of the reduced risks to the mother. The net likelihood is that women who deliver by caesarean section have more complications in number and seriousness than have other parturients.

Accordingly, if without adjustment for these other complications, and despite the greater risk associated with caesarean section, this operation yields a lower estimated relative risk of perinatal death than do alternative methods of delivery, the caesarean section is likely to be safe in the described circumstances. This method of proceeding, which has the additional advantage of not losing surveyed women through adjustment for confounding, is followed in sections 6.6 to 6.8.

These, and all other, sections include congenital anomalies in data analyses because small total numbers do not permit otherwise. However, at least in the following sections of this chapter, where any resultant bias is most likely to occur, the effect is favourable. The inclusion of congenital anomalies helps to compensate for overestimation of the risk of perinatal mortality associated with caesarean section when adjustment is not made for other indications for this operation.
If the infant born by caesarean section for, say, a breech presentation is not at lower risk in the presence of other indications for the caesarean section, then it becomes necessary to consider caesarean sections only for a breech delivery - so as to exclude the confounding effects of other complications. However, this is shown not to be necessary. Although congenital anomalies are not removed from the following tables, the results of doing so for cells that have sufficient subjects to be interpretable are reported in the text, and these findings are consistent with those based on the tables presented without adjustment for this confounding effect.

6.6 INDUCTION

When, as in most inductions, labour is induced by amniotomy,19 and typically intravenous oxytocin infusion, the medical indications20 should be strong enough also to warrant a caesarean operation should the induction fail.21 In practice, both induction and elective caesarean sections are sometimes undertaken for the convenience of the patient or obstetric team and, more seriously, for flimsy medical indications such as a gestational age of 40 weeks without other medical reason; this risks prematurity since menstrual data are unreliable in 20 to 30 per cent of patients (Beischer and Mackay, 1983).22 However, assuming the correctness of any assessed

19 Induction is defined in chapter 2, footnote 37. Amniotomy or artifical rupture of the membranes is the usual means of surgical induction (the initiation of labour through mechanical means). Rupture is of the forewaters (low amniotomy) with a forceps such as Kocher's.
20 These usually include major disorders, for example, the hypertensive disorders of pregnancy, postmaturity (42 weeks or more) and intrauterine growth retardation. A sound indication may be produced by a combination of relative factors such as advanced maternal age, previous obstetric history and previous subfertility (Beischer and MacKay, 1983; New South Wales Department of Health, 1989d).
21 From 24 hours following an amniotomy, the risks of intrauterine infection to the mother and baby increase rapidly.
22 However, the risk of iatrogenic prematurity has been reduced by the increased use of ultrasonic parameters, and the availability of liquor studies to assess foetal pulmonary maturity.
desirability of delivery\textsuperscript{23} in the woman in whom spontaneous labour has not begun, an important issue is the comparative safety of induction vis-à-vis elective caesarean section.\textsuperscript{24}

Of specific interest is whether induction produces risks of perinatal death that are greater than the risks associated with elective caesarean section, the latter procedure being the more likely to be resorted to in situations when delivery is most urgent. Conversely, should induction be more often performed in preference to caesarean delivery? These questions should not be considered provocative. The intention in asking and in seeking to resolve them is not necessarily to support a reduction or elevation in the incidence of caesarean sections,\textsuperscript{25} but rather to elucidate the specific circumstances under which caesarean delivery should or should not take place.

Manifold arguments were earlier cited as evidence of the risks of caesarean section compared with vaginal delivery (see section 2.4.3). However, it is necessary also, by way of comparison, to appreciate the risks of induction and the management options available to the obstetrician in deciding on an appropriate method of delivery. With reference to the latter issue, it is unusual to induce labour because an elective caesarean section cannot be performed.

In essence, the number of possible caesarean sections depends for each woman on the amount of damage previously done to her uterus. For example, if a woman's lower uterine segment is damaged, an upper segment caesarean section is likely to be possible, although in

\textsuperscript{23} That is, the risks of delivery are considered to outweigh the risks of permitting the pregnancy to continue.

\textsuperscript{24} An elective caesarean section is one performed before the onset of labour. This precludes the possibility of a failed induction (that is, uterine contractions cannot be induced within 12 hours of starting the induction), which is uncommon.

\textsuperscript{25} Impassioned opposition could no doubt be expected to any attempt to promote elective recourse to a surgical operation that, despite recent, mixed publicity, some consider is already too prevalent.
reality this operation is rarely done.\textsuperscript{26} It brings increased risks of subsequent uterine rupture with vaginal delivery, and most women cease childbearing after, or before, three caesarean births - which helps to explain why vaginal delivery is rare certainly after two caesareans.

The second issue relates to the risks of induction. Most immediately, there are clear absolute contraindications to an induction, such as true foetopelvic disproportion, as well as other relative contraindications.\textsuperscript{27} Yet disproportion can be difficult to diagnose (without an X-ray pelvimetry or preferably a CAT scan of the pelvis in selected cases),\textsuperscript{28} and in the majority of women a carefully monitored trial of labour is considered safe management practice (Blakemore and Petrie, 1988). Of course, any induction introduces risks, particularly when the cervix is unripe. Rupture of the membranes and labour can both be prolonged which can lead to maternal exhaustion, an increased rate of instrumental and caesarean delivery, intrauterine infection and an elevated risk of perinatal asphyxia (New South Wales Department of Health, 1989d). Yet the key questions are how do these risks compare with those of elective caesarean section? And how prevalent is exposure to each procedure? The latter question is addressed first.

Table 6.8 focuses on the management of the confinement in which labour does not start spontaneously and which results in a live birth. The table shows that inductions are resorted to mainly from 37 weeks onwards when a good Bishop score\textsuperscript{29} is less difficult to achieve than

\textsuperscript{26} This technique is most often performed for extreme prematurity, some malpresentations or for an anterior low-lying placenta.

\textsuperscript{27} Relative contraindications to induction are conditions, such as a breech presentation, that may benefit from this procedure if they are individualized and given expert medical supervision.

\textsuperscript{28} For example, the cervix may dilate only slightly and the accoucheur may have no prior record of any earlier parturition.

\textsuperscript{29} The Bishop score assesses the ripeness of the cervix on the basis of five criteria: dilation, consistency, length, position and the station of the head.
before term, and then in approximately three-quarters of the weighted sample of women in whom labour did not start spontaneously. By contrast, after weighting, an estimated 2.4 times more elective caesarean sections than inductions are performed before 37 gestational weeks at low birthweights. Before term, the percentages of infants born by caesarean section are 84.8 per cent (n=112) at birthweights 500 to 1,499 grams and 64.3 per cent (n=166) at 1,500 to 2,499 grams.

Table 6.8: Relation of Neonatal Death to the Management of Non-Spontaneous Labour in Women Aged 20 to 34 Years according to Gestational Age and Birthweight, 1986

<table>
<thead>
<tr>
<th>Period of Gestation (completed weeks)</th>
<th>Type of Labour</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>500-1,499</td>
</tr>
<tr>
<td></td>
<td>P.D. Ctl. θ</td>
<td>P.D. Ctl. θ</td>
</tr>
<tr>
<td>&lt; 37</td>
<td>Induction</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>No labour*</td>
<td>14</td>
</tr>
<tr>
<td>37 or more</td>
<td>Induction</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>No labour*</td>
<td>0</td>
</tr>
</tbody>
</table>

P.D.: Perinatal Death; Ctl: Control
a The term no labour, as defined by the New South Wales Department of Health, implies an elective caesarean section and no failed induction
b Reference group for Mantel-Haenszel estimated odds ratios
c Reference group for other odds ratios
*** Statistically significant at the 0.005 level
(Source: Maternal and Perinatal Collection, 1986)

The timing of the delivery is more hazardous than the delivery management option chosen: preterm births record higher estimated relative risks of perinatal death than do births from 37 weeks onwards. Gestational age is also revealed to have an important influence on the comparative risks of each management approach. After adjustment for birthweight, there is little difference at, or after, term in estimates of the relative risks of induction (θ mh=0.96) and of the reference group of no labour. However, before term, induction is associated with a conspicuously higher relative odds of death in relation to the ischial spines. Each criterion is given a score of 0, 1 or 2 and the total is the Bishop score.
(\theta_{mh}=2.13) \text{ than is no labour (}\theta_{mh}=1.31). \text{ After adjustment for birthweight and gestational age, the risk of neonatal death following an induction, compared to an elective caesarean section, is elevated at 1.55.}

Before 37 gestational weeks, induction is associated with higher estimated relative risks of neonatal death than is no labour at low and, especially, very low birthweights. At birthweights 500 to 1,499 grams, the estimated odds ratio attending an induction (\theta=9.02) is almost four times higher than that associated with no labour (\theta=2.39), the reference group being term, normal birthweight babies delivered by elective caesarean section. At birthweights 1,500 to 2,499 grams, the differential is approximately two times. Despite these increased risks associated with induction before term, 15.2 per cent (n=20) and 35.7 per cent (n=30) of preterm babies at these respective low birthweights were born following an induction rather than no labour. Moreover, in a separate analysis that removed all congenital anomalies (see section 6.5), the increased risk of induction was reduced but still apparent at these birthweights. It was 3.23 at birthweights 500 to 1,500 grams and 1.31 at birthweights 1,500 to 2,499 grams.

The implication is that before term and at low birthweights, either induction is more dangerous than an elective caesarean section, or the induced women are at greater risk of perinatal death than are the women who deliver by elective caesarean section. In either case, the result is disturbing. If some of the induced women are at higher risk than those who delivered by elective caesarean section, why did more of the induced women not receive a caesarean section which is designed for the highest risk deliveries? If, as is more likely, an induction itself presents the greater risks for the small preterm baby, why was induction rather than caesarean section resorted to in almost
one-third (n=50) - or, after weighting, 35.8 per cent - of the preterm, non-spontaneous labours leading to low birthweight babies? Notwithstanding the inclusion of congenital anomalies, the levels of prevalence seem high. Indeed the answers to both the questions posed, support the conclusion that an elective caesarean section may be a safer option than an induction when, before 37 weeks gestation, the delivery of a low birthweight baby is deemed necessary and labour has not started.

After 36 weeks gestation, induction is possibly less hazardous (0=0.46) than is no labour (0=2.39) in babies weighing 1500 to 2499 grams at birth. However, at normal birthweights, the induced women have a higher estimated relative risk of neonatal death.

Given the apparent, increased perinatal mortality risks of an induction before term at low birthweights, it may be helpful to contrast the exposure and risk patterns attending different methods of induction against no labour (see Table 6.9). Of 50 inductions, 30 per cent (n=15) were by medical means30 and 20 per cent were surgical inductions (n=10). The remaining 50 per cent (n=25) of inductions were combined surgical and medical inductions. Relative to no labour, all three methods are shown to be hazardous, although least so is the comparatively uncommon surgical induction, which also yields the least stable relative risk estimate. The highest estimated odds ratio is associated with induction by medical means (0=2.81), while for the most widely resorted to, combined medical and surgical inductions, it is 0=2.44.

Again, it is uncertain to what extent these risk estimates refer to the differential safety of the methods themselves, and to differences

30 Medical induction of labour is the initiation of labour with drugs, namely syntocinon given intravenously or prostaglandin administered vaginally.
in the risk statuses of the women who are induced by each method. At
the least, the former possibility should not be too readily dismissed,
as each method brings its own risks. Inductions by medical means,
using synthetic oxytocin or the prostaglandins (for cervical
preparation and/or induction of labour), yield the highest odds ratio
estimated in Table 6.9; and the next highest odds ratio (θ = 2.44) also
involves medical induction (in combination with amniotomy). Also,
both oxytocin and the prostaglandins present the risks of uterine
hyperstimulation31 which can lead to foetal hypoxia and uterine
rupture.

Table 6.9: Relation of Neonatal Death of Low Birthweight
Infants to Induction Method and No Labour before Term
among Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Induction method</th>
<th>P.D.</th>
<th>Ctl.</th>
<th>θ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical Induction</td>
<td>4</td>
<td>11</td>
<td>2.81</td>
</tr>
<tr>
<td>Surgical Induction</td>
<td>2</td>
<td>8</td>
<td>1.93</td>
</tr>
<tr>
<td>Combined Surgical and Medical Induction</td>
<td>6</td>
<td>19</td>
<td>2.44</td>
</tr>
<tr>
<td>No Labour</td>
<td>19</td>
<td>147</td>
<td>1.00a</td>
</tr>
<tr>
<td>Total</td>
<td>31</td>
<td>185</td>
<td></td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for odds ratios
(Source. Maternal and Perinatal Collection, 1986)

Oxytocin infusion is less widely used before 35 to 37 weeks than is
intravaginally administered prostaglandin in women with, for example,
low Bishop scores. However, oxytocin, given that it is sometimes
used, and that it can have a number of adverse risks, requires
elaboration. The preterm uterus has decreased sensitivity and is more
resistant to the stimulus effect of the higher doses of oxytocin

31 Uterine hyperstimulation, or uterine hypertonus, is an abnormal uterine
action characterized by excessive muscle tone. The contractions are frequent
but ineffective producing a painful and prolonged labour.
required for adequate contractility (Blakemore and Petrie, 1988).

However, oxytocin sensitivity varies widely from woman to woman
(Chamberlain, 1984), and, as argued by Brindley and Sokol (1988:
730), high incidences of hyperstimulation and foetal distress may be
related to the 'use of oxytocin at doses higher than currently appear
appropriate'; moreover, according to the same authors (1988, 730) in
the United States 'protocols for the use of oxytocin currently used in
many hospitals seem to be based on outdated pharmacologic data'.

Oxytocin can have further adverse maternal cardiovascular and
anti-diuretic effects, while increasing the incidence of
hyperbilirubinaemia.

Amniotomy is the safest and most effective method of inducing labour
(Beischer and MacKay, 1983), a point supported by Table 6.9, but even
low amniotomy is not without risks, for example, of cord prolapse,
placental abruption and intrauterine infection (also see section
2.4.2). Combining amniotomy with the medical control of labour gives
exposure to the risks of both methods, although this may be dictated
by the state of the cervix (amniotomy may be deferred until cervical
ripening is achieved with a prostaglandin E\textsubscript{2} pessary or gel) or by the
inability of amniotomy alone to establish labour. Failure to wait
about four hours before commencing medical induction increases the
risks of stimulation of uterine activity that might have been avoided
had amniotomy itself been sufficient to establish labour (Beischer and
MacKay, 1983). The most useful finding is that induction may be more
hazardous than elective caesarean section at low birthweights before
term.

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32 The effectiveness of oxytocin depends 'on the extent to which the
underlying control mechanism for parturition has already been activated' (New
33 There is an excess of bilirubin (a bile pigment) in the bloodstream.
34 Rupture of the hindwaters (high amniotomy), using a Drew-Smythe Catheter,
is contraindicated in modern obstetric practice (New South Wales Department of
Health, 1989d).
Of medical and demographic significance is how parity influences the management of the nonprogressive labour and delivery. When labour starts, but proceeds too slowly, the appropriate course of action should take into account whether the patient is nulliparous or multiparous. In the latter woman, augmentation is less frequently needed than in the nullipara. The multiparous uterus is a highly efficient organ, and any delay in labour is assumed most often to result from foetopelvic disproportion. Augmentation would introduce the risks of foetal distress and uterine rupture, and, if done in the multipara, must be monitored especially carefully. With epidural anaesthesia, women can better tolerate prolongation of their labours, although the anaesthesia may increase the need for forceps assistance.

Among nulliparae, failure to progress, which is more common in these women than in multiparae, is likely to result from inefficient uterine action. Since the nulliparous uterus is virtually immune to rupture, the nonprogressive labours of these women, claims Chamberlain (1986), can usually be augmented by amniotomy or syntocinon. According to Seitchik et al. (1985), only when the period of maximum rate of cervical dilatation is already in force does amniotomy facilitate rapid, efficient labour. In other words, in nulliparous women, 'time should not be spent awaiting the cure of an inefficient labour by amniotomy before getting on with an intravenous oxytocin supplement' (Kirschbaum, 1987: 183). However, this increases the risks of uterine hypertonus that may be avoided by a more conservative approach to the induction of labour (see page 300).

35 Exact timing of the duration of labour is difficult. For example, false diagnosis of prolonged labour is associated with premature identification of the active phase of labour (National Health and Medical Research Council, 1984).
It has been further reported that the active management of dystocia\textsuperscript{36} can diminish the importance of two principal contributors to current high rates of caesarean section: dystocia in nulliparous women, and repeat caesarean sections in these women (Akoury et al., 1984; O'Driscoll et al., 1984). However, increasing caesarean section rates for dystocia are usually attributed to the observation that, while labour should rarely exceed 24 hours (hence the dictum 'the sun should not set twice on the labouring woman'), augmentation of labour is often followed by foetal heart rate changes suggestive of foetal distress.

Continued study is required of the safety and efficacy of active labour management in the dystocic nullipara. The recommendation may also need to be qualified if augmentation, as would seem to be true of induction in section 6.6, is hazardous before term at low birthweights. Below are the results of efforts to estimate the prevalence and risks of parity, and of different methods of managing the nonprogressive labour in nulliparae.

\section*{6.7.1 AUGMENTATION}

That multiparae are less often augmented than nulliparae, and then at increased risk, can be regarded as a truism. According to Table 6.10, except at very low birthweights, fewer multiparae are augmented than are nulliparae, particularly at birthweights of 2 500 grams and over. Although the form of the relation is appropriate, the magnitude of the exposure by multiparae seems high. The problem is made clearer by the observation that approximately half of all augmentations, irrespective of the birthweight, are to multiparous women. Further, 16.9 per cent (n=11 759) of the weighted sample of women were augmented, which

\textsuperscript{36} See chapter 2, footnotes 56 and 57.
approaches the half-yearly estimate of the prevalence of augmentations, 17.2 per cent, made by the State Department of Health (New South Wales Department of Health, 1988a).

The estimated odds ratios do not show augmentation to be more hazardous for multiparae than for nulliparae except at birthweights 1 500 to 2 499 grams, whilst among multiparous women, augmentation is less hazardous than is no augmentation (only) at very low birthweights. These results, especially the former one, do not fit uncomfortably with the knowledge that for the treatment of nonprogressive labour, oxytocin 'should be confined to primigravidae, with malpresentations, hydrocephalus and twins specifically excluded' (Chamberlain, 1988: 15).

Table 6.10: Relation of Neonatal Death to Augmentation of Women Aged 20 to 34 Years according to Parity and Birthweight, 1986

<table>
<thead>
<tr>
<th>Parity</th>
<th>Augmentation</th>
<th>Birthweight (grams)</th>
<th>500-1 499</th>
<th>1 500-2 499</th>
<th>2 500-3 999</th>
<th>4 000+</th>
<th>0 P.D. Ctl. 0</th>
<th>P.D. Ctl. 0</th>
<th>P.D. Ctl. 0</th>
<th>P.D. Ctl. 0</th>
<th>0 _mh</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nulliparae</td>
<td>No</td>
<td></td>
<td>38</td>
<td>181 2.40</td>
<td>12 233 0.82</td>
<td>25 286 1.38</td>
<td>2 7 4.52</td>
<td>0 0.87</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td></td>
<td>9</td>
<td>12 11.87</td>
<td>2 43 0.74</td>
<td>10 75 2.11</td>
<td>0 7 -</td>
<td>1.53</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiparae</td>
<td>No^a</td>
<td></td>
<td>60</td>
<td>155 6.13</td>
<td>20 265 1.19</td>
<td>29 459 1.00</td>
<td>4 54 1.17</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td></td>
<td>4</td>
<td>21 3.01</td>
<td>4 44 1.44</td>
<td>10 75 2.11</td>
<td>0 7 -</td>
<td>1.14</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
\^a Reference group for Mantel-Haenszel estimated odds ratios
\^b Reference group for other odds ratios
** Statistically significant at the 0.01 level
*** Statistically significant at the 0.005 level
(Source. Maternal and Perinatal Collection, 1986)

In practice, when the disproportion is borderline, at least some obstetricians do, in fact, carefully augment labour rather than, or before, if necessary, resorting to caesarean section. But it is difficult to accept that the augmentation of multiparae is as widespread as Table 6.10 suggests, and if measurement of the frequency
of the procedure is erroneous, so too may be the estimation of its safety at the two parities.

Most likely, the results reveal the difficulty of knowing when labour begins, and thus of distinguishing between induction and augmentation. Unfortunately, labour is a presumptive diagnosis, which is not incontrovertible, for example in the woman who reaches some kind of 'labour' but fails early to progress. In such circumstances, in which a prolonged latent phase is commonly confused with false labour (Niswander, 1986), administration of oxytocin would seem to have been quite often recorded by the midwives as an augmentation. It should have been recorded as an induction. Moreover, in the hospitals worked in by the researcher, only vague reference is made in some patient records to the timing of the onset of labour. Both problems lend themselves to errors in recording ex post facto an event the exact timing of which depends on an informed opinion. Confusion over the onset of labour also casts doubt on the success of attempts to distinguish between elective and emergency caesarean section.

6.7.2 OPERATIVE DELIVERY OF THE NULLIPARA

It has been suggested in the recent literature (see section 6.7) that dystocia in first labours can be safely and effectively managed through a policy of active management. This possibility is tested in Table 6.11, which relates neonatal survival to augmentation and to delivery by forceps operation vis-à-vis emergency caesarean section when dystocia is the stated indication for operative delivery.

---

37 Following the onset of labour, the latent phase is characterized by slow dilation of the cervix. The latent phase ends when the cervix begins to dilate rapidly. This active phase continues until the end of the first stage of labour.

38 Another problem with the measurement of augmentation is the failure of some hospitals to include under this procedure artificial rupture of the membranes after the onset of labour (N.S.W. Department of Health, 1989b).
Dystocia is defined operationally as foetopelvic disproportion, obstructed labour, abnormality of the forces of labour and long labour.

The data suggest that dystocia is comparatively rare at low birthweights, and that most nulliparous women, especially at these birthweights (80.7 per cent, n=46), do not have their labours augmented for dystocia. Emergency caesarean section is most often performed (39.1 per cent, n=18) when labour is not augmented and leads to a low birthweight baby. By contrast, the forceps operation is undertaken for 83.3 per cent (n=65) of non-augmented labours leading to the delivery of a baby weighing 2 500 grams or more at birth.

Table 6.11: Relation of Neonatal Death to Type of Operative Delivery in Nulliparous, Dystocic women Aged 20 to 34 years according to Augmentation and Birthweight, 1986

<table>
<thead>
<tr>
<th>Augmtn.</th>
<th>Type of Operative Delivery</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>500-2499</td>
</tr>
<tr>
<td></td>
<td></td>
<td>P.D. Ctl. 0</td>
</tr>
<tr>
<td>No</td>
<td>Emergency caes. section</td>
<td>1 17 1.85</td>
</tr>
<tr>
<td></td>
<td>Forceps</td>
<td>2 26 2.42</td>
</tr>
<tr>
<td>Yes</td>
<td>Emergency caes. section</td>
<td>0 0 -</td>
</tr>
<tr>
<td></td>
<td>Forceps</td>
<td>1 10 3.15</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group
(Source. Maternal and Perinatal Collection, 1986)

Relative risk estimates lack stability. There are too few caesarean sections to yield meaningful estimates for comparison with those attending the forceps operation. However, one finding is that at low birthweights, forceps may be more hazardous (0=2.42) than a caesarean delivery (0=1.85) relative to the reference group of newborn weighing
over 2 499 grams who were delivered by forceps operation after no augmentation.

It is thus uncertain whether or not the forceps operation represents a safe alternative to caesarean section in the management of dystocia. The issue deserves research attention, which could also consider the effect of early gestation, a variable which, extrapolating from section 6.6, could make augmentation inadvisable at low birthweights.

Two further points should be made about the finding that dystocia is uncommon at low birthweights. The first is that it meshes nicely with the finding of Silbar (1986) that, in Chicago, the increased incidence of caesarean section for cephalopelvic disproportion may be secondary to an increase in birthweights; as noted in section 6.7, this is not, however, the usual explanation for rising disproportion-specific caesarean section rates. The second point is that since small babies tend not to be associated with dystocia, this indication for caesarean section is less likely to be strongly related to neonatal death than are other key indications for caesarean section like foetal distress. Moreover, because of the sampling design, low birthweight babies are also under-represented in the control group in Table 6.11.

Thus, even if it were safe to perform fewer emergency caesarean sections for dystocia, such a change, though it may be desirable, could be expected to have little effect on perinatal mortality. At the same time, caesarean sections themselves would become associated with increased rates of perinatal death. Whereas rising levels of caesarean section have appeared safe because arguably they subsume less valid indications, so first eliminating the least hazardous indications for this procedure would inevitably result in more perinatal deaths associated with caesarean section.
Because reducing primary caesarean incidence for dystocia, through increased augmentation and forceps assistance, will not eliminate many perinatal deaths, an important problem remains: to identify aetio logically important indications for caesarean section that, when associated with other delivery methods, yield outcomes better, or at least no worse, than caesarean section. In the group of perinatal deaths, two indications, previous uterine scar and malpresentation (see Table 6.12), may least justify elective and emergency caesarean section respectively. The data are not available to assess the safety of vaginal delivery following a previous caesarean section, but management of the breech presentation is now discussed in section 6.8.

**Table 6.12:** Most Common Indications for Caesarean Section among Neonatal Deaths

<table>
<thead>
<tr>
<th>Indication for Caesarean Section</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Emergency caesarean section</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early onset of labour</td>
<td>18</td>
<td>20.0</td>
</tr>
<tr>
<td>Foetal distress</td>
<td>17</td>
<td>18.9</td>
</tr>
<tr>
<td>Malpresentation</td>
<td>15</td>
<td>16.7</td>
</tr>
<tr>
<td>Other</td>
<td>40</td>
<td>44.4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>90</td>
<td>100.0</td>
</tr>
<tr>
<td><strong>Elective cesarean section</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Malpresentation</td>
<td>8</td>
<td>18.6</td>
</tr>
<tr>
<td>Hypertensive disease of pregnancy</td>
<td>8</td>
<td>18.6</td>
</tr>
<tr>
<td>Previous uterine scar</td>
<td>6</td>
<td>14.0</td>
</tr>
<tr>
<td>Other</td>
<td>21</td>
<td>48.8</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>43</td>
<td>100.0</td>
</tr>
</tbody>
</table>

*a* Each woman could have more than one indication. The mean numbers by caesarean type are 2.0 for the 45 women who had an emergency caesarean section, and 1.5 for the 29 women whose caesarean section was elective. These indications have been edited to ensure that multiple indications do not fall into the same disease category.

(Source. Maternal and Perinatal Collection, 1986)
There is a continuing debate on the optimal method of delivery for the breech baby. Some obstetricians incline toward caesarean section, whilst others advocate vaginal delivery in the absence of added complications. What some of these complications are, however, is a source of further controversy. Viewpoints differ over whether prematurity and multiple pregnancy, for example, contraindicate vaginal breech delivery.

Both complications increase the risks associated with vaginal breech delivery of perinatal death or morbidity caused by umbilical cord prolapse or compression, and by entrapment of the aftercoming head. Breech extractions performed quickly to avoid birth asphyxia can produce trauma. The caesarean section may avoid these dangers (although it requires manipulations on the foetus which can also cause damage and trauma) but the operation involves, for example, maternal risks such as infection, haemorrhage and, very rarely, death (and see section 2.4.3). There are thus potential conflicts between maternal and foetal interests, and these are compounded by uncertainty about the most appropriate method of delivering the baby.

Most studies (for example, Main et al., 1983; Doyle et al., 1985) demonstrate caesarean section for the very low birthweight breech to be associated with reduced perinatal mortality. However, other investigations, for example by Effer et al. (1983) and Bodmer et al. (1986), report no differences between the outcomes of vaginal and caesarean deliveries. Greater disagreement surrounds the management of twin pregnancies. Cetrulo et al. (1980) recommend caesarean section, especially to avoid the greater hazards sometimes imputed to breech extraction of the second twin. But studies have not consistently found added risks to attend the vaginal breech delivery
of first or second twins, compared either to singleton breech babies (Buekens et al. 1985) or, when the birthweight is above 1 499 grams,\(^{39}\) to delivery by caesarean section.

Table 6.13 contributes to these uncertainties by considering whether prematurity and plurality, and, with regard to the latter, also rank, compromise the safety and efficacy of the vaginal delivery of breech infants. Prematurity is defined operationally by birthweights below 2 500 grams, although, within this range, very low birthweight infants demand special consideration. The caesarean sections are indicated by complications in the mother or baby other than always only a breech delivery.

The decision not to adjust for confounding by other complications was based on two reasons, which exemplify the argument presented in section 6.5. First, the sample numbers would become too small; few of the breech babies that died were delivered by caesarean section only because of their breech presentations. Secondly, the caesarean-delivered women, who, because of their other complications (which frequently also contraindicate vaginal delivery) are expected a priori to be at usually higher perinatal mortality risk than the women having a vaginal breech delivery, still yield the lower estimated relative odds of perinatal death. However, this is withstanding the delivery of some babies per vaginam because, for example, of lethal congenital anomalies which are more frequent in breech than in non-breech babies (Confino, 1985). Small numbers do not permit adjustment for this effect except for very low birthweight singleton breeches, for whom estimates of relative risk confirm the finding of Table 6.13.

\(^{39}\) However, the second twin should not be significantly larger than the first twin, the maternal pelvis should be adequate and the foetal head, flexed.
Examination of this table reveals that among all breech births, caesarean section is the chosen method of delivery by a factor of 1.83 (77/42) at very low birthweights. No difference is found in the prevalence of the two delivery methods at birthweights 1 500 to 2 499 grams, while two-thirds (n=31) of the babies with birthweights 2 500 to 3 999 grams are delivered by caesarean section. An aggressive approach is thus apparently being taken toward salvaging the smallest babies, presumably because of improvements in neonatal care. However, some obstetricians in small or intermediate-sized hospitals may still be reluctant to perform a caesarean section for an extremely small baby, say 500 to 600 grams, that in their expectations would not survive (Suidan and Sayegh, 1989).

Table 6.13 Relation of Neonatal Death to the Type of Delivery of Breech Infants of Women Aged 20 to 34 Years according to Plurality, Rank and Birthweight, 1986

<table>
<thead>
<tr>
<th>Plurality</th>
<th>Type of Delivery</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-1 499</td>
<td>1 500-2 499</td>
</tr>
<tr>
<td></td>
<td>P.D. Ctl. θ</td>
<td>P.D. Ctl. θ</td>
</tr>
<tr>
<td>Singleton</td>
<td>Vag. Breech</td>
<td>19 6 14.97***</td>
</tr>
<tr>
<td></td>
<td>Caes. Section</td>
<td>11 52 1.00^a</td>
</tr>
<tr>
<td>Multiple</td>
<td>Vag. Breech</td>
<td>0 2 -</td>
</tr>
<tr>
<td></td>
<td>Caes. Section</td>
<td>1 3 1.58</td>
</tr>
<tr>
<td>Rank 1</td>
<td>Vag. Breech</td>
<td>7 8 4.14*</td>
</tr>
<tr>
<td></td>
<td>Caes. Section</td>
<td>2 8 1.18</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group
b Reference group for Mantel-Haenszel estimated odds ratios
* Statistically significant at the 0.05 level
*** Statistically significant at the 0.005 level
(Source. Maternal and Perinatal Collection, 1986)

This overall preference for caesarean delivery is not shown to extend to multiple breech births, about half of which involve caesarean section irrespective of the birthweight category. This same proportion of breech infants of multiple gestations has birthweights
500 to 1499, and 1500 to 2499 grams respectively. Moreover, multiple breech births account for 37.3 per cent (n=31) of all breech births at birthweights 1500 to 2499 grams, but only about one-quarter (n=31) of the very smallest breech neonates. The second twin or other multiple of second rank composes four-fifths (n=25) of the very low birthweight breeches resulting from multiple gestations, and is no less likely to be delivered vaginally than are larger breeches of multiple plurality. With weighting of the birthweight strata, the ratio of caesarean section to vaginal delivery is 2.1.

Coupled with these levels of exposure is evidence that caesarean section represents the safer method of delivery at all birthweights and pluralities, but most conspicuously for singleton neonates of very low birthweight. The odds ratio associated with these babies is 14.97 (p < 0.005) with vaginal delivery but 1.00 (the reference group) with caesarean delivery. Even after adjustment for plurality and rank, vaginal birth of the smallest breech baby yields a Mantel-Haenszel estimate almost eight times higher (TH=7.92) than the reference level of caesarean-delivered very low birthweight infants. When congenital anomalies are excluded, the estimated relative risk of perinatal death rises to 10.2 times higher with vaginal birth than caesarean section.

The danger of vaginal delivery at very low birthweights is also especially apparent for multiple births of second rank; the increase in risk, compared to caesarean-delivered breech babies of equivalent plurality and rank is about three and a half times, the risk differential decreasing to 2.6 after adjustment for birthweight. This suggests the possible error of the preference to deliver vaginally multiple very low birthweight breeches, or at least not to deliver them by caesarean section.
Increases in birthweight improve survival chances with vaginal delivery, yet at no birthweight do these risks fall below those associated with caesarean section, which also fall, more consistently so, with increasing birthweights. Among singleton babies, vaginal delivery is 2.4 and 4.6 times more hazardous than is caesarean delivery at birthweights 1 500 to 2 499 grams and more than 2 499 grams respectively. Both vaginal and caesarean deliveries appear safe for neonates with birthweights above 1 499 grams. After adjustment for birthweight, delivery by caesarean section yields estimated relative odds of perinatal death that are still almost eight times lower ($\theta_{mn}$=7.85) for singletons and 2.6 times lower for multiple breeches of second rank.

The evidence tentatively supports an ameliorative effect of caesarean section on the outcome chances, in particular, of very low birthweight breech babies, irrespective of their plurality although especially when singletons. Caesarean section is also apparently safer for singleton breech neonates at all birthweights and for very low birthweight multiple breeches of second rank.

6.9  CONCLUSION

The health care received by pregnant women and their babies is an important determinant of risks for an adverse perinatal outcome. In this chapter such care was conceptualized, first, by focusing on the place of birth and on geographic mobility and then, in more explicitly temporal terms, by emphasizing the amount and type of care received during pregnancy and confinement.

The success of a regionalized perinatal health system, as in New South Wales, depends on the abilities of hospitals to match patients with appropriate levels of maternity and neonatal care. One outcome of
screening and referral decisions is the place of birth; another is the unit providing postnatal care. In terms of the former outcome, this chapter found the smallest hospitals to manage comparatively few births, especially at very low birthweights and, except at these birthweights, to yield neonatal outcomes that are as safe as at higher level hospitals.

Level 2 hospitals were estimated to manage almost one-third of all deliveries of very low birthweight babies, also at a higher risk than at level 3 units. The latter finding was discussed in terms of constraints to antenatal transfer, the most important apparently being insufficient specialist neonatal services for critically ill neonates, and high demand for confinements in level 3 units of the mothers who could be effectively managed elsewhere. An important mechanism underlying the locus of infant care is postnatal transfer which was found to be surprisingly safe.

The second part of the chapter began by considering the perinatal mortality risks associated with a small amount of antenatal care. Collection data showed that fewer than two visits was uncommon and, at ages 20 to 34 years, also hazardous for newborn weighing at least 1 500 grams. At other maternal ages, 0 or 1 visit increased the relative risk of perinatal death for very low birthweight babies.

Results based on survey data estimated that a comparatively small percentage (17.1 per cent, n=37) of the mothers of low birthweight babies made 10 or more antenatal visits but that for these women, this largest amount of care attended the highest relative risk of perinatal death. These women received much care probably because they are high-risk candidates for an adverse outcome and because some antenatal care may be inefficacious. At normal birthweights fewer than six visits appeared particularly hazardous.
For neonates, comparisons were made between selected methods of managing confinements. First, the risks of induction were compared with no labour, that is, with elective caesarean section. From 37 gestational weeks, the women who received inductions recorded lower estimated relative risks of neonatal death if their newborn weighed 1 500 to 2 499 grams but not 2 500 to 3 999 grams. Before term, higher neonatal mortality was associated with the 30 per cent (n=50) of low birthweight infants whose mothers were induced than with the heavier babies electively delivered by caesarean section. It appears that either the procedure of induction is hazardous or, much less likely, the women receiving inductions were at greater risk than those delivered by caesarean section. Induction by medical means, alone or in combination with amniotomy, is possibly less safe than amniotomy alone.

Attention was subsequently given to assessment of the effect of parity on appropriate management of the nonprogressive labour. Results suggested over-reporting of multiparae. Results neither confirmed nor denied that in nulliparae, the active management of labour is a safe alternative to caesarean section for dystocia; consideration was not given to the effect of preterm gestation which, extrapolating from the discussion of induction, may make augmentation inadvisable at low birthweights. However, at these birthweights, dystocia was shown to be comparatively rare, suggesting that reducing caesarean sections for this indication would have little effect on perinatal mortality. Finally, for breech infants, caesarean section was estimated to yield lower relative odds of neonatal death than did vaginal delivery, especially at very low birthweights. Caesarean section was also shown, on less convincing evidence, to be safer than vaginal delivery for singleton breeches at higher birthweights and for very low birthweight multiple breech babies of second rank.
7.0 INTRODUCTION

Pregnancy health factors are psychological and behavioural factors, or stressors, describing maternal pregnancy health (see sections 2.8 to 2.8.5). This chapter relates perinatal death to three sets of these stressors: psychological stress, physical stress through exercise and work, and chemical stress via tobacco smoking. For each set, as it operates in the expectant mother, the mediating role of birthweight is first briefly considered (see section 2.2.1). Then, consideration is given to whether the aetiological effects of the stressors persist only among low birthweight babies.

There are a priori reasons for hypothesizing persistence of the relation between these stressors and perinatal death in low birthweight babies. In any foetus, severe diminution in uterine and umbilical blood flow can lead to acute asphyxia possibly causing death or, less commonly, brain injury (Adamsons and Myers, 1975). Usually, because the foetus possesses an excellent supply line, eliciting such effects requires substantial interference with the uterine circulation. However, foetal stress is unlikely to be an absolute event determined only by the character of the insult. Presumably the status and responsiveness of the foetus are also important (Reece et al., 1986) which may be less than ideal when growth retardation or preterm birth yield low birthweights.
The growth retarded and thus chronically stressed foetus is less able to withstand ‘normal’ stresses during pregnancy. A moderate reduction in blood flow may induce acute hypoxia which, superimposed on a subclinically chronic hypoxic state, may lead to severe metabolic and heart rate abnormality (Reece et al., 1986). The preterm foetus is at similar risk because the relative lack of foetal reserves may be rapidly depleted by repetitive stress (Gimovsky and Bruce, 1986).

Moreover, there are two other mediating variables through which pregnancy health factors may contribute to the perinatal deaths of babies with low birthweights. The first variable is the teratogenic effect, especially during the first trimester of pregnancy, of, for example, hyperthermia produced by unsafe exercise, and perhaps tobacco smoking. Some embryos and foetuses exposed thereto may abort spontaneously; others may die during the perinatal period or later, or suffer, for example, neurological damage. A second intermediate variable is obstetric complications other than those typically associated with a reduction in birthweight. For example, horseback riding has been linked to antepartum haemorrhage (pers comm., M. O’Neill, 1989).

Nevertheless, treating low birthweight as a categorical variable introduces the possibility that exposures affect the distribution of low birthweights within birthweight categories. Resulting outcome differences, produced by moderate reductions in uterine and placental blood flow over a long period, would then, at least in part, be mediated through these distributional differences. As in previous chapters, this observation is not empirically tested, except in section 7.3 on smoking.

1 For example, exercise is likely to be contraindicated in the pre-eclamptic woman.
2 This is defined in sections 2.8.2 and 7.2.2.
The interest in low birthweight is a result of the high incidence of low birthweight among the interviewed women. The mean birthweights are 1209 grams in the case group (s=600 grams) and 1525 grams in the control group (s=504). It follows that almost all the low birthweight babies in both groups are preterm, this outcome characterizing 86.3 per cent (n=44) of the case group and 90 per cent (n=36) of the control group. The mean gestational ages of the case and control groups are 29 and 31 weeks respectively.

Focusing, for the most part, only on infants weighing less than 2 500 grams, and also on women aged 20 to 34 years (see section 3.5), reduces the already small sample sizes; thus some potentially important covariates such as the gravidity of smokers cannot be adjusted for. As noted above, newborn weighing above 2 499 grams enter precursory discussions that relate individual exposures broadly to birthweight. But perinatal outcome in low birthweight babies, or at least the contribution of stressors thereto, is the primary focus.

7.1. PSYCHOLOGICAL STRESS

Psychological stress (see section 2.8.1) has two components each of which may lack change or have dynamic aspects: these components are exposure to stimuli in the immediate social environment, and response to these stimuli in terms of personality factors and social context. Life-events research is one way of relating exposures in the environment to the stress response, which may be conceptualized, for example, as state anxiety or trait anxiety (see section 2.8.1).

Contradictory findings hamper efforts to evaluate the life events-pregnancy outcome relation. Some studies find that obstetric complications attend no increase in life events (Chalmers, 1983; Stein et al., 1987) or fewer such events (Jones, 1978; Omer et al., 1986b).
Other researchers show life events to contribute to obstetric complications such as, usually, conditions associated with low birthweight (Newton and Hunt, 1984) or preterm labour (Newton et al., 1979; Berkowitz, 1983).

Life events have also been linked, for example, to congenital malformations (McNeil et al., 1988), but not directly to perinatal death even though this last outcome can follow sudden, marked interference in the maternal blood supply to the foetus (Adamsons and Myers, 1975), perhaps secondary to acute psychological stress. In poorly compensated foetuses, even chronic stress of lesser magnitude may lead to death (Reece et al., 1986).

The mixed findings of existing studies may reflect methodological differences. However, inconsistent results are found, first, among as well as across retrospective and prospective investigations, even by the same author (e.g., Omer et al., 1986b). Results differ, secondly, although to a lesser extent, when the pregnant woman's social support is accounted for. Stress buffering effects are commonly linked to received and especially perceived support (Aaronson, 1989), despite some studies such as Omer et al. (1986a) showing even perceived support to produce no difference in obstetrical outcome. Disparities may be better explained by the substantial methodological problems inherent in life-event research. These problems, which also affect the reporting of other pregnancy health factors, are discussed in section 3.7.2.

In this investigation a life events inventory was developed from the scale produced for obstetric groups by Barnett et al. (1983). A total list was developed of 45 pregnancy-specific and general, but non-trivial, items (see Appendix 5). Of these items, 36 relate to all women, irrespective of perinatal outcome. Nine items, which pertain
to labour and delivery, do not apply to the women who experienced a stillbirth.

All the items seek to represent the potential experiences of women during pregnancy\(^3\) and confinement. Through its diversity and intended relevance, the inventory further aspires to limit selectivity effects associated with favouring events to which some subjects may be predisposed. To the extent that these effects occur, they may reflect social differences in the probability of experiencing life events (Henderson et al., 1981).

The inventory was read to every subject who tried to recall, first, whether she had experienced each event during her most recent pregnancy ending in a 1986 confinement. Secondly, if the event did happen to her, she was asked to recall its month(s) of occurrence during the pregnancy. In this manner a matrix of life-events was constructed for each respondent, identifying event frequency and timing.

Subjects were also asked to rate the importance of each reported event using a visual analogue scale of increasing distress\(^4\) from 0 to 10. The rating measures the most distress suffered at the time the event occurred, irrespective of the duration of the event. No attempt was made to evaluate changing levels of distress over time; subjective measurements are especially liable to recall inaccuracies and the subject's immediate affective state.

Besides other potential sources of contamination (see section 3.7.2), three assumptions underlie the number, and the distress rating, of reported items. First, many life events are not fortuitous; they are

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\(^3\) Chalmers (1983) suggests that life events before the pregnancy can also influence birth outcome.

\(^4\) Of course, distress is just one manifestation of stress; another may be anger.
responses to continuing human behaviour (Byrne, 1983). Secondly, all life events are not equally and intrinsically noxious. Their emotional impact depends on interpretative factors that operate in cognitive and affective frameworks unique to the individual. Thirdly, distress rather than life change best represents the mediating nexus between life events and illness. That is, the emotional undesirability of life events, rather than the extent to which these events demand personal adjustment of the individual, is the more suitable measure of impact (Mueller et al., 1977; Henderson et al., 1981).

Perinatal death among low birthweight babies is first related to one measure of subjects' overall levels of psychological stress: their total distress score on pregnancy items. This measure, which is the sum of individual distress ratings on these items, excludes items pertaining to labour and delivery; these items, as noted above, were not asked of women who experienced a stillbirth.

In Table 7.0, which uses three arbitrary categories of distress, proportions of women fall with progressively higher total distress scores and, taking each score separately, with increasing birthweight. After weighting, about half (n=33 065) of the women have distress scores 0-15, this proportion rising slightly with increasing birthweights. Concomitantly, the proportion with the highest total distress scores (30+) is about two times greater in each birthweight stratum below 2 500 grams than at normal birthweights. In the weighted sample, the highest percentage of low birthweight babies, 17.9 per cent (n=1 151), is associated with scores of 30 or more, 10 per cent (n=6 444) of women being estimated to occupy this highest category of the total distress score.
The case group is conspicuous by its low distress scores, while scores greater than 15 are a salient feature of the control group. The result is an inverse relation between estimated odds ratios and distress. Taking the reference group as having the low total distress scores of 0-15, and adjusting for birthweight, scores above 15 reduce estimates of the relative risk of death by more than half.

Table 7.0: Relation of Perinatal Death to Total Distress Score\(^a\) of Women Aged 20 to 34 Years according to Birthweight, 1986

<table>
<thead>
<tr>
<th>Total Distress Score</th>
<th>Birthweight (grams)</th>
<th>500-1 499</th>
<th>1 500-2 499</th>
<th>2 500-3 999</th>
<th>4000+</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P.D. Ctl. (\theta)</td>
<td>P.D. Ctl. (\theta)</td>
<td>P.D. Ctl. (\theta)</td>
<td>P.D. Ctl. (\theta_{mh})</td>
<td></td>
</tr>
<tr>
<td>0-15(^b)</td>
<td>20</td>
<td>9</td>
<td>2.67</td>
<td>12</td>
<td>6</td>
</tr>
<tr>
<td>16-29</td>
<td>8</td>
<td>7</td>
<td>1.35</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>30+</td>
<td>7</td>
<td>6</td>
<td>1.40</td>
<td>2</td>
<td>6</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
\(a\) These scores do not include items about the labour and delivery - which do not apply to stillbirths.
\(b\) Reference group for Mantel-Haenszel odds ratios
\(c\) Reference group for other odds ratios
(Source. Interview Data, 1986; Hospital Patients' Records, 1986)

It is at low birthweights that death appears most strongly associated with minimal distress in the mother. Compared with scores above 15, low distress increases estimates of the odds ratios by a factor of two at birthweights less than 1 500 grams, and by a factor of six at birthweights 1 500 to 2 499 grams; the reference group is babies of normal birthweight born to women with low distress scores. Sparse data show newborn of normal birthweight more, and at least no less, likely to die if their mothers are in the highest distress score category (\(\theta=1.20\)).

Overall, the higher total distress scores of the control group add to existing uncertainties about the life events-obstetric complications
relation. However, this distress-survival finding is less aberrant than novel, for existing studies, for example by Newton and Hunt (1979), have focused on physiological immaturity rather than perinatal death as the outcome variable. Moreover, for newborn weighing 2 500 grams or more, minimal distress is not shown to increase the relative risk of death.

Of course, this does not explain why low total distress occurs even among low birthweight newborn, and why this smallest amount of distress is most hazardous for low birthweight babies. Physiological mechanisms at work, influenced, but not mediated, by birthweight, were earlier postulated. There may be unknown confounding factors, whilst higher, if still low, maternal distress levels may stimulate foetal growth and maturity. Curet et al. (1983) postulated a similar mechanism of chronic stress in the foetus to account for a reduced incidence of respiratory distress syndrome when the woman smoked tobacco during pregnancy.

Some light is cast by comparing levels of life event duration between the case and control groups. Despite similar mean durations in months - 2.84 for the case group and 2.72 for the control group - the between-women variance for the control group, 2.29, exceeds that for the case group by 1.11. Events of shorter and longer duration than each mean may thus offer some survival protection to low birthweight babies. Little assistance is given by group differences in the mean total distress score. For small babies, the total distress score in the mother averages at 4.60 for the case group and 4.75 for the control group, the between-women variances being 4.82 and 4.68 respectively. Among low birthweight babies, the highest total distress scores yield higher mean (low) birthweights than do lower
distress scores, but the difference is not statistically significant at the 0.05 level.

A further possibility to be entertained is that of data contamination owing to denial of events by the case group or 'effort after meaning' by the control group. Yet perinatal death, or at least its stated cause, is often inexplicable ipso facto because nothing out of the ordinary precedes it. Also, it is unclear why over-reporting, to the extent it occurs, should characterize the control group much more than the case group.

Finally, no account is taken of differences in the timing of these aggregated events, or of the unique effects of individual events. Table 7.1 explores the latter ingredient, exclusively for low birthweight babies by relating item-specific distress scores to the frequency of events reported by women. Selected items are later examined with respect to when and for how long they occurred during the pregnancy.

Table 7.1 shows only three items that are frequently reported and distressing, with both characteristics being more strongly associated with perinatal death relative to reference groups signifying the occurrence and reported distress of the procedure of an ultrasound examination. The three items meeting these criteria are almost miscarrying - which, of course, would reflect an underlying medical problem - and the eventuation during labour of medical complications and pain.

5 At least 10 per cent of women in one or both groups report an item with a mean distress rating of 5 or more.
Table 7.1: Relation of Perinatal Death of Low Birthweight Infants to Maternal Life Experiences according to their Frequency and Mean Distress Ratings by Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Maternal Life Experiences</th>
<th>Times reported(^a)</th>
<th>Distress rating</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P.D. No.</td>
<td>%</td>
</tr>
<tr>
<td>Work situation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>You were unemployed or seeking work</td>
<td>5</td>
<td>9.8</td>
</tr>
<tr>
<td>You started a new job</td>
<td>3</td>
<td>5.9</td>
</tr>
<tr>
<td>There was a big change in the people, duties, hours or responsibilities at your work</td>
<td>4</td>
<td>7.9</td>
</tr>
<tr>
<td>You were sacked or laid off</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Your husband/partner became unemployed</td>
<td>5</td>
<td>9.8</td>
</tr>
<tr>
<td>Your husband's/partner's business failed</td>
<td>2</td>
<td>3.9</td>
</tr>
<tr>
<td>Living conditions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A new person came to live in your household</td>
<td>3</td>
<td>5.9</td>
</tr>
<tr>
<td>You moved house</td>
<td>7</td>
<td>13.7</td>
</tr>
<tr>
<td>You had financial problems</td>
<td>11</td>
<td>21.6</td>
</tr>
<tr>
<td>Someone close to you developed a serious illness</td>
<td>3</td>
<td>5.9</td>
</tr>
<tr>
<td>You were involved in a traffic accident in which someone was badly injured</td>
<td>2</td>
<td>3.9</td>
</tr>
<tr>
<td>You were involved in a legal action that could have damaged, or did damage, your reputation</td>
<td>0</td>
<td>0.0</td>
</tr>
<tr>
<td>Relationships</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Your husband/partner did not want you to become pregnant</td>
<td>4</td>
<td>7.8</td>
</tr>
<tr>
<td>The behaviour of your husband/partner was a problem to you</td>
<td>9</td>
<td>17.6</td>
</tr>
</tbody>
</table>

Continued overleaf
<table>
<thead>
<tr>
<th>Maternal Life Experiences</th>
<th>Times reporteda</th>
<th>Distress rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serious arguments developed between you and your husband/partner</td>
<td>9 17.6 8 20.0 0.93</td>
<td>7.6 2.4 7.3 2.5 1.19</td>
</tr>
<tr>
<td>Your husband/partner told you that he no longer loved you</td>
<td>2 3.9 1 2.5 1.64</td>
<td>7.5 0.7 9.0 - 0.95</td>
</tr>
<tr>
<td>You had sexual difficulties</td>
<td>6 11.8 5 12.5 0.98</td>
<td>2.8 1.5 4.8 3.0 0.67</td>
</tr>
<tr>
<td>Your husband/partner was unfaithful</td>
<td>0 0.0 0 0.0 -</td>
<td>- - - - -</td>
</tr>
<tr>
<td>You were unfaithful</td>
<td>0 0.0 0 0.0 -</td>
<td>- - - - -</td>
</tr>
<tr>
<td>Serious arguments developed between you and someone close to you (not husband/partner)</td>
<td>6 11.8 8 20.0 0.62</td>
<td>5.8 3.5 6.8 2.3 0.97</td>
</tr>
<tr>
<td>Separations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>You separated from, or divorced your husband/partner</td>
<td>2 3.9 4 10.0 0.41</td>
<td>9.5 0.7 6.5 4.4 1.67</td>
</tr>
<tr>
<td>You were separated from a close family friend</td>
<td>1 2.0 1 2.5 0.82</td>
<td>5.0 - 4.0 - 1.43</td>
</tr>
<tr>
<td>Bereavements</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Your child died (not baby)</td>
<td>0 0.0 0 0.0 -</td>
<td>- - - - -</td>
</tr>
<tr>
<td>Your husband/partner died</td>
<td>0 0.0 0 0.0 -</td>
<td>- - - - -</td>
</tr>
<tr>
<td>Someone close to you died (not child or husband/partner)</td>
<td>9 17.6 7 17.5 1.06</td>
<td>7.9 2.0 8.9 2.0 1.01</td>
</tr>
<tr>
<td>Pregnancy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Your pregnancy was not planned</td>
<td>10 19.6 13 32.5 0.63</td>
<td>1.7 2.9 2.5 3.3 0.78</td>
</tr>
<tr>
<td>You took something during the pregnancy (e.g., medication, alcohol, cigarettes) that might harm your baby</td>
<td>12 23.5 13 32.5 0.76</td>
<td>3.4 2.0 2.0 2.6 1.94</td>
</tr>
<tr>
<td>You had an X-ray during the pregnancy</td>
<td>2 3.9 2 5.0 0.82</td>
<td>2.0 2.8 1.0 1.4 2.29</td>
</tr>
</tbody>
</table>

Continued overleaf
<table>
<thead>
<tr>
<th>Maternal Life Experiences</th>
<th>Times reported&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Distress rating</th>
<th>P.D.</th>
<th>Ctl.</th>
<th>P.D.</th>
<th>Ctl.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. %</td>
<td>No. %</td>
<td>gb</td>
<td></td>
<td>gb</td>
<td></td>
</tr>
<tr>
<td>You had an ultrasound investigation during the pregnancy</td>
<td>45 88.2</td>
<td>37 92.5</td>
<td>1.00&lt;sup&gt;d&lt;/sup&gt;</td>
<td>0.7</td>
<td>2.2</td>
<td>0.8</td>
</tr>
<tr>
<td>You were in contact during the pregnancy with someone who had an infectious disease that might harm your baby</td>
<td>2 3.9</td>
<td>5 12.5</td>
<td>0.33</td>
<td>0.5</td>
<td>0.7</td>
<td>5.4</td>
</tr>
<tr>
<td>You almost miscarried</td>
<td>11 21.6</td>
<td>8 20.0</td>
<td>1.13</td>
<td>8.5</td>
<td>2.6</td>
<td>8.5</td>
</tr>
<tr>
<td>You were seriously ill during the pregnancy</td>
<td>2 3.9</td>
<td>5 12.5</td>
<td>0.33</td>
<td>9.0</td>
<td>1.4</td>
<td>8.8</td>
</tr>
<tr>
<td>You had severe morning sickness</td>
<td>10 19.6</td>
<td>10 25.0</td>
<td>0.82</td>
<td>6.4</td>
<td>3.2</td>
<td>4.8</td>
</tr>
<tr>
<td>You had blood pressure problems during the pregnancy</td>
<td>10 19.6</td>
<td>16 40.0</td>
<td>0.51</td>
<td>6.6</td>
<td>3.4</td>
<td>6.8</td>
</tr>
<tr>
<td>Your doctor said you were going to have twins</td>
<td>4 7.8</td>
<td>5 12.5</td>
<td>0.66</td>
<td>2.0</td>
<td>2.2</td>
<td>0.4</td>
</tr>
<tr>
<td>You had difficulty arranging for someone to look after your family whilst in hospital</td>
<td>3 5.9</td>
<td>4 10.0</td>
<td>0.62</td>
<td>5.7</td>
<td>3.1</td>
<td>6.5</td>
</tr>
<tr>
<td>Labour and delivery</td>
<td>2 11.8</td>
<td>3 7.5</td>
<td>1.64</td>
<td>4.5</td>
<td>2.1</td>
<td>4.3</td>
</tr>
<tr>
<td>The doctor made your baby come early</td>
<td>6 35.3</td>
<td>7 17.5</td>
<td>2.11</td>
<td>9.3</td>
<td>1.6</td>
<td>6.0</td>
</tr>
<tr>
<td>Your labour was very painful</td>
<td>2 11.8</td>
<td>6 15.0</td>
<td>0.82</td>
<td>1.0</td>
<td>0.0</td>
<td>3.0</td>
</tr>
<tr>
<td>Your husband/partner was not present at the labour</td>
<td>2 11.8</td>
<td>2 5.0</td>
<td>2.47</td>
<td>10.0</td>
<td>0.0</td>
<td>4.5</td>
</tr>
<tr>
<td>Medical complications arose during the labour</td>
<td>5 29.4</td>
<td>18 45.0</td>
<td>0.69</td>
<td>4.0</td>
<td>5.1</td>
<td>1.4</td>
</tr>
<tr>
<td>Your doctor was not present at the labour</td>
<td>3 17.6</td>
<td>9 22.5</td>
<td>0.82</td>
<td>0.0</td>
<td>0.0</td>
<td>0.2</td>
</tr>
</tbody>
</table>

Continued overleaf
Hospitals’ Patients’ Records, 1986)

Pain during labour, for example, is typically distressing for the parturient woman, irrespective of perinatal outcome. However, relative to the mean distress attending an ultrasound, the distress produced by a painful labour is more strongly associated with perinatal death ($\theta=1.77$). The item is also frequently reported, especially by the women who lost their baby; the differential exceeds that for the reference group who had an ultrasound by two times ($\theta=2.11$).

Similar characteristics are associated with starting a new job, and the failure of the husband’s or partner’s business. Distressing, especially to the case group, these life experience are uncommon. But they are more so in the control group, yielding odds ratios of 2.47 and 1.64 respectively, relative to having an ultrasound. Other,
frequent life events, which likewise produce high absolute and relative levels of mean distress, occur more often in the control group. Focusing on their distress ratings, preterm birth ($\theta=2.16$) and caesarean section ($\theta=1.79$) record the two highest estimates of the relative risk of death. Also conspicuous are three items describing the marriage. These items are separation from, serious arguments with, and problematic behaviour by the husband or partner.

Of further note are low mean distress scores that also produce high estimates of the relative odds of death. Differences among these items relate to their frequency of occurrence. More strongly characteristic of the case group are house-moving ($\theta=1.15$), being unemployed or seeking work ($\theta=1.37$), and induction of labour ($\theta=1.64$). The second of these items does not fulfil the criterion of frequent occurrence. Other events such as the doctor being absent from the labour were commonly reported but no more so by the case group. Relatively unusual and less prevalent in the case group are expecting twins ($\theta=0.66$) and having an X-ray ($\theta=0.82$).

Compared to the ultrasound procedure, almost all life events cause the case group more distress than the control group. Events that are rarely reported, such as deaths, permit no such assessment or possibly unreliable, low odds ratios - as also, those above 1.0. However, some, if limited, evidence suggests that the main locus of less distress to the case group is items describing personal relationships: for example, the husband or partner not wanting the pregnancy, and sexual difficulties.

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6 Because starting a new job yields a mean distress rating of zero in the control group, no odds ratio is estimated based on the magnitude of distress reported for this item relative to the reference group. However, as the indicated zero is a structural zero and not a sampling zero, an increased relative risk of death is apparent. For the same reason, a reduction in risk may be noted for an episiotomy.
The remaining discussion of life events centres on items collectively describing the family work situation. The foregoing discussion identified their possibly important contribution to perinatal death, and maternal work is studied further in section 7.2.1 with reference to physical stress. Given this larger interest in work during pregnancy, the duration and timing of aggregated work events are now examined.

Table 7.2 shows that about half of the work events (n=16) receive ratings of 7 to 10. This proportion differs little by perinatal outcome, and is increased by events of longer than three months duration compared to shorter events. Most work events (n=24, 72.7 per cent) last less than three months, and these events show an inverse U pattern of risks with increasing distress ratings: the safety of scores between 0 and 3 (the reference group) is diminished especially by scores from 4 to 6 (0=42.0). Higher ratings yield a lower but still elevated odds ratio of 4.8 relative to the reference group, and this estimate is 3.1 times lower than that for longer-lasting events rated 7 to 10 (0=15.0).

Categories describing the timing of work events are not mutually exclusive; events may occur in one category, or in two or three categories. Work events are slightly more likely to happen in the last trimester, especially for the case group (59.3 per cent, n=16) than in the first trimester, while events continuing throughout the complete pregnancy are least common and affect only the case group.

Compared to scores greater than 3, smaller ratings lower the risk of death in the last trimester (the reference group) and especially the first trimester (0=0.25). Higher ratings, particularly between 4 and 6, are most hazardous, more so in the last trimester. Greater danger
attends scores of 7 to 10 in the first trimester ($\theta=2.00$) than in the last trimester ($\theta=1.40$).

Distressing life events were uncommon, especially among the women who lost babies, and minimal distress was found more hazardous than higher levels of reported distress. Threatened miscarriage and difficulties during the confinement were most frequent and distressing in the group of women that experienced perinatal deaths.

Table 7.2: Relation of Perinatal Death of Low Birthweight Infants to the Duration and Timing of Reported Work Events according to their Distress Ratings by Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Work Events</th>
<th>Distress Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Duration (months)</td>
</tr>
<tr>
<td></td>
<td>P.D. Ctl. $\theta$</td>
</tr>
<tr>
<td>First trimester</td>
<td>1-3</td>
</tr>
<tr>
<td></td>
<td>4+</td>
</tr>
<tr>
<td>Last trimester$^a$</td>
<td>1-3</td>
</tr>
<tr>
<td></td>
<td>4+</td>
</tr>
<tr>
<td>Complete trimester</td>
<td>1-3</td>
</tr>
<tr>
<td></td>
<td>4+</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
$^a$ Reference group for other odds ratios
* Statistically significant at the 0.05 level
(Source. Interview data, 1986; Hospital Patients' Records, 1986)

7.2 PHYSICAL STRESS

Two agents of maternal physical stress during pregnancy - work and exercise - may contribute to perinatal death. Work, in the labour force or involving domestic duties, is usually non-leisure based and subsumes an economic imperative. Exercise, ostensibly salubrious, is typically a recreational endeavour undertaken through choice. Contrasting physiological effects on the foetus, consequent on these
differences, are manifest most importantly in the effects of each agent of reducing the uterine and placental blood flow.

Exercise is more likely than work to cause a reduction in blood flow of greater magnitude but, almost certainly, shorter duration. However, exercising too frequently can also produce chronic stress, whilst acute stress can result from work. Possible sequelae are interference of foetal growth and development, and especially in the foetus so afflicted, death in utero.

7.2.1 MATERNAL WORK

As discussed in section 2.8.2, work per se during pregnancy does not appear to affect perinatal outcome adversely. Particularly relevant to this conclusion is that the women who do physically arduous or tiring work are also likely to be exposed to other risk factors such as tobacco smoking and low socioeconomic status. In the analysis below, small interview numbers do not permit statistical adjustment for most of these confounding influences.

However, this should not detract too much from its ability to identify paid work as a risk factor for perinatal death, multivariate analyses (e.g. Saurel-Cubizolles et al., 1984) having elsewhere shown various exposures to physical stress in the paid workforce to accompany increased perinatal risks, for example, of preterm birth and reduced birthweights; that is, after adjustment for factors such as the husband's social class, and maternal age and parity. Accordingly, failure to adjust for such factors probably increases the danger of overestimating the magnitude rather than of mis-stating the direction of risks. The following discussion of the effects of work in the home has considerably less literature to substantiate its conclusions but,
to some extent, evidence of the effects of physical stress in the workforce should be generalizable to work at home.

Moreover, what is at issue here is not only whether, but also why, specific work conditions, which may produce physical fatigue, are hazardous for the baby: is foetal immaturity sometimes less the result of work than a modulating influence on its impact? That is, is some work hazardous when only small babies are selected for study? This theme of the chapter guides the discussion of the risks for perinatal death of exposure to the conditions in the paid workforce as well as to the often additional work women perform in the home. Both issues are explored in turn.

First, participation in the workforce during pregnancy is related in Table 7.3 to birthweight-specific estimates of the prevalence and relative risks of perinatal death. While Glezer (1988) found that 45.8 per cent (n=921) of her sample worked during pregnancy, in the present study, workforce participation characterizes 71.7 per cent (n=44 362) and 64.3 per cent (n=72) of the weighted and unweighted samples respectively. On a priori grounds the high proportion of low birthweight babies is itself suggestive of more pregnant women working, but paid work is actually more prevalent among the small number of women bearing normal birthweight infants (68.4 per cent, n=13) than among the mothers of low birthweight newborn (63.7 per cent, n=58). Out of all working women, the proportion delivering newborn in each birthweight category approximates the relative contribution of that category to the overall sample.

Relative to no maternal work, Mantel-Haenszel estimates are in the expected direction. They suggest for all babies, a slight reduction

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7 This comprises different occupations or types of work, which each involve sets of common jobs or tasks performed by workers in given establishments.
in risk ($\Theta_{mh}=0.96$), and for newborn of low birthweight, a small elevation in risk ($\Theta_{mh}=1.05$), associated with maternal work. The latter finding appears to hold only for newborn weighing 1 500 to 2 499 grams at birth. To help clarify any aetiological effects of work, less obviously mediated by birthweight, different working conditions are related to perinatal outcome for babies of low birthweight only.

Table 7.3: Relation of Perinatal Death to Maternal Work by Women Aged 20 to 34 Years according to Birthweight, 1986

<table>
<thead>
<tr>
<th>Work during Pregnancy</th>
<th>Birthweight (grams)</th>
<th>P.D.</th>
<th>Ctl.</th>
<th>P.D.</th>
<th>Ctl.</th>
<th>P.D.</th>
<th>Ctl.</th>
<th>P.D.</th>
<th>Ctl.</th>
<th>$\Theta_{mh}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>500-1 499</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>23</td>
<td>15</td>
<td>1.53</td>
<td>10</td>
<td>10</td>
<td>1.00</td>
<td>5</td>
<td>8</td>
<td>0.63</td>
<td>1</td>
</tr>
<tr>
<td>No$^a$</td>
<td>12</td>
<td>7</td>
<td>1.71</td>
<td>6</td>
<td>8</td>
<td>0.75</td>
<td>3</td>
<td>3$^b$</td>
<td>1.00</td>
<td>1</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
$^a$ Reference group for Mantel-Haenszel estimated odds ratios
$^b$ Reference group for other odds ratios
(Source: Interview data, 1986; Hospital Patients' Records, 1986)

Conditions of work are measured, first indirectly, by relating different occupational groups to perinatal outcome, and then directly, by comparing occupational groups by outcome across work circumstances, and work conditions shown to be potentially hazardous by the literature. The former measurements are shown in Table 7.4, based on eight major groups which form the broadest level of occupational aggregation in the Australian Standard Classification of Occupations (ASCO). Boundaries between these groups are set mainly in terms of skill level.$^8$

$^8$ Skill levels describe the range and complexity of duties involved in an occupation. The ability to perform these duties is a function of formal education, on-the-job training and previous experience (Department of Employment and Industrial Relations, and Australian Bureau of Statistics, 1987).
The table shows a much greater mix of occupational groups in the case group. One result is that clerks, the most common group irrespective of perinatal outcome, account for three-fifths \( (n=15) \) of the control group, but one-third \( (n=11) \) of the case group. This is reflected in an odds ratio of 0.73, taking the reference group as managers and administrators. Professionals, the second most frequent group, cannot account for the deficit; so that with clerks, they describe 80 percent \( (n=20) \) of the mothers of surviving babies, but approximately half \( (n=17) \) of the mothers of dead babies. Para-professionals \( (0=5.00) \) and labourers and associated workers are the conspicuous occupations in the case group.

Table 7.4: Relation of Perinatal Death of Low Birthweight Infants to the Main Occupation of Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Occupation</th>
<th>P.D.</th>
<th>Ctl.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Managers and administrators</td>
<td>1</td>
<td>3.0</td>
</tr>
<tr>
<td>Professionals</td>
<td>6</td>
<td>18.2</td>
</tr>
<tr>
<td>Para-professionals</td>
<td>5</td>
<td>15.2</td>
</tr>
<tr>
<td>Tradeswomen</td>
<td>1</td>
<td>3.0</td>
</tr>
<tr>
<td>Clerks</td>
<td>11</td>
<td>33.3</td>
</tr>
<tr>
<td>Saleswomen and personal service workers</td>
<td>4</td>
<td>12.1</td>
</tr>
<tr>
<td>Plant and machine operators, and drivers</td>
<td>1</td>
<td>3.0</td>
</tr>
<tr>
<td>Labourers and related workers</td>
<td>4</td>
<td>12.1</td>
</tr>
<tr>
<td>Total</td>
<td>33</td>
<td>100.0</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control

\(^a\) Reference group for odds ratios

(Source. Interview data, 1986; Hospital Patients' Records, 1986)

The group of para-professionals is especially interesting because four of the five women in the case group are nurses, as is the single control member. A laboratory assistant is the fifth person in the case group who, like health care workers, may be exposed to chemicals potentially hazardous to the foetus (see section 2.8.4). At
birthweights over 2,499 grams, health care workers gave birth to three babies that died and two that survived. A final observation relates to saleswomen and personal service workers: two of the four women in the case group are pharmacy assistants. None of the women in the control group has health-related occupations. These facts may be coincidence, but they deserve noting.

Besides specific occupations associated with different groups of occupations, two ingredients of the work experience during pregnancy may influence perinatal outcome: work circumstances comprising the timing and duration of work, and work conditions such as standing for a long time. Both components are presented in Table 7.5 for specific occupational groups and for all these groups collectively defined.

Data for three individual groups of occupations are presented in the table. For para-professionals, and labourers and related workers, the sample sizes are very tiny and do not permit comparison on the basis of perinatal outcome. A small amount of cross-sectional information can, however, be gained from the case women in these seemingly most hazardous occupational groups; although almost none of this information is reported in the written text. More, if still limited, insights come from the apparently safe and most common occupational group of clerks.

The table considers in turn two work circumstances: the timing and duration of work. Every woman, employed at some time during her pregnancy, is shown to work in the first trimester. Thereafter, a slight attrition of numbers occurs, with the result that work for the complete pregnancy is more common in the case group than in the control group. This is found for clerks, with \( \theta = 1.20 \) for all working women, taking the reference group as work in the first trimester.
<table>
<thead>
<tr>
<th>Work Circumstances and Work Conditions</th>
<th>Occupational groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Para-professionals</td>
</tr>
<tr>
<td></td>
<td>P.D. No. %</td>
</tr>
<tr>
<td>Work during</td>
<td></td>
</tr>
<tr>
<td>(a) first trimester</td>
<td>5 100.0 1 100.0 4 100.0 0 0.0 11 100.0 15 100.0 33 25 1.00&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>(b) last trimester</td>
<td>3 60.0 1 100.0 3 75.0 0 0.0 10 90.9 13 86.7 27 21 0.97</td>
</tr>
<tr>
<td>(c) last two months of pregnancy</td>
<td>3 60.0 1 100.0 2 50.0 0 0.0 10 90.9 12 80.0 25 19 1.00</td>
</tr>
<tr>
<td>(d) complete pregnancy</td>
<td>3 60.0 1 100.0 1 25.0 0 0.0 7 63.6 8 53.3 19 12 1.20</td>
</tr>
<tr>
<td>Work for</td>
<td></td>
</tr>
<tr>
<td>(a) 1-6 months</td>
<td>5 100.0 1 100.0 4 100.0 0 0.0 7 63.6 10 66.7 27 16 1.00&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
<tr>
<td>(b) 7-9+ months</td>
<td>2 0.0 0 0.0 0 0.0 0 0.0 4 36.4 5 33.3 6 9 0.40</td>
</tr>
<tr>
<td>Work conditions</td>
<td></td>
</tr>
<tr>
<td>Operate a machine</td>
<td>1 20.0 0 0.0 3 75.0 0 0.0 9 81.8 11 73.3 15 13 1.00&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>Work with a vibrating machine</td>
<td>0 0.0 0 0.0 2 50.0 0 0.0 0 0.0 1 6.7 2 1 1.73</td>
</tr>
<tr>
<td>Lift heavy objects</td>
<td>4 80.0 0 0.0 2 50.0 0 0.0 2 18.2 2 13.3 11 3 3.17</td>
</tr>
<tr>
<td>Stand for a long time</td>
<td>3 60.0 1 16.7 3 80.0 0 0.0 3 27.3 1 6.7 20 7 2.48</td>
</tr>
<tr>
<td>Do repetitive tasks</td>
<td>1 20.0 0 0.0 2 50.0 0 0.0 5 45.5 7 46.7 11 9 1.06</td>
</tr>
<tr>
<td>Work more than 8 hours per day</td>
<td>1 20.0 0 0.0 0 0.0 0 0.0 1 9.1 2 13.3 10 3 2.89</td>
</tr>
<tr>
<td>Work with a loud noise</td>
<td>0 0.0 0 0.0 1 25.0 0 0.0 0 0.0 2 13.3 4 3 1.16</td>
</tr>
<tr>
<td>Use public transportation</td>
<td>0 0.0 0 0.0 1 25.0 0 0.0 3 27.3 4 26.7 6 4 1.30</td>
</tr>
<tr>
<td>Commute for more than 90 minutes per day</td>
<td>1 20.0 0.0 0.0 0 0.0 0 0.0 1 9.1 2 13.3 7 3 2.02</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
<sup>a</sup> The data presented for para-professionals and laborers and related workers are for information only.
<sup>b,c,d</sup> Reference groups for timing and duration of work, and for work conditions, respectively.
(Source: Interview data, 1986; Hospital Patients' Records, 1986)

Yet at least among clerks, the case women who did stop work during pregnancy were more likely than the control women with this characteristic to do so before the final two months of pregnancy. No such difference is found for all working women. About one-quarter of them, irrespective of perinatal outcome, did not work at any time during the last two months of pregnancy, leaving the estimated odds ratio equivalent to that of the reference group. How many women took maternity leave is unknown, but taking maternity leave was found by Glezer (1988) to be associated with fewer problems during the
pregnancy and more difficulties during the confinement, without apparent adjustments for confounding factors.

Explicit focus on the duration of work during pregnancy reveals that most women, with some variation in the size of this majority, work for no more than six completed months. Amongst all working women, yet not, for example, for clerks, this is more characteristic in relative terms of the case group than the control group: thus, the estimated odds ratio associated with work for 7 to 9 months is 0.40, work for 1 to 6 months being the reference group. Related to the discussion above, shorter work periods in the case group reflect lower gestational ages. Preterm delivery, by definition, means fewer months of work and increased rates of perinatal death.

Consideration is also given to the effects of nine work conditions, suggested by the literature to contribute to prematurity, on the perinatal outcome of low birthweight babies. For the group of total women that worked, operating a machine is the work condition most commonly reported (48 per cent, n=28), but the difference in exposure by perinatal outcome is less than for any other work modality. Almost as prevalent overall is standing for a long time (47 per cent, n=27), which is interesting because the condition characterizes 60 per cent (n=20) of all the women who worked and whose baby died, but only about one quarter of the corresponding control group; the differential produces an odds ratio of 2.48 where operating a machine is the reference group.

The same pattern is shown for clerks, while standing for a long time is a salient work condition of para-professionals and labourers in the case group. Although this work exposure has been linked to increased preterm birth (Murphy et al., 1984; Saurel-Cubizolles et al., 1985),
this association, and outcome differences by gestational age, are not supported by the data. When women stood at work for long periods, the case group did give birth at an earlier mean gestational age, 29.5 weeks, than the control group, 31.1 weeks; the sample standard deviations were 5.2 months and 2.8 months respectively. But these statistics differ little from those associated either with other working conditions or with not working during pregnancy.

Repetitive tasks, although frequently reported by the group of all working women, (36.4 per cent, n=20), yield an estimated relative risk of death of 1.06. Less prevalent, but much more hazardous for perinatal outcome, are lifting heavy objects (0=3.17); working more than eight hours each working day (0=2.89); and commuting times exceeding 90 minutes daily (0=2.02). These experiences affect about one-fifth of women giving birth to low birthweight babies.

Confounding many studies of the relation between participation in the workforce and prematurity or reduced birthweights has been the failure to consider unpaid work performed by women in the home (Chavkin, 1986). In this study a total housework score was calculated for every woman. This first involved allocating to each valid household task, from a list of seven such tasks, a score of 2, 1 or 0, according respectively to whether it was done mostly by the woman; shared jointly by herself and her husband or partner, children or other household members; or done mostly by these other individuals. Summation of the scores then yielded for each woman a total housework score, taking a value from 0, where the woman contributed labour to none of the household duties, to 2 where she did all of them herself.

---

9 Valid duties are those applicable to the household. For example, lawn mowing is not relevant when, as for example in Chippendale, Sydney, there may be no lawn to mow.

10 These tasks around the home are shopping and other errands; cooking meals; washing dishes and drying up; house cleaning; washing clothes and ironing them; lawn mowing; and other gardening.
In Table 7.6, two categories of these scores are related to whether work in the paid workforce was also done. Almost three-quarters (72.7 per cent, n=24) of non-workforce participants recorded housework scores above 1.00. Scores of equivalent magnitude are recorded by fewer, but still a high percentage (53.4 per cent, n=31) of women with paid jobs. An estimated 60.4 per cent of all women (n=55) occupy the higher category of housework scores.

Adjusting for paid work yields a Mantel-Haenszel odds ratio of 1.28 for scores 1.1 to 2.0 relative to lower housework scores. Closer examination shows more housework to increase the relative risk of perinatal death only for women also in the workforce. Taking lower housework scores by these women as the reference group, higher scores yield an odds ratio of 1.47. This exceeds each odds ratio estimated for women not in paid employment. Yet for these women, more housework (θ=1.10) appears slightly less hazardous than less housework (θ=1.16).

In sum, the risk of perinatal death is most increased for women in the workforce who also perform a large proportion of the housework.

<table>
<thead>
<tr>
<th>Total Housework Score</th>
<th>Paid work</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes P.D.</td>
<td>No P.D.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ctl θ</td>
<td>Ctl θ</td>
<td></td>
</tr>
<tr>
<td>0.0-1.0a</td>
<td>14 13 1.00b</td>
<td>5 4 1.16</td>
<td>1.00 1.00</td>
</tr>
<tr>
<td>1.1-2.0</td>
<td>19 12 1.47</td>
<td>13 11 1.10</td>
<td>1.28</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
(Source. Interview data, 1986; Hospital Patients’ Records, 1986)
The total physical load of the pregnant woman probably also rises with increasing household size. Although the housework scores take into account the help provided by other household members, these members, such as usually children but also relatives of either partner, or, for example, boarders, can add extra burdens to the physical loads of women. Notwithstanding this, the individual load per household member probably decreases with each new member (Chamberlain, 1984a). Table 7.7 attempts to measure the effects of parity and housework on perinatal risks according to labour force participation.

Table 7.7: Relation of Perinatal Death of Low Birthweight Infants to Home Duties according to Work in the Paid Labour Force and Parity of Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Parity</th>
<th>Total Housework Score</th>
<th>Paid work</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes P.D. Ctl. θ P.D. Ctl. θ</td>
<td>No P.D. Ctl. θ</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiparae</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.0-1.0</td>
<td>5 6 1.00b</td>
<td>5 1 6.00 1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.1-2.0</td>
<td>6 4 1.80</td>
<td>11 9 1.47 0.80</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nulliparae</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.0-1.0</td>
<td>9 7 1.54</td>
<td>0 3 - 0.68</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.1-2.0</td>
<td>13 8 1.95</td>
<td>2 2 1.20 1.17</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
(Source. Interview data, 1986; Hospital Patients' Records, 1986)

According to this table, only 36.2 per cent (n=21) of the women in paid work during pregnancy have previous children, but multiparae compose almost four-fifths (n=26) of the women without paid jobs. High housework scores, indicating the least help with home duties, are less prevalent among multiparous (47.6 per cent, n=10) than nulliparous women (56.8 per cent, n=21) in the paid labour force. The opposite holds for women not in paid employment: 76.7 per cent (n=20) of multiparae receive little help with housework, compared with 57.1
per cent (n=4) for nulliparae. Qualified by the small size of the last estimate, paid work for pregnant women appears to promote sharing of household tasks. Yet because the majority of the interviewed women undertook paid work during their studied 1986 pregnancy, multiparae were more likely to perform household duties independently or with little assistance than to receive much help in the home.

Irrespective of parity, the estimated relative risks of perinatal death are generally higher for paid work especially in the presence of elevated housework scores. However, multiparity is not shown to compound the physical stresses of paid work and unaided household duties. This is revealed by making the reference group multiparous women in the paid work force who record low housework scores.

The most hazardous combination (θ=6.00), based on a cell size of six, is then for multiparae who were not, however, in the paid work force and who recorded low housework scores. The second identifiable cluster of high odds ratios is for women employed in the labour force who did much work in the home, but the estimate for multiparae (θ=1.80) is not statistically significantly different from θ=1.95 for multiparae. The most interesting result produced by the Mantel-Haenszel estimates is the low odds ratio, adjusted for paid work, associated with no previous children and minimal housework duties. Paid work during pregnancy was reported by the majority of mothers. It appears safe except possibly for some small babies and for women exposed to specific circumstances or conditions of work in the labour force and at home.
After testing for a simple relation between perinatal death and exercise, mediated by birthweight, again without developing this focus of inquiry, a second issue is addressed for low birthweight babies. The hypothesis underlying this issue is that when physical exercise during pregnancy is not contraindicated for health reasons\(^{11}\), the safety of particular exercises may depend to a large extent on previous levels of these exercises (see section 2.8.2). Often less important than the activity per se is whether it represents a new endeavour or a continuation of normal living for that person (Paisley and Mellion, 1988). To give a controversial example, scuba diving may possibly, if conservatively undertaken\(^{12}\), be safe for experienced pregnant divers (Newhall, 1981), but it is very unlikely to be recommended as a pregnancy activity for novices.

During pregnancy, some new forms of exercise may be initiated, for example cycling because it is not a weight-bearing activity, even by previously sedentary women\(^{13}\). However, other things being equal, the potential risks of exercise to the foetus and mother\(^{14}\) are likely to be magnified if the exercise is new and especially if it is commenced in the second or, worse, the third trimester. This is not to refute the hazard of first trimester exercise of a type, frequency or intensity that markedly raises the level of exertion over preconceptional levels. The point is that the later trimesters present the added

\(^{11}\) Contraindications may be general, such as heart disease, or relative, such as essential hypertension. Relative contraindications are conditions that may benefit from exercise if it is individualized and medically supervised.

\(^{12}\) Dives should probably not exceed 1 atm in pressure (10 metres) or last longer than 30 minutes (Bergfeld et al., 1987). The potential for teratogenic effects should be recognized (Gorkai, 1985).

\(^{13}\) These women should start at a very low intensity, only gradually increase their level of activity and obey signals to stop exercising. These signals may be as innocuous as breathlessness, dizziness and headache.

\(^{14}\) Possible risks include biomechanical damage to the pregnant woman and foetus, and alteration of the haemodynamic responses to exercise that permit homeostasis through physiological adaptations (Leaf, 1989).
problems of decreased exercise tolerance owing to physiological changes of pregnancy.

As foetal needs increase, cardiovascular reserve is reduced and hypovolaemia may, for example, contribute to preterm labour. Decreased pulmonary reserve can be a limiting factor when attempting exercise at maximal intensity, and as pregnancy progresses, weight gain increases the energy cost and physical discomforts of weight-bearing activities like jogging or walking. Musculoskeletal changes, viz. connective tissue laxity and joint instability, can inhibit the tolerance of activities that involve jumping, for example netball, or sudden changes in direction, such as tennis. The energy demands of pregnancy rise after the first trimester and they are further increased by exercise, which may demand adjustment in the exercise regime or diet.

These considerations may, in sum, appear least pertinent to the small foetus. It may be less likely to make necessary, intense maternal haemodynamic responses (for example, selective peripheral vasoconstriction) than a larger foetus of more advanced gestational age. But these responses may be at least as hazardous in, for example, a preterm baby whose relative lack of foetal reserves can be rapidly depleted with repetitive stress (Gimovsky and Bruce, 1986). In any event, most pregnancies in this study reach at least the seventh month, which is the start of what is typically the last trimester.

Before this second issue is empirically analysed, Table 7.8 permits consideration of the extent to which birthweight mediates the effect of exercise on perinatal outcome, exercise being defined as physical activity of a recreational nature. The table shows that during pregnancy, exercise is 1.3 times more common in the weighted sample
than is no exercise. In the individual birthweight strata, the ratio of exercise to no exercise, while close to unity at very low birthweights, is 2.4 and 2.2 among women delivering babies of 1 500 to 2 499 grams and 2.500 to 3 999 grams, respectively.

Accounting for perinatal outcome, and adjusting for the effect of birthweight, exercise during pregnancy yields a Mantel-Haenszel estimated odds ratio of 0.83, relative to not exercising. Contrary to expectations, the safety of exercise is shown in both low birthweight categories, but not at normal birthweights. Explanations may lie at least partly in the small cell sizes, especially at birthweights 2 500 to 3 999 grams, and in confounding variables including possibly such exercise variables as duration and timing. The last factor, or specifically the potential danger of exercising without suitable previous levels of activity, is, as described above, now explored for low birthweight babies.

Table 7.8: Relation of Perinatal Death to Exercise among Women Aged 20 to 34 Years according to Birthweight, 1986

<table>
<thead>
<tr>
<th>Exercise During Pregnancy</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-1 499</td>
</tr>
<tr>
<td>Yes</td>
<td>17</td>
</tr>
<tr>
<td>No</td>
<td>18</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
(Source. Interview data, 1986; Hospital Patients' Records, 1986)

Table 7.9 compares perinatal outcomes according to the concurrence of the same physical exercise in the first and last trimesters. Because multiple exercises were sometimes reported by the same woman (see Table 7.10), cell entries refer to exercises rather than to women.
Three possible exercises are considered for each woman in the case and control groups, yielding 153 and 120 exercise combinations respectively.

Treating these combinations as independent does not take account of overall levels of physical activity. However, some measure of these is given in Table 7.10, and the approach taken in Table 7.9 retains the integrity of individual exercise types — to the extent these make different demands on the practitioner. In addition, although the data do not consider preconceptional exercise, last trimester exercise can be compared with doing the same activity earlier in the pregnancy.

Exercise in the first trimester has no prior reference level.

Table 7.9: Relation of Perinatal Death of Low Birthweight Infants to Exercise in the Last Trimester according to Exercise in the First Trimester by Women Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Exercise in the Last Trimester</th>
<th>Exercise in the First Trimester</th>
<th>P.D. Ctl.</th>
<th>No Ctl.</th>
<th>$\theta_{mh}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>Yes</td>
<td>30</td>
<td>19</td>
<td>1.17</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>7</td>
<td>4</td>
<td>1.30</td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>37</td>
<td>23</td>
<td>2.36</td>
</tr>
<tr>
<td>No</td>
<td>Yes</td>
<td>16</td>
<td>0.33*</td>
<td>1.00b</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>81</td>
<td>1.00</td>
<td>1.00</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control  
a Reference group for Mantel-Haenszel estimated odds ratios  
b Reference group for other odds ratios  
* Statistically significant at the 0.05 level  
(Source. Interview data, 1986; Hospital Patients' Records, 1986)

Of 273 possible activities, only 30.4 per cent (n=83) took place. The reasons for this, presented in Table 7.10, are twofold. On a per woman basis, first, a sizable group of women (40.7 per cent, n=37) did not exercise during the first or last trimester. This was especially characteristic of the case group, so that no exercise, taken as the
reference group, records the highest estimated odds ratio. However, some women especially in the case group might have been advised not to exercise if they were identified by their doctors as being at high risk for an adverse pregnancy outcome. Only one-quarter (n=23) of all women did two or three different types of exercises. Doing two exercises (θ=0.99) was shown to be just as hazardous as doing no exercises, whilst one type of exercise was apparently most safe (θ=0.39).

Table 7.10: Relation of Perinatal Death of Low Birthweight Infants to Number of Types of Exercises by Women aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Number of Types of Exercises</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>P.D.</td>
</tr>
<tr>
<td>No. %</td>
<td>No. %</td>
</tr>
<tr>
<td>0</td>
<td>24 47.1</td>
</tr>
<tr>
<td>1</td>
<td>13 25.5</td>
</tr>
<tr>
<td>2</td>
<td>11 21.6</td>
</tr>
<tr>
<td>3</td>
<td>3 5.9</td>
</tr>
<tr>
<td>Total</td>
<td>51 100.1</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group
* Statistically significant at the 0.05 level
(Source. Interview data, 1986; Hospital Patients’ Records, 1986)

Focusing on the exercises reported in Table 7.9, more than half (59.0 per cent, n=49) occurred in the first and last trimesters. The relative risk of this combination is 1.17, taking the reference group to be not exercising in either trimester. Doing an exercise in the first but not the last trimester yields the lowest odds ratio (θ=0.33), while doing an exercise in the last trimester without having done that exercise in the first trimester is the most hazardous
combination (θ=1.30). Fortunately it accounts for only 13.3 per cent (n=11) of exercises done.

Differences by perinatal outcome in the types of exercises started in the last trimester permit interesting speculations; although the numbers, being tiny, are strictly of information value. For two women in both the case and control groups, a new activity was swimming. An excellent aerobic exercise, this can be safely initiated during pregnancy. Yet swimming in hot water or, as reported by one of the two women who lost her baby, very cold water, is not advisable (St. John et al., 1986). Three women in the case group and one in the control group commenced prenatal exercises in the last trimester— which should not be too readily dismissed as a chance finding.

According to Artal and Gardin (1986) the safety and imputed benefits of prenatal exercise programs, involving, for example, pelvic floor exercises, are unknown. Such programs ‘typically ignore the aortocaval compression syndrome'15 [for example in the bridging exercise], the increased laxity of joints and ligaments, and exaggerated lumbar lordosis’ (page 4).

Guidelines for exercise during pregnancy, prepared by the American College of Obstetricians and Gynecologists,16 recommend no exercise be performed in the supine position after the fourth month. Dissenters, including the American College of Sports Medicine (1985), argue that even from this time the supine position is generally well tolerated. Ironically, most women during labour and delivery continue to spend extended periods on their backs (Paisely and Mellion, 1988).

15 Hypotension and decreased blood flow to the placenta can result from compression of the aorta and inferior vena cava by the enlarged uterus in the supine position.
16 No equivalent set of guidelines has been prepared by the Australian College of Obstetricians and Gynaecologists.
Further postulated by Artal and Gardin (1986) is that many women are pushed into an exercise program, which they are more equipped to handle mentally than physically, by motivational arguments not always supported by strong scientific evidence. For example, these programs promote the expectation of an easier labour and delivery, when verification of this claim is lacking. Exercise is common during pregnancy and is not found to increase the risks of a perinatal death even for low birthweight babies, except perhaps when continued into the last trimester.

7.3 CHEMICAL FACTORS

Tobacco smoking contributes to perinatal death, mainly by reducing birthweights through intrauterine growth retardation (see section 2.8.4). However, the relation between smoking and perinatal death is much less clear than that between smoking and birthweight. Of particular interest is the paradox that among low birthweight babies, perinatal death is apparently less common among the women who smoke during pregnancy than among those who do not smoke (Werler et al., 1985). The overall probability of a perinatal death is, of course, the product of the probability of being of low birthweight and the probability of a perinatal death associated with being of low birthweight.

These issues are explored in Table 7.11, which relates smoking to perinatal death in different birthweight categories. Smoking is least common (26.3 per cent, n=5) at normal birthweights, and most prevalent at birthweights 1 500 to 2 499 grams (44.1 per cent, n=15). After weighting, the prevalence of low birthweights is 15 percentage points higher in the offspring of smokers than of non-smokers during pregnancy. More than one-third of the women smoked at some time
during pregnancy, this weighted estimate exceeding the Behrens et al. (1987) finding that one-quarter of 140 000 women in Lower Saxony smoked while pregnant.

The data further suggest that after adjustment for birthweight, smoking slightly reduces the estimated relative risk of perinatal death ($\theta_{mh}=0.87$). However, across different birthweight strata, this effect of smoking is apparent only at normal birthweights ($\theta=0.28$). Smoking does not influence perinatal outcome at birthweights 1 499 to 2 500 grams, and at very low birthweights, maternal smoking ($\theta=1.71$) is more hazardous than not smoking ($\theta=1.53$).

These results differ from reports that smoking reduces perinatal mortality among low birthweight newborn. To elucidate this relation, Table 7.12 expands on the preceding table by relating the consumption and timing of smoking during pregnancy to the perinatal death of low birthweight babies. Before examining the table, reference is made to three a priori reasons for expecting smoking to contribute to perinatal death among these small newborn.

### Table 7.11: Relation of Perinatal Death to Tobacco Smoking by Women Aged 20 to 34 Years according to Birthweight, 1986

<table>
<thead>
<tr>
<th>Smoking During Pregnancy</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-1,499</td>
</tr>
<tr>
<td></td>
<td>P.D. Ctl. $\theta$</td>
</tr>
<tr>
<td>Yes</td>
<td>12 7 1.71</td>
</tr>
<tr>
<td>No ($a$)</td>
<td>23 15 1.53</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control

a Reference group for Mantel-Haenszel estimated odds ratios

b Reference group for other odds ratios

(Source. Interview data, 1986; Hospital Patients' Records, 1986)
First, the foetus that is already compromised, for example by growth retardation, may die if its oxygen supply is reduced, secondary to the effects of tobacco smoking on the placenta and on umbilical and uterine vasculature. Secondly, in addition to influencing foetal growth and development, smoking is associated with increased incidences of obstetric complications such as antepartum haemorrhage (Naeye, 1980), and exacerbation of existing maternal conditions like hypertension (Abel, 1983). Thirdly, tobacco may be a teratogen, causing, for example, neural tube defects (Christensen, 1980) or chromosomal exchange-type aberrations (Abel, 1983).

Table 7.12 compares the effect of smoking in the first and third trimesters of pregnancy on perinatal outcome among low birthweight babies. The table shows that 37.1 per cent (n=33) of women smoked cigarettes in one trimester or both trimesters, comparatively few of them (15.2 per cent, n=5) smoking fewer than 10 cigarettes per day. Nevertheless, 42.4 per cent (n=14) of smokers cut back their daily number of cigarettes by the last trimester, but approximately half of all smokers maintained consumption levels of 10 or more cigarettes per day.

Taking the reference group as no cigarettes in either trimester, continuing to smoke at least 10 cigarettes per day is estimated to reduce the relative risk of perinatal death by 44 per cent. Reporting this dosage in the first trimester, but no cigarette smoking in the last trimester, also yields a lower odds ratio of 0.75. The opposite effect attends cutting back to 1 to 9 cigarettes (θ=1.13), or from this dosage to 0 cigarettes (θ=1.50).

17 This is controversial, but the possibility cannot be dismissed that smoke products contribute to congenital malformations.
Table 7.12: Relation of Perinatal Death of Low Birthweight Infants to Cigarettes Per Day in the First Trimester according to Cigarettes Per Day in the Last Trimester by Smokers Aged 20 to 34 Years, 1986

<table>
<thead>
<tr>
<th>Cigarettes per Day in the First Trimester</th>
<th>Cigarettes per Day in the Last Trimester</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1-9</td>
</tr>
<tr>
<td>P.D. Ctl. θ</td>
<td>P.D. Ctl. θ</td>
</tr>
<tr>
<td>0</td>
<td>32</td>
</tr>
<tr>
<td>1-9</td>
<td>2</td>
</tr>
<tr>
<td>10+</td>
<td>3</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control

a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios

(Source. Interview data, 1986; Hospital Patients' Records, 1986)

Smoking more than ten cigarettes per day in the first and last trimesters yields the most reliable estimate of an effect attributable to smoking; the combination yields the largest, although still small, cell sizes and indicates continued high doses. For these persistently heavy smokers, the diminution in relative risk (θ=0.66) reverses the earlier finding from Table 7.11 that linked smoking to increased perinatal death at very low birthweights. The revised finding is due, according to the 'displacement hypothesis', to higher mean birthweights among the low birthweight babies of the heaviest smokers than among the babies of other smokers and non-smokers (Goldstein, 1972). This is found to be true. Mean birthweights, among infants weighing 500 to 2 499 grams at birth, were higher when their mothers smoked at least 10 cigarettes per day in the first and last trimesters (1 399 grams) than when they did not do so (1 345.5 grams).

However, the difference in means is not statistically significant at the 0.05 level. Also, since the displacement hypothesis is not usually applied only to heavy smoking, an opposite shift in mean
birthweights should attend the increased risk of perinatal death found
either in Table 7.11 or by collapsing over timing and consumption
variables in Table 7.12. This reversal is not found. Irrespective of
perinatal outcome, mean birthweights remain higher for the infants of
all maternal smokers, 1439 grams, than for the infants of
non-smokers, 1295.2 grams (p < 0.05), which suggests that the
apparent safety associated with heavy smoking at low birthweights may
not be due to its effect on birthweight within this group.

Fortunately, there is a second explanation for this last, paradoxical
result. Smoking contributes to low birthweights mainly by retarding
growth in utero, more in boys than in girls (Wertelecki et al., 1987),
whereas among non-smokers, other things being equal, low birthweight
tends mainly to result from preterm birth (see sections 2.6.1 and
5.1.1). Consequently, at the same low birthweight, say 1500 grams,
the infants of heavy smokers are more likely to be of higher
gestational ages than are the infants of women who smoked much less or
not at all during pregnancy; that is, the babies of heavy smokers are
likely to be the more mature physiologically (see also section 5.2.1).
The same emphasis on maturity, rather than on birthweight per se as in
the Goldstein hypothesis, is supported by the reported association
between smoking and stimulation of foetal lung maturation (Curet et
al., 1983). One beneficial effect of smoking may be a reduction in
psychological stress.

Maternal smoking, although a known risk factor for low birthweight and
thus perinatal death, has previously been observed to improve survival
chances for babies of low birthweight. In this study, only heavy
maternal smoking reduces estimated relative risks of perinatal death,
and the reduction is not explicable in terms of higher mean (low)
birthweights exclusively among the babies of heavy maternal smokers.
An alternative explanation was offered and a multivariate analysis is recommended.

7.4 CONCLUSION

This chapter, more than any other in the thesis, is essentially exploratory because of small sample sizes. Analyses are inconclusive but they address fresh, important issues, while adding to existing knowledge about better understood relations. In the latter context, most research to date has shown that so-called pregnancy health stressors can diminish physiological maturity of the conceptus and contribute to perinatal death through this mediating variable. For each stressor in the chapter, this mechanism was discussed without adjustment being made for effects other than maternal age and birthweight.

However, subsequent considerations are novel in exploring possibly direct aetiological effects. This takes cognizance of the probability that stressors may contribute to death not by causing low birthweight, but by affecting a foetus that, because of its low birthweight, is severely compromised and vulnerable. Also mentioned were other mechanisms that may act independently of (as well as influence) foetal growth and development, such as some obstetrical complications and teratogens.

Three types of stress were examined: psychological, physical and chemical. Psychological stress was operationally defined by reported life events and self-rated distress. Approximately half of all the women had low total distress scores, irrespective of birthweight and survival status, although low scores were more prevalent in the case group. At low birthweights, the women reporting the lowest total distress scores recorded the highest estimated odds ratios.
One approach taken to elucidate these findings was to relate item-specific distress scores to the frequency with which particular events were reported. Compared to the reference group, only a threatened miscarriage, and the confinement being painful or bringing new complications, were, especially in the case group, frequently reported and distressing. Items describing personal relationships appeared the main locus of lower distress to the case group. Distress scores for work events were examined with respect to their timing and duration, the last trimester being shown to be especially hazardous.

Physical stress was measured by work and exercise. Paid work was reported by almost two-thirds of the weighted sample of women. It was not conspicuous among the mothers of low birthweight babies, and was not found to increase perinatal mortality risks except possibly at birthweights 1 500 to 2 499 grams. Perinatal outcomes for small infants were compared across occupational groups, and different circumstances and conditions of work.

Small numbers suggested that the babies of para-professionals, and labourers and related workers were the least likely to survive. The most hazardous work circumstances were working during the complete pregnancy but, paradoxically, for fewer than six months. Unfavourable work conditions include standing for a long time, lifting heavy objects and working long hours. Paid work did not prevent a preponderance of high total housework scores which elevated odds ratios, if not more so for multiparous than nulliparous women.

Exercise is common among the mothers not only of low birthweight babies, but also of babies at higher birthweights. Exercise reduces the relative odds of perinatal death after adjustment for birthweight, and appears safe at low birthweights. On a per-woman basis, the safest number of exercise types between 0 and 3 is 1. On a
per-exercise basis, most hazardous is exercising during the last trimester, possibly especially after not exercising during the first trimester. More than half of all exercises are in both of these trimesters.

The empirical referent for chemical stress is tobacco smoking which was reported by about one-third of the weighted sample of interviewed women, most conspicuously when babies weighed 1 500 to 2 499 grams at birth. Initial analyses revealed that contrary to evidence in the literature, smoking did not reduce odds ratios at low birthweights, even though estimated odds ratios were depressed at birthweights 2 500 to 3 999 grams. Smoking more than 10 cigarettes per day in the first and last trimester produced the expected attenuation of risk, but the displacement hypothesis could not account for the reversal.
CHAPTER 8
BACKGROUND FACTORS

8.0 INTRODUCTION

This chapter explores mainly how empirical referents of three sets of factors in the expectant mother - medical history, sociocultural and socioeconomic factors - may operate at the micro-level to influence the occurrence of perinatal death. In section 2.0, causal chains between background factors and perinatal death were conceptualized to be always, rather than usually (as with proximate factors) indirect. For operational purposes, however, it is heuristically useful and convenient to estimate the 'direct' as well indirect aetiological effects of background factors, and not explicitly to distinguish between these. The analytical format of preceding chapters is thus continued into the present chapter which ends by fitting a logistic regression model to estimate concurrently the relative risks of multiple background and proximate factors. Again, because of the small sample sizes, analyses are fundamentally exploratory and novel rather than definitive.

8.1 MEDICAL HISTORY FACTORS

The expectant mother's medical history is empirically represented in this study by her reproductive history, which, of course, forms only part of her medical history. Estimates are made of the effect on perinatal outcome of two elements of a woman's reproductive history: her gravidity, which provides a useful summary of the history, and her pregnancy outcomes composing this gravidity.
As described in section 2.10.2, the relation of perinatal death to gravidity or to parity is characteristically J-shaped. This is possibly due to confounding, especially at high gravidities or high parities, by factors including maternal age and the constituent reproductive history. The latter factor is probably the more important (Yudkin and Baras, 1983), and exploring how it operates is fundamental to understanding the gravidity-perinatal death nexus.

The effects of gravidity, or parity, on perinatal outcomes can be distorted by compositional and timing characteristics of women’s reproductive histories. Compositional factors pertain mainly to numbers of good and poor outcomes, the relative importance of each of which varies with two perspectives. According first to the ‘recurrence risk’ hypothesis, applied initially to spontaneous abortions (Kline, 1978), only the number of previous losses is important: mortality risks increase with each death irrespective of the numbers of pregnancies or births.

An alternative gravidity effect may result from heterogeneous risk and selective fertility. This recognizes that women differ in their innate reproductive capacities, while good and poor previous outcomes affect the occurrence and outcome chances of subsequent pregnancies (Santow and Bracher, 1988). Just as poor outcomes increase numbers of pregnancies, to replace or compensate for prior losses, as well as reproductive risks at successive gravidities, so past good outcomes discourage additional pregnancies and improve risks. These countervailing forces interplay to influence gravidity and reproductive success in the index, or most recent, pregnancy.
The second, timing mechanism expands on the latter perspective. Not only may good outcomes protect against future losses at successive pregnancies, but this safety is hypothesized to be especially likely when a good outcome immediately precedes the index pregnancy. Likewise, poor outcomes are most likely to occur consecutively (Yudkin and Baras, 1983).

Table 8.0 has been designed to address all these issues by relating gravidity to perinatal outcome in the index, or most recent 1986, pregnancy. The total number of pregnancies is expressed as a function, first, of the number of good outcomes, that is parity defined as the number of live births surviving the neonatal period; secondly, of the number of poor outcomes, this being a neonatal death, stillbirth or spontaneous abortion, or an induced abortion; and thirdly, the outcome of the immediately preceding pregnancy.

<table>
<thead>
<tr>
<th>Table 8.0: Relation of Perinatal Death of Low Birthweight Babies to Gravidity and Reproductive History of Women Aged 20 to 34 Years, 1986</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of Poor Outcomes</td>
</tr>
<tr>
<td>----------------------</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>0</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>4+</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

P.D.: Perinatal Death; Ctl: Control
a Since a preceding outcome cannot be good for nulliparae, cells entries for these women refer instead to not applicable preceding outcome
b Reference group for odds ratios
*** Statistically significant at the 0.005 level
** Statistically significant at the 0.01 level
* Statistically significant at the 0.025 level
(Source. Maternal and Perinatal Collection, 1986)
The sum of good and poor outcomes yields the total number of previous pregnancies, where adding a one to the index pregnancy yields the gravidity for that pregnancy. The uppermost left corner describes the women with no reproductive history who, therefore, enter the 1986 pregnancy as primigravidae. Represented beneath these women in a stepwise pattern are the multigravidae who, having previously had between one and three pregnancies, are now at gravidities 2 to 4. Occupying the lowest series of steps are 'grand multigravidae' who have been pregnant four or more times and so enter the 1986 pregnancies being studied at gravidities 5 and above.

Each group of multigravidae subsumes three categories representing the reproductive history underlying the multigravidity. This can occur mainly or completely through prenatal or neonatal losses to yield poor reproducers, or, alternatively, through more good than poor past outcomes to yield successful reproducers. The third group of women have equal numbers of good and poor reproductive outcomes.

By disaggregating reproductive histories into good and poor outcomes, with attention paid also to the outcome of the immediately preceding pregnancy, the table facilitates exploration of the relation between perinatal death and especially multigravidity effects. It should be noted that some women may not admit to particular pregnancies. However, reasonably accurate clinical means are available for detecting multigravidae (Beischer and Mackay, 1981).

Restricting the analysis to women aged 20 to 34 years is especially important here because it excludes high gravidities at advanced maternal ages, as well as some first pregnancies to adolescent mothers. Only low birthweight newborn are examined, partly to simplify the tabular presentation yet also because birthweight has

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1 These are based, for example, on the status of the reproductive organs.
been found to increase from parity 1 to parity 3, before decreasing at higher parities (MacLeod and Kiely, 1988).

Before exploring the relations between perinatal death, gravidity and reproductive history, some overall measures of prevalence and risk are useful. Almost one-third (n=422) of all the women are primigravidae. Of 914 multigravidae, 23.9 per cent (n=218) have no good outcomes, and 44.2 per cent (n=404) have no losses. Grand multigravidae constitute 9.1 per cent (n=121) of gravid women, almost half (47.9 per cent) having more good than poor reproductive outcomes, and 30.6 per cent (n=37) having mainly reproductive failures.

Perinatal mortality appears to increase directly with gravidity. Relative to primigravidae, the estimated relative risk of perinatal death is 1.56 at gravidities 2 to 4, rising to 0=2.41 at gravidities 5 and above. However, study is required of the compositional and timing characteristics of reproductive histories constituting these gravidities in order to understand how these histories may confound the gravidity-perinatal death relation.

According to the recurrence risk hypothesis, estimated relative risks of perinatal death at each gravidity should rise with the number of poor outcomes, irrespective of the number of good outcomes and of the immediately preceding outcome. If this holds, to measure the effect of gravidity per se is to assess differences in estimated odds ratios for women with the same number of losses.

Table 8.0 supports at least the first part of the hypothesis, namely that increased numbers of past losses attend rising gravidities and elevated mortality risks therein, but not its nonconditional tailpiece. First, however, the hazard of prior reproductive failure is shown to be confirmed. Taking primigravidae as the reference
group, estimated odds ratios for gravidae 2 to 4 are increased from 1.40, with no poor outcomes, to 1.70 and 2.00 with one and two poor outcomes respectively. Three prior losses yield an odds ratio of 1.18 possibly owing to small numbers, although these poor reproducers may have been especially likely to have received the finest health care available in their index pregnancy. The direct association of mortality risk and reproductive failure is even clearer for grand multigravidae. Although two losses appear safer than one loss, but not safer than no losses ($\Theta=1.06$), three and four losses produce the highest odds ratios of 2.83 and 4.50 respectively.

Yet it does not appear, as expected under the hypothesis of heterogeneous risks and selective fertility, that reproductive failure affects gravidity differences in perinatal mortality independently of reproductive success. Perinatal mortality risks among multigravidae increase with the ratio of poor to good outcomes. Among grand multigravidae, successful reproducers, that is women with more good than poor outcomes, have an odds ratio of 1.86 while poor reproducers, who have more poor than successful outcomes, have an odds ratio of 3.73. Equal numbers of good and poor outcomes yield the relative odds of 2.21.

While poor reproducers have higher relative odds of perinatal death than successful reproducers, within these categories a more complex pattern emerges. Increases in parity, meaning numbers of neonatal survivors, are actually hazardous in the presence of more good than poor outcomes. Consider first, grand multigravidae.

When they are poor reproducers, movement from three to four losses is associated with increased odds ratios at parity 1, although not, according to very small numbers, at parity 2. In strata representing the same number of poor outcomes, whether three or four, rising parity
yields increased estimated relative risks of perinatal death. Among successful reproducers, movement from one loss to two losses yields reduced odds ratios at parity 3, whilst at parity 4 and above, there is no discernible trend. At each level of poor outcomes, which should be noted to be low for these women, increasing parity is associated with a reduction in odds ratios. The suggestion is that among women having at least their fifth pregnancy, past good outcomes afford some protective effect in the index pregnancy to successful reproducers but not to poor reproducers.

A similar pattern is found at gravidities 2 to 4. Among poor reproducers, movement to a higher parity increases the estimated relative risk of perinatal death. However, there are almost no good outcomes to measure. Among the women with more good than poor outcomes increasing parity remains hazardous. For the women with no poor outcomes, odds ratios rise at parity 2, but fall at parity 3. It can nevertheless be noted that for the women with one poor outcome, parity 2 is more hazardous for successful reproducers than nulliparity is for poor reproducers. Obviously, among primigravidae, the recurrence hypothesis is not testable.

Last considered is the contribution of the immediately prior outcome to perinatal death in the index pregnancy. Compared with a poor last outcome, a good preceding outcome does give some subsequent protection. This is most salient for grand multigravidae with recurrent prior losses. For example, with four or more poor outcomes at parity 1, the estimated relative odds of perinatal death is increased fourfold to 10.61 by a poor last outcome vis-à-vis a successful antecedent outcome, 0=2.65.

In consequence, to assess the contribution of gravidity per se to perinatal death, comparisons should be based on gravidities derived
from common reproductive histories as defined, first by the ratio of
good to poor previous outcomes, and secondly by the outcome of the
preceding pregnancy. Gravidity effects can thus be measured, for
example, for successful reproducers whose immediately preceding
outcome was successful, and for poor reproducers whose last pregnancy
failed, but not across these groups.

In these groups, collapsing over the numbers of good and poor
pregnancy outcomes, and taking primigravidae as the reference group,
yields the following relative risk estimates, first for women with
more good than poor outcomes. When these women have a good preceding
outcome, the odds ratios are 1.39 for women at gravidities 2 to 4, and
1.34 for grand multigravidae. A poor preceding outcome yields
estimated relative odds of 3.54 for both multigravid groups.

Secondly, consider women with more poor than successful outcomes. A
successful last outcome produces for women who have had up to three
pregnancies an odds ratio of 4.72 but for grand multigravidae an odds
ratio of 1.92. By contrast, a poor last outcome is much more
hazardous for grand multigravidae (Θ=7.08) than for women at
gravidities 2 to 4 (Θ=1.62). With an equal number of good and poor
previous pregnancy outcomes, a good immediately preceding outcome
produces no difference in mortality risk across the two multigravid
groups, however, a poor last outcome yields estimated odds ratios of
3.54 for grand multigravidae and 1.60 for women at gravidities 2 to 4.

In sum, women having their first pregnancies record consistently the
lowest estimated relative risks of perinatal death, which is in
contrast to other studies. Among women who have been pregnant before,
the greatest difference in perinatal mortality risks by gravidity
occurs when the number of good outcomes is equal to or less than the
number of poor outcomes. A poor reproductive outcome immediately
preceding the index pregnancy is then especially hazardous for grand multigravidae.

8.2 SOCIOCULTURAL FACTORS

Three variables describing maternal attributes are used to assess empirically the contribution of sociocultural factors to perinatal death. The first two variables, whether the mother is Aboriginal, and her region of birth, are ascribed maternal characteristics and emphasize the race and culture of the mother. Her marital status, which is the third variable, relates to an acquired social role.

8.2.1 ABORIGINALITY

According to the Better Health Commission (1986), the Australian perinatal death rate in 1984, expressed per 1 000 total births, was 29.9 for Aboriginal women and 11.9 for non-Aboriginal women. This was after spectacular falls in Aboriginal rates of perinatal mortality and especially neonatal mortality since the 1970s. By comparison, Aboriginal stillbirth rates have tended to fall much more slowly (Thomson, 1983; Gray, 1989).

However, this subsumes State and Territorial differences in especially Aboriginal perinatal mortality. The highest rates of Aboriginal perinatal death and, in particular, Aboriginal stillbirth have continued to occur in the Northern Territory (Julienne et al., 1983; Musgrave, 1984; Northern Territory Department of Health, 1986). Indeed, on Elcho Island, Aboriginal women recorded a perinatal mortality rate of 59 per 1 000 for the period 1979 to 1982 (Watson, 1984).

\[\text{However, in Western Australia, stillbirth rates 1975-1978 showed a larger decline than did rates of neonatal or perinatal death (Seward and Stanley, 1981).}\]
Persistent, high rates of Aboriginal perinatal mortality are largely accounted for by higher incidences of low (and lower) birthweights among Aboriginal infants than among non-Aboriginal infants (Seward and Stanley, 1981). However, Kliewer and Stanley (1989) observed that, among liveborn singletons, Aboriginal infants were lighter than non-Aboriginal infants only at term and beyond. Before term, Aboriginal infants tended to be heavier than white infants. Despite a possible race differential in the recall of the last menstrual period, similar results were cited from the United States for black and white infants.

That overall, low birthweight is more common among Aboriginal than non-Aboriginal infants has often been attributed to intrauterine growth retardation among Aboriginal babies.\(^3\) With preterm birth being the more frequent cause of low birthweights among non-Aboriginals, this could help to explain Seward’s and Stanley’s (1981) finding of lower rates of perinatal mortality among Aboriginals than Caucasians at low birthweights; preterm birth is more likely to result in perinatal death than is growth retardation \textit{in utero} (see sections 5.1.1, 5.2.1 and 7.3).

However, preterm birth has also been observed to account for low birthweights among Aboriginals (Kliewer and Stanley, 1989). Among singletons born to Aboriginal teenagers, Stanley and Mauger (1986) found preterm birth to be the more important cause of low birthweight, although there were difficulties in estimating gestational age and in classifying growth retardation \textit{in utero}.

\(^3\) This growth retardation \textit{in utero} has been shown most reliably by paediatric examination (Gogna et al., 1986). Watson (1984) cautions that intrauterine growth charts based on Caucasians may be of limited value in assessing Aboriginal foetal maturity and development.
Also more common in Aboriginal than non-Aboriginal women are medical complications such as anaemia, urinary tract infections, diabetes and cardiac disorders (Hart et al., 1985). These conditions often reflect maternal malnutrition (Gracey et al., 1984); high incidences of tobacco smoking and alcohol consumption (O’Connor, 1982); and inadequate antenatal care (Julienne, 1983; Hart et al., 1985; Gray, 1987). Aboriginal pregnant women are likely to be younger and to deliver at higher parities than Caucasian pregnant women, but these factors are not important reasons for the high perinatal mortality rate among Aboriginals (Seward and Stanley, 1981).

Helping to redress the paucity of literature on the risks of congenital malformations in Aboriginals is the study by Bower et al. (1989). This found a significantly higher birth prevalence of microcephaly, specific cardiovascular defects; cleft lip and palate; and talipes in Aboriginal infants than non-Aboriginal infants in Western Australia between 1980 and 1987. Significantly less prevalent in Aboriginals than non-Aboriginals were pyloric stenosis and two urogenital defects: undescended testis and hypospadias. Differences in the prevalence of some malformations are possibly explained by chance and diagnostic, treatment or notification biases. For other differences, for example in pyloric stenosis, explanation in environmental or genetic factors is indicated (Bower et al., 1989; Lancaster, 1989b). The prevalence of all congenital malformations was

4 Microcephaly is a nervous system defect characterized by arrest of brain growth and an abnormally small head.
5 These cardiovascular defects were tetralogy of Fallot, patent ductus arteriosus and ventricular septal defects.
6 There is a fissure in the upper lip and palate (roof of the mouth) resulting from the failure of the two sides to fuse in embryonic development.
7 Talipes or club-foot is a congenital deformity of one or both feet that prevents standing with the sole flat on the ground.
8 Pyloric stenosis is a narrowing of the muscular outlet of the stomach.
9 The testes do not descend into the scrotum but remain in the abdomen.
10 With hypospadias, the opening of the urethra is on the underside of the penis.
3.5 per cent for both populations, a result similar to that of Hart et al. (1985) for South Australia.

According to Seward and Stanley (1981), genetic racial differences may also contribute, for example, to variations in birthweight. Yet Cooper (1986) argues that 'no discrete "package" of gene differences has ever been described between two races' (p.101). Still, no-one has contested the belief that high perinatal death rates among Aboriginals are explained better by social than biological contexts of race. The nature of this social inequality is, however, a source of debate.

The consensus perspective emphasizes the failure of many Aboriginals to cope with modernization and the general lack of understanding by Aboriginals and white Australians of each other's culture and value structures. Conflict approaches target poverty, racism and powerlessness as the root of Aboriginal suffering (Bates and Linder-Pelz, 1987). Elements of each variety of structuralism are fused below into one argument.

The structural relationship of Aboriginals to the larger Australian society is fundamentally one of a marginalized minority group pressed to accept dominant, Anglo-European modes of living, but at the same time hindered often from doing so by two sets of factors. The first of these is dispossession of land and of traditional rights by European colonists with attendant Aboriginal depopulation and dispersal. Today, dispossession continues under the guise of institutionalized racism. Racial differences cannot be explained by social class since race is not confounded by class variables: it is antecedent to them.

The second set of factors influencing Aboriginal and non-Aboriginal behaviour is the lack of knowlege and the cultural discomfort of both
groups with the other's customs, economy and polity. For example, many Aboriginals dislike hospitalization. They feel uncomfortable in this setting because care is removed from community supports; the hospital may be perceived to provide unequal treatment, and represents an alien, male-dominated, 'white' obstetrics (Health Commission of New South Wales, 1980).

The most damaging consequences of both sets of factors have been to restrict Aboriginal access to, and use of, white Australian institutions, and to undermine Aboriginal self-esteem, and health and other rights (Thomson, 1983; Gray, 1985, 1989). Increasing numbers of Aboriginals have come to use separate, Aboriginal community-controlled health care services, yet these operate on a small amount of the Aboriginal health budget (Franklin, 1982; Copeman, 1988). Moreover, for high-risk obstetric patients, they are no substitute for large hospitals and do not absolve hospitals of their responsibilities to Aboriginal communities. Hospitals must develop types of care that are more sensitive to Aboriginal needs even if, for example, these value individual well-being less highly than community cohesion and welfare.

These ideas are not further developed here, but serve rather to contextualize the subsequent empirical analysis which relates perinatal death to Aboriginal and non-Aboriginal status according to birthweight. More adventurous associations are not explored because of deficiencies in the reporting of Aboriginal data by the Maternal and Perinatal Collection. First, the race of the woman only is recorded on the Collection form, so that a 'Caucasian' newborn may have an Aboriginal father and a white mother. Secondly, no attempt is made to distinguish between full-blood and mixed-blood Aboriginals even though the latter group, whose size has probably increased in recent decades, has presumably fewer risk factors (Lancaster, 1989b).
Thirdly, as is now discussed, the number of Aboriginals reported by the Maternal and Perinatal Collection is likely to be less than the real number.

According to Table 5.0, just 1.6 per cent (n=1,254) of the total births reported on the 1986 Collection form were to Aboriginal women. The data needed to confirm that this understates the true percentage are not available, but some under-reporting seems likely given evidence that this has occurred with respect to perinatal deaths. Specifically, in Table 8.1, the percentage of perinatal deaths to Aboriginals is 1.9 per cent (n=11), yet, using the New South Wales definition of perinatal death, the 1986 prevalence of perinatal death in New South Wales was 2.6 per cent; and this latter percentage itself suggests some under-reporting, especially of stillbirths (pers. comm., L. Coleman).

Table 8.1: Relation of Perinatal Death to Aboriginality of Women Aged 20 to 34 Years according to Birthweight, 1986

<table>
<thead>
<tr>
<th>Aboriginal Birthweight (grams)</th>
<th>500-1 499</th>
<th>1 500-2 499</th>
<th>2 500-3 999</th>
<th>4 000+</th>
<th>P.D. Ctl. θ</th>
<th>P.D. Ctl. 0</th>
<th>P.D. Ctl. θ</th>
<th>P.D. Ctl. 0</th>
<th>P.D. Ctl. 0</th>
<th>θ_{nh}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>7</td>
<td>5</td>
<td>7.11***</td>
<td>0</td>
<td>13</td>
<td>-</td>
<td>4</td>
<td>5</td>
<td>4.06</td>
<td>0</td>
</tr>
<tr>
<td>No^a</td>
<td>261</td>
<td>364</td>
<td>3.64***</td>
<td>117</td>
<td>570</td>
<td>1.04</td>
<td>175</td>
<td>889</td>
<td>1.00^b</td>
<td>14</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
*** Statistically significant at the 0.005 level
(Source. Maternal and Perinatal Collection, 1986)

The reasons for the under-reporting in the Collection are presumably found in the method of defining Aboriginal persons. The Collection defines Aboriginals, including Torres Strait Islanders, as women who

11 This inclusion of Torres Strait Islanders follows the guidelines of the National Committee on Health and Vital Statistics (New South Wales Department of Health, 1988b).
identify themselves as Aboriginals, or who are observed to be Aboriginal by the midwife completing the Collection form (New South Wales Department of Health, 1988b). Yet identifying Aboriginals by observation could be unreliable when midwives do not know subjects well, which presumably occurs most often in urban areas. As Coaby (1987) notes, Aboriginal people from the city look quite different from those in country areas and even more so, from tribal people.

This potential for error would seem best to explain occasional disagreement in the Collection between the country of birth of the mother and whether or not she is Aboriginal: three women in the control group and one woman in the case group were reported to be Aboriginal but to have been born outside Australia, most commonly in Pacific islands. Observational evaluations of race remove, moreover, the element of choice which is assumed by existing definitions of Aboriginality to belong to the individual being evaluated.

Discussions with race relations personnel suggest that many health workers do not ask patients whether they are Aboriginal for fear of causing these patients, or themselves, embarrassment. No legal barriers control the ascertainment of this information - only its use. Asking women explicitly whether or not they are full or mixed blood Aboriginals would improve Aboriginal health statistics.

The indicated under-reporting and associated small numbers of Aboriginals in Table 8.1 make Aboriginal estimates of prevalence and relative risk unstable. However, as the non-Aboriginal group is comparatively large, including a small number of Aboriginal women in

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12 Self-identification is part not only of the New South Wales Health Department definition of Aboriginality, but also, for example, of the Commonwealth definition.

13 The 1989 Privacy Act prohibits the asking of questions not necessary for the rendering of a service. However, this Act affects only Commonwealth departments and, therefore, not State hospitals.
it should not greatly distort the findings for non-Aboriginals.

Against this background, the table estimates that Aboriginal women account for 1.9 per cent (n=12 and n=25) of babies of very low and low birthweights respectively, but only for 0.8 per cent (n=9) of infants of normal birthweight. After weighting, low birthweights characterize almost one fifth of Aboriginal births yet only about five per cent of non-Aboriginal births.

Estimated relative risks of perinatal death are greater for Aboriginals than non-Aboriginals. After adjustment for birthweight, Aboriginal women aged 20 to 34 years experience relative odds of perinatal death 2.41 times higher than for non-Aboriginal women at the same maternal ages. Estimated relative risks of perinatal death are lower for non-Aboriginals than Aboriginals at very low and, in particular, normal birthweights, but not at birthweights 1 500 to 2 499 grams (see page 365). Collapsing over the low birthweight strata yields similar odds ratios for Aboriginals (θ=1.98) and non-Aboriginals (θ=2.06). Babies with normal birthweights show the greatest differential between Aboriginal and non-Aboriginal mortality of approximately four times, and indeed the estimated odds ratio for Aboriginal infants of normal birthweight (θ=4.06) is higher than that for non-Aboriginal babies with very low birthweights (θ=3.64). Being Aboriginal in New South Wales is a risk factor for perinatal death.

8.2.2 MATERNAL REGION OF BIRTH

In the discussion to date, non-Aboriginal people have been depicted as a homogeneous entity which, of course, they are not: of the industrialized countries, Australia houses the largest percentage (20.6 per cent) of overseas born persons in the world (United Nations, 1989). This is important, for one, because national origins by
birthplace, which also descend through parentage and ancestry, can produce distinctive ethnic and class behaviours notwithstanding the potentially distorting effects of selective migration, duration of residence and generational time. Of interest in this study is the extent to which perinatal mortality risks differ with membership of particular birthplace groups.

Successive historical streams and counterstreams of migration provide a useful framework for understanding the ethnic composition of Australian, and specifically, New South Wales, society. Compositional changes have occurred in two broad periods of net migration. First, from World War 2 to the early 1970s, migration was predominantly British, although increased proportions of migrants arrived initially from Eastern, Northern and Western Europe - mainly from Germany and the Netherlands - and subsequently from Southern Europe, especially from Italy, Greece and Yugoslavia.

The second period, continuing since the mid-1960s, has witnessed in terms of absolute numbers, continuing high migration from the United Kingdom until the mid-1980s, and an increase in immigration from Poland and non-European regions. The latter regions include West Asia, initially Egypt and then especially Lebanon; Southeast Asia supplemented by refugees from Indo-China; and New Zealand, South Africa and the United States (Hugo, 1986; Young, 1989).

Table 8.2 relates perinatal death to these national origins as measured by the mother’s region of birth. Within the individual regions, percentages of low birthweight infants range, after weighting, from 10.2 per cent for Africa (n=45) to 4.3 and 3.1 per cent for Northern and Western Europe (n=196) and America (n=17) respectively. About five per cent of the babies born to native Australian women are estimated to have low birthweights.
Irrespective of the birthweight, more than three-quarters of the births are to native Australian women. The next most common birthplace region is Northern or Western Europe which contributes 4.7 per cent (n=28) of very low birthweight babies yet 7.0 per cent of the infants of normal birthweight. In descending order of the frequency of births, the remaining birthplace regions are West Asia followed by Oceania excluding Australia; Southeast Asia; Southern and Eastern Europe; and South and East Asia. No more than one per cent of births at any birthweight are to mothers born in Africa or America.

Table 8.2: Relation of Perinatal Death to Maternal Region of Birth of Women Aged 20 to 34 Years according to Birthweight, 1986

<table>
<thead>
<tr>
<th>Maternal Birthplace</th>
<th>Birthweight (grams)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-1 499</td>
</tr>
<tr>
<td>Australia</td>
<td>201</td>
</tr>
<tr>
<td>Oceania</td>
<td>11</td>
</tr>
<tr>
<td>Other</td>
<td>11</td>
</tr>
<tr>
<td>Northern and Western Europe</td>
<td>4</td>
</tr>
<tr>
<td>Southern and Eastern Europe</td>
<td>10</td>
</tr>
<tr>
<td>West Asia</td>
<td>5</td>
</tr>
<tr>
<td>South and East Asia</td>
<td>7</td>
</tr>
<tr>
<td>Southeast Asia</td>
<td>2</td>
</tr>
<tr>
<td>Africa</td>
<td>1</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ct1: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
*** Statistically significant at the 0.005 level
** Statistically significant at the 0.01 level
* Statistically significant at the 0.025 level

For each maternal birthplace, estimated relative risks of perinatal death are compared and interpreted with regard to existing knowledge and to selected characteristics of the complete sample of 20 to 34 year old mothers of low birthweight babies (see Table 8.3). After
Table 8.3: Number and Percentage of 20 to 34 Year Old Mothers of Low Birthweight Infants with Selected Characteristics by Birthplace Region, 1986

<table>
<thead>
<tr>
<th>Maternal Birthplace</th>
<th>Single¹</th>
<th>Primigravida</th>
<th>1st Ant-11+ wks</th>
<th>Clinic Patient</th>
<th>&lt; 2 Antl. visits</th>
<th>HDPb</th>
<th>Caesarean Section</th>
<th>Birthweight (grams)</th>
<th>Maternal Age (yrs.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
<td>No. %</td>
</tr>
<tr>
<td>Australia</td>
<td>165</td>
<td>17.0</td>
<td>298</td>
<td>30.3</td>
<td>1254</td>
<td>26.59</td>
<td>3</td>
<td>19.6</td>
<td>34</td>
</tr>
<tr>
<td>Other Oceania</td>
<td>7</td>
<td>17.1</td>
<td>18</td>
<td>42.9</td>
<td>16</td>
<td>39.01</td>
<td>9</td>
<td>45.2</td>
<td>2</td>
</tr>
<tr>
<td>Northern and Western Europe</td>
<td>8</td>
<td>11.9</td>
<td>19</td>
<td>28.3</td>
<td>18</td>
<td>27.32</td>
<td>2</td>
<td>32.8</td>
<td>0</td>
</tr>
<tr>
<td>Southern and Eastern Europe</td>
<td>0</td>
<td>0.0</td>
<td>14</td>
<td>37.8</td>
<td>11</td>
<td>31.41</td>
<td>3</td>
<td>35.1</td>
<td>1</td>
</tr>
<tr>
<td>West Asia</td>
<td>3</td>
<td>5.8</td>
<td>13</td>
<td>24.1</td>
<td>21</td>
<td>38.92</td>
<td>9</td>
<td>53.7</td>
<td>8</td>
</tr>
<tr>
<td>South and East Asia</td>
<td>1</td>
<td>4.2</td>
<td>10</td>
<td>41.7</td>
<td>6</td>
<td>25.0</td>
<td>9</td>
<td>37.5</td>
<td>0</td>
</tr>
<tr>
<td>Southeast Asia</td>
<td>3</td>
<td>8.1</td>
<td>20</td>
<td>52.6</td>
<td>17</td>
<td>45.9</td>
<td>20</td>
<td>52.6</td>
<td>2</td>
</tr>
<tr>
<td>America</td>
<td>0</td>
<td>0.0</td>
<td>1</td>
<td>14.3</td>
<td>3</td>
<td>50.0</td>
<td>1</td>
<td>14.3</td>
<td>0</td>
</tr>
<tr>
<td>Africa</td>
<td>2</td>
<td>11.8</td>
<td>9</td>
<td>52.9</td>
<td>3</td>
<td>17.6</td>
<td>5</td>
<td>29.4</td>
<td>2</td>
</tr>
</tbody>
</table>

¹ This includes divorced, separated and widowed women, as well as never married women.

b HDP means hypertensive disorder of pregnancy.

c A single sample size for each maternal birthplace cannot be given because it varies with each covariate according to the presence or absence of missing values.

(Source: Maternal and Perinatal Collection, 1986)
adjustment for birthweight, and taking maternal Australian birth as the reference group, the estimated relative risks of perinatal death are greatest for the women born in America ($\theta_{mh}=4.11$) and Africa ($\theta_{mh}=2.17$), although these regions are ethnically very heterogeneous. Also, the Mantel-Haenszel estimates are especially unstable for these regions because of the small numbers of women representing them. Although none of the adjusted odds ratios is statistically significant at the 0.05 level, each estimate is assumed to be correct in the following discussion.

South American countries are the most frequently reported birthplaces of mothers born in America. The case group evinces no clear, regional birthplace pattern but migrants from North America and the South American 'southern cone' countries of Uruguay, Chile and Argentina are conspicuous in the control group. These latter nations are economically advanced by South American standards and refugees have come from the countries' more advantaged sectors (Schneider, 1980).

One explanation for the estimated overall high perinatal mortality of Australian mothers born in South America is that Catholicism is the stated religion of almost two-thirds of South American-born women aged 15 to 44 years and living in New South Wales (Australian Bureau of Statistics, 1986b).\textsuperscript{14} If, for religious reasons, these women are less likely to terminate pregnancies with known foetal defects, more 'handicapped' foetuses would be included in perinatal statistics than otherwise. The argument depends, of course, on the assumption that religion is a powerful social force for most South Americans, and this assumption has been questioned, for example, by Moraes-Goraeki (1988).

\textsuperscript{14} According to the 1986 Census of Population and Housing, 63.5 per cent (n=7055) of women with the stated characteristics identified themselves as Catholics.
Table 8.3 suggests that antenatal care is received later by the group of all American mothers of low birthweight babies than by the women born in other regions - which could contribute to the increased perinatal mortality of the women born in America. Further discussion of these women is probably not helpful because of the ethnic diversity of their regional birthplace and its representation by so few women.

The African-born women are mainly from Egypt, Mauritius and South Africa. Small numbers show these mothers' babies to be at high risk of perinatal death particularly at birthweights 2 500 to 3 999 grams. Contributing perhaps to these estimated risks of Africans is the increased proportion of Muslim women (\(\text{vis-à-vis}\) Coptic Christians) that has recently migrated to Australia, especially from Egypt.

In parts of South and West Sydney, many Muslim women have attended late for antenatal care rather than be examined by male doctors, so jeopardizing perinatal well-being (Bennett and Shearman, 1989; New South Wales Department of Health, 1989b). However, this argument is also relevant to West Asian women, mainly from Lebanon, whose Mantel-Haenszel relative odds of perinatal death was half as low \((\theta_{mh}=1.09)\) as that for women born in Africa. Table 8.3 suggests a high proportion of primigravidae among selected African women, which is usually considered a perinatal risk factor.

Other odds ratios appear much smaller, being highest for Southeast Asian-born women \((\theta_{mh}=1.51)\), especially at very low birthweights. Most of these women have come from the Philippines or mainly as refugees from Indo-China, especially Vietnam. Chan et al. (1988), who presented South Australian data, for 1981 to 1983, found that Filipino women were mainly primigravidae and comparatively old. The same characteristics, which were observed for Vietnamese women by Cauchi (1986), are shown in Table 8.3 for the Southeast Asian mothers of low
birthweight babies. The mean age of these women, 27.4 years (s=3.8), is high compared to four of the other, large birthplace groups, and approximately half of the Southeast Asian women are primigravidae.

The age factor and high incidences of both antepartum haemorrhage and prolonged labour were presented by Chan et al. (1988) as partial explanations for a disconcertingly high rate of caesarean section (36 per cent) among Filipino women. If, as their paper implies, cephalopelvic disproportion was an important indication for what were mainly emergency caesarean sections, an additional reason for these caesarean operations can be suggested: larger babies owing to increases over the last decade in Filipino women marrying Australian men (Watkins, 1982). The data used to produce Table 8.3 support the hypothesis, for the mean (low) birthweight for Filipino women is very high at 1992.5 grams (s=616.9).

Vietnamese women not only begin childbearing comparatively late but they tend to be of short stature and low body weight and, partly for these reasons, typically deliver smaller babies than, for example, Australian-born women (Ward et al., 1981; Cauchi, 1986). This last finding, which was not reproduced by the present study, is exemplified by Chan et al (1988) who found the mean birthweight for Vietnamese women to be 263 grams lower than for native Australians.

In addition, many Vietnamese women have been noted not to receive antenatal care until after the first trimester (Ward et al., 1981; Chan et al., 1988), this likewise being found in the present study for women having babies of low birthweight: half (n=7) of the Vietnamese women did not seek antenatal care during the first ten weeks of their pregnancies. As reported in Table 8.3, late antenatal attendance (45.9 per cent, n=17) is typical of Southeast Asian women generally,
who also appear especially likely to receive care from a hospital antenatal clinic.

Vietnamese women have further been observed to be uncertain of the dates of their last menstruation, a problem which is exacerbated when Vietnamese patients follow the Chinese lunar calendar. The Vietnamese, like some other groups in developing countries (for example, Sri Lanka), are discouraged from breastfeeding by their belief that colostrum is hazardous for babies (Duke, 1988) and by reasons relating to the Asian humoral system (Fishman et al., 1988).

Further affecting the survival chances of newborn of Southeast Asian women, as of women from Africa and from Mediterranean countries, are the haemoglobinopathies\textsuperscript{15} in pregnancy, which can be broadly divided into thalassaemia\textsuperscript{16} and variant haemoglobins. For example, high incidences of \( \beta \) thalassaemia, as well as non-thalassaemic anaemia, have been reported for Greek- and Italian-born women (Dunt et al., 1973; Cauchi, 1986), even though women born in Southern Europe record a Mantel-Haenszel estimated odds ratio of 0.71 relative to native Australian women. Cauchi (1986) found Australian women born in Southeast Asia to have a much higher incidence of severe anaemia (haemoglobin below 100 gm/dl) than the women born in Australia.

Possibly also contributing to perinatal mortality risks are economic or psychological hardships suffered by refugees. Speculations on economic grounds are perhaps less fruitful. Many Indo-Chinese refugees were disadvantaged economically, but they were 'political', not 'economic', refugees who embraced diverse economic backgrounds (Kelly, 1988). Middle class Vietnamese at first faced long periods in low paying jobs, yet considerable continuity of occupational class has

\textsuperscript{15} These are genetic disorders of haemoglobin.

\textsuperscript{16} In this hereditary blood disease there is an abnormality in the protein part of the haemoglobin molecule leading to anaemia.
occurred in their movement between Vietnam and Australia (Lewins, 1984).

Probably less uncertain is the extent to which the physical and psychological stresses of refugees' experiences affect perinatal survival. Imprisonment, forced labour and 're-education' of Indo-Chinese refugees have often produced depressive and trauma-related syndromes generically described by the 'post-traumatic stress disorder'. This, in turn, has been compounded by forced withdrawal from the home country and by the stresses of adjustment to life in a new country. Because, in New South Wales, few sources of treatment and rehabilitation for refugees have been available, many victims in the absence of help have suffered enduring hardships (Kalucy, 1988; Reid and Strong, 1988). It does not seem improbable that, in some refugees, perinatal complications could be late sequelae of persecution and migration stresses (see section 2.8.1).

Women born in Other Oceania record the birthweight-adjusted odds ratio of 1.32, this being due entirely to newborn with very low birthweights recording a higher estimated relative risk of death ($\Theta=8.59$) than other babies of similar birthweight. Relative to Australian-born women, the Mantel-Haenszel relative odds of perinatal death is also elevated for women born in South or East Asia, West Asia, or Northern or Western Europe, but it is lower for women born in Southern or Eastern Europe.

According to Table 8.3, when low birthweight babies have mothers born in Other Oceania or especially in West Asia, salient adverse characteristics are poor antenatal records, including late and too few

17 Military dictatorships in Latin America have used trained torturers to inflict somatic and psychological damage.
18 Between 5 and 30 per cent of incoming refugees are estimated to have been severely traumatized, suggesting individual differences in vulnerability to traumatic effects (Reid and Strong, 1988).
attendances frequently at hospital clinics, and low mean birthweights. High incidences of caesarean sections are also reported for women born in West Asia (43.4 per cent, n=23) or in Northern or Western Europe (41.8 per cent, n=28), while South or East Asian women are conspicuously primigravidae.

The low relative risk of perinatal death among Australian-born women is noteworthy even though it does not occur at very low birthweights, 0=4.02 (see Table 8.2), and in Table 8.3, the low birthweight newborn of Australian women record the lowest mean birthweight of any birthplace group. An Australian birthplace also brings high proportions of non-married women (17.0 per cent, n=165) and women with hypertensive disorders of pregnancy (15.6 per cent, n=339). Nevertheless, the overall perinatal survival advantage of infants born to native Australian women attracts notice for empirical and theoretical reasons.

Empirical evidence shows the opposite pattern among adults. Young (1989) found that at adult ages (15 to 74 years), standardized mortality ratios for 1980 to 1982 were higher for Australian-born persons than for most other birthplace groups. Apart from the different ages being looked at, the contrasting results cannot be explained by the regional focus of the present study vis-à-vis Young's country-specific analysis. For example, the women born in developed countries, in regions such as America and Other Oceania that contain countries at different levels of development, still record higher perinatal mortality risks than do Australian-born women. This is exemplified by New Zealand women in Australia whose perinatal
mortality is also higher than that for all New Zealanders (United Nations, 1988).\(^{19}\)

This is surprising since, in terms of poorly developed migration selectivity theory, it can be postulated that perinatal mortality risks should be lower for migrants born in developed regions (including developed countries) than for Australian-born women. When the 'pull stimulus' is greater than the 'push stimulus', as is perhaps more likely when the source area is developed, migrants will more usually be positively selected (Lee, 1966, Lewis, 1982), for example in terms of education, than non-migrants and the population of the destination area. This should also result from Australia's policy of selecting immigrants from developed (and developing) areas according to their skills and health before coming to Australia (Young, 1987).

However, besides the difficulties already noted to affect migrants from particular - especially developing - birthplace regions (hence hypothesis 15, section 3.1), migrants generally may have problems adapting to Australian culture and society, and these problems could also increase migrants' risks of perinatal death - irrespective of their home countries' levels of development. For example, Eisenbruch (1989) notes that 'cultural bereavement', resulting from a loss of social structure and cultural meaning, can affect migrants, and not just refugees, long after resettlement. A further potential complication is difficulties of language and communication between non-English speaking migrants and health professionals. Labour wards have lacked suitable interpreter services out-of-hours (Duke, 1988) despite the many languages and dialects found in urban migrant communities.

\(^{19}\) Perinatal mortality rates in 1985, calculated for the purpose of international comparisons but including neonatal deaths, were 10.9 for Australia and 9.5 for New Zealand (United Nations, 1988).
The magnitude of the language 'problem' is revealed by approximately ninety birthplace groups (which include still other countries) that are listed by the Australian Bureau of Statistics and for which English is not the main language. The communication problem is thought to be a further reason why migrant groups, for example from Vietnam and the Philippines, generally commence antenatal care later than do Australian-born women (Chan et al., 1988; Duke, 1988).

The superior perinatal outcomes of women born in Southern and Eastern Europe, except perhaps at birthweights 1 500 to 2 499 grams, are consistent with very low rates of overall mortality among especially Australian Greeks and Italians (Young, 1989). In terms of perinatal survival, both communities have been observed to have low levels of pre-eclampsia, plus favourable birthweight distributions and early attendance for antenatal care compared with other birthplace groups in Australia (Dunt and Le Moine Parker, 1973; Cauchi, 1986).

Table 8.3 shows mothers born in Southern or Eastern Europe to have a high incidence of the hypertensive disorders of pregnancy (21.6 per cent, n=8) and a fairly low mean birthweight. However, the group evinces, for example, a good level of antenatal care which, among other things, would contribute to its estimated low perinatal mortality. The apparent survival advantage may be due to selective migration, cultural factors such as diet including lower levels of alcohol and cigarette consumption than the national average (Lee et al., 1987), and a generally longer duration of residence in Australia.

20 However, Chan et al. (1988) found slightly elevated proportions of Yugoslav women to make fewer than seven antenatal visits; the difference was not statistically significant.

21 The continuing high intake of fruits and vegetables by Southern Europeans has been linked to low all-cause mortality (Kromhout et al., 1982; Kahn et al., 1984). However, high levels of non-thalassaemic anaemia among these women might also be partly explained by diet, including perhaps a low red meat intake, as well as by failure to take iron supplements (Dunt and Parker 1973) and the high parities and by implication close birth intervals of Southern European women (Chan et al., 1988).
than characterizes most other immigrant groups; this would promote a knowledge of English and available services. Mental health problems among Southern and Eastern European refugees (Lee et al., 1987) are now probably confined to persons older than the reproductive ages. Lower risks of perinatal death are found for native Australian women than for women born in other regions except Southern and Eastern Europe.

8.2.3 MARITAL STATUS

Typically, births are studied either for the collectivity of all unmarried women, or for the group of never married, especially teenage, women. Despite commonalities between non-marital groups, for example in adverse economic and psychosocial environments (see section 2.11.2), it is important especially now, following recent increases in marital breakdown and remarriage, to explore distinctive features of the formerly-married component, as well as of the never-married component, of births to single women.

This is done in Table 8.4 without, owing to data limitations, adjusting for mediating factors describing economic and social supports. The data, drawn from Maternal and Perinatal Collection, also use categories of marital status that are potentially unreliable. Marriages are defined by the Collection to include de facto relationships, but whether or not stable cohabiting unions are recorded as marriages depends on how these unions are reported by patients and are interpreted by doctors and the midwives who complete the Collection form. Misclassifying married women in groups of presumably higher risk 'single' women, would thus understate the dangers faced by the latter women. Potential for bias also lies with different degrees of misclassification in the non-married groups of
never married and formerly married women, yet there is no evidence to suggest this happens.

After weighting, about 11 per cent (n=7 774) of all the women reported in Table 8.4 are estimated to be non-married, approximately 16 per cent (n=1 227) of them being formerly married. Among both the never married and formerly married, proportionately more births occupy the strata of low birthweights or, specifically the birthweight stratum 1 500 to 2 499 grams, than of higher birthweights. In proportionate terms, an estimated 1.4 times more non-married women deliver babies weighing 1 500 to 2 499 grams than 2 500 to 3 999 grams.

Table 8.4: Relation of Perinatal Death to the Marital Status of Women Aged 20 to 34 Years according to Birthweight, 1986

<table>
<thead>
<tr>
<th>Marital Status</th>
<th>Birthweight</th>
<th>500-1 499</th>
<th>1 500-2 499</th>
<th>2 500-3 999</th>
<th>4 000+</th>
<th>θ_{mh}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Married</td>
<td>P.D. Ctl. 0</td>
<td>34</td>
<td>47</td>
<td>3.67***</td>
<td>26</td>
<td>102</td>
</tr>
<tr>
<td>Never Married</td>
<td>P.D. Ctl. 0</td>
<td>31</td>
<td>44</td>
<td>3.58***</td>
<td>20</td>
<td>74</td>
</tr>
<tr>
<td>Formerly Married</td>
<td>P.D. Ctl. 0</td>
<td>3</td>
<td>3</td>
<td>5.07</td>
<td>2</td>
<td>18</td>
</tr>
<tr>
<td>Married*</td>
<td>P.D. Ctl. 0</td>
<td>229</td>
<td>316</td>
<td>3.68***</td>
<td>94</td>
<td>480</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
*** Statistically significant at the 0.005 level
(Source. Maternal and Perinatal Collection, 1986)

Taking married women as the reference group and adjusting for birthweight, the estimated relative risk of perinatal death for non-married women is 1.14. For never married and formerly married women, the Mantel-Haenszel estimated odds ratios are 1.12 and 1.23 respectively. Both marital and non-marital births are most hazardous at very low birthweights. Only babies 1 500 to 2 499 grams born to never married women are at higher risk of death than are heavier
newborn. Infants born to formerly married women are conspicuously safe at birthweights 1 500 to 2 499 grams, but at increased risk of perinatal death at very low birthweights ($\Theta=5.07$) and at normal birthweights ($\Theta=2.18$).

When single parenthood is identified as a risk factor for perinatal death, the concern, as noted above, is invariably with never married, and especially teenage, women. Births to never married women are more frequent than births to formerly married women, according to Table 8.4 more than five times so among women aged 20 to 34 years – but the coping abilities of formerly married women are not necessarily superior to those of never married women. Given that the highest estimated odds ratios in Table 8.4 are for formerly married women, among whom all perinatal deaths are to separated or divorced women, discussion is required of the economic and social consequences of marital breakdown for women.

Divorce has been observed to bring a drop in household income (McLanahan, 1985), this being most damaging for the women who did not earn independent incomes immediately before the separation, or who had not yet repartnered22 (McDonald, 1985). Pregnant, divorced women may be especially likely to have repartnered and, therefore, not to have been financially disadvantaged. However, it may still be relevant that the wife's share of property on divorce has tended to be reduced by her leaving the matrimonial home and not being the custodial parent, as well as by total wealth that includes a high percentage of non-basic assets. Separation and divorce, with attendant determinations of a property settlement and maintenance, and custody

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22 Before cohabitation is classified as de facto marriage, a 'trial' period may be necessary for the couple, or for the record holder. On settlement of the total property, the wife’s share is diminished if she is not the custodial parent, if non-basic assets are a high proportion of total wealth, and if the wife left the matrimonial home in the initial period following separation.
of, and access to, any children, are also ingredients for distress and trauma (McDonald, 1985).

Several mainly cross-sectional studies have shown separated and divorced people to be especially susceptible to emotional and physical illnesses (Goetting, 1981; Tcheng-Laroche and Prince, 1983); while marital dissolution is positively associated with female suicide (Stack, 1980, 1981; Trovato, 1988). Discord and unhappiness are likely to precede marital breakdowns, but a prospective study by Menaghan and Lieberman (1988) found individual well-being also to be a consequence of marital termination.

There are reasons supporting this. More than the never married, separated and divorced people associate being alone with loneliness and unhappiness (Cargan, 1981). This may be because formerly married people must adjust to losing a longer-standing and possibly more truly caring relationship than that leading to a birth among the never married. Social stigmas against non-marital birth may be severe against formerly married women who, usually being older than never married women, are expected to be 'wiser', especially by peers and hence primary affiliates from the same generation. The marital status with the highest estimated risk of perinatal death is being formerly married.

8.3 SOCIOECONOMIC FACTORS

One of the three explanations cited in section 2.11.3 for the widely reported association between low income and increased rates of perinatal death is diminution of access to items of consumption including health insurance. It has been suggested, for example by Grosse (1979), that a problem with this argument is that low income and poverty are not necessarily the same: low income and a high level
of health care can coexist. Whether or not they do, depends on the
distribution of income within households and on how that income is
used. What is important is the relative proportions of income spent
on items such as food, clothing and health care.

Countervailing this logic is the proposition that low income
households, by definition, have less income to distribute, and ceteris
paribus, spend less income in absolute, if possibly not proportionate,
terms on basic expenditure items such as health. Supporting this view
is the 1984 Household Expenditure Survey in Australia (Australian

The survey indicates that compared to low income households, medium
income and high income households spent 2.2 and 3.6 times more in
dollar terms of their average weekly gross incomes on medical care and
health expenses. In proportionate terms, these outlays amount to
3.83 per cent of the weekly expenditures of low income households, and
3.85 and 3.59 per cent of the weekly expenditures of middle income and
high income households respectively. Only absolute spending is
reported for specific health expenditures, dollar expenditures on
health and accident insurance being 6.8 times greater for households
with high incomes than with low incomes.

To develop the argument, the purchase of equivalent types of care
would require low income households to spend proportionately much more
than higher-income households on health care, which has not happened
widely in New South Wales. To the extent that relative over-spending

23 High income and low income households are the highest and lowest 20 per
cent of households respectively after they have been ranked in ascending
order. In New South Wales these households equated to less than $169.00 per
week and more than $695.00 per week respectively. Middle income households
fall between these limits.
24 This includes accident and health insurance; practitioners' fees;
medicines, pharmaceutical products, therapeutic appliances and equipment; and
other health charges.
on health has typified individual low income households, other expenditures are likely to have been forgone or reduced since resources are limited; and this could have had mixed consequences for perinatal outcome.

Accordingly, it is hypothesized that low income households in the present survey are also less likely than higher income households to have health insurance. Without regard for the type or amount of insurance, there are two assumptions underlying this hypothesis. First, low income households more often forego this expenditure because secondly, they are forced to direct their health dollars elsewhere: toward, for example, recouping any Medicare deficit. Of further interest is how insurance affects perinatal outcomes for different income households.

With a very small table size, Table 8.5 relates perinatal death to terciles of 1986 gross household income and to health insurance. The lowest-income households, earning less than $20,000 dollars per annum constitute 12 per cent (n=112) of the weighted sample of households. One quarter (24.4 per cent, n=22) of low birthweight deliveries were to low income households, and 36.4 per cent (n=8) of these households did not have health insurance.

Three-quarters (n=717) and about 13 per cent (n=119) of households received gross incomes in 1986 of $20,000 to $50,000 and more than $50,000 respectively. Of all households, those with middle incomes were the most likely to deliver newborn weighing more than 2,499 grams. Low birthweight babies were born into the highest-income households (93.1 per cent) even more frequently than into the lowest-income households (88 per cent, n=22), although this is almost certainly an artefact of the small numbers. At low birthweights, health insurance was not reported by 19.5 per cent (n=8) of middle
income and 14.8 per cent (n=4) and high income households whilst, weighting and then collapsing over both birthweights, the ratio of insurance to no insurance takes a J-shaped pattern with increasing income.

Table 8.5: Relation of Perinatal Death to Household Income of Women Aged 20 to 34 Years according to Health Insurance and Birthweight, 1986

<table>
<thead>
<tr>
<th>Health Insurance</th>
<th>Gross Annual Income ($)</th>
<th>Birthweight (grams)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>500-2 499</td>
<td>2 500-3 999</td>
<td></td>
</tr>
<tr>
<td>P.D. Ctl.</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>P.D. Ctl.</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>θ_{mh}</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>10</td>
<td>4</td>
<td>4.40</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1</td>
<td>3.50</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>1</td>
<td>3.10</td>
</tr>
<tr>
<td>&lt;20 000</td>
<td>4</td>
<td>4</td>
<td>1.75</td>
</tr>
<tr>
<td>20-000-50 000</td>
<td>15</td>
<td>15</td>
<td>1.46</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>7</td>
<td>1.00b</td>
</tr>
<tr>
<td>&gt;50 000</td>
<td>15</td>
<td>8</td>
<td>3.28</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>1</td>
<td>1.75</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>1</td>
<td>2.19</td>
</tr>
<tr>
<td>No</td>
<td>4</td>
<td>4</td>
<td>1.75</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>2</td>
<td>1.57</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>2</td>
<td>1.20</td>
</tr>
</tbody>
</table>

P.D: Perinatal Death; Ctl: Control
a Reference group for Mantel-Haenszel estimated odds ratios
b Reference group for other odds ratios
(Source. Hospital patients' records, 1985-86)

After adjustment for maternal age, birthweight and health insurance, the estimated relative odds of perinatal death decline from 2.10 for the lowest-income households to 1.00 for middle income households before rising unexpectedly to 1.89 for households earning more than $50 000 in 1986. Without adjustment for health insurance, health insurance is beneficial only for middle income households. Insurance is least effective within low income households (θ_{mh}=3.10), middle income and insured households providing the reference group. For the highest-income households, θ_{mh} is 2.19 with insurance and 1.20 without health insurance, and the combination of no health insurance and the
lowest incomes also appears surprisingly safe. Birthweight-specific estimates mirror these trends. The highest estimated odds ratios are generally associated with low birthweight newborn, estimated relative risks of perinatal death being greatest for low income households and often not being improved by health insurance.

Selection for health insurance by women likely to be at increased risk of perinatal complications may explain the lesser ability of health insurance vis-à-vis no health insurance to minimize estimated relative risks of perinatal death for low income and high income households. However, this result may be an artefact of small numbers since middle income households, who are the most adequately represented income group in Table 8.5, are apparently immune from any such selectivity effect.

8.4 REGRESSION ANALYSIS OF RISK FACTORS FOR PERINATAL DEATH

A conditional logistic regression model was fitted to estimate the relative odds of perinatal death for each level of six categorical factors, net of the effects of other factors contained in the model. This was done after adjustment for the main and interactive effects of the two other factors, birthweight and maternal age, used earlier to group match cases and controls. Included in the regression analysis were 2,607 subjects, of whom 614 belonged to the case group and 1993 to the control group.

The selection of predictors for inclusion in the multivariate analysis was based on five criteria, the first two being coverage in the Maternal and Perinatal Collection, and relevance to stillborn foetuses as well as to neonates. These criteria excluded factors describing maternal behaviour or relating to the labour and delivery. Thirdly, a good mix of proximate and background variables was required, as was,
fourthly, evidence from this or other studies of factors contributing to perinatal death. The final criterion was that variables could not have substantial missing values. When this happened, as with the mother’s country of birth, the complete record was itself frequently atypical and removed from the analysis.

The method of model selection was described in 2.10. Suffice it here to say that eleven models were fitted to yield a change in the scaled deviance, subsequent to including birthweight and maternal age, of 143.1 for a loss of 11 degrees of freedom. The goodness of fit of the models was most improved by taking into account the main and interactive effects of gestational age and obstetric complications. Adding antenatal care had almost no effect. This was also true for two background factors, marital status and Aboriginality, although a statistically significant (p < 0.005) interaction occurred between obstetric complications and gravidity. The otherwise weak contribution of the background variables did not change when they were entered before other, more proximate variables. This supports the conceptualization of background variables as affecting perinatal death through other mediating factors, that is indirectly.

Table 8.6 presents the main effects of four variables: antenatal care, gravidity, marital status and Aboriginality, where coefficients estimated for each level of a factor are adjusted for all other factors in the model. As shown by the t values, none of the estimated odds ratios is statistically significant, which may be due, in part, to small numbers. Relative risks estimated through logistic regressions are for the most part not inconsistent with estimates obtained through tabular analyses. Thus adjusting for large numbers of factors through regression has often a minimal effect.
Relative to two or more antenatal visits, fewer than two visits yields an estimated odds ratio of 1.19, which is similar to the birthweight adjusted Mantel-Haenszel odds ratio of 1.09 for women aged 20 to 34 years (see Table 6.5). Primigravidae have approximately the same estimated relative risk of a perinatal death as have multigravidae, while that for Aboriginal women, 1.12, is much smaller than $\theta_{mh}=2.41$ from Table 8.1. This is possibly because the very small number of identified Aboriginal women does not permit adjustment for the seven other variables.

Table 8.6: Parameter Estimates for the Main Effects of Suspected Risk Factors for Perinatal Death after Adjustment for Birthweight and Maternal Age, 1986

<table>
<thead>
<tr>
<th></th>
<th>Maximum Likelihood Estimate</th>
<th>Standard Error</th>
<th>T value</th>
<th>$\theta^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>-1.775</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antenatal Care</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fewer than 2 visits</td>
<td>0.170</td>
<td>0.260</td>
<td>0.654</td>
<td>1.19</td>
</tr>
<tr>
<td>2 or more visits</td>
<td>0.000</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gravidity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primigravidae</td>
<td>-0.044</td>
<td>0.140</td>
<td>0.314</td>
<td>0.96</td>
</tr>
<tr>
<td>Multigravidae</td>
<td>0.000</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never married</td>
<td>-0.037</td>
<td>0.158</td>
<td>0.234</td>
<td>0.96</td>
</tr>
<tr>
<td>Formerly married</td>
<td>0.237</td>
<td>0.330</td>
<td>0.718</td>
<td>1.27</td>
</tr>
<tr>
<td>Married</td>
<td>0.000</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aboriginality</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aboriginal</td>
<td>0.115</td>
<td>0.380</td>
<td>0.303</td>
<td>1.12</td>
</tr>
<tr>
<td>Non-Aboriginal</td>
<td>0.000</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$a$ Odds ratio adjusted for birthweight and maternal age.
(Source. Maternal and Perinatal Collection, 1986)

Being formerly married is the marital status most hazardous for perinatal outcome. Taking currently married as the reference group, formerly married women record an odds ratio of 1.27 which is nearly
identical to the Mantel-Haenszel estimate of 1.23 reported in Table 8.4. However, in contrast to this earlier analysis, never married women are now shown not to be at increased risk of perinatal death. The reversal is interesting because, although only some never married women are adolescents, it accords with findings that apparently high perinatal mortality risks associated with adolescence are removed by adjustment for a large number of variables.

Table 8.7: Estimated Odds Ratios for Statistically Significant Interactions between Suspected Risk Factors for Perinatal Death, 1986

<table>
<thead>
<tr>
<th>Gestational Age</th>
<th>Gestational Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preterm Birth</td>
<td>Birth from 37 weeks</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Birthweight (grams)</th>
<th>Gestational Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>500-1 499</td>
<td>8.18 1.80</td>
</tr>
<tr>
<td>1 500-2 499</td>
<td>2.46 0.61</td>
</tr>
<tr>
<td>2 500+</td>
<td>10.90 1.00</td>
</tr>
<tr>
<td>Obstetric complications</td>
<td>Selected complications</td>
</tr>
<tr>
<td>No or other complications</td>
<td>10.90 1.00</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Obstetric complications</th>
<th>Obstetric complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Selected complications</td>
<td>Gestational Age</td>
</tr>
<tr>
<td>No or other complications</td>
<td>1.97 1.00</td>
</tr>
</tbody>
</table>

a See the main text
(Source. Maternal and Perinatal Collection, 1986)

Estimated odds ratios for factors included in statistically significant interactions are reported in Table 8.7. Gestational age interacts strongly with obstetric complications (p < 0.005) and with birthweight (p < 0.05). The latter interaction yields higher estimated odds ratios for preterm birth than for birth from 37 gestational weeks, irrespective of birthweight. Newborn weighing at
least 2 500 grams ($\theta=10.90$) are at greater risk of perinatal death if they are preterm than are very low birthweight newborn who are also preterm ($\theta=8.18$).

Among preterm births, the relative odds of perinatal death ($\theta=2.58$) associated with four selected obstetric complications – antepartum haemorrhage, the hypertensive disorders of pregnancy, premature rupture of the membranes and multiple birth – is more than three times lower than for less common, or no, obstetric complications. For non-preterm births, the estimated relative risk of perinatal death is higher in the presence of the selected complications than of other complications but, irrespective of complications, lower than for preterm births.

However, taking as the reference group multigravidae without the complications named, the relative odds of perinatal death are highest for primigravidae with the stated complications ($\theta=1.97$). It will be recalled from sections 5.3 to 5.3.3 that more prevalent among primigravidae than multigravidae are hypertensive disorders of pregnancy and placenta praevia, but not premature rupture of the membranes.

8.5 CONCLUSION

The main body of this chapter investigated the separate abilities of three sets of maternal background factors to explain perinatal deaths. These factors were medical history factors, sociocultural factors and socioeconomic factors. Gravidity and interpregnancy intervals were taken as empirical referents of the expectant mother's medical history.
The relation of gravidity to perinatal death was studied by focusing on compositional and timing characteristics of women's reproductive histories. A method was presented for evaluating these histories' potential confounding effects whilst using them, at the same time, to describe the expectant mother's gravidity and parity. Analyses supported the hypothesis of heterogeneous risk and selective fertility in finding perinatal mortality risks at different gravidities to be influenced by good and poor previous pregnancy outcomes, as well as by the outcome of the immediately preceding pregnancy.

Successful reproducers recorded lower estimated odds ratios than did poor reproducers. Among the latter group, and successful reproducers at gravidities 2 to 4, increasing parity was shown to be hazardous. However, some protection was afforded by a last pregnancy outcome that was good rather than poor. By taking these compositional and timing factors into account, an attempt was made to measure the effect of gravidity per se. In contrast to known risk patterns, primigravidae recorded the lowest estimated relative risk of perinatal death. Among multigravidae, a history of fewer good than poor outcomes was less safe, first, at gravidities 5 and above when the last preceding outcome was bad, and secondly, at gravidities 2 to 4 when the last previous outcome was good.

Measurement was undertaken of three sociocultural influences, the first being Aboriginality. Undercounting of Aboriginal women might have biased the estimates toward unity. Aboriginal women were 2.2 times more likely to have had a baby of low than normal birthweight, and relative risks of perinatal death were estimated to be higher for Aboriginal than non-Aboriginal newborn at very low and, in particular, normal birthweights. Taking non-Aboriginals as the reference group, the birthweight-adjusted Mantel-Haenszel odds ratio was 2.41.
Adjustment for seven variables through logistic regression reduced the odds ratio for Aboriginals to 1.12.

The second sociocultural factor studied was the woman's region of birth. Slightly more than one-fifth of the births, after weighting, were to women born outside Australia, particularly in Europe. No increased tendency was identified for non-Australian born women to deliver low birthweight babies. The highest relative odds of perinatal death characterized women with American and African birthplaces. Other odds ratios were smaller, being highest for Southeast Asian born women, mainly from the Philippines and Vietnam. Women born in Southern or Eastern Europe recorded the lowest estimated relative risk of perinatal death.

Marital status was the third investigated sociocultural variable. Infants born to the minority of non-married women were more likely to be of low birthweight (a) than were the infants of married women and (b), especially at birthweights 1,500 to 2,499 grams, than of normal birthweight. Relative to married women, the birthweight-adjusted odds ratio for non-married women was 1.14; for never married, it was 1.12 and for formerly married women, 1.23. Logistic regression confirmed the elevated risk for formerly married women (0=1.27), but indicated almost no difference in the relative odds for never married women.

To study socioeconomic influences on perinatal death, the hypothesis was tested that low income households are likely to have no health insurance and, in consequence of both characteristics, increased risks of perinatal death. The results confirmed a positive association between household income and the ownership of health insurance. However, such insurance made the least difference for low income households. After adjustment for health insurance, low income households had the highest estimated relative risk of perinatal death.
To complete the chapter, a logistic model was fitted to estimate the relative risk of perinatal death associated with each level of six categorical factors after prior adjustment for birthweight and maternal age. Background factors contributed little to the fit of the model, and odds ratios for Aboriginality and marital status were given above. The relative odds were effectively the same for primigravidae and multigravidae. Except for antenatal care, fewer than two visits recording $\theta=1.19$, the other proximate factors were included in statistically significant interactions.
This thesis has fulfilled its aims of developing a conceptual model of risk factors for perinatal death, and of testing hypotheses, extracted from this model, by subsequently collecting and analysing 1985 and 1986 New South Wales data in a case-control research framework. In this chapter, these hypotheses are re-examined in turn in the light of the empirical findings of the thesis, and an evaluation is made of the strengths and limitations of the study's overall contribution to interdisciplinary knowledge about perinatal mortality in New South Wales and in other developed contexts. Research needs for the future are identified.

9.1 RE-EVALUATION OF HYPOTHESES

This thesis is less concerned with verification than exploration, but it is incumbent on the researcher to re-evaluate the hypotheses specified in section 3.1, on the basis of the empirical evidence reported in chapters 5 to 8, for the studied 1985-86 infants of New South Wales women aged 20 to 34 years.

A general hypothesis, which is considered aetiologically new, identified five groups of proximate determinants of perinatal death. Empirical study of the effect of chromosomal and genetic factors on perinatal outcome was not possible. But with this omission, the study confirmed the hypothesis, finding each group, taken separately, to have a strong influence on perinatal death. Analyses mainly focused within these groups, although multivariate investigations are also
needed of the relative aetiological contributions of each group. The identification of proximate determinants was conceptually useful in giving structure to the investigation.

The results of testing four sets of specific hypotheses are re-examined separately. The first set of hypotheses relates to the physiological maturity of the foetus and infant, and was examined in the context of the inter-relations between birthweight, gestational age and the appropriateness of birthweight for gestational age. Growth retardation in utero was first hypothesized, on the basis of findings from elsewhere, to yield better perinatal outcomes than preterm birth - since early gestation retards not only growth but development of the foetal physiological systems. It was found that babies born before term, but with birthweights appropriate for gestational age, yielded higher relative risks of perinatal death than did term babies who were small for gestational age.

A slightly more ambitious hypothesis was that at low birthweights, intrauterine growth retardation often precedes preterm birth, especially as the gestational duration increases, and that concurrence of both complications produces worse perinatal outcomes than does either of the two complications when it occurs independently. All parts of this hypothesis were broadly confirmed. Half of all the preterm babies of low birthweight were also growth retarded and, with advancing gestational age, the proportion of growth retarded infants increased; almost two-thirds (n=403) of growth retarded babies at low birthweights were born before term. When both causes of low birthweight occurred together, the baby was more likely to die than when only one cause was found. However, at very low birthweights,

1 A comprehensive summary of all the main results can be found in the individual empirical chapters.
preterm birth was not made more hazardous by the coincidence of growth retardation in utero.

The second set of hypotheses responds to uncertainty in the literature about so-called obstetric factors. Support was found for the hypothesis that low Apgar scores at one minute are better associated with neonatal mortality risk than are Apgar scores at five minutes: the risk of perinatal mortality associated with the comparatively rare scores of 0 to 2 was 1.6 times higher at the earlier time. Higher scores, especially 3 to 6, were less often associated with neonatal death than were comparable scores at five minutes. Low neonatal ponderal indices were hypothesized to be a useful predictor of neonatal death. They showed an increased risk of death, but this increase was not appreciably large, and unstable relative risk estimates suggested that these indices have low sensitivity.

The next hypothesis was that perinatal death was less frequent at low birthweights among multiples than among singletons of equivalent preterm gestational ages. The study supported this comparatively new hypothesis especially between 28 and 36 weeks but in fact almost exclusively at birthweights 1 500 to 2 499 grams.

The final hypotheses relating to obstetric factors postulated that male births predispose to selected risk factors for perinatal death: preterm birth, hypertensive disorders of pregnancy, placenta praevia and preterm rupture of the membranes. These hypotheses, which derive from very little earlier research, were tested for the singleton babies of non-Aboriginal women. The strongest support was found for a positive association between proportions of boys and preterm births at birthweights 1 500 to 2 499 grams in particular, but also at normal birthweights. Resulting differences in exposure by males and females to the mortality risks associated with different birthweight and
gestational age combinations help to account for the generally higher male than female perinatal mortality. An increased incidence of boys was not associated with the other three specified obstetric complications except unclassified hypertension for which reported numbers were small. Placenta praevia and most reliably – because of the larger cell sizes – preterm membrane rupture brought higher estimates of perinatal mortality for girls than for boys born especially to multiparous women. Both unclassified hypertension and pre-eclampsia yielded higher male than female perinatal mortality.

Health care factors form the subject of the third group of hypotheses, which have important monitoring implications. It was postulated that the babies at highest risk of perinatal death are born in the hospitals most equipped to care for them. In 1986, the majority of very low birthweight infants were indeed liveborn in units providing the highest (level 3) neonatal care. However, almost one-third of these smallest infants were born in hospitals providing level 2 neonatal care at which estimated relative risks of perinatal death were higher than at level 3 hospitals. The high-risk character of the maternity and infant populations of these latter units is reflected in a slightly elevated odds ratio. Overall, small maternity hospitals and hospitals providing level 2 neonatal care were safe, suggesting effective risk assessment and compliance with referral protocols.

A positive association was hypothesized between the perinatal outcome and the quantity of antenatal care received by women. This hypothesis, which accords with most current thought, was borne out only at normal birthweights. Increased risks of perinatal death were associated with more than 10 antenatal visits by the women who gave birth to infants weighing less than 2 500 grams and especially 1 500 to 2 499 grams. These women received the most care presumably because
they were identified to be at higher risk of perinatal complications than the other women who delivered low birthweight babies. A different explanation is that some antenatal care may be ineffective. In either event, the receipt of much antenatal care could not reduce perinatal mortality sufficiently to yield a low relative odds of perinatal death.

Only for low birthweight infants was the hypothesis upheld that, before term, induction attends a higher risk of neonatal death than does no labour and thus elective caesarean section. The comparative risks of induction and caesarean section for these infants were considered to explain the result better than differences in the risk statuses of the groups undergoing the procedures. Other evidence presented could neither confirm nor reject the new hypothesis that in nulliparae, the active management of dystocia is a safe alternative to emergency caesarean section. However, results rejected the hypothesis that the incidence of caesarean section can be safely reduced in the delivery of breech infants of very low birthweight and, at least at these birthweights, of multiple plurality. At birthweights 500 to 1,500 grams, caesarean section was the safer method of delivery especially for singletons, but also for the four-fifths (n=25) of these smallest multiple infants born of second rank.

The fourth set of specific hypotheses pertains to pregnancy health factors and was tested using interview data mainly for infants of low birthweight because of the high incidence of these infants among the interviewed women. Contrary to expectations, the relative risk of perinatal death was not positively associated with psychological stress as retrospectively measured using a life-events inventory. However, estimated relative odds of perinatal death were increased by measures of physical stress. In the paid labour force, these
measures, for women delivering low birthweight babies, included working throughout the pregnancy and exposure to conditions such as lifting heavy objects and, more commonly, standing for a long time. The highest risks of perinatal death were recorded by the one-third (n=31) of women in paid employment who received little help with household tasks. These last risks, which have received scant attention elsewhere, were not further augmented for multiparous women. Starting a new type of exercise during pregnancy was hypothesized to be potentially dangerous. On a per-exercise basis, most hazardous was exercising in the last trimester especially after not doing the same exercise during the first trimester. Further distinctions were not made by exercise type.

Continued, intensive use of non-therapeutic drugs was hypothesized to increase perinatal mortality risks by, for example, reducing birthweight. Yet once the subject of study became babies exclusively of low birthweight, smoking was not expected to be a risk factor (see section 7.3). This latter hypothesis, which was suggested by a small amount of existing research, was confirmed for the small infants of the almost one-third (n=28) of smokers who smoked more than ten cigarettes per day during the first and last trimesters.

The final set of hypotheses relates to background factors. Perinatal mortality was indeed found to be most prevalent at the highest gravidities, especially when these were composed of more poor than successful outcomes and an immediately prior reproductive failure. The analysis used a novel method of inferring the gravidity by means of compositional and timing characteristics of women's reproductive histories. A subsequent, new hypothesis was that Aboriginality is a risk factor for perinatal death at all birthweights, this being endorsed only at very low and normal birthweights by data with
identified deficiencies. It was further, adventurously hypothesized that births to the women born in developing regions outside Australia were at higher risk of perinatal death than the births to Australian-born women. Perinatal survival in the latter women was observed in actuality to exceed that for all birthplace regions except Southern and Eastern Europe.

The last hypothesis associated with background factors was that never married women experience higher risks of perinatal death than do formerly married or currently married women. The analysis instead showed the estimated 17.8 per cent (n=1227) of women who were formerly married to be most at risk of perinatal death, never married women being at higher risk of perinatal death than were married women. These results, which were confirmed by logistic regressions, subsume the classification problem that some never or formerly married women might have been in stable cohabiting relationships during their 1986 pregnancies (see section 8.2.3). However, the analysis should remain useful within the constraints of this caveat, in particular because being formerly married has received almost no attention from perinatal researchers as a possible, aetiological risk factor. Indeed, the level of risk associated with former marriage has become a question of growing importance in the face of the rising fertility attending recent increases in divorce and remarriage, and childbearing at advanced maternal ages.

9.2 THE CONTRIBUTION OF THE STUDY: RETROSPECT AND PROSPECT

These results do not exist in a vacuum but in the contexts of existing knowledge and the constraints affecting the ability of this study to answer the questions that were considered important to investigate. Some obstacles have defied resolution, but most have presented
themselves as opportunities. At the least, the need for the research has not been dissolved by handicap, its voice only tempered and denied confident enunciation.

The key obstacle limiting this investigation was early identified to be the lack of an existing, suitable database in New South Wales. To help minimize the problem, multiple sources of data were drawn upon. Population-based information was collected from the State's Maternal and Perinatal Collection and vital registrations, while survey data were extracted from the hospital patient records and the interviews with former patients of six large maternity hospitals in New South Wales.

These sources furnished information about a rich diversity of variables and helped collectively to overcome the deficiencies of each individual source of data. However, the insoluble problem remained that the numbers of individuals recording different characteristics were not sufficiently large to yield results with as much confidence as was desirable. The reasons for this difficulty are briefly restated. The Maternal and Perinatal Collection provided the best available data source, furnishing relevant details about mothers and babies for the complete State. But Collection data were available only for 1986, earlier collections being deficient in completeness and quality, and lacking final editing checks; of course in the years to come, the emergence of a longitudinal database will permit the study of many more subjects with rare characteristics, such as the experience of a perinatal death. The 1986 Collection was itself flawed in ways that limited the number of subjects for study. It did not identify neonatal deaths occurring in hospitals after babies had earlier been discharged, and this problem was only partly reduced by the linkage of individual records from the Collection with perinatal
death registrations. In addition, some data, such as about antenatal care, were poorly reported by the Collection; were inadequately defined, for example, marital status: guidelines fail to distinguish adequately between de facto marriage and singleness; or were not collected at all: behavioural information about pregnant women had to be collected with other details from hospital patient records and interviews, necessarily for small numbers of women.

The result was an exploratory analysis that has traversed a vast spectrum of variables, and that has sought to address fresh and, for the most part, relatively unexplored issues rather than always those already well canvassed. Analyses, structured by a conceptual model of the proximate determinants of perinatal death, have most usefully related to the effects of exposures to suspected or known factors in low and other birthweight strata. Least reliable are proportions derived from the aggregation of birthweight strata owing to the need for weighting.

Against this background, the study has contributed to perinatal research in two geographical arenas: New South Wales which lacks any other large-scale epidemiological inquiry of this type, and the larger Australian and international scenes. The main contributions of the 'case study' to both theatres have been to add to existing knowledge, especially, as noted above, when this is least developed, and also perhaps in a small way to assist in agenda-setting. The study has identified possible risks for perinatal death among selected groups in Australia's largest State, but the certainty of these risks and their extrapolability to other developed contexts requires clarification and substantiation. The thesis has asked questions and identified issues for which confident resolution requires other, larger projects that will also benefit from an interdisciplinary approach.
In section 1.6 a number of research priorities were identified. It is apparent now, at the end of the study, that two broad issues demand attention, and they are discussed with reference mainly to New South Wales. The first issue, which elaborates on the discussion above, considers the quality of existing data and the information that should perhaps be collected. While there is much to commend the institutional sources used in this study (see sections 3.4.1 to 3.6.2), they all bear common deficiencies. In particular they report little useful information about the maternal lifestyle, which prevents stable and rigorous measurements being made of behavioural impacts on perinatal outcome. Strong behavioural data must become available at the population level, although large-scale sample survey efforts, directed at specific questions, would also be invaluable.

At the State level, the best option is a prospective register initiated during pregnancy and detailing maternal and infant characteristics. In New South Wales, retrospective collections of enhanced behavioural data require more thorough reporting by doctors on patient medical records of the lifestyle practices of pregnant women. For example, whilst existing medical records might state that women are social drinkers or that they smoke ten cigarettes per day, the timing and duration of these consumptions, according to which the amounts consumed may also change, should also be reported. Nevertheless, there is behavioural information already in records that could and should be reported mainly on the New South Wales Maternal and Perinatal Collection form, as on the Notification of Birth Form in Tasmania and on the mother's Perinatal Morbidity Statistics Form in the Australian Capital Territory. Behavioural information could also be reported on birth and death certificates. Such changes would help to communicate to doctors the need for, and value of, pregnancy health information, especially as part of a large longitudinal database.
Further improvements are needed in the collection and reporting of non-behavioural information. This includes the holding and results of autopsy examinations which are inadequately detailed on death certificates (see section 4.8.2). With reference to the Maternal and Perinatal Collection form, the race of the father should be recorded to improve the identification of Aboriginals (see section 8.2.1). Also, clear, precise definition should be given to what constitutes a stable consensual union and this definition should be consistently applied; only then will truly 'single' women be reliably distinguished from those in de facto marriages. It would be useful to know the indications for intrapartum procedures like induction, augmentation and the administration of anaesthesia, while the Health Department should replace its use of the term 'essential hypertension' with 'unclassified hypertension' (see section 5.3.1).

The second broad priority identified by this investigation, and it is related closely to the collection of improved, especially behavioural, data, is the need for increased participation by demographers and other social scientists in perinatal mortality research. Whether demographers accept and apply epidemiological methods or use their own approaches is of less consequence than the valuable insights and contributions they could make, ideally alongside their medical colleagues. At present, few demographers publish on perinatal topics in their own journals. However, in part this reflects barriers to interdisciplinary acceptance and cooperation in perinatal research by some social and medical scientists, and by institutional structures that affect the type of data and their availability even to medical professionals. In Victoria, strict confidentiality provisions debar all but selected individuals from using non-aggregated data at the State level. The result of this segmentation of academic responsibilities is that in New South Wales, social scientists are
visibly absent from the Ministerial Task Force that in 1989 presented its report on maternity services in the State. Yet this study is testimony to the assistance that the medical profession and record holders will give a persistent and grateful social scientist.

With these sorts of changes, perinatal epidemiologists and demographers will be able to direct their future researches increasingly toward behavioural characteristics of the pregnant woman and her family, and the relations that orchestrate the symphony of factors affecting perinatal mortality and morbidity. It is in this context, and perhaps increasingly for high-risk groups such as, in Australia, Aboriginal women, that new investigations may be most likely to prosper. In this country and overseas, our challenge and responsibility is to optimize collective use of the available resources to ensure that we do not deny life to the seeds of humanity's future.
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Dear

The Hospital and the Area Health Service has recently given approval to the conduct of a research study by Mr. Stephen Buetow, a Ph D student from the Australian National University. This study, which is approved by the Area's Ethics Committee is important in seeking to discover why some babies do not survive, while others do. The project, which is absolutely confidential, involves many hospitals and a large number of women who gave birth in 1986.

You are asked to participate in this study and subject to your consent, this will mean allowing Mr. Buetow to collect information from your hospital records and/or most importantly to conduct an interview with you at home.

You may consent to one or both, or neither, of the requests. Certainly you are not obliged to participate and you may withdraw from the project at any time. However, the hospital does consider the project a most worthy one and I would urge you to help Mr. Buetow with this research.

If you are willing to be involved, please complete the two consent forms attached and return them in the enclosed envelope free of charge. If you have any questions, please do not hesitate to contact Mr. Beutow on (02) 498 6457.

Your co-operation in assisting with this research program will be appreciated.

Yours sincerely,
Appendix 2
Letter from Principal Investigator to Selected Women

The Australian National University
Department of Demography

Research School of Social Sciences
GPO Box 4, ACT 2601

TO WHOM IT MAY CONCERN

There is much society does not understand about why some babies do not survive. I am undertaking research that may help to answer this question so that future losses may be prevented. The completed research will be presented in a Ph.D. thesis to the Australian National University.

However, I need your help to do my study. I require information about all kinds of pregnancies and to obtain this, I must ask you and several hundred other women, who have all been randomly selected, two very important questions.

First, may I please use the statistical information relating to your pregnancy ending in 1986 from your hospital patient records? Secondly, would you please consider being interviewed once in your home about this pregnancy?

I wish you to know that my study has been approved by your hospital and meets the ethical standards required by the National Health and Medical Research Council. For example, all the information I collect will be treated confidentially, and I guarantee the anonymity of every person involved in the project.

To elaborate, each individual's information will be grouped with that from other women. Only from the aggregated data, will it be possible to identify those factors, which at the time were considered safe by women and/or their doctors, that in fact may not be safe.

I must explain that if you are one of the women who has lost her baby, the interview, which takes approximately forty minutes, may cause you some distress. On the other hand, you may find it helpful to review the pregnancy, birth and subsequent events. Follow-up professional counselling has been arranged for the women who would like this.

Please understand that you are free not to participate in the study, or to withdraw at any time. However, may I emphasize that my research, which could help to save lives, depends on as many women as possible helping in the manner described. If you are willing to help, please complete the consent forms and return them freepost as soon as possible. Your assistance will be greatly appreciated.

Yours sincerely

Stephen Buetow
Appendix 3
Sample Form of Consent to Participate in Research Project

FORM OF CONSENT TO PARTICIPATE IN RESEARCH PROJECT

I, ......................................................... of ..............................................................
Postcode ................................ hereby consent to taking part in a research project entitled Perinatal Deaths in South-East Australia.
The aim of the project is to obtain further information to help prevent perinatal deaths.
The procedure will involve the extraction and use of statistical data from my hospital patient records.
I understand that I can refuse to take part in this project, or withdraw from it at any time.
I agree after considering all the above factors to participate in the project.
SIGNATURE OF VOLUNTEER ....................................................... DATE ........................................
WITNESS (Print Name) ..............................................................................................................
SIGNATURE OF WITNESS ..........................................................................................................
## APPENDIX 4
Schedule for Hospital Patient Record Extractions

### SECTION A: THE MOTHER

#### PERSONAL FACTORS

<table>
<thead>
<tr>
<th>01. Health region of usual residence</th>
</tr>
</thead>
<tbody>
<tr>
<td>02. Country of birth</td>
</tr>
<tr>
<td>03. Aboriginal</td>
</tr>
<tr>
<td>01 Yes</td>
</tr>
<tr>
<td>02 No</td>
</tr>
<tr>
<td>04. Marital Status</td>
</tr>
<tr>
<td>01 Single</td>
</tr>
<tr>
<td>02 Married</td>
</tr>
<tr>
<td>03 Other</td>
</tr>
<tr>
<td>05. Date of birth (mother)</td>
</tr>
<tr>
<td>06. Age at delivery (completed years)</td>
</tr>
<tr>
<td>07. Height (in centimetres)</td>
</tr>
<tr>
<td>08. Health insurance</td>
</tr>
<tr>
<td>01 Yes</td>
</tr>
<tr>
<td>02 No</td>
</tr>
</tbody>
</table>

#### ANTENATAL FACTORS

<p>| 09. Earliest recorded pregnant weight (to nearest kilogram) |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>10. Week recorded</td>
<td></td>
</tr>
<tr>
<td>11. Last weight prior to delivery</td>
<td></td>
</tr>
<tr>
<td>12. Week recorded</td>
<td></td>
</tr>
<tr>
<td>13. Number of antental visits</td>
<td></td>
</tr>
<tr>
<td>14. Week of first antenatal visit</td>
<td></td>
</tr>
<tr>
<td>15. Responsibility for obstetric care</td>
<td></td>
</tr>
<tr>
<td>01 Hospital clinic</td>
<td></td>
</tr>
<tr>
<td>02 Specialist obstetrician</td>
<td></td>
</tr>
<tr>
<td>03 General practitioner</td>
<td></td>
</tr>
<tr>
<td>04 Registered midwife</td>
<td></td>
</tr>
<tr>
<td>05 Shared</td>
<td></td>
</tr>
<tr>
<td>06 Other</td>
<td></td>
</tr>
<tr>
<td>16. Lowest antenatal haemoglobin</td>
<td></td>
</tr>
<tr>
<td>17. Week recorded</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Antenatal investigations</strong></td>
<td></td>
</tr>
<tr>
<td>18. Ultrasound</td>
<td></td>
</tr>
<tr>
<td>20. Cardiotocography</td>
<td></td>
</tr>
<tr>
<td>21. Glucose tolerance test</td>
<td></td>
</tr>
<tr>
<td>22. Amniocentesis</td>
<td></td>
</tr>
<tr>
<td>01 Yes</td>
<td></td>
</tr>
<tr>
<td>02 No</td>
<td></td>
</tr>
<tr>
<td>Drugs prescribed during pregnancy</td>
<td></td>
</tr>
<tr>
<td>----------------------------------</td>
<td></td>
</tr>
<tr>
<td>23 Antiemetics</td>
<td></td>
</tr>
<tr>
<td>24 Antibiotics</td>
<td></td>
</tr>
<tr>
<td>25 Haematinics</td>
<td></td>
</tr>
<tr>
<td>26 Tocolytics</td>
<td></td>
</tr>
<tr>
<td>27 Sedatives/antidepressants</td>
<td></td>
</tr>
<tr>
<td>28 Anticonvulsants</td>
<td></td>
</tr>
<tr>
<td>29 Anticoagulants</td>
<td></td>
</tr>
<tr>
<td>30 Hypotensives</td>
<td></td>
</tr>
<tr>
<td>01 Yes</td>
<td></td>
</tr>
<tr>
<td>02 No</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Antenatal transfer</th>
</tr>
</thead>
<tbody>
<tr>
<td>01 Yes</td>
</tr>
<tr>
<td>02 No</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>If yes: reason for transfer</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>LABOUR AND DELIVERY FACTORS</th>
</tr>
</thead>
<tbody>
<tr>
<td>33. Most qualified attendant at labour</td>
</tr>
<tr>
<td>01 Specialist obstetrician</td>
</tr>
<tr>
<td>02 General practitioner</td>
</tr>
<tr>
<td>03 Qualified nurse or midwife</td>
</tr>
<tr>
<td>04 Student (nurse/midwife/medical)</td>
</tr>
<tr>
<td>05 None or no trained person</td>
</tr>
<tr>
<td>06 Other</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Labour induced</th>
</tr>
</thead>
<tbody>
<tr>
<td>01 Yes</td>
</tr>
<tr>
<td>02 No</td>
</tr>
<tr>
<td>Question</td>
</tr>
<tr>
<td>-------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| 35. Labour augmented                                                    | 01 Yes  
|                                                                       | 02 No                                                                 |
| 36. Analgesia and/or anaesthesia used                                  | 01 None or psychoprophylaxis only  
|                                                                       | 02 Analgesic, systemic or inhalation  
|                                                                       | 03 Local  
|                                                                       | 04 Paracervical or pudendal  
|                                                                       | 05 Epidural or spinal  
|                                                                       | 06 General  
|                                                                       | 07 02 and 03 or 02 and 04  
|                                                                       | 08 Other combination  
|                                                                       | 09 Other                                                                 |
| 37. Presentation                                                        | 01 Vertex  
|                                                                       | 02 Breech  
|                                                                       | 03 Other                                                                 |
| 38. Foetal monitoring                                                  | 01 External heart rate monitoring only  
|                                                                       | 02 Internal heart rate monitoring only  
|                                                                       | 03 Internal heart rate monitoring plus scalp pH                           |
| 39. Method of delivery                                                 | 01 Spontaneous cephalic  
|                                                                       | 02 Ventouse  
|                                                                       | 03 Low forceps  
|                                                                       | 04 Mid forceps  
|                                                                       | 05 Vaginal breech  
|                                                                       | 06 Elective caesarean section  
|                                                                       | 07 Emergency caesarean section  
|                                                                       | 08 Other                                                                 |
### 40. If 03 or 04 to Q.39, indication for forceps delivery

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>01</td>
<td>Delay in second stage of labour</td>
</tr>
<tr>
<td>02</td>
<td>Foetal distress</td>
</tr>
<tr>
<td>03</td>
<td>Prematurity</td>
</tr>
<tr>
<td>04</td>
<td>Maternal disease</td>
</tr>
<tr>
<td>05</td>
<td>Other</td>
</tr>
</tbody>
</table>

### 41. If 06 or 07 to Q.39, indication for caesarean delivery

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>01</td>
<td>Repeat caesarean</td>
</tr>
<tr>
<td>02</td>
<td>Breech presentation</td>
</tr>
<tr>
<td>03</td>
<td>Other malpresentation</td>
</tr>
<tr>
<td>04</td>
<td>Failure to progress</td>
</tr>
<tr>
<td>05</td>
<td>Foetal distress</td>
</tr>
<tr>
<td>06</td>
<td>Toxaemia</td>
</tr>
<tr>
<td>07</td>
<td>Antepartum haemorrhage</td>
</tr>
<tr>
<td>08</td>
<td>Other</td>
</tr>
</tbody>
</table>

### 42. Duration of labour (hours and minutes)

- **First stage:**
- **Second stage:**

### 44. Number of babies delivered

### MEDICAL HISTORY FACTORS

#### Family history

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>01</td>
<td>Yes</td>
</tr>
<tr>
<td>02</td>
<td>No</td>
</tr>
</tbody>
</table>

#### Diabetes

#### Cardiac disease

#### Hypertension
<table>
<thead>
<tr>
<th><strong>Family History</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>48. Multiple births</td>
<td></td>
</tr>
<tr>
<td>49. Low birth weights</td>
<td></td>
</tr>
<tr>
<td>50. Congenital malformations</td>
<td></td>
</tr>
<tr>
<td>01 Yes</td>
<td></td>
</tr>
<tr>
<td>02 No</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Past Medical History</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>51. Previous gynaecological surgery</td>
<td></td>
</tr>
<tr>
<td>52. Chronic renal disease</td>
<td></td>
</tr>
<tr>
<td>53. Gestational diabetes</td>
<td></td>
</tr>
<tr>
<td>54. Diabetes mellitus</td>
<td></td>
</tr>
<tr>
<td>55. Cardiac disease</td>
<td></td>
</tr>
<tr>
<td>56. Other medical disorders</td>
<td></td>
</tr>
<tr>
<td>01 Yes</td>
<td></td>
</tr>
<tr>
<td>02 No</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Past Obstetrical History</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>57. Previous pregnancies</td>
<td></td>
</tr>
<tr>
<td>01 Yes</td>
<td></td>
</tr>
<tr>
<td>02 No →SECTION B</td>
<td></td>
</tr>
<tr>
<td>58. Toxaemia</td>
<td></td>
</tr>
<tr>
<td>59. Antepartum haemorrhage</td>
<td></td>
</tr>
<tr>
<td>60. Diabetes (class A or B)</td>
<td></td>
</tr>
<tr>
<td>61. Rhesus iso-immunization</td>
<td></td>
</tr>
<tr>
<td>01 Yes</td>
<td></td>
</tr>
<tr>
<td>02 No</td>
<td></td>
</tr>
<tr>
<td>Past obstetrical history cont.</td>
<td></td>
</tr>
<tr>
<td>-------------------------------------------------------------------</td>
<td>---</td>
</tr>
<tr>
<td>62. Mid-forceps delivery</td>
<td></td>
</tr>
<tr>
<td>63. Caesarean section</td>
<td></td>
</tr>
<tr>
<td>64. Birth-weight less than 2 500 grams</td>
<td></td>
</tr>
<tr>
<td>65. Birth-weight more than 4 000 grams</td>
<td></td>
</tr>
<tr>
<td>66. Major congenital malformation</td>
<td></td>
</tr>
<tr>
<td>01 Yes</td>
<td></td>
</tr>
<tr>
<td>02 No</td>
<td></td>
</tr>
<tr>
<td>Total number of</td>
<td></td>
</tr>
<tr>
<td>67. Live-births only</td>
<td></td>
</tr>
<tr>
<td>68. Still-births</td>
<td></td>
</tr>
<tr>
<td>69. Spontaneous abortions</td>
<td></td>
</tr>
<tr>
<td>70. Induced abortions</td>
<td></td>
</tr>
<tr>
<td>71. Neonatal deaths</td>
<td></td>
</tr>
<tr>
<td>72. Completed weeks since end of last pregnancy</td>
<td></td>
</tr>
<tr>
<td>73. Completed weeks since last birth</td>
<td></td>
</tr>
<tr>
<td>74. Outcome of last previous pregnancy</td>
<td></td>
</tr>
<tr>
<td>01 Live-birth full-term</td>
<td></td>
</tr>
<tr>
<td>02 Live-birth pre-term</td>
<td></td>
</tr>
<tr>
<td>03 Still-birth</td>
<td></td>
</tr>
<tr>
<td>04 Spontaneous abortion</td>
<td></td>
</tr>
<tr>
<td>05 Induced abortion</td>
<td></td>
</tr>
<tr>
<td>06 Neonatal death</td>
<td></td>
</tr>
</tbody>
</table>
SECTION B: THE BABY

75. Place of birth
   01 Home
   02 Hospital
   03 Other

76. If 02 to Q.76, name of hospital

77. Estimated gestational age (completed weeks)

78. Date of birth

79. Birth-weight (grams)

80. Body-length (centimetres crown-heel)

81. State at birth (1 Minute APGAR)

82. State at birth (5 Minute APGAR)
   None taken CODE 95

83. Sex
   01 Male
   02 Female

84. Birth order
   01 Singleton
   If multiple, this infant
   02 First
   03 Second
   04 Other
APPENDIX 5
Interview Schedule

SECTIONS A-D: PREGNANCY AND POSTPARTUM HISTORY MATRICES.

SECTION A. EXERCISE HISTORY.

First, I would like to ask you some questions about the exercises you did during pregnancy. That is, any physical activities you did for fun or for their beneficial effect. They may have been activities you also did before your pregnancy—or, they may have been new ones.

RECORD THE FOLLOWING EVENTS AGAINST THE APPROPRIATE MONTH(S) OF PREGNANCY.

1. Did you exercise at any time during your pregnancy?
   CODE: 00 YES.
   01 NO. ---> SECTION B.

2a. At which month did you start/restart exercising?

2b. After this month, how many months did you continue exercising?

2c. PROBE: That is, you continued exercising until the ... month of pregnancy. Is this correct?
   IF YOU HAVE REACHED THE END OF THE PREGNANCY ---> Q.3a

2d. After the ... month, did you ever again exercise during your pregnancy?
   IF "YES": REPEAT Q.2a-2d UNTIL EXERCISING ENDS.

3a. During your complete pregnancy, what types of exercise did you do?
   IF ONE EXERCISE ONLY ---> 3C, THEN ---> SECTION B.

3b. What was the first/nth type of exercise you did?

3c. At which month of pregnancy did you start /again start ...?

1 Respondents' answers are entered on the matrices shown on pages 483-488.
3d. For how many months did you ...?

3e. PROBE: That is, you exercised by .... until the .... month of pregnancy. Is this correct?

IF YOU HAVE REACHED THE END OF THE PREGNANCY, REPEAT Q. 3b-4 FOR EACH OTHER TYPE OF EXERCISE.
THEN ---> SECTION B.

4. After the .... month, did you ever again start .... during your pregnancy?

IF "NO": REPEAT Q. 3b-4 FOR EACH OTHER TYPE OF EXERCISE.

IF "YES": REPEAT Q. 3c-4 FOR THE SAME EXERCISE UNTIL YOU REACH THE END OF THE PREGNANCY.
THEN REPEAT Q. 3b-4 FOR EACH OTHER TYPE OF EXERCISE.

SECTION B. OCCUPATION HISTORY:

In this section I have some questions about the occupation (or occupations) you may have had during pregnancy.

RECORD THE FOLLOWING EVENTS AGAINST THE APPROPRIATE MONTH(S) OF PREGNANCY.

5. Did you work outside the home at any time during your pregnancy?
CODE: 00 YES.
01 NO. ----> SECTION C.

6a. At which month did you start/again start work?

6b. For how many months did you continue working?

6c. PROBE: That is, you stopped working at the ....... month. Is this correct?


6d. After this month, did you ever again work during your pregnancy?

IF "YES": REPEAT Q. 6a-6d UNTIL WORK ENDS.

7. During your complete pregnancy, did you have the same
occupation?

IF "NO": ---> Q.8b.

8a. What was your occupation?

--- > Q.10.

8b. What was the first/nth occupation you had during pregnancy?

8c. At which month of pregnancy, did you start/again start this occupation?

8d. For how many months did you have this occupation?

8e. PROBE: That is, you worked in your first/nth occupation until the ..... month of pregnancy. Is this correct?

IF YOU HAVE REACHED THE END OF THE PREGNANCY, REPEAT Q.8b-8e FOR EACH OTHER OCCUPATION. THEN ---> Q.10.

9. After the .... month, did you ever again work as a ... during your pregnancy?

If "NO": REPEAT Q.8b-8e FOR EACH OTHER OCCUPATION.

IF "YES": REPEAT Q.8c-8e FOR THE SAME OCCUPATION UNTIL YOU REACH THE END OF THE PREGNANCY. THEN REPEAT Q.8b-8e FOR EACH OTHER OCCUPATION.

10. During your complete pregnancy, did you continue to work the same number of hours each week?

CODE: 00 YES.
01 NO. ---- > Q.11b.

11a. How many hours did you work each week?

CODE EXACT NUMBER. THEN: ---> Q.12.

11b. How many hours per week did you first work during pregnancy/then work?

11c. How many months did you work this number of hours?

11d. PROBE: That is, you worked ..... hours per week until the ..... month of pregnancy. Is this correct?

REPEAT Q.11b-11d UNTIL WORK ENDS.
12. During your pregnancy, were you required in any occupation to ... ?
(READ NTH. WORKING CONDITION - VARIABLES 07-15).
CODE: 00 YES.
01 NO. ---> NEXT VARIABLE AND, WHEN LIST COMPLETED, SECTION C.

13a. Which occupation(s) required you to do this?

FOR THE FIRST OCCUPATION ASK:

13b. Did you have to ... the whole time you worked as a ....? ...

IF "NO": ----> Q.13d-13f.

13c. PROBE: That is, when you worked as a ...., you had to ...
untill the .... month of pregnancy. Is this correct?

REPEAT Q.13b FOR EACH OTHER OCCUPATION INVOLVING THIS WORKING CONDITION.
THEN REPEAT Q.12-14 FOR EACH OTHER WORKING CONDITION.

13d. From which month of pregnancy did you have to .... /again have to ....

13e. How many months did you have to do this for?

13f. PROBE: That is you had to ... until the .... month of pregnancy. Is this correct?

IF YOU HAVE REACHED THE END OF THE OCCUPATION, REPEAT Q.13b FOR EACH OTHER OCCUPATION INVOLVING THE SAME WORKING CONDITION.
THEN REPEAT Q.12-14 FOR EACH OTHER WORKING CONDITION.

14. After the .... month, did you ever again have to ... during your pregnancy?

IF "NO": REPEAT 13b FOR EACH OTHER OCCUPATION INVOLVING THE SAME WORKING CONDITION.
THEN REPEAT Q.12-14 FOR EACH OTHER WORKING CONDITION.

IF "YES": REPEAT Q.13d-13f UNTIL YOU REACH THE END OF THE OCCUPATION.
THEN REPEAT 13b FOR EACH OTHER OCCUPATION INVOLVING THIS WORKING CONDITION.
SECTION C. CHEMICAL HISTORY:

Now, I will ask you about some of the medication or drugs which you may have taken just before, or during your pregnancy/and possibly, while breastfeeding.

RECORD THE FOLLOWING EVENTS AGAINST THE APPROPRIATE MONTH(S) OF PREGNANCY/INFANT LIFE.

15. Either in the month before you became pregnant, or during the pregnancy itself, did you use any drug (including tobacco and alcohol) without a doctor's knowledge or approval?

CODE: 00 YES.
01 NO. ----> Q.19 IF A NEONATAL DEATH.
       ----> SECTION D IF A STILL BIRTH.

IN THE FIRST READING, USE THE PREGNANCY WORDING OPTION.

16a. What was the name of each drug you used in the month before, or during, your pregnancy/ while you were breastfeeding?

16b. At which month did you first use/again start using .... (EACH DRUG NAMED IN 16a)?

16c. For how many months did you use this drug?

16d. PROBE: That is, you used .... until the ... month of pregnancy/ after giving birth. Is this correct?

IF YOU HAVE REACHED THE END OF THE PREGNANCY/NEONATAL PERIOD, REPEAT Q.16b-16e FOR EACH OTHER DRUG.
THEN ---> Q.17.

16e. After this month, did you ever again, during pregnancy/ your baby's life, use .... without a doctor's knowledge or approval?

IF "NO": REPEAT 16b-16e FOR EACH OTHER DRUG.

IF "YES": REPEAT Q.16b-16e UNTIL YOU REACH THE END OF THE PREGNANCY.
THEN REPEAT Q.16b-16e FOR EACH OTHER DRUG.
IF USING THE NEONATAL WORDING OPTION, AFTER ALL DRUGS HAVE BEEN COVERED --> SECTION D.

17. During the whole time you were using ... (EACH DRUG NAMED IN 16a) did you use the same number?

IF "NO": ----> Q.18b.

18a. How much/many ... did you use per day?

REPEAT Q.17 FOR EACH OTHER DRUG.
WHEN LIST ENDS: FOR A STILL BIRTH --> SECTION D.
FOR A NEONATAL DEATH --> Q.19.

18b. How much/many ... did you use per day in the ... month before/of your pregnancy?

18c. For how many months did you use this number?

18d. PROBE: That is, you used ... (NUMBER) ... (DRUG) per day until the ... month of your pregnancy. Is this correct?

18e. How much/many ... did you use per day after the ... month of your pregnancy?

18f. For how many months did you use this number?

18g. PROBE: That is, you used ... (NUMBER) ... (DRUG) per day until the ... month of your pregnancy. Is this correct?

REPEAT Q.18e-18g UNTIL YOU HAVE REACHED THE END OF THE PERIOD OF USING THE DRUG.
THEN REPEAT Q.17-18g FOR EACH OTHER DRUG.
WHEN LIST ENDS FOR A STILL BIRTH --> SECTION D.

19. Did you breastfeed your baby?

IF "NO": ----> SECTION D.

20. While you were breastfeeding, did you take any drug without your doctor's knowledge or approval?

IF "YES", REPEAT Q.16a-16e ONLY, USING THE WORDING OPTION FOR THE NEONATAL PERIOD.
SECTION D. PREGNANCY EVENTS HISTORY.

This fourth section looks at pregnancy events. I will read to you a list of things that might happen to any woman during or just after her pregnancy. As I read each item, please say whether this happened to you. This is important no matter how trivial or unimportant you think any item may be.

READ THE COMPLETE LIST OF LIFE EVENTS AND RECORD EACH RESPONSE AGAINST THE APPROPRIATE MONTH(S) OF PREGNANCY.

CODE: 01 YES.
02 NO.

Now, please consider in turn each of the events that happened to you. You said ............ (READ EVENT AGAIN).

21. At which month during pregnancy (or after birth) did this event occur/occur again?

22. For how many months did the event occur?

22a. PROBE: That is, the event lasted until the .......... month of pregnancy/after birth.

IF YOU HAVE NOT REACHED THE END OF THE PREGNANCY OR POSTPARTUM PERIOD, ASK

23. After this month, did this event, that is ...., ever occur again during your pregnancy?

IF YES, REPEAT Q. 21-23.

FOR EACH EVENT, SHOW RATING SCALE & SAY: Please study this card. The numbers on the card show how much distress an event causes a person when it occurs. The number 0 shows no distress; the number 10 shows considerable distress; and the other numbers show intermediate amounts of distress.

For the event ............, (READ EVENT AGAIN) select the number on the line which best shows the amount of distress you felt when the event happened.
<table>
<thead>
<tr>
<th>CARD</th>
<th>EXERCISE HISTORY</th>
<th>MONTHS OF PREGNANCY</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>VARIABLE NAME</td>
<td>CODE</td>
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<tr>
<td></td>
<td>00 Exercise Status</td>
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<td></td>
<td>EXERCISE</td>
<td>01</td>
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<td>EXERCISE</td>
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<td>TYPE</td>
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<td>TYPE</td>
<td>05</td>
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<tr>
<td>CARD</td>
<td>OCCUPATIONAL HISTORY.</td>
<td>MONTHS OF PREGNANCY.</td>
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<tr>
<td></td>
<td>VARIABLE NAME CODE</td>
<td>01 02 03 04 05 06 07 08 09 10 11</td>
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<tr>
<td></td>
<td>01 Work status</td>
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<td></td>
<td>06 Hours worked per week</td>
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<td></td>
<td>07 Operate a machine</td>
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<td></td>
<td>08 Work with a vibrating machine</td>
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<td></td>
<td>09 Lift heavy objects</td>
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<td>10 Stand for a long time</td>
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<td></td>
<td>11 Do repetitive tasks</td>
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<td></td>
<td>12 Work more than 8 hours per day</td>
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<td></td>
<td>13 Work with a loud noise</td>
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<td>14 Use public transportation</td>
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<td></td>
<td>15 Commute for more than 90 minutes per day.</td>
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<tr>
<td>CARD</td>
<td>CHEMICAL HISTORY</td>
<td>MONTHS OF ANTEPARTUM AND POSTPARTUM LIFE</td>
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<td></td>
<td>DRUG NAME</td>
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<td>06</td>
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<tr>
<td>C A D</td>
<td>MATERNL LIFE EXPERIENCES</td>
<td>MONTHS OF PREGNANCY AND NEONATAL LIFE</td>
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<tr>
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<td>WORK SITUATION</td>
<td>APPLIC. 01 02 03 04 05 06 07 08 09 10 11 02 04 05 06 07 08 09 10 11 01</td>
</tr>
<tr>
<td>01</td>
<td>You were unemployed or seeking work</td>
<td></td>
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<td>02</td>
<td>You started a new job</td>
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<tr>
<td>03</td>
<td>There was a big change in the people, duties, hours or responsibilities at your work</td>
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<tr>
<td>04</td>
<td>You were sacked or laid off</td>
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<tr>
<td>05</td>
<td>Your husband/partner became unemployed</td>
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<td>06</td>
<td>Your husband/partner's business failed</td>
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<td>07</td>
<td>A new person came to live in your household</td>
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<td>08</td>
<td>You moved house</td>
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<td>09</td>
<td>You had financial problems</td>
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<td>10</td>
<td>Someone close to you developed a serious illness</td>
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<td>11</td>
<td>You were involved in a traffic accident in which someone was badly injured</td>
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<td>12</td>
<td>You were involved in a legal action that could have or did damage your reputation</td>
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<tr>
<td>13</td>
<td>Your husband/partner did not want you to become pregnant</td>
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<tr>
<td>14</td>
<td>The behaviour of your husband/partner was a problem to you</td>
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<tr>
<td>15</td>
<td>Serious arguments developed between you and your husband/partner</td>
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<tr>
<td>16</td>
<td>Your husband/partner told you that he no longer loved you</td>
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<td>17</td>
<td>You had sexual difficulties</td>
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<td>18</td>
<td>Your husband/partner was unfaithful</td>
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<td>NO.</td>
<td>ITEM</td>
<td>APPLIC.</td>
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<tr>
<td>19</td>
<td>You were unfaithful</td>
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<tr>
<td>20</td>
<td>Serious arguments developed between you and someone close to you (not husband/partner)</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>SEPARATIONS. You separated from, or divorced your husband/partner</td>
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<tr>
<td>22</td>
<td>You were separated from a close family friend</td>
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<tr>
<td>23</td>
<td>Bereavements. Your child died (not baby)</td>
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<tr>
<td>24</td>
<td>Your husband/partner died</td>
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<td>25</td>
<td>Someone close to you died (not child or husband/partner)</td>
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<tr>
<td>26</td>
<td>FERTILITY. Your pregnancy was not planned</td>
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<td>27</td>
<td>You took something during the pregnancy (e.g. medication, alcohol, cigarettes) that might harm your baby</td>
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<td>28</td>
<td>You had an X-ray during the pregnancy</td>
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<td>29</td>
<td>You had an ultrasound investigation during the pregnancy</td>
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<td>30</td>
<td>You were in contact during the pregnancy with someone who had an infectious disease that might harm your baby</td>
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<tr>
<td>31</td>
<td>You almost miscarried</td>
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<tr>
<td>32</td>
<td>You were seriously ill during the pregnancy</td>
<td></td>
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<td>33</td>
<td>You had severe morning sickness</td>
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<td>34</td>
<td>You had blood pressure problems during the pregnancy</td>
<td></td>
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<td>35</td>
<td>Your doctor said you were going to have twins</td>
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<tr>
<td>C A N D NO.</td>
<td>MATERNAL LIFE EXPERIENCES.</td>
<td>MONTHS OF PREGNANCY AND NEONATAL LIFE</td>
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<tr>
<td>36</td>
<td>You had difficulty</td>
<td>APPLIC. 01 02 03 04 05 06 07 08 09 10 11 01 RATING</td>
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<td></td>
<td>arranging for someone to</td>
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<td></td>
<td>look after your family</td>
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<tr>
<td></td>
<td>whilst in hospital</td>
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<tr>
<td>37</td>
<td>LABOUR AND DELIVERY.</td>
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<td></td>
<td>The doctor made your</td>
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<td></td>
<td>baby come early.</td>
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<td>38</td>
<td>Your labour was very</td>
<td></td>
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<td></td>
<td>painful</td>
<td></td>
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<tr>
<td>39</td>
<td>Your husband/partner</td>
<td></td>
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<td></td>
<td>was not present at the</td>
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<td></td>
<td>labour</td>
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<td>40</td>
<td>Medical complications</td>
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<td></td>
<td>arose during the labour</td>
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<td>41</td>
<td>Your doctor was not</td>
<td></td>
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<td></td>
<td>present at the labour</td>
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<tr>
<td>42</td>
<td>You had stitches during</td>
<td></td>
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<tr>
<td></td>
<td>the labour</td>
<td></td>
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<td>43</td>
<td>You had an anaesthetist</td>
<td></td>
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<td></td>
<td>during the labour</td>
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<td></td>
<td>and were not awake when</td>
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<tr>
<td></td>
<td>your baby arrived</td>
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<td>44</td>
<td>You had a caesarean</td>
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<tr>
<td></td>
<td>operation</td>
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<td>45</td>
<td>THE BABY.</td>
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<td></td>
<td>Your baby arrived before</td>
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<td></td>
<td>the expected date</td>
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<tr>
<td>46</td>
<td>Your baby was very</td>
<td></td>
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<td></td>
<td>small at birth</td>
<td></td>
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<tr>
<td>47</td>
<td>Your baby was abnormal</td>
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<tr>
<td>48</td>
<td>Your baby had a birth</td>
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<td></td>
<td>mark or something</td>
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<td></td>
<td>similar spoiling his/her</td>
<td></td>
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<tr>
<td></td>
<td>appearance</td>
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<tr>
<td>49</td>
<td>Your baby needed special</td>
<td></td>
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<tr>
<td></td>
<td>treatment after the</td>
<td></td>
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<tr>
<td></td>
<td>birth</td>
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<tr>
<td>50</td>
<td>Breast feeding was</td>
<td></td>
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<tr>
<td></td>
<td>difficult to establish</td>
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</tbody>
</table>
### SECTION E. SHORT ANSWER QUESTIONS

Now, I would like to ask you some short questions, mostly about your pregnancy.

<table>
<thead>
<tr>
<th>Question</th>
<th>Options</th>
<th>Answer</th>
</tr>
</thead>
<tbody>
<tr>
<td>24. How would you describe your general health at the start of your pregnancy?</td>
<td>01 Excellent, 02 Good, 03 Fair, 04 Poor</td>
<td></td>
</tr>
<tr>
<td>25. What was your weight just before you became pregnant?</td>
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<tr>
<td>26. Did you attend prenatal classes?</td>
<td>01 Yes, 02 No</td>
<td></td>
</tr>
<tr>
<td>I shall read a list of jobs around the home. Please say whether, during the last three months of your pregnancy, each of these jobs was</td>
<td>01 Done mostly by yourself, 02 Done mostly your husband/partner, children or other household members, 03 Shared jointly between yourself and your husband/partner, children or other household members</td>
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<tr>
<td>27. Shopping and other errands</td>
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<tr>
<td>28. Cooking meals</td>
<td></td>
<td></td>
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<tr>
<td>29. Washing dishes and drying up</td>
<td></td>
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<tr>
<td>30. House cleaning</td>
<td></td>
<td></td>
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<tr>
<td>31. Washing clothes and ironing them</td>
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<tr>
<td>32. Lawn mowing</td>
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</tr>
<tr>
<td>33. Other gardening</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Question</td>
<td>Answer</td>
<td></td>
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<tr>
<td>-------------------------------------------------------------------------</td>
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<tr>
<td>35. During these last three months, first, how many hours sleep did you usually get each night?</td>
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<tr>
<td>36. Second, how many hours rest did you usually get each day?</td>
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<tr>
<td>37. Which of the following best describes your family background?</td>
<td></td>
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<tr>
<td>01 You were born overseas</td>
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<tr>
<td>02 You were born in Australia, but at least one of your parents was born overseas</td>
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<tr>
<td>03 You were born in Australia and your parents were born in Australia, but at least one grandparent was born overseas</td>
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<tr>
<td>04 You were born in Australia and your parents and grandparents were born in Australia</td>
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<tr>
<td>38. If 01 to Q.37, in which year did you arrive in Australia?</td>
<td></td>
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<tr>
<td>39. In which Australian state or territory did you go to school? If more than one, name these also?</td>
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<tr>
<td>40. At the time you gave birth, what was your highest educational achievement?</td>
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<tr>
<td>REFER TO MANUAL TO SELECT APPROPRIATE OPTION(S) FOR CATEGORIES 03 AND 04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>01 No schooling</td>
<td></td>
<td></td>
</tr>
<tr>
<td>02 Some primary school</td>
<td></td>
<td></td>
</tr>
<tr>
<td>03 Some secondary school</td>
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<td></td>
</tr>
<tr>
<td>04 Intermediate/School/Proficiency Certificate</td>
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<tr>
<td>05 Year 12/Leaving/Higher School/Matriculation Certificate.</td>
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<td>06 Technical, teaching or other qualification</td>
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<tr>
<td>07 University degree</td>
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<td></td>
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<tr>
<td>Question</td>
<td>Options</td>
<td></td>
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<tr>
<td>-------------------------------------------------------------------------</td>
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<td></td>
</tr>
<tr>
<td>41. What was your marital status when you became pregnant?</td>
<td>01 Married</td>
<td></td>
</tr>
<tr>
<td></td>
<td>01 Lived with your partner but not married</td>
<td>02 Other</td>
</tr>
<tr>
<td>42. What is your husband's date of birth?</td>
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<td></td>
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<tr>
<td>43. During your pregnancy? what was your husband's/partner's usual occupation?</td>
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<tr>
<td>44. During your pregnancy, did your husband/partner regularly use the following drugs. First, tobacco.</td>
<td>01 Yes 02 No</td>
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<td></td>
<td></td>
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<td>45. Second, alcohol.</td>
<td>01 Yes 02 No</td>
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<td>46. Did you have parents, or other family or non-family members, living in your household for all, or part of, your pregnancy?</td>
<td>01 Yes 02 No</td>
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<td></td>
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<td>47. During your pregnancy, what was the usual number of people living in your household?</td>
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<tr>
<td>48. In the year of your pregnancy, what was your household's gross annual income?</td>
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</tbody>
</table>
49. *Approximately what percentage of your household's net annual income did you spend on health?*

50. *During your pregnancy, how many bedrooms were there in your house?*

*ENID OF INTERVIEW FOR CONTROL WOMEN.*
SECTION F: DISCUSSION QUESTIONS.

Finally, I would like simply to talk with you about the time since your baby's death and about your feelings and ideas concerning this loss. Please let me stress that you may say as little or as much as feels comfortable for you.

1. How did you feel when your baby died?

2. What kind of help have you received to cope with your loss?

3. What do you now think caused your baby's death?

4. Have you always felt this, or have your reasons changed over time?
5. In what ways would you have liked to receive a different kind of maternity care?

6. How has your baby's death affected your life?

7. How well do you feel you have adjusted to your loss?

IF NECESSARY, PROBE: Why is this?

7. What are your feelings now about your loss?