



**The Relevance of Concepts of
Hyperalgesia to "RSI"**

**Milton L. Cohen, Jesus F. Arroyo and
G. David Champion**

WORKING PAPER NUMBER 31

NATIONAL CENTRE FOR EPIDEMIOLOGY AND POPULATION HEALTH

WORKING PAPERS

**Discussion Papers on the Pathology of Work-Related
Neck and Upper Limb Disorders and the Implications
for Diagnosis and Treatment**

Gabriele Bammer
editor and coordinator

QUINTNER
39 Mayfair Street
Mt Claremont 6010

**The Relevance of Concepts of
Hyperalgesia to "RSI"**

**Milton L. Cohen, Jesus F. Arroyo and
G. David Champion**

WORKING PAPER NUMBER 31

Commentaries:

Trevor Beswick and Annette Cursley
Earl Owen
Michael Patkin
John Quintner
Gisela Sjøgaard
Richard Wigley
Max Zusman

November 1992

**NATIONAL CENTRE FOR EPIDEMIOLOGY AND POPULATION HEALTH
THE AUSTRALIAN NATIONAL UNIVERSITY**

ISBN 0 7315 1447 5
ISSN 1033-1557

Contents

Introduction	v
Gabriele Bammer	
The Relevance of Concepts of Hyperalgesia to “RSI”	1
Milton L. Cohen, Jesus F. Arroyo and G. David Champion	
Commentaries	9
Trevor Beswick and Annette Cursley.....9	
Earl Owen10	
Michael Patkin11	
John Quintner.....13	
Gisela Sjøgaard19	
Richard Wigley.....19	
Max Zusman20	
Response to Commentaries	25
Milton L. Cohen	

INTRODUCTION

Gabriele Bammer BSc BA PhD

Research Fellow

National Centre for Epidemiology and Population Health

The Australian National University

GPO Box 4

Canberra ACT 2601 Australia

There is continuing debate about work-related neck and upper limb disorders, also commonly referred to as repetition strain injuries (RSI), cervicobrachial disorders and cumulative trauma disorders (CTDs). One focus of the debate concerns the exact nature of the physical basis of the disorders. Health professionals who are involved in this area generally have a working hypothesis about the underlying pathology; some practitioners emphasise trigger points, others muscle fibre changes, others irreversible irritability of nerves and so on. In most cases these hypotheses have not been clearly expounded or discussed, let alone confirmed or refuted.

In late 1989 I invited some of the leading exponents of different viewpoints to write detailed accounts of their hypotheses regarding the underlying pathology. These were to be circulated amongst a panel of people with a range of different expertise, to encourage discussion from various perspectives. The original authors were also to be invited to respond to these commentaries.

The following paper, *The Relevance of Concepts of Hyperalgesia to "RSI"*, by Milton Cohen, Jesus Arroyo and David Champion is the second in this series. It provides a view on the role the nervous system may play in these disorders, which is an alternative to that proposed by John Quintner and Robert Elvey in the first paper in this series (1). There are

seven commentaries highlighting important points for debate and further work.

The debate about underlying pathology is taking place within a broader sociopolitical debate about these disorders. In the latter debate the disorders are most commonly referred to as RSI. As Cohen and colleagues point out elsewhere (2), at one end of the socio-political debate, RSI is seen as the medicalisation of a social problem. At the other, RSI is seen as a physical disorder (or, more accurately, a set of physical disorders), but where physiological and ergonomic aspects are modulated by sociopolitical factors.

In the broader sociopolitical debate understandings from the social construction of medical knowledge and from consideration of social problems as social movements have been influential. In a recently published paper (3) Brian Martin and I point out that both of these ways of analysing RSI tend to delegitimize the position that RSI is work related and has an organic basis. This results from the context in which the sociopolitical debate has occurred, rather than being a reflection of the veracity of competing claims.

The sociology of medical knowledge is founded on deconstructing, and thereby opening to social explanations, the origins, development and deployment of medical knowledge. By its very nature such analysis threatens the dominant position in a medical

debate. In the case of RSI, understandings from the sociology of medical knowledge were brought into play when the dominant position was that RSI is an organic work-related condition and were thereby used to undermine that position. If the dominant position had been that RSI is a form of mass hysteria, a sociology of knowledge analysis would have undermined that claim.

The social problems as social movements perspective is useful in understanding the widespread public attention given to RSI in Australia in the mid-1980s. This situation was unique. More recently there has been public recognition of these disorders in the United Kingdom and the United States of America but it has not replicated the intensity of the Australian situation. Brian Martin and I argue that the rise of RSI in Australia depended on the generation of a movement by core activists and a range of supporters and that they used a variety of resources, including the media, to mobilise concern. Their interpretation of the problem justified social action, particularly improvements in the physical and organisational aspects of work. A social movement explanation tends to delegitimise RSI because it is commonly assumed - except by analysts of social problems - that a real, organic condition will be recognised as a social problem without the entrepreneurial activities of a social movement.

Another way of looking at the broader sociopolitical debate is that it offers an opportunity to analyse the competition between a range of forces seeking to provide the dominant and enduring explanation for RSI. The competition is predominantly between a range of medical disciplines*. The competition has two poles. At one end is psychiatry, whose exponents have effectively captured the sociology of medical knowledge and social problems as social movements understandings to aid their claims that these disorders have no organic base. At the other end of the pole are a range of other disciplines who maintain that there is a predominant physiological underpinning, although there is

no agreement about its precise organic nature. The main aim of these working papers is to ascertain if some agreement can be reached about the physical underpinnings.

An improved understanding will allow people with these disorders to be diagnosed more accurately and treated with more success.

I am grateful to Drs Cohen, Arroyo and Champion and to the commentators for generously devoting time to this project.

The long-term aim is to publish this working paper, along with the other papers in this series, in a book. Further contributions to the debate, either commentaries on this paper or expositions of a particular hypothesis, are invited. Please contact me for details.

REFERENCES

1. Quintner J & Elvey R. The neurogenic hypothesis of RSI. In Bammer G (ed) *Discussion papers on the pathology of work-related neck and upper limb disorders and the implications for diagnosis and treatment*. NCEPH Working Paper Number 24 Canberra: National Centre for Epidemiology and Population Health, 1991.
2. Cohen ML, Arroyo JF, Champion GD & Browne CD. In search of the pathogenesis of refractory cervicobrachial pain syndrome: a deconstruction of the RSI phenomenon *Med J Aust* 1992; 156: 432-436.
3. Bammer G & Martin B. Repetition strain injury in Australia: medical knowledge, social movement and de facto partisanship. *Social Problems* 1992; 39: 219-237.

* The position of individual specialists does not necessarily coincide with that of their discipline. Thus, for example, some surgeons support psychiatric explanations, while some psychiatrists support explanations arising from rheumatology.

THE RELEVANCE OF CONCEPTS OF HYPERALGESIA TO "RSI"

Milton L. Cohen MB BS MD FRACP

Jesus F. Arroyo MD FMH

G. David Champion MB BS FRACP

Department of Rheumatology
St Vincent's Hospital
Sydney, NSW 2010

The clinical phenomenon of RSI or, as preferred by the present authors, refractory cervicobrachial pain (RCBP) has been presented by Quintner and Elvey (22). Before developing any hypothesis it is necessary carefully to define the problem, in this case the clinical presentation to which the label RCBP can be attached. In order to avoid the problems of imprecise nosology and of tautology which have bedevilled the debate concerning this entity, this chapter will begin with a detailed clinical description from which will be developed the basis for a neuropathic interpretation of pathogenesis.

THE UNDERLYING CLINICAL PROBLEM

The authors have appended the diagnostic label of RCBP after firstly, exclusion of known (nociceptive) entities (eg. "tendonitis", "epicondylitis", "carpal tunnel syndrome", cervical radiculopathy) and secondly clinical elicitation of features suggesting a neuropathic aetiology. Lest this be considered a circular process, it must be emphasised that, in these patients in whom after repeated assessments over time no disease-based diagnosis could be found, groups of phenomena suggesting perturbation of neural functioning were consistently able to be demonstrated. Paramount among these were the "positive"

signs of neural dysfunction, generically "hyperaesthesiae", including in particular hyperalgesia, or "tenderness", which includes both an increased algescic response to a stimulus which normally evokes pain and pain in response to a non-noxious stimulus.

Historically, in reaching this formulation the authors were impressed by three clinical findings in particular: the diffuse distribution of both the complaints of pain and the clinical hyperalgesia elicitable; the frequent complaint of pain worsened by the operation of vibrating tools (for example, a vacuum cleaner) and its correlate of allodynia on percussion; and by subtle vasomotor disturbances.

This collection of patients, from the severe end of the spectrum by nature of the referral process, numbers in excess of 1000 accrued over a five-year period. It is characterised by a number of clinical observations which constitute a homogeneous profile. There is a predominance of females, aged usually between 20 and 50 years, working either as manual process workers in industry or as office employees, in whom symptoms progressively develop over periods from some months to more than five years with bilateral affection in about one quarter of cases.

The history of such patients was of persistent pain, experienced at first in a discrete area later diffusely in the neck, pectoral girdle and arms, of a deep, burning, electrical quality, accompanied by hyperpathia, cramp, loss of muscle strength and by vasomotor abnormalities. The pain had occurred in the context of keyboard operation or of repetitive process work. A variety of therapies, predicated on conventional disease models, had been unsuccessful, whether physical or pharmacological (usually non-steroidal anti-inflammatory drugs and muscle relaxants). Many patients had undergone carpal tunnel release surgery, again often without benefit.

On examination, the apparent paradox of a hypoaesthetic painful limb presented, with perturbation of cutaneous sensation, allodynia, hyperalgesia of muscles and joints, mechanosensitivity of peripheral nerves, abnormal vaso- and sudomotor phenomena and impaired motor function, especially weakness without wasting. The patients typically presented with an antalgic attitude of the affected upper limb, with half-flexion of the elbow, wrist and fingers, without evidence of muscular atrophy or formal myotomal deficit. Consistently seen were lack of spontaneous movement, difficulty in performing fine movements, rapid onset of fatigue, spasms and cramps, all suggesting dystonic phenomena (2). All patients experienced difficulties throughout the affected limb in determining touch-pinprick, two-point discrimination and vibration intensity. These tests frequently induced characteristic dysaesthesiae and allodynia. Position sense, stereognosis and temperature appreciation were not affected. Painful hypomobility of the cervical and upper thoracic spine with associated hyperalgesia of the vertebrae and related soft tissues was frequent (1).

Many patients showed clinical signs suggestive of presumed local sympathetic dysfunction (discrete swelling of the hand, unilateral cold sweating, often blue

discoloration, more rarely piloerection). On clinical, radiological and scintigraphic grounds (13) only a minority of this subgroup fulfilled accepted criteria for "reflex sympathetic dystrophy syndrome" (17), itself a tautologous concept. However this subgroup did fulfill criteria for "sympathetically-maintained pain" as proposed by Roberts (9,25). Also observed were warm skin and exaggerated wheal/flare response, especially as part of a hyperpathic response to examination.

Laboratory investigations pursuing evidence for inflammatory disease of joints or muscles were invariably negative. Radiographs of upper limb joints were normal; those of cervical and thoracic spines revealed age-related changes only. Nerve conduction studies and/or electromyographic examinations had been performed in many patients: none was confidently interpretable as indicating neuropathy or myopathy.

At the affective level the majority of patients showed changes of anxiety/depression, more marked when the painful symptoms had persisted for a long time. Fifty of our patients were asked, on several occasions, to record their pain using the McGill Pain Questionnaire (20). In contrast to a comparison group with rheumatoid arthritis, the adjectives favoured by the RCBP patients were: shooting, pulling, burning, tingling, numb and penetrating; those are sensory words suggesting neural mechanisms. Furthermore these patients tended to avoid the affective and evaluative word groups. No significant influences were found on the responses to the McGill Pain Questionnaire by social class, cultural or ethnic origin and the presence or otherwise of anxiety or depressive features (5).

Relevant also to discussion of the role of psychogenic factors in RCBP is the reported effect of cognitive-behavioural interventions (28). Forty-five subjects were randomly assigned to individual or group cognitive-behavioural programs or to a

control group which received no intervention. Those patients receiving therapy experienced significant improvement in measures of pain and psychopathology which were maintained at six-month follow-up. However this intervention was associated with enhanced ability to cope rather than being "curative", as most subjects were still experiencing pain and restrictions in normal functioning.

CHOICE OF PARADIGM

Faced with this clinical problem the authors were reluctant to accept a primary psychogenic interpretation. By contrast it was considered that RSI may be best processed through the framework of pain itself, in particular that of chronic pain. It became apparent to us as physicians that it was necessary first to study potential abnormalities of nociception in RCBP. In this we were influenced by the guideline of Wall: "We need to proceed step by step from the periphery through the afferent nerves and through the neuronal circuits of the central nervous system before assuming a psychiatric diagnosis for those patients whose peripheral tissues seem to provide an inadequate basis for their complaint" (30).

By definition pain is a subjective experience which is the integrated expression of afferent neurophysiological mechanisms and affective-emotional phenomena susceptible to modulation by environmental and cultural, including sociopolitical, issues (16). Over recent years chronic pain has come to be appreciated by workers in the field as a syndrome, that is, a medical problem in its own right, not merely a symptom of injury or disease, and distinct in many ways from acute pain. Concepts of the biology of pain were revolutionised by the stimulus of the gate-control theory of Melzack and Wall (19), published twenty-five years ago. This theory has provided the substrate for greater neuroanatomical and

neurophysiological understanding of the known influences of somatic and "emotional" factors in the expression of pain, by nominating the dorsal horns of the spinal cord as their focus of convergence.

It is relevant at this stage to present a framework for chronic pain which identifies three levels, not mutually exclusive, of analysis: nociceptive, neuropathic and psychogenic (34). The nociceptive level is familiar as that of tissue disease or damage, areas of medical epistemology served well by the biomedical (or disease-illness) model. Nociception however strictly refers to the signalling of tissue damage or the threat thereof and may reflect altered function as well as or instead of altered structure. The neuropathic level acknowledges that between the soma and the psyche is interposed the nervous system, itself a plastic structure the function of which may change in response to afferent barrage (11,31,32). This level of pathogenesis of chronic pain may be invoked to explain the pain of phantom limbs or of the deafferented arm following brachial plexus avulsion as well as painful peripheral neuropathies and reflex states such as sympathetically-maintained pain (25). The psychogenic level acknowledges that the behavioural expression of pain (including suffering, affective disturbance, real and imagined loss) depends on the interaction of the organism with its past experience, culture and environment (14). This biopsychosocial view itself then provides a framework onto which the arguments of both medical scientists and social theorists may be projected.

BASES FOR A NEUROPATHIC HYPOTHESIS

The homogeneity of presentation of our patients implies a common pathophysiology. Certain pathophysiological inferences were made from our observations and are summarised in the following table:

Clinical feature	Physiological inference
History of neck pain Cervico-thoracic hypomobility	Anatomical origin of pain
Referred phenomena	Convergence-projection/muscle contraction theories of referred pain
Burning/electrical quality	Coactivation of different fibre types
Hypoaesthesia	Loss of myelinated afferent function
Paraesthesiae/dysaesthesiae	Ectopic impulse formation
Hyperalgesia) Allodynia)	Sensitisation of nociceptors, peripherally or centrally
Local pain becoming diffuse	Receptive field enlargement
Hyperpathia	Consistent with known neuropathic pain states
Failure of treatment based on disease model	Not primarily a nociceptor (tissue pathology) phenomenon
Vasomotor and sudomotor changes	Sympathetic dysfunction
Dermatographia	Exaggerated axon reflex
Tendency to bilaterality	Spinal cord reflex
Weakness without wasting) Pseudocramp) Characteristic posture)	?Reflex ?antalgic ?dystonic
Persistence) Influence of emotion) Past pain history)	CNS plasticity including defective descending inhibition

These features define pain of neuropathic origin, that is, attributable to dysfunction of the nervous system itself. The main theme is of hyperalgesia, especially to mechanical stimulation, manifest by definition as lowered threshold to noxious stimulation (including the operationally similar phenomenon of pain in response to non-noxious stimuli, or allodynia), increased response to noxious stimulation and spontaneous pain. Both peripheral and central mechanisms have been proposed for hyperalgesia (7,8,11,15,23,24). In these patients, where peripheral disease or damage has not been demonstrated, the hyperalgesia is more likely to be secondary and thus to imply changes in spinal cord (dorsal horn) function. This invokes the concept of spinal cord plasticity, that set of changes in dorsal horn neurons which may be induced by ongoing nociceptive barrage and is characterised by increased ongoing activity of dorsal horn cells, increased responsiveness to peripheral stimuli and enlarged receptive

fields (11,21,26,31,32,33). In particular this phenomenon has been shown in response to the stimulation of articular nociceptors in the cat (21,26).

Thus on clinical grounds we inferred that RCBP is characterised by secondary hyperalgesia. The challenges were then to relate this to physiological or psychophysical investigation and to address the question of the origin of the inferred plasticity.

In a study performed by our group, non-noxious electrical cutaneous stimulation was used to determine threshold for minimal sensation and tolerance for pain with respect to amplitude of current and duration of pulse (3). Fifteen patients with typical RCBP and ten normal volunteers were studied. The sensory profiles obtained were reproducible over time in patients and controls and were able clearly to distinguish between the affected and non-affected limbs. The threshold for minimal sensation and

tolerance for pain in the unaffected limbs of patients did not differ from those in normal subjects. Reduction in pain tolerance in affected limbs was accompanied by prolongation and spread of dysaesthesiae thus reflecting the observations evoked on clinical examination. These results were considered to define affected limbs as regions of secondary hyperalgesia and were interpreted as reflecting a state of sensitisation of afferent neurons, more probably at a central than peripheral level.

In a series of 5 such patients, injection under fluoroscopic control of depot corticosteroid into those cervical apophyseal joints clinically determined to be hyperalgesic and to induce pain remote from the site of mechanical stimulus was associated with reproduction not only of local and referred pain but also of distal vasomotor phenomena (unpublished). This might be explained by a systemic sympathetic response to a painful procedure but it was confined to the clinically painful limb. Although primarily conceived as a therapeutic manoeuvre, the procedure was so poorly tolerated that its use as a diagnostic probe was abandoned. However this experience did recall the feline experimental work (21,26) and thus provided *prima facie* evidence for a source of ongoing nociceptive activity which might be relevant to dorsal horn dysfunction.

A positive feedback loop through the spinal cord involving primary afferents and sympathetic and motor efferents, to explain chronic pain following a noxious stimulus to the periphery, was first proposed by Livingston (18). In critically reviewing the concept of *reflex* sympathetic dystrophy (emphasis added), Roberts (25) also focussed on the dorsal horn in suggesting that there is persistent sensitisation of spinal wide-dynamic-range (WDR) neurons, which results in an abnormally high rate of firing in response to afferent input. The burning pain and allodynia were then considered to result from tonic activity in low-threshold, myelinated mechanoreceptors which project

to these sensitised spinal WDR neurons, when stimulated by sympathetic efferents. The origin of the sensitisation of WDR neurons remains conjectural.

However there is indirect evidence for the source of this sensitisation. Myelinated (mechanoreceptive) afferents have been shown to mediate the secondary hyperalgesia associated with peripheral injury to nerve (4) or skin (29). In addition there is pharmacological evidence that low threshold afferent (mechanoreceptor) input may be controlled by local modulation in the spinal cord, loss of which results in mechanoreceptor input being encoded as a noxious event (35).

HYPOTHESIS

In our patients, it was considered reasonable to infer from both clinical observations and limited psychophysical studies that a state of secondary hyperalgesia exists, which in turn implies changes in the function of dorsal horn neurons similar to those described above. The concept of reflexly evoked motor and autonomic phenomena, as addressed by Livingston and Roberts, appears attractive in the present context. If mechanisms of plasticity are relevant to the pathophysiology of RCBP it is necessary also to identify the source(s) of ongoing nociceptive barrage.

We propose therefore that RCBP is a reflex neuropathic state consequent upon continuing afferent barrage from nociceptors and mechanoreceptors in anatomically relevant sites. These sites may be the spinal zygapophyseal joints or related structures, or muscles, tendons and joint capsules in the upper limb and/or the dorsal root, the dorsal root ganglion or peripheral nerves. Hypotheses based on the latter sites have been proposed, invoking the heuristic of entrapment neuropathy in which mechanical tension on those structures generates ectopic impulses in nociceptive afferents (6, 22). However we suggest that sustained afferent activity in nociceptive fibres supplying the somatic structures above may be itself

primary. This afferent barrage can readily be related to the constrained work postures and movements executed by our subjects and may be sufficient to sensitise WDR neurons such that mechanoreceptive afferent information is processed as nociceptive and thus induce the consequences envisaged by Roberts and Livingstone.

Thus, of the "afferent" clinical phenomena in these patients, pain may be referred from somatic and/or neural structures, paraesthesiae may be projected into extended receptor fields, whilst allodynia, hyperalgesia and hyperpathia reflect amplification (spatial and temporal summation) of normal sensory input at the dorsal horn level. Meanwhile the "efferent" motor and sympathetic phenomena are reflexly mediated. All three schemata provide a neurophysiological basis at a spinal cord level for the persistence of these phenomena, their potential to be influenced by descending pathways and the later involvement of the contralateral upper limb.

This hypothesis is not simply a restatement that pain in the arm may be referred from the neck (27), although that phenomenon also does focus on dorsal horn function. It explains why therapies which block sympathetic efferents will be only partially successful, as there has been no relief from the primary afferent nociceptive input, and accounts for the frequency of carpal tunnel release procedures performed, as the distal mechanosensitive phenomena could be mistaken for nerve entrapment.

CONCLUSION

A range of clinical evidence, spontaneous and evoked, has been presented to suggest that refractory cervicobrachial pain is of neuropathic pathogenesis. The "window" through which this hypothesis has been formulated is the pathophysiology of hyperalgesia, in particular secondary hyperalgesia which is considered to reflect changes in central nociceptive function.

Clinical findings in the established syndrome, especially the positive sensory phenomena of hyperalgesia and persistent dysaesthesiae, suggest sensitisation of nociceptive afferents, probably at a central level, whilst the apparent sympathetic signs may be reflex epiphenomena, spinally mediated. The motor dysfunction – weakness, cramp and focal dystonia – may also be centrally mediated. That RCBP is frequently associated with stereotyped upper limb movements from constrained postures suggests that musculoskeletal structures and/or proximal peripheral neural tissue may be candidates *prima facie* for the origin of persistent afferent barrage, initially via nociceptors and later sustained via mechanoreceptors. By focussing on the function of the dorsal horn in the spinal cord, this schema also allows for the influences by descending pathways of factors in the personality, culture and environment of the individual. Early testing of the hypothesis has not led to its refutation; further testing proposed will need to assess changes in spinal cord nociceptive function less indirectly.

REFERENCES

1. Arroyo JF, Cohen ML, Champion GD. Les cervicobrachialgies occupationnelles chroniques: éléments sémiologiques et mécanismes nociceptifs. *Douleur et Analgésie*, 1989;11:137–146.
2. Arroyo JF, Champion GD, Cohen ML. Focal dystonia in association with upper limb pain of presumed neuropathic aetiology. *Aust NZ J Med* 1990; 20 Suppl 1:494.
3. Arroyo JF, Cohen ML, Champion GD. Evidence for neuropathic mechanisms in diffuse upper limb (occupational cervicobrachial disorder) and diffuse musculoskeletal (fibromyalgia) pain syndromes. *Pain* 1990; Suppl. 5: S465.

4. Campbell JN, Raja SN, Meyer RA, Mackinnon SE. Myelinated afferents signal the hyperalgesia associated with nerve injury. *Pain* 1988; **32**: 89–94.
5. Champion GD, Cairns D, Spence S, Arroyo JF. Correspondence analysis of the adjectives of the McGill Pain Questionnaire in occupational cervicobrachial disorders. *Abstracts of the Eleventh Annual Scientific Meeting, Australian Pain Society, Sydney, March 1989*.
6. Champion GD, Cornell J, Browne CD, Garrick R, Herbert TJ. Clinical observations in patients with the clinical syndrome "repetition strain injury". *J Occup Health Safety – Aust NZ* 1986; **2**: 107–113.
7. Cline MA, Ochoa J, Torebjork HE. Chronic hyperalgesia and skin warming caused by sensitised C nociceptors. *Brain* 1989; **112**: 621–627.
8. Coderre TJ, Melzack R. Cutaneous hyperalgesia: contribution of the peripheral and central nervous systems to the increase in pain sensitivity after injury. *Brain Res* 1987; **404**: 95–106.
9. Cohen ML, Gibb DB. Does the sympathetic nervous system play a role in the pathogenesis of diffuse regional upper limb pain? *Aust NZ J Med* 1988; **18** Suppl 1:216.
10. Cohen ML, Arroyo JF, Champion GD & Browne CD. In search of the pathogenesis of refractory cervicobrachial pain syndrome: a deconstruction of the RSI phenomenon. *Med J Aust* 1992; **156**: 432–436.
11. Cook AJ, Woolf CJ, Wall PD. Dynamic receptive field plasticity in rat spinal cord dorsal horn following C–primary afferent input. *Nature* 1987; **325**: 151–153.
12. Culp WJ, Ochoa J, Cline M, Dotson R. Heat and mechanical hyperalgesia induced by capsaicin. *Brain* 1989; **112**: 1317–1331.
13. Demangeat J-L, Constantinesco A, Brunot B. Three-phase bone scanning in reflex sympathetic dystrophy of the hand. *J Nucl Med* 1988; **29**: 26–32.
14. Fordyce W. Pain and suffering. A reappraisal. *Amer Psychol* 1988; **43**: 276–283.
15. Hardy TD, Wolff HG, Goodell H. Experimental evidence on the nature of cutaneous hyperalgesia. *J Clin Invest* 1950; **29**: 115–140.
16. International Association for the Study of Pain, Subcommittee on Taxonomy. Pain terms: a list with definitions and notes on usage. *Pain* 1979; **6**: 249–252.
17. Kozin F. The painful shoulder and reflex sympathetic dystrophy syndrome. In: McCarty DJ, ed. *Arthritis and Allied Conditions*, 10th ed. Philadelphia: Lea & Febiger, 1985: 1322–1355.
18. Livingston WK. *Pain mechanisms, a physiologic interpretation of causalgia and its related states*. New York: Macmillan, 1943.
19. Melzack R, Wall PD. Pain mechanisms: a new theory. *Science* 1965; **150**: 971–978.
20. Melzack R. The McGill Pain Questionnaire: major properties and scoring methods. *Pain* 1975; **1**: 277–299.

21. Neugebauer V, Schaible H-G. Peripheral and spinal components of the sensitization of spinal neurons during an experimental arthritis. *Agents Actions* 1988; **25**: 234-237.
22. Quintner J, Elvey R. The neurogenic hypothesis of RSI. In Bammer G, ed. *Discussion papers on the pathology of work-related neck and upper limb disorders and the implications for diagnosis and treatment*. NCEPH Working Paper Number 24 Canberra: National Centre for Epidemiology and Population Health, 1991.
23. Raja SN, Campbell JN, Meyer RA. Evidence for different mechanisms of primary and secondary hyperalgesia following heat injury to the glabrous skin. *Brain* 1984; **107**: 1179-1184.
24. Raja SN, Meyer RA, Campbell JN. Peripheral mechanisms of somatic pain. *Anaesthesiol.* 1988; **68**: 571-590.
25. Roberts WJ. A hypothesis on the physiological basis for causalgia and related pains. *Pain* 1986; **24**: 297-311.
26. Schaible HG, Schmidt RF, Willis WD. Enhancement of the responses of ascending tract cells in the cat spinal cord by acute inflammation of the knee joint. *Exp Brain Res* 1987; **66**: 489-499.
27. Smythe H. The "repetitive strain injury syndrome" is referred pain from the neck. *J Rheumatol* 1988; **15**: 1604-1608.
28. Spence SH. Cognitive-behavioural therapy in the management of chronic, occupational pain of the upper limbs. *Behav Res Ther* 1989; **27**: 435-446.
29. Torebjörk E, Lundberg L, LaMotte R. Neural mechanisms for capsaicin-induced hyperalgesia. *Pain* 1990; Suppl. 5:S114.
30. Wall PD. Causes of intractable pain. *Hosp Update* 1986; (Dec):969-974.
31. Woolf CJ. Evidence for a central component of post-injury pain hypersensitivity. *Nature* 1983; **306**: 686-688.
32. Woolf CJ. Recent advances in the pathophysiology of acute pain. *Brit J Anaesth* 1989; **63**: 139-146.
33. Woolf CJ, Thompson SLN, King AE. Prolonged primary afferent induced alteration in dorsal horn neurons: intracellular analysis in vivo and in vitro. *J Physiol (Paris)* 1988; **83**: 255-266.
34. Working Party on the Management of Severe Pain. *Management of severe pain*. Canberra: AGPS 1988, 10.
35. Yaksh TL. Behavioural and autonomic correlates of the tactile evoked allodynia produced by spinal glycine inhibition: effects of modulatory receptor systems and excitatory amino acid antagonists. *Pain* 1989; **37**: 111-123.

COMMENTARIES

Trevor Beswick BAppSc (Phty) MAPA MESA
Physiotherapist
Annette Cursley BAppSc (Phty) MAPA MESA
Physiotherapist

Sport & Work Injuries Centre
PO Box 454
Jamieson ACT 2614 Australia

The term chosen by the authors, Refractory Cervicobrachial Pain (RCBP), is a no more diagnostic term than RSI and adds another name to the already long list of pseudonyms. We also feel that it is unwise to exclude such entities as tendonitis, epicondylitis, etc as they are frequently found in RSI sufferers.

We would agree with the authors' general findings of the history provided by RSI patients. These findings closely parallel our own clinical findings and observations. We would also agree that treatments based on conventional disease models are seldom successful. In our own paper for this series (Beswick & Cursley, forthcoming) we discuss forms of treatment outside the conventional methods, which we have found effective.

The clinical findings noted are an interesting counter to the many previous authors in the RSI debate who have remarked

consistently on the absence of any observable or demonstrable physical signs.

The concept of using cognitive-behavioural interventions as a coping rather than curative strategy is valid. Some authors chose to label RSI as a purely psychogenic entity to which the application of psychotherapeutic programmes should be successful. Where they are not successful, the client is often labelled as a malingerer. Cohen and coworkers have identified the appropriate use of psychotherapeutic intervention.

We find the paper exciting in the concepts it proposes and, whilst not being in a position to critically evaluate the neurophysiology described, our own clinical findings closely parallel those described. Some of the treatment successes and failures which we have experienced would appear to mesh with and be partially explicable within the general hypothesis of this paper.

Earl Owen AO MD MBBS FRCS FRACS FRCSE FICS
Consultant Microsurgeon & Medical Director

The Microsurgery Centre
1 Esther Street
Surrey Hills, Sydney NSW 2010 Australia

If you really know your subject you should be able to explain it in such a way that another interested person could understand what you are on about.

"Overuse Syndrome", "R.S.I." and other inappropriately vague names do not help us to understand these conditions where patients genuinely suffer with pain and debility which does not fit into more easily labelled and more specific musculo/tendinous/joint/nerve disorders.

I am hopeful that one day someone can learn why one person can rapidly and repeatedly twist his or her forearm and extended wrist without much effort, without any pain and without obvious short or long term sequelae, while another individual, doing precisely the same movements, ends up with such pain and debility that he or she seeks medical assistance.

Surgeons, who tend to have active interventionist personalities, like to surgically treat specific conditions and so favour exact diagnoses. As yet there appear to be no definite reasons to operate in these conditions, so surgeons feel frustrated that all their surgical skills are wasted on these individuals who can recover quite well if you just judiciously supervise their rest, and support them with heat/cold, exercises, physiotherapy, show them concern and support them, and withhold harmful movement. Some surgeons, suitably surgically thwarted, even deny such conditions exist!

Psychiatrists are on record as believing that these conditions do not fall into regular psychiatric symptom complexes, and as they do not respond to these physicians' ministries,

therefore the symptomatology does not really exist either, and so these patients must be labelled as malingerers.

There is, however, no one cause yet established for what is now known as "Overuse Syndrome" or "Cervico-Brachial Pain" or "R.S.I.", not to mention other names for a very difficult-to-classify condition. Every case is different, just as every individual is different, but there are enough similarities in certain groups of cases to influence sincere workers in this field to postulate sensible arguments, and indeed in some cases even some scientific evidence, in favour of some genuinely helpful treatments.

Cohen and co-workers are physicians specialising in rheumatology (which is accepted as a more passive or contemplative speciality) of widely differing general views to those of surgeons. They too are very well trained, and just as concerned and interested in these conditions, and they come to conclusions based on their association with over 1,000 patients over five years and their own assiduous research. Cohen et al, however, do not write clearly and seem to be so confused about these conditions that they feel compelled to give them yet another name - refractory cervicobrachial pain (R.C.B.P)! Calling this symptom complex virtually "a pain in the neck" could shed some light on the subject if only they could present some evidence to back up their involved but inaccurate name for a condition that more commonly is seen in forearms, hands, wrists and around joints. They labour hard to try to justify the whole thing as being due to the patients' concept of pain mediated by a dysfunction of their nervous systems.

Unfortunately, Cohen et al have confused the issue further with their new name "R.C.B.P." as their diagnosis of what they think they see, and string together a ladder of important sounding "newspeak" terms to prop up their generalised but unconvincing vision of the usual clinical picture. They then state, without mentioning how the condition is treated, that "those patients receiving therapy experienced significant improvement" but such a claim is unscientific when not backed by any evidence at all.

Having failed to adequately establish the pathogenesis of the condition, we are then led up their garden path hypothesis via their interpretation of the patients' interpretation of chronic pain - and we all know that the assessment of pain is a notoriously deceptive path indeed.

I am disappointed that Cohen et al's rambling and confused paper fails to contribute anything scientific to help our patients with this distressing condition.

Michael Patkin MBBS FRCS FRCSEd FRACS
Surgeon

PO Box 735
Whyalla SA 5600

Drs Cohen and Champion are both respected senior consulting rheumatologists at a leading Australian teaching hospital with experience in clinical research. They describe 1000 patients, mostly female workers in offices and factories, and a very typical "attitude of the affected upper limb, with half-flexion of the elbow, wrist, and fingers, without evidence of muscle atrophy ...".

Nowhere do they suggest that this posture is learned, whether consciously or not. Its pattern of incidence is like complaints of "koro" in South East Asian Chinese (sudden anxiety about recession of the penis into the surrounding skin), hysterical overbreathing in teenage girls at pop-concerts, or several conditions described in detail in "Mass Psychogenic Illness: A Social Psychological Analysis", written in 1982 by Colligan, Pennebaker, and Murphy.

This book should be compulsory reading for all those involved in the RSI debate. In Chapter 2, W. H. Phoon describes 6 separate outbreaks of mass hysteria at workplaces in Singapore. In the next chapter Colligan and Murphy, then working for the National Institute

of Occupational Safety and Health in the USA, describe 23 separate outbreaks with various symptoms. If it were brought up to date, it would have included "facial dermatitis" in computer operators in Sweden, described in detail at several large recent international conferences, where the "rash" could not be identified by dermatologists, and occurred even when computers were not turned on.

Such reports are ignored in references by writers with a monoculture approach to aetiology, whether their background is academic or, worse still, political and manipulative. They ignore early reports like those of "angina" described in computer operators in North Carolina in 1983. Instead, these bibliographies consist largely of reports of unproven pathology or the results of subjective questionnaires among populations of subjects whose interest it is to promote the concepts of "RSI" and "injury".

Cohen et al describe "Many patients [who] showed clinical signs of ... discrete swelling of the hand ... blue coloration". No reference to actual measurements of this swelling is given. In my own series of

approximately 200 patients examined personally, only two had swollen fingers on measurement of the circumference of one hand compared with the other, and both seemed within the limits of observer error. There are no published volumetric studies of patients with unilateral hand swelling, although it was the rule for doctors and others to describe this kind of swelling when the RSI epidemic was at its height in the mid-1980's.

The proposal by Cohen et al of a "neuropathic" basis for chronic pain is an attractive one. Half-way between purely mental and purely physical pain is a phenomenon in dorsal horn cells in the spinal cord, whose threshold falls in response to repeated noxious stimuli, provided this is related to facilitatory messages from higher nervous centres - the messages given through the media to individual victims that they are going to feel pain, and that they are suffering injury. It fits in with modern ideas on pain, such as the gate theory of Melzack and Wall (1965). It recalls the frightening sensation of mild pressure on the gum from the dentist's apparatus which is going to turn into excruciating pain, but doesn't. It is like the woman who is scared witless by feeling the normal bony bump of a rib underlying breast tissue after hearing a television warning about breast cancer, until reassured by the examining surgeon when she is likely to burst into tears.

Neuropathic hyperalgesia is presented as a credible hypothesis to explain SOME of the pain in SOME patients. It doesn't explain the pain in thirty to fifty per cent of workers in some industries (Telecom switchboard operators in Perth in 1983, library assistants using "unergonomic" bar code readers in Adelaide public libraries in 1984 where the investigator privately admitted the influence of political pressures on his findings). It would go down as an explanation for Quintner and Elvey (1991), for whom practically all cases start off with Brachial Plexus Tension, for those early enthusiasts in Australia in 1982 who called every case "tenosynovitis" as they now do "carpal tunnel syndrome" in North America, and it wouldn't do for Hunter Fry (1992), whose theme

has been muscle damage in musicians, detectable on electron microscope study.

Reviewers come to their task biased by their own training, experience and individual values and judgements. As I have criticized the views offered, it should help to put this criticism into perspective if I present, briefly, my own view of RSI: RSI is a label used to refer to many different conditions. In the Australian epidemic most complainants had a combination of some initial minor muscular problem with a large psychological problem they were talked into by the prevailing public climate. They were talked into believing they had structural injuries in their arms or neck by people with strong ideas on the pathology - whether clinical or political, or people searching for a sense of mission in their lives, especially journalists.

There is a myth that science is an objective pursuit, typified by researchers in white coats, and with blank minds before they start to make or interpret observations. The sociology of science suggests otherwise.

REFERENCES

- Colligan MJ, Pennebaker JW and Murphy LR
Mass Psychogenic Illness: A Social Psychological Analysis, New Jersey: Lawrence Erlbaum, 1982.
- Fry HJH Overuse syndrome and the overuse concept. In Bammer G (ed) *Discussion papers on the pathology of work-related neck and upper limb disorders and the implications for diagnosis and treatment*. NCEPH Working Paper Number 32 Canberra: National Centre for Epidemiology and Population Health, 1992.
- Melzack R, Wall PD. Pain mechanisms: a new theory. *Science* 1965; **150**: 971-978.

Quintner J, Elvey R The neurogenic hypothesis of RSI. In Bammer G (ed) *Discussion papers on the pathology of work-related neck and upper limb disorders and the implications for diagnosis and*

treatment. NCEPH Working Paper Number 24 Canberra: National Centre for Epidemiology and Population Health, 1991.

John Quintner MBBS MRCP
Rheumatologist

St John of God Medical Centre
175 Cambridge Street
Wembly WA 6014

OVERVIEW OF HYPOTHESIS

Cohen *et al.* propose that the nexus between repetitive manual work performed in constrained postures, and the development of refractory cervicobrachial pain (RCBP), is the sustained afferent activity *per se* in mechanosensitive and nociceptive fibres supplying the relevant somatic and/or neural tissues. At the spinal level, this activity constitutes a barrage which sensitises the nociceptive sensory transmission neurons known as wide dynamic range (WDR) neurons. These neurons then respond inappropriately, or in an exaggerated manner, to low intensity stimuli (A beta afferents). With this response, physiologically-induced pain changes to neuropathic pain. Motor and sympathetic phenomena of RCBP are then reflexly induced as a result of the abnormal response characteristics of sensitised WDR neurons.

Although Cohen *et al.* accept that the origin of the sensitisation of WDR neurons remains conjectural, their explanatory model (EM) denies the necessary primacy of tissue damage (neural or somatic) as a prelude to the neuropathic pain state. Persistence of RCBP, in the absence of even a low level of pathological input from tissue damage (peripheral nociceptor sensitisation), infers the existence of an as yet undiscovered mechanism to explain the persistent state of central excitability. The important issue of whether RCBP is an example

of sympathetically maintained pain is not discussed. In addition, their favoured EM does not offer an explanation for the paraesthesiae and hypoaesthesia.

REFLEX MECHANISMS

We propose therefore that RCBP is a reflex neuropathic state consequent upon continuing afferent barrage from nociceptors and mechanoreceptors in anatomically relevant sites.

The hypotheses of Livingstone¹⁸ and Roberts³⁰ are used to explain the reflex induction of the neuropathic pain state in the absence of demonstrable peripheral disease or damage. According to Price,²⁶ who has recently reviewed the theories of pain mechanisms, "Livingstone recognized the fact that long-duration pains could sometimes be triggered by brief duration stimuli and he postulated the existence of reverberating circuits within the gray matter of the spinal cord." Livingstone¹⁸ also proposed that different forms of damage to large and small peripheral nerves may give rise to irritative nerve lesions which could, in turn, initiate activity in these reverberatory circuits. By this means, additional nociceptive impulses summate to create a "vicious circle" in a closed loop between the central and the peripheral processes that maintain the abnormal spinal cord activity. If the disturbance so created continues to operate, it

may become self-sustaining and likely to disrupt nearby and distant portions of the nervous system:

The reflex disturbance first spreads beyond the distribution of the sensory nerve originally involved by the lesion; then it may spread to the opposite limb, to an ipsilateral limb, or even to affect the functioning of one side of the body ... Once a new area is involved, the process may continue after the original lesion has lost its sustaining effect. It is difficult to escape the conviction that some dynamic process has been initiated within the spinal cord that may persist after the original stimulus has been withdrawn.¹⁸

The EM of Livingstone was enlarged upon by Kelly,¹⁵ who, in his discussion of the functional organisation of the spinal cord, was ahead of his time:

In other words, stimulation and inhibition are continually proceeding side by side in the spinal cord, both on the motor and the sensory side. It seems not unreasonable, therefore, to suggest that a succession of abnormal sensory impulses, such as may proceed from diseased or damaged tissue, could set up in the cord a disorder of function, a functional disturbance in the truest sense of the word, which would manifest itself by *abnormal* (my italics) sensory or motor function.¹⁵

The neural mechanisms postulated by Livingstone have not been substantiated.²⁶ However, within the dorsal horn, there are mechanisms which explain the phenomena of slow temporal summation, spatial recruitment and afterresponse.^{8,33}

Kelly¹⁵ set out to explain the pain of a condition then known as interstitial neuritis, characterised by: (a) radiating pain; (b) tenderness in the vicinity of a nerve trunk; (c) paraesthesiae or objective sensory loss (uncommon); (d) muscle wasting or paresis (uncommon), or loss of deep reflexes

(uncommon). Finding unsatisfactory the conventional theory that non-specific inflammatory changes in a nerve trunk cause neuralgic pain by exerting pressure upon the axis cylinders, Kelly¹⁵ argued that a focal fibrositic lesion must be responsible for the manifestations of interstitial neuritis via an "antidromic nervous reflex". This explanation is not required now that a number of peripheral neural mechanisms underlying chronic pain have been discovered - pathological spontaneous activity in the nociceptors, cross-talk between large and small (nociceptive) fibres, sensitivity of damaged nociceptor afferents to circulating adrenergic compounds.⁷

Roberts³⁰ put forward his hypothesis to explain "sympathetically maintained pain". According to his model, the initiating event is any trauma sufficient to activate C-nociceptors. Although his hypothesis required "Neither dystrophic tissue nor nerve injury", Roberts³⁰ acknowledged that sensitisation of nociceptive afferents, dystrophic or ischaemic tissues or nerve damage "may exist and may contribute to the pain".

Retreating somewhat from the position originally taken by Roberts, Roberts and Foglesong³¹ acknowledged that in most people, activation of low threshold mechanoreceptors through non-damaging pressure or sympathetic arousal does not result in painful sensation and that a precipitating injury is usually necessary to produce the nociceptive barrage necessary to sensitise WDR neurons. Herein lies the main difficulty I have in accepting the proposed somatogenic, as opposed to neurogenic, initiation of RCBP.

SOMATIC AFFERENT INPUT AS THE REFLEX TRIGGER

However we suggest that sustained afferent activity in nociceptive fibres supplying the somatic structures ... may itself be primary. This afferent barrage can readily be related to the constrained work postures and movements executed by our subjects and may be sufficient to sensitise WDR neurons...

In the presence of tissue damage and inflammation, unmyelinated C-fiber afferents are readily activated and appear to play an important role in the associated pain and hyperalgesia, to which both peripheral and central mechanisms contribute.³⁶ Chemical stimuli are known to be of utmost importance for the chronic excitation of nociceptors.^{20,37}

In animal experimental models of chronic arthritic pain, both structural and functional changes have been shown to occur within the spinal cord, the thalamus and the cerebral cortex (reviewed by Helme *et al.*¹⁴).

Also from animal experiments, it is known that most nociceptors (small myelinated A-delta and unmyelinated C-fiber afferents) in joint capsule respond to intense mechanical strain, such as heavy local pressure or to joint movements beyond the physiologically normal range.²⁸ They could therefore be responsible for local and/or referred pain induced by mechanically overloaded cervical spinal apophyseal joints. Approximately half of the nociceptors also respond to non-noxious movements and may possibly contribute to a sensation of deep pressure.

The observation that injection of depot steroid into hyperalgesic cervical apophyseal joints leads to reproduction of both local and referred pain together with distal vasomotor phenomena could incriminate these joints as the source of on-going nociception, particularly if the initial pain of these patients was axial. But some caution is in order before coming to this conclusion in the clinical setting of neuropathic pain, where there is likely to be considerable expansion of the receptive fields of WDR neurons to mechanical stimuli.^{8,33} It must, at present, remain problematic whether these nociceptive inputs from apophyseal joint capsule(s) are commonly generated by work performed in constrained postures (as seems likely) and whether they can, under certain circumstances, constitute the type or pattern of barrage sufficient to sensitise WDR neurons.

Muscle nociceptive afferent fibres are known to respond to strong localised pressure, but not to muscle stretch or contractions. Some

units are excited by ischaemia combined with contraction of the muscle.^{7,28}

Upper limb muscle injury (albeit subtle) from occupational overuse has been proposed as the primary underlying pathology in RCBP.^{10,11,35} This EM received some support from the findings of a muscle biopsy study.⁵

Unable to make a tissue-specific clinical diagnosis, and finding 2 or more Smythe tender points in the majority of their patients, Miller and Topliss¹⁹ noted that the changes in muscle reported by Dennett and Fry⁵ were similar to those found in the fibrositis syndrome. On these grounds they saw an analogy between the diagnosis of repetitive strain injury (RCBP) and the fibrositis syndrome. The same question, "Can muscle injury at a single site be the cause of a generalized form of nonarticular rheumatism compatible with a diagnosis of fibrositis", has previously been raised but not answered.²

Littlejohn¹⁷ classified RCBP as a localized fibrositis syndrome (regional pain syndrome). Although (for medico-legal reasons) he downplayed the importance of physical causative or precipitating factors in the workplace, the EM which he, together with Reilly,²⁹ put forward for fibrositis/fibromyalgia syndrome resembles closely the neuropathic pain model for RCBP proposed by Cohen *et al.*

The clinical features of fibromyalgia syndrome (FS) appear ... to be mediated peripherally through activation of the dorsal horn deep pain system in its association with the sympathetic nervous system and its automatic reflex activation of regional motor nerve pools. ... Thus, we have a syndrome characterized by peripheral sensorineural activation, with important central modulatory factors in which it is likely that there is a resetting of controls of peripheral pain perception through dorsal horn and other central mechanisms.²⁹

Ochoa *et al.*²⁵ theorised that selective injury to muscle fascicles could result in sympathetic dependent muscle pain. Considering the rarity of neuropathic pain states after well-defined limb muscle injury on the playing field or after continuous and repetitive upper limb exertion undertaken without fixed or constrained head/neck postures, one is forced to look towards the spinal axial (cervical and shoulder girdle) musculature for the possible source of the initial nociceptive barrage in RCBP.²¹

In the occupational health literature, it has been taught that neck and shoulder pain are more likely to occur in those workers who develop a high static load in the musculature of their neck and shoulder girdle.^{12,13} A recent controlled experimental study from Finland has challenged this hypothesis.³² The researchers failed to show an association between increased shoulder muscle activity (as assessed by surface electromyography over the upper trapezius and rhomboids/erector spinae muscles) and neck-shoulder pain. In addition, the matched asymptomatic controls reported taking fewer rest periods than those with frequent neck-shoulder pain.

PARAESTHESIAE

The hypotheses of Livingstone¹⁸ and Roberts³⁰ offer no explanation for the paraesthesiae frequently experienced by patients with RCBP.^{4,9,19} Paraesthesiae are commonly experienced following minor mechanical traumata to peripheral nerves. They can also be elicited from patients with injuries or diseases involving sensory pathways, in either the peripheral or the central nervous system.²²

As shown by Cohen *et al.* in their table, paraesthesiae are thought to result from ectopic impulse formation in sensory units with myelinated fibres.^{6,24}

The normal spatio-temporal pattern of impulses in populations of sensory units is upset during paraesthesiae, and replaced by high-frequency bursting discharges, appearing asynchronously in multiple sensory units of different types

and with different central connections. This would create a chaotic percept consisting of an assortment of sensations referred in irregular succession to multiple areas. This is the essential feature of paraesthesiae.²⁴

Having correctly inferred the pathophysiological basis for paraesthesiae, Cohen *et al.* fail to discuss the significance of this symptom which, of course, points towards a neurogenic basis for RCBP. An alternative explanation, using their hypothesis, requires that sensitised WDR neurons retain the capacity to process afferent non-noxious stimuli as either painful, or non-painful but unfamiliar sensations (paraesthesiae).

HYPOAESTHESIA

In their reported study,¹ patients with RCBP were found to have a normal threshold for minimal sensation as measured by non-noxious electrical cutaneous stimulation. This finding is at variance with the hypoaesthesia reported in these patients. In addition, the finding of a normal cutaneous threshold using electrical stimuli differs from the finding by Procacci and Maresca²⁷ of an abnormal difference in cutaneous sensory thresholds between the affected limb and the contralateral one in patients with reflex dystrophies. This difference requires explanation.

Cohen *et al.* correctly point out in their tabulation that hypoaesthesia is said to infer loss of myelinated afferent function. However, hypoaesthesia may also be due to a functional block at spinal or higher levels associated with neuralgic pain.¹⁶

Hypoaesthesia, in association with allodynia and hyperpathia has been documented in patients with chronic pain as a consequence of nerve trauma.^{16,23} Although an inordinate amount of research has been devoted to the better understanding of allodynia and hyperpathia (reviewed by Bonica³), the mechanism of hypoaesthesia remains obscure.

CONCLUSION

When describing an outbreak amongst women of arm pain and other sensory symptoms, Sir Francis Walshe,³⁴ one of the great British neurologists, showed remarkable prescience: "No one can doubt that the syndrome will shortly be conscripted into the swelling ranks of so-called psychoneurotic disorders, from which it will with difficulty be rescued for medicine proper".

Cohen, Champion and Arroyo have rescued RCBP "for medicine proper" by providing a thought-provoking hypothesis based upon their own research into this poorly understood area of medicine. Through their efforts, RCBP can now be further studied as yet another clinical model of neuropathic pain.

REFERENCES

1. Arroyo JF, Cohen ML, Champion GD. Evidence for neuropathic mechanisms in diffuse upper limb (occupational cervicobrachial disorder) and diffuse musculoskeletal (fibromyalgia) pain syndromes. *Pain* 1990; Suppl. 5: S465.
2. Bennett RM. Current issues concerning management of the fibrositis/fibromyalgia syndrome. *Am J Med* 1986; 81(Suppl 3A): 15-18.
3. Bonica JJ. The Management of Pain, 2nd ed. Philadelphia: Lea & Febiger, 1990: 220-243.
4. Champion GD, Cornell J, Browne CD, Garrick R, Herbert TJ. Clinical observations in patients with the clinical syndrome "repetition strain injury". *J Occup Health Safety-Aust NZ* 1986; 2(2): 107-113.
5. Dennett X, Fry HJH. Overuse syndrome: a muscle biopsy study. *Lancet* 1988; i: 905-908.
6. Devor M. Neuropathic pain and injured nerve: peripheral mechanisms. *British Medical Bulletin* 1991; 47: 619-630.
7. Dostrovsky JO. The neural basis of pain. In: Kennard C, et al., eds. *Recent advances in clinical neurology*. No. 6. Edinburgh: Churchill Livingstone, 1990: 1-22.
8. Dubner R. Hyperalgesia and expanded receptive fields [editorial]. *Pain* 1992; 48: 3-4.
9. Elvey RL, Quintner JL, Thomas AN. A clinical study of RSI. *Aust Fam Physician* 1986; 15: 1314-1322.
10. Ferguson D. The 'new' industrial epidemic [editorial]. *Med J Aust* 1984; 140: 327-329.
11. Fry HJH. Overuse syndrome in musicians: prevention and management. *Lancet* 1986; ii: 728-731.
12. Hagberg M. Occupational musculoskeletal stress and disorders of the neck and shoulder. A review of possible pathophysiology. *Int Arch Occupat Environ Health* 1984; 53: 269-278.
13. Hagberg M, Wegman DH. Prevalence rates and odds ratios of shoulder-neck diseases in different occupational groups. *Br J Ind Med* 1987; 44: 602-610.
14. Helme RD, Gibson S, Khalil Z. Neural pathways in chronic pain. *Med J Aust* 1990; 153: 400-406.
15. Kelly M. Interstitial neuritis and the pressure theory of pain: a critical review and a reflex hypothesis. *Med J Aust* 1946; ii: 325-334.
16. Lindblom U, Verrillo RT. Sensory functions in chronic neuralgia. *J Neurol Neurosurg Psychiatry* 1979; 42: 422-435.

17. Littlejohn GO. Repetitive strain syndrome: an Australian experience [editorial]. *J Rheumatol* 1986; **13**: 1004-1006.
18. Livingstone WK. *Pain mechanisms, a physiologic interpretation of causalgia and its related states*. New York: McMillan, 1943.
19. Miller MH, Topliss DJ. Chronic upper limb pain syndrome (repetitive strain injury) in the Australian workforce: a systematic cross sectional rheumatological study of 229 patients. *J Rheumatol* 1988; **15**: 1705-1702.
20. Nakamura-Craig M, Smith TW. Substance P and peripheral inflammatory hyperalgesia. *Pain* 1989; **38**: 91-98.
21. Newham DJ. Skeletal muscle pain and exercise. *Physiotherapy* 1991; **77**: 66-70.
22. Nordin M, Nystrom B, Wallin U, Hagbarth K-E. Ectopic sensory discharges and paraesthesiae in patients with disorders of peripheral nerves, dorsal roots and dorsal columns. *Pain* 1984; **20**: 231-245.
23. Ochoa J, Noordenbos W. Pathology and disordered sensation in local nerve lesions: an attempt at correlation. In: Bonica JJ et al., eds. *Advances in Pain Research and Therapy, vol 3*. New York: Raven Press, 1979: 67-90.
24. Ochoa JL, Torebjork E. Paraesthesiae from ectopic impulse generation in human sensory nerves. *Brain* 1980; **103**: 835-853.
25. Ochoa JL, Torebjork, Marchettini P, Sivak M. Mechanisms of neuropathic pain: cumulative observations, new experiments, and further speculation. In: Fields HL et al., eds. *Advances in pain research and therapy, vol 9*. New York: Raven Press, 1985: 431-450.
26. Price DD. *Psychological and neural mechanisms of pain*. New York: Raven Press, 1988: 212-231.
27. Procacci P, Maresca M. Reflex sympathetic dystrophies and algodystrophies: historical and pathogenic considerations. *Pain* 1987; **31**: 137-146.
28. Raja SN, Meyer RA, Campbell JN. Peripheral mechanisms of somatic pain. *Anaesthesiology* 1988; **68**: 571-590.
29. Reilly PA, Littlejohn GO. Fibrositis/fibromyalgia syndrome: the key to the puzzle of chronic pain [editorial]. *Med J Aust* 1990; **152**: 226-228.
30. Roberts WJ. A hypothesis on the physiological basis for causalgia and related pains. *Pain* 1986; **24**: 297-311.
31. Roberts WJ, Foglesong ME. I. Spinal recordings suggest that wide-dynamic-range neurons mediate sympathetically maintained pain. *Pain* 1988; **34**: 289-304.
32. Takala E-P, Viikari-Juntura E. Muscular activity in simulated light work among subjects with frequent neck-shoulder pain. *Int J Indust Ergon* 1991; **8**: 157-164.
33. Wall PD. Neuropathic pain and injured nerve: central mechanisms. *British Medical Bulletin* 1991; **47**: 631-643.
34. Walshe FMR. On "acroparasthesia" and so-called "neuritis" of the hands and arms in women. *Br Med J* 1945; **2**: 596-598.
35. Wigley RD. Repetitive strain syndrome - fact not fiction. *NZ Med J* 1990; **103**: 75-76.

36. Woolf CJ, Thompson SWN. The induction and maintenance of central sensitization is dependent on *N*-methyl-D-aspartic acid receptor activation: implications for the treatment of post-injury pain hypersensitivity states. *Pain* 1991; **44**: 293-299.
37. Zimmermann M. Pain mechanisms in osteoarthritis. *Sem Arth Rheum* 1989; **18**: 22-29.

Gisela Sjøgaard PHD DR MED

Professor
Department of Physiology
National Institute of Occupational Health

Lerso Parkalle 105, DK-2100 Copenhagen,
DENMARK

This paper illustrates how complex the mechanisms for RSI or RCBP may be and that several concepts may be involved. A whole cascade of events may be involved in the development into the final stage of irreversible disorder and pain. Which tissues the morphological or biochemical changes occur in is still a matter of discussion. Even the mechanisms of sensitisation of nociceptive afferents are to be explained on a subcellular level. Focussing on the function of the dorsal horn in the spinal cord may add

important information to this area. However, changes in this location are likely to be induced by other processes, which precede the sensitisation. Basic knowledge is still missing regarding the aetiology of work related musculoskeletal disorders but epidemiological studies indicate strongly a causal relationship. However, in order to develop optimal strategies for prevention of these disorders myogenic (nociceptive), neuropathic, as well as psychogenic mechanisms need further attention.

Richard D. Wigley MB CHB FRCP FRACP FACRM

Director
WHO Collaborating Centre for the Epidemiology of Rheumatic Disease Research Laboratory

Public Hospital
Palmerston North New Zealand

The authors coin their own term "refractory cervicobrachial pain" to encompass "the clinical phenomenon of RSI". They rightly warn against imprecise nosology and the danger of tautology but the new title sets new nosological traps. In introducing the word pain they exclude

those with discomfort and/or movement disorder. By including the word refractory they exclude the less severe cases which are free of secondary and tertiary features which are more likely to be the result than the cause of the symptoms. Such cases are therefore less likely

to yield clues to control and treatment and this must be the primary object of research into the mechanism of disease.

They then exclude cases with "features suggesting a neuropathic aetiology". They consider this not to be a circular process since over time no disease based diagnosis could be found. But, is this not teleological since disease according to the Oxford English and other dictionaries is "dis-ease" so by this definition all subjects presenting with symptoms have a disease? If the authors have their own definition of disease which, one reads between the lines, is "a complaint in which there is macro, micro, ultra-microscopic or biochemical abnormality" there is a non sequitur. Migraine and schizophrenia then have to be considered non diseases not to mention disorders, such as the one in question, which may, perhaps next year, be explained by magnetic resonance imaging (MRI), non invasive real time biochemistry. Further the authors go on to say "In these patients where peripheral disease or damage has not been demonstrated" we have to ask "Was it looked for?" since Fry (1992, e.g.) states that there is demonstrable abnormality. Clearly both cannot be right. We are back in a whirlpool of circular thought.

The high proportion of cases at "the severe end of the spectrum by nature of the referral process" will inevitably show bias towards features reflecting the authors' interest in neuropathic pain. So their evidence may

reflect that bias rather than justifying the conclusion that "the homogeneity of the presentation of our patients implies a common patho-physiology".

Again the need for agreed diagnostic criteria followed by a formal epidemiological approach is highlighted.

I have questioned the philosophical approach to this problem but am not qualified to comment on the neurophysiology. However, the hypothesis proposed does explain the self sustaining pain cycle generally considered to be responsible for the prolonged course of this syndrome and is quite consistent with experience with the less severe cases seen by the writer.

REFERENCE

- Fry HJH Overuse syndrome and the overuse concept. In Bammer G (ed) *Discussion papers on the pathology of work-related neck and upper limb disorders and the implications for diagnosis and treatment*. NCEPH Working Paper Number 32 Canberra: National Centre for Epidemiology and Population Health, 1992.

Max Zusman M App Sci
Lecturer

Department of Physiotherapy
Curtin University of Technology
GPO Box U1987
Perth WA 6001

Cohen, Arroyo and Champion have proposed that the clinical syndrome known as RSI (here termed refractory cervicobrachial pain) is a centrally mediated neuropathic state. Spinal cord neurones are said to be sensitized

(rendered hyperresponsive) by a barrage of nociceptive input from one or more pathological peripheral tissues. As a consequence these neurones are able to pick up, encode as 'painful' and relay incoming

information that would normally be inappropriate and inaccessible to them in terms of both modality and connections. The relay may be to contralateral as well as ipsilateral sites at segmental, intersegmental and supraspinal levels of the central nervous system. In this way the central nervous system is believed to be capable of making a contribution to spontaneously occurring and mechanically provoked clinical signs and symptoms (Dubner, 1991; Zusman, 1992a).

That both the sensory and motor responses observed clinically for this syndrome might be, at least in part, accounted for by spinal cord neurone hyperresponsiveness is consistent with current thinking regarding the mechanisms of acute and chronic pain (Dubner, 1991; Woolf, 1991). There is an abundance of neurological and behavioural experimental evidence which implicates central nervous system mechanisms in the expression of, for example, hyperalgesia, allodynia, hyperpathia, after and referred pain. Recent evidence from Berberich et al (1989) also suggests a central basis for the frequently observed muscle weakness without wasting. Activity in gamma motoneurons, hence presumably spindle afferent drive to alpha motoneurons, was found to be reduced following a barrage of nociceptive input (Mense, 1991). Some clinical support for spinal cord neurone hyperresponsiveness with acute inflammatory and chronic neuropathic conditions has come from the area of post operative pain management (Cousins, 1991). Wall (1991) considers a central contribution to be a component with the peripheral nerve pathology hypothesis of RSI proposed by Quintner and Elvey (1991).

Clinical observations with their large sample of patients has led Cohen, Arroyo and Champion to conclude that RSI is characterised by secondary hyperalgesia. Centrally mediated secondary hyperalgesia is generally accepted as being referral of input from normal large diameter mechanoreceptive afferents which supply the healthy area surrounding some pathological site (Raja, Meyer and Campbell, 1988). Hyperresponsive nociceptive specific

and wide dynamic range spinal cord neurones are thought to either gain abnormal access and/or respond with an abnormal pattern, to such usually innocuously encoded information (Dubner, 1991; Yaksh, 1990). This being the case, perhaps the authors could have commented on the "apparent paradox" of clinically observed hypoesthesia which they attribute in the table to loss of (large) myelinated afferent fibre function. Also, several references to "ongoing" and "continuous" nociceptive (presumably small fibre) input confuses the exclusive role movement evoked large fibre input is proposed to play in the subsequent maintenance of central sensitization.

In essence the authors have used the RSI syndrome to discuss some current research and views for a central nervous system contribution to clinically observed symptoms and signs which accompany peripheral deep tissue (joint, muscle) and nerve pathology. On the other hand, the evidence presented does not completely exclude a significant peripheral basis for their origin with a syndrome such as RSI (Rappaport and Devor, 1990; Zusman, 1992b). Indeed the array of tissues proposed to be implicated - facet joints and surrounding structures, muscles, tendons, joint capsules of the upper limb, dorsal roots, dorsal root ganglia and peripheral nerves - is of little value in helping resolve the aetiological, diagnostic and therapeutic controversies which surround this syndrome.

The central nervous system probably does contribute to the spontaneously occurring and mechanically provoked sensory and motor responses observed with many peripheral deep tissue and nerve pathology syndromes. Moreover, in some instances and for whatever reason (? genetic factors, repeated triggering, neurotoxicity), peripheral input of any sort may not be essential for (although this may enhance) ongoing symptoms and signs (Devor and Raber, 1991; Dubner, 1991). Sustained abnormal activity in central neurones may in itself be sufficient for their maintenance.

At the present time direct confirmation and successful management of any central

contribution to chronic syndromes such as RSI await clarification of the proposed biochemical events believed responsible and the development of acceptable therapeutic strategies for their modification.

REFERENCES

- Berberich P, Hoheisel U, Mense S and Skeppar P (1987): Fine muscle afferent fibres and inflammation: changes in discharge behaviour and influence on gamma-motoneurons. In R F Schmidt, H-G Schaible and C Vahle-Hinz (Eds): *Fine Afferent Fibres and Pain*. Weinheim: VCH Verlagsgesellschaft. pp 165-175.
- Cousins M J (1991): Prevention of postoperative pain. In M R Bond, J E Charlton and C J Woolf (Eds): *Proceedings of the VIIth World Congress on Pain*. Amsterdam: Elsevier. pp 41-52.
- Devor M and Raber P (1991): Heritability of neuropathic pain: evidence from an experimental model. In M R Bond, J E Charlton and C J Woolf (Eds): *Proceedings of the VIIth World Congress on Pain*. Amsterdam: Elsevier. pp 343-347.
- Dubner R (1991): Neuronal plasticity and pain following peripheral tissue inflammation or nerve injury. In M R Bond, J E Charlton and C J Woolf (Eds): *Proceedings of the VIIth World Congress on Pain*. Amsterdam: Elsevier. pp 262-276.
- Mense S (1991): Considerations concerning the neurobiological basis for muscle pain. *Canadian Journal of Physiology and Pharmacology* 69: 610-616.
- Quintner J and Elvey R (1991): The neurogenic hypothesis of RSI. In G Bammer (Ed): *Discussion papers on the pathology of work-related neck and upper limb disorders and the implications for diagnosis and treatment*. NCEPH Working Paper Number 24 Canberra: National Centre for Epidemiology and Population Health pp 1-22.
- Raja S N, Meyer R A and Campbell J N (1988): Peripheral mechanisms of somatic pain. *Anaesthesiology* 68: 571-590.
- Rappaport Z H and Devor M (1990): Experimental pathophysiological correlates of clinical symptomatology in peripheral neuropathic pain syndromes. *Stereotactic Funct Neurosurg* 54-55: 90-95.
- Wall P D (1991): Commentary on Quintner J & Elvey R L: The Neurogenic Hypothesis of RSI. In G Bammer (Ed): *Discussion papers on the pathology of work-related neck and upper limb disorders and the implications for diagnosis and treatment*. NCEPH Working Paper Number 24 Canberra: National Centre for Epidemiology and Population Health p.50.
- Woolf C J (1991): Central mechanisms of acute pain. In M R Bond, J E Charlton and C J Woolf (Eds): *Proceedings of the VIIth World Congress on Pain*. Amsterdam: Elsevier. pp 25-34.
- Yaksh T L (1990): Pharmacological studies suggesting the role of spinal receptor systems in the anomalous encoding of high and low threshold cutaneous stimuli. In M R Dimitryevic and U Lindblom (Eds): *Altered Sensation and Pain. Recent Achievement in Restorative Neurology, vol 3*. Basel: Karger. pp 132-142.
- Zusman M (1992a): The central nervous system contribution to mechanically produced motor and sensory responses following peripheral tissue pathology. *Australian Journal of Physiotherapy*. In press.

Zusman M (1992b): Some neurologically based effects of mechanical stimulation of deep tissue. *Physical Therapy*. In press.

RESPONSE FROM M. COHEN

TO BESWICK AND CURSLEY

Generally supportive, although their objection to the term RCBP is spurious. RCBP does not claim to be a diagnostic term, in contradistinction to RSI. Furthermore we have been strenuous in defining the clinical syndrome denoted by the term RCBP, whereas they are content to include other "entities" such as tendonitis, etc. This aspect, fundamentally one of terminology, was addressed in our paper published in the Medical Journal of Australia in March this year (1) but not spelled out in the present contribution.

TO OWEN

Remarkable for its thinly veiled pejorative remarks which play the person and not the ball. Obviously Dr Owen has difficulty in coping with our attempts at precise terminology. Dr Owen also betrays ignorance of current thinking concerning pain ("...the patients' concept of pain...?").

TO PATKIN

His perspective of the "sociology of science" is indeed appropriate and we would freely admit the role of bias that comes from training, experience, etc. Again, our paper in the Medical Journal of Australia (1) paid appropriate respect to the sociopolitical influences on the RSI phenomenon (as well as supporting Dr Patkin's view of the unsatisfactory connotation of that term). We have in this discussion presented an alternative hypothesis based on neurophysiology which should be subject to all the strings and arrows of comment and criticism. At least it is testable.

TO QUINTNER

Dr Quintner's position in this debate is very close to our own. His detailed response is welcome, although we do not consider that tissue damage is necessarily primary. Dr Quintner also throws out a challenge to better explain the cutaneous hypoaesthesia which, incidentally, is always associated with deep tissue hyperaesthesia (including hyperalgesia), which again focusses on dorsal horn function. Testing of the proposed hypothesis will help to meet this challenge.

TO WIGLEY

Dr Wigley is concerned by possible circular reasoning which we have strenuously avoided (although not in as much detail here as elsewhere). In fact we did not exclude cases suggesting a neuropathic aetiology: it was precisely the recognition of these features which stimulated our thinking. We acknowledged that our cases are, by definition, refractory. If our hypothesis has any value, then cases may be identified at an earlier stage by specifically seeking the clinical phenomena as reported. Dr Wigley's epidemiologically inspired comments are relevant but his claim of our bias is not correct: we started with unexplained pain, in some cases of which disturbed neural functioning was suspected, not vice versa. The need for "agreed diagnostic criteria" is supported - but what is to be the starting point for the agreement? (Compare the so-called "fibromyalgia syndrome" argument.)

TO ZUSMAN

Again, generally supportive. It is true that peripheral contributions cannot be excluded, nor did we seek to do so. A close reading of our proposal reveals that RCBP may be the end result of nociceptive input from diverse sources, that is, a reflex phenomenon. It is difficult to agree with Zusman's conceptualisation of secondary hyperalgesia as "...being referral of input from normal large diameter afferents...": that is not a fair reference to Raja et al (2). Secondary hyperalgesia is characterised by enhanced response to mechanical stimuli to nociceptors and/or mechanoreceptors, thought to be due to altered dorsal horn stimulus-response functions. The fibres responsible for the alteration have not been confidently identified. The end result is that innocuous stimuli, as signalled by large-diameter afferents, are processed as noxious. Zusman is helpful in drawing attention to the potential role of muscle afferents.

REFERENCES

1. Cohen ML, Arroyo JF, Champion GD & Browne CD. In search of the pathogenesis of refractory cervicobrachial pain syndrome: a deconstruction of the RSI phenomenon *Med J Aust* 1992; **156**: 432-436.
2. Raja SN, Meyer RA, Campbell JN. Peripheral mechanisms of somatic pain. *Anaesthesiol*. 1988; **68**: 571-590.

National Centre for Epidemiology and Population Health

Working Paper Series

ISSN 1033-1557

WORKING PAPER NUMBER 1*

Noor A. Khalidi *Aboriginal mortality in Central Australia, 1975-77 to 1985-86: a comparative analysis of levels and trends*
April 1989 ISBN 0 7315 0801 7
Published in A. Gray (ed) *A Matter of Life and Death: Contemporary Aboriginal Mortality*, Aboriginal Studies Press, 1990 ISBN 0 85575 291 X

WORKING PAPER NUMBER 2*

Alan Gray *Discovering determinants of Australian Aboriginal population health*
May 1989 ISBN 0 7315 0629 4

WORKING PAPER NUMBER 3*

P.J. Solomon, J.A. Doust and S. R. Wilson *Predicting the course of AIDS in Australia and evaluating the effect of AZT: a first report*
June 1989 ISBN 0 7315 0660 X

WORKING PAPER NUMBER 4*

John Deeble *The Medicover proposals*
August 1989 ISBN 0 7315 0737 1

WORKING PAPER NUMBER 5

J.R.G. Butler *Intergovernmental relations and health care*
August 1989 ISBN 0 7315 0740 1
Published as 'Health Care' in B. Galligan, O. Hughes & C. Walsh (eds), *Intergovernmental Relations and Public Policy*, Allen & Unwin, Sydney, 1991, pp 163-89

WORKING PAPER NUMBER 6

Alan Gray *Aboriginal fertility: trends and prospects*
October 1989 ISBN 0 7315 0809 2
Published in Journal of the Australian Population Association, vol 7, 1 May 1990, pp 57-77

WORKING PAPER NUMBER 7

John McCallum *The public versus the policies: the ethical basis of Australian retirement income policy*
November 1989 ISBN 0 7315 0798 3

WORKING PAPER NUMBER 8

Noor A. Khalidi *Aboriginal fertility in Central Australia*
November 1989 ISBN 0 7315 0825 4

WORKING PAPER NUMBER 9

Dr John McCallum *The dynamics of community involvement in old age: the syndrome of underuse*
November 1989 ISBN 0 7315 0826

WORKING PAPER NUMBER 10

Dr John McCallum *Noncontributory pensions for developing countries: rehabilitating an old idea*
December 1989 ISBN 0 7315 0838 6

WORKING PAPER NUMBER 11

M. Mackisack, A.J. Dobson & C.R. Heathcote *Predicting the prevalence of a disease in a cohort at risk*
December 1989 ISBN 0 7315 0839 4

WORKING PAPER NUMBER 12

C.R. Heathcote, C. Keogh, T.J. O'Neill *The changing pattern of coronary heart disease in Australia*
December 1989 ISBN 0 7315 0840 8

WORKING PAPER NUMBER 13*

Alan Gray *Analysis of components of demographic change*
March 1990 ISBN 0 7315 0877 7
Published in *Mathematical Population Studies* 1991, 3:1, pp 21-38

WORKING PAPER NUMBER 14

J.R.G. Butler *Day surgery: cost-reducing technological change?*
April 1990 ISBN 0 7315 0882 3

WORKING PAPER NUMBER 15

Stephen J. Kunitz *Disease and the destruction of indigenous populations*
April 1990 ISBN 0 7315 0889 0

WORKING PAPER NUMBER 16*

P.J. Solomon, C. Fazekas de St Groth, S.R. Wilson *Projections of Acquired Immune Deficiency Syndrome in Australia using data to the end of September 1989*
April 1990 ISBN 0 7315 0890 4

WORKING PAPER NUMBER 17

Alison M. Hill, Robert M. Douglas *Fluoridation of public water supplies and public health: an old controversy revisited*
June 1990 ISBN 0 7315 0910 2

WORKING PAPER NUMBER 18*

J.R.G. Butler, C.M. Furnival, R.F.G. Hart *Do treatment costs vary by stage of detection of breast cancer?*
July 1990 ISBN 0 7315 1000 3

WORKING PAPER NUMBER 19*

Gabriele Bammer, Brian Martin *Repetition Strain Injury in Australia: Medical Knowledge and Social Movement*
August 1990 ISBN 0 7315 1095 X

WORKING PAPER NUMBER 20*

Gabriele Bammer *Occupational Disease and Social Struggle: The Case of Work-related Neck and Upper Limb Disorders*
August 1990 ISBN 0 7315 1096 8

WORKING PAPER NUMBER 21*

John McCallum *Australian Mandatory Retirement Challenged*
November 1990 ISBN 0 7315 1144 1

WORKING PAPER NUMBER 22*

P.J. Solomon & D.R. Cox *Nonlinear component of variance models*
January 1991 ISBN 0 7315 1148 4

WORKING PAPER NUMBER 23

J.R.G. Butler & R.F.G. Hart *A model for estimating the incremental cost of breast cancer screening programs*
February 1991 ISBN 0 7315 1169 7

WORKING PAPER NUMBER 24

J. Quintner & R. Elvey *The Neurogenic Hypothesis of RSI*. With 11 commentaries in G. Bammer (ed) *Discussion Papers on the Pathology of Work-Related Neck and Upper Limb Disorders and the Implications for Diagnosis and Treatment*.
May 1991 ISBN 0 7315 1197 2

WORKING PAPER NUMBER 25

S. Keiko and J. McCallum *The Means Test on the Japanese Kosei Nenkin Pension: Wages versus hours adjustment*
August 1991 ISBN 0 7315 1268.5

WORKING PAPER NUMBER 26

J.R.G. Butler and J.P. Smith *Tax expenditures on health in Australia: 1960-61 to 1988-89*
August 1991 ISBN 0 7315 1269 3

WORKING PAPER NUMBER 27

M.E. Jones and P.J. Solomon *Blood donation and Human Immunodeficiency Virus infection: Do new and regular donors present different risks?*
December 1991 ISBN 0 7315 1328 2

WORKING PAPER NUMBER 28*

J.R.G. Butler and Amanda L. Neil *Fiscal Stress and Health Policy in the A.C.T.*
December 1991 ISBN 0 7315 1329 0

WORKING PAPER NUMBER 29

P.J. Solomon, R.G. Attewell, E.B. Freeman and S.R. Wilson *AIDS in Australia: Reconstructing the epidemic from 1980 to 1990 and predicting future trends in HIV disease*
December 1991 ISBN 0 7315 1355 X

WORKING PAPER NUMBER 30*

S.J. Kunitz, M.G. Santow, R. Streatfield and T. de Craen *The health of populations on North Queensland Aboriginal Communities: change and continuity*
June 1992 ISBN 0 7315 1402 5

WORKING PAPER NUMBER 31

M. Cohen, J. Arroyo and D. Champion *The Relevance of Concepts of Hyperalgesia to "RSI". With 7 commentaries in G. Bammer (ed) Discussion Papers on the Pathology of Work-Related Neck and Upper Limb Disorders and the Implications for Diagnosis and Treatment.*
November 1992 ISBN 0 7315 1447 5

* out of print

Copies of Working Papers still in print may be obtained from:
The Publications Officer,
NCEPH, The Australian National University,
GPO Box 4, CANBERRA A.C.T. 2601, AUSTRALIA