Influences of posture and task on cognitive performance, physiological activity and negative affect

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DECLARATION

Other than where reference is made, this thesis is a report of my own original research.

Some results from Study 1 have been published as conference proceedings:

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ABSTRACT

Both electrophysiological and behavioural evidence indicate that there is greater cortical arousal in a more upright posture. Animal experiments suggest that this may be an effect of enhanced central noradrenergic system activity, stemming in turn from there being less baroreceptor activity when posture is closer to vertical. This thesis investigates the corollary that higher order psychological processes modulated by central noradrenaline are also modulated by posture. Recent studies demonstrate that central noradrenergic activity impairs the ability to solve anagrams. To test the hypothesis that a less upright posture would be more conducive to solving anagrams, 20 participants performed anagram and mental arithmetic tasks in both standing and supine conditions (Study 1). Supporting the hypothesis, anagrams were solved more rapidly in the supine condition; non-specific cognitive effects were excluded as mental arithmetic performance was unaffected by posture. Recordings of heart rate and blood pressure indicated greater physiological reactivity associated with mental arithmetic than with anagrams. This was reinforced by the findings of Study 2, in which 36 participants performed both problem types while seated. Generally greater physiological reactivity for mental arithmetic was not because this was more difficult or stressful than anagrams (as indicated by subjective ratings); rather, it may be associated with expending more mental effort. Theoretically, mental effort may increase central noradrenergic activity and (in keeping with Study 1) thus, paradoxically, be detrimental to solving anagrams. A positive correlation between blood pressure reactivity and self-reported stress was also found, which may have implications for a theory of learned hypertension (for which a stress reducing effect of blood pressure is critical). Central noradrenaline contributes to the development of psychological stress and anxiety; given greater central noradrenergic activity, it was anticipated that a stressor would generate more negative affect in a more upright posture. To investigate this, 20 participants provided ratings of

stress and anxiety, before and both immediately and ten minutes after performing a stressful mental arithmetic task in both standing and supine conditions (Study 3). In contrast to expectations, there was a trend for greater negative affect after performing mental arithmetic in the supine condition; this may have resulted from compensatory mental effort being required to overcome relatively low arousal in that condition (the presence of which was supported by lower skin conductance than when standing). However, in line with theoretical reasoning, the presence of anticipatory anxiety in the standing (though not the supine) condition supports the facilitation of negative affect in a more upright posture. Heart rate increased more during mental arithmetic in the supine condition; also of interest were time-related changes in physiological activity (possibly a Finapres device recording artefact for blood pressure). The major conclusion from these studies is that a simple change in posture is sufficient to significantly influence higher order psychological processes that are modulated by central noradrenaline, including certain cognitive abilities and negative affect. There are implications for experiments in which participants are supine (e.g., during brain scans) and for any other situation in which baroreceptor activity is altered (e.g., low gravity environments).

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CHAPTER 1

GENERAL INTRODUCTION

Thesis outline

More than one hundred years have passed since both William James (1884) and Carl Lange (1885/1912) proposed theories in which the physiological state of the body was afforded an important, if not critical, role in the production of emotional experience. Today, the idea of a relationship between bodily state and psychological processes remains extant. In fact, there is currently substantial interest (facilitated by brain scanning technology) in how feedback from the viscera and other internal structures may affect both emotion and cognition (recent reviews include Berntson, Sarter & Cacioppo, 2003; Cameron, 2001; Craig, 2002, 2003; also see Damasio, 2003).

This thesis explores relationships between bodily state and psychological processes, with one aspect on which there is a focus being how differences in posture may influence cognition and affect; the current chapter is largely an account of the theory and research pertinent to this idea. Briefly, due to gravity, changes in posture affect the distribution of blood throughout the body. This is detected by stretch-sensitive baroreceptors (located primarily in major arteries and the heart), whose signals to the central nervous system typically evoke a homeostatic mechanism that, via manipulating autonomic activity, maintains an adequate blood pressure. To illustrate, upon standing blood is drawn towards the lower body, this decreases the stretch of baroreceptors and thereby reduces their firing rates, in turn inducing compensatory increases in heart rate and vascular resistance. In addition to regulating this homeostatic blood pressure mechanism, baroreceptors are associated with extra-homeostatic effects that impact upon higher cortical centres. At least some of these extra-homeostatic effects could stem

from a modulation of central noradrenergic activity, which has been shown to be regulated by baroreceptors. This idea is consistent with the existence of a global sympathetic system that controls parallel changes in peripheral and central sympathetic activity.

Because of the extra-homeostatic effects of baroreceptor activity, it could be expected that changes in posture affect psychological processes. Indeed, the performance of some cognitive tasks has been shown to be influenced by posture; however, there are only a small number of relevant studies. Two of the studies in this thesis were conducted to expand upon the known psychological effects of posture, and specifically, to investigate the influence of posture on higher-order psychological processes. This was achieved in one study by comparing performance on an anagram task in different postures: standing and supine. Based on previous (non-postural) findings, it was hypothesised that the ability to solve anagrams would be greater when participants were supine than when they were standing.

The extra-homeostatic effects associated with baroreceptors may extend to a modulation of negative affect (such as psychological stress and anxiety); an increase in baroreceptor activity is thought to reduce negative affect. Implications of this idea were investigated in the second postural study of this thesis (Study 3), in which both the anxiety and psychological stress associated with performing a stressful cognitive task were compared between standing and supine conditions; it could be expected that because of greater baroreceptor activity, less negative affect would develop in a supine condition. A finding that either the affective response to mental stress, or the performance on an anagram task, differs between standing and supine conditions would support the idea that posture influences higher-order psychological processes and would contribute to

existing knowledge regarding the role of the peripheral body in regulating psychological phenomena.

In addition to implications for posture, the idea that baroreceptor activity reduces negative affect is important to a theory of learned hypertension, in which elevations in blood pressure are proposed to be reinforced by this rewarding effect (via a presumed rise in baroreceptor activity). The idea that baroreceptor activity reduces stress and anxiety was investigated in one of the postural studies of this thesis (mentioned above). In a further study, the relationship between changes in blood pressure (as distinct from imposed changes in baroreceptor activity) and psychological stress was investigated; this was achieved by correlating blood pressure reactivity during attempts to solve both mental arithmetic and anagram problems (while seated) with ratings of perceived stress that participants associated with these tasks (Study 2). In keeping with the theory of learned hypertension, it might be thought that larger blood pressure responses would be associated with less psychological stress.

Study 2 was also conducted in relation to another focus of this thesis, the neurophysiological mechanisms that influence and subserve cognitive processes, with particular attention given to these as they relate to solving anagrams. The motivation for this is that an understanding of the neurophysiology responsible for a cognitive process is critical for understanding how and why changes in bodily state (including posture) affect this process. In accordance with the idea of a global sympathetic system that co-ordinates central and peripheral sympathetic activity (e.g., in relation to posture), cues to the central mechanisms associated with a cognitive process may be provided by peripheral physiological (e.g., cardiovascular) recordings. For this reason, in Study 2 the physiological reactivity associated with performing anagrams was compared with the

physiological reactivity associated with performing mental arithmetic (which has dissimilar cognitive processing requirements to solving anagrams). This helped to expand upon existing knowledge regarding the mechanisms, both neurophysiological and psychological, that underlie the ability to solve anagrams, and in doing so complemented Study 1, in which modulation of this ability by a postural manipulation was investigated.

In broad terms, research into the relationships between bodily state and higher-order psychological processes has multiple applications. These extend to the development of theories of emotion and assisting to facilitate an understanding of psychological effects that may be associated with the diverse range of situations in which bodily state is altered, including low-gravity environments and chronic medical conditions such as hypertension.

Body posture, baroreceptors and the baroreflex[1]

As body posture becomes more upright (from lying down to sitting to standing, or by an increase in the degree to which head up tilt approaches vertical), gravity displaces blood towards the lower body; this reduces the central venous pool (which "corresponds roughly to the volume enclosed by the right atrium and the great veins in the thorax", Mohrman & Heller, 2003, p.146). A reduced central venous pool leads in turn to falls in stroke volume, cardiac output (the product of stroke volume and heart rate) and mean blood pressure (the product of cardiac output and total peripheral resistance). A significant fall in blood pressure could reduce brain perfusion sufficiently to induce unconsciousness. Normally however, adjustments in autonomic nervous system activity maintain a relatively constant mean blood pressure across a range of postural

[1] General references for this section are Harkin & Warltier (1995), Levick (2000), Mohrman & Heller (2003), Smith & Ebert (1990) and Wieling & Shepherd (1992).

conditions; this homeostatic mechanism is the baroreflex.

The baroreflex depends upon specialised receptors, baroreceptors, to provide information on blood pressure and blood volume to the central nervous system. There are primarily two types of baroreceptors, distinguished (in part) by their anatomical locations: arterial and cardiopulmonary. Arterial baroreceptors are present in the walls of the aorta (specifically in the aortic arch) and carotid sinuses (which are associated with the carotid arteries on either side of the neck). Rather than gauging blood pressure directly, arterial baroreceptors are sensitive to arterial stretch, which increases their activation. Thus, typically the firing rate of arterial baroreceptors increases when blood pressure and/or arterial stretch rises, and decreases when blood pressure and/or arterial stretch falls. Cardiopulmonary baroreceptors are dispersed predominantly throughout the atria and ventricles. Like their arterial counterparts they are stretch sensitive, responding to changes in heart chamber distension; firing rates are enhanced and reduced by increases and decreases in the central venous pool respectively.

As shown in Figure 1.1, baroreceptor afferents from the aortic arch travel in the aortic depressor nerve (not in all species, see Sapru, 1991) before joining those from cardiopulmonary sites in the vagus. From the carotid sinus, afferents are conveyed by the carotid sinus nerve, which joins the glossopharyngeal nerve. Regardless of their anatomical origins, baroreceptor afferents terminate at synapses in the nucleus tractus solitarius (NTS) of the medulla.

Within the NTS, information from baroreceptors is integrated with, or modulated by, signals from other sources. For example, as shown in rats, inputs from skeletal muscle to the NTS can reduce the saliency of arterial baroreceptor signals; this probably occurs

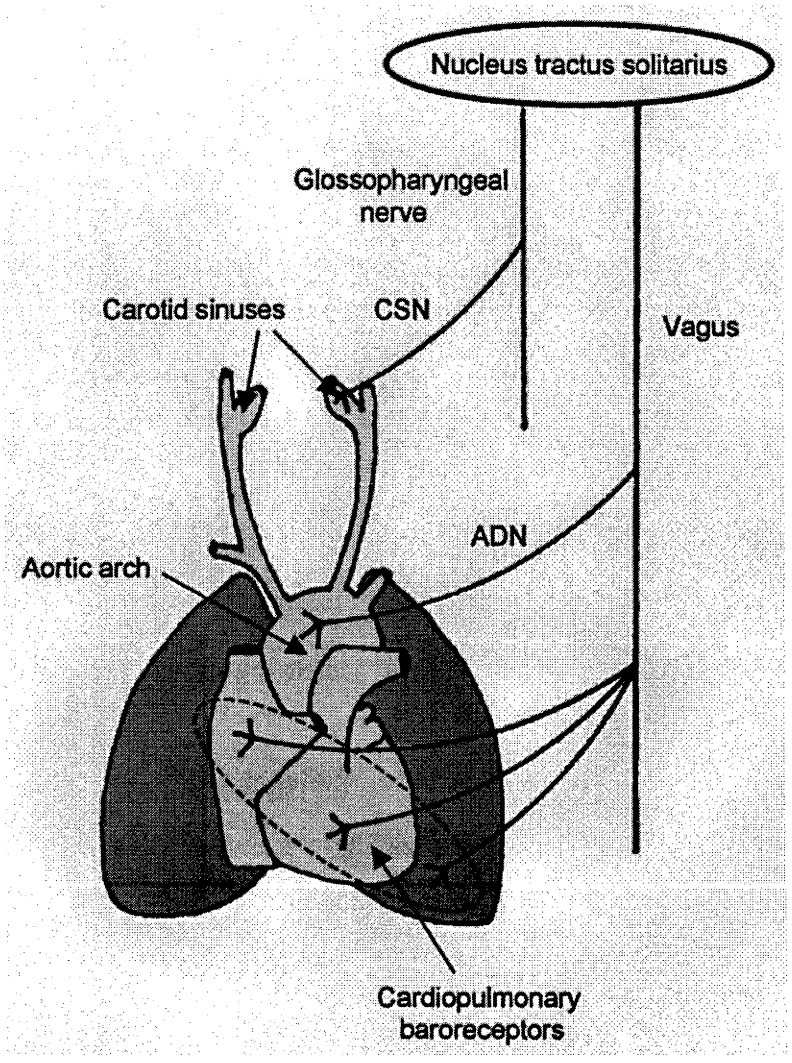

Figure 1.1. Baroreceptor locations and nerves conveying baroreceptor afferents to the nucleus tractus solitarius. CSN, carotid sinus nerve; ADN, aortic depressor nerve. Portion of the figure showing baroreceptor locations adapted from "*Neuro-cardiovascular regulation: from molecules to man*" by M. W. Chapleau & F. M. Abboud, 2001, Annals of the New York Academy of Sciences, 940, p. xvi.

when skeletal muscle is contracting heavily, as in during exercise (Potts et al., 2003). As shown in Figure 1.2, output from the NTS activates neurons in the caudal ventrolateral medulla (CVLM), which in turn provide inhibitory input to the rostral ventrolateral medulla (RVLM), a nucleus with sympathetic efferents. Cardiopulmonary baroreceptor information from the NTS appears to reach the RVLM by a route other than the CVLM, at least in rabbits (Shafton, Ryan, McGrath & Badoer, 1999); nevertheless, as with arterial baroreceptors, signals from cardiopulmonary baroreceptors inhibit the RVLM.

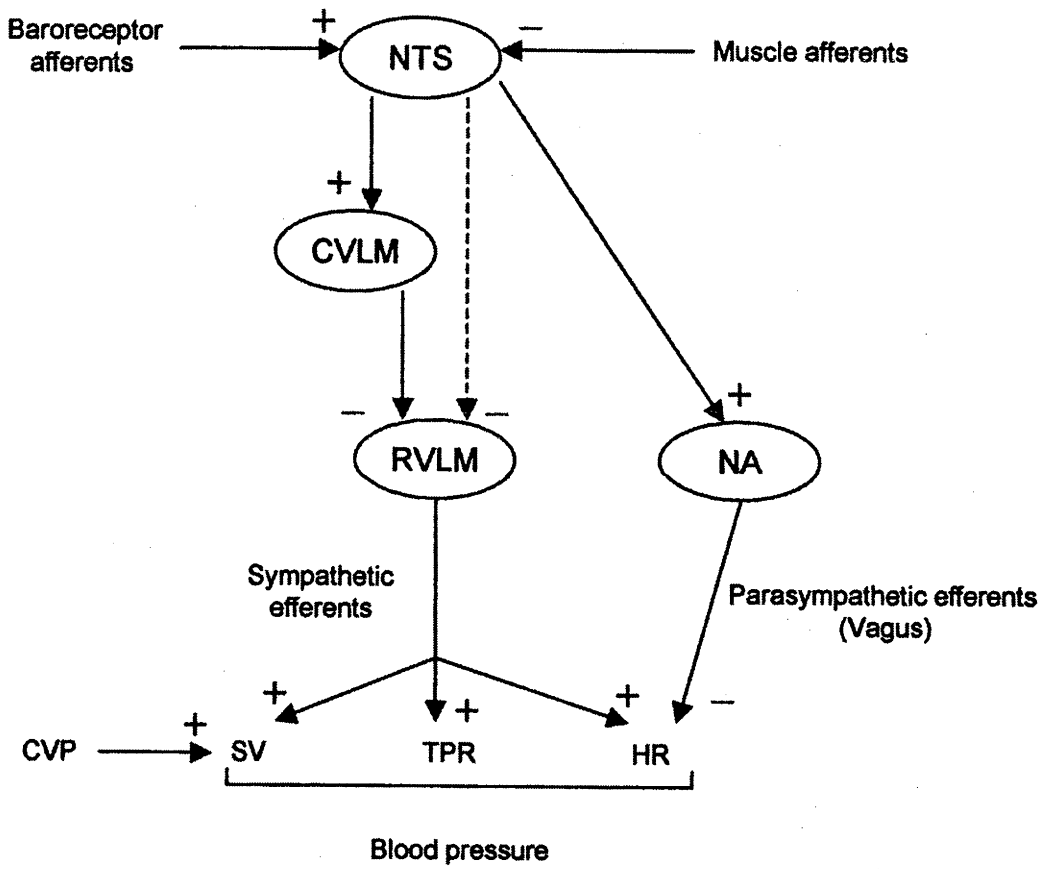

Figure 1.2. Central and peripheral pathways by which stroke volume (SV), total peripheral resistance (TPR) and heart rate (HR) are varied in the maintenance of blood pressure by the baroreflex. NTS, nucleus tractus solitarius; CVLM, caudal ventrolateral medulla; RVLM, rostral ventrolateral medulla; NA, nucleus ambiguus; CVP, central venous pool. Muscle afferents illustrate one of the non-baroreceptor inputs to the NTS.

The RVLM provides tonic activation of the sympathetic nervous system; therefore, NTS activity reduces sympathetic outflow. Information from the NTS is also sent to the nucleus ambiguus of the vagus, activation of which enhances parasympathetic outflow to the periphery. Consequently, the end result of an increase in baroreceptor activity (in the absence of competing inputs from other sources, such as skeletal muscle) is to reduce the prevailing level of sympathetic nervous system activity, while facilitating activity of the parasympathetic nervous system.

Changes in baroreceptor activity associated with changes in posture underlie the maintenance of similar mean blood pressure levels across postural conditions. When adopting a more upright posture (e.g., moving from supine to standing) the displacement of blood towards the lower extremities reduces the degree to which upper body arteries and the chambers of the heart are stretched or distended; this decreases the activity of both arterial and cardiopulmonary baroreceptors. Consequently, and via the central pathways outlined in Figure 1.2, there is less inhibition of sympathetic outflow to the vasculature and heart: the resultant vasoconstriction in skeletal muscle, splanchnic and renal beds increases total peripheral resistance, while increases in both heart rate (augmented by reduced vagal outflow) and contractility mitigate the initial fall in cardiac output (stroke volume and cardiac output typically remain lower than in the less upright condition however). While maintaining adequate mean blood pressure, these autonomic changes result in higher diastolic blood pressures with more upright postures; systolic blood pressure may decrease slightly or be left unchanged. Needless to say, opposite changes (i.e., decreases in heart rate, total peripheral resistance and diastolic blood pressure, and increases in stroke volume and cardiac output) are induced by moving to a less upright posture; these are the result of there being relatively more blood in the upper body and therefore relatively greater baroreceptor activity. However,

depending on the particular baroreceptor population, changes in activity may not last the duration for which the new posture is maintained. For example, while carotid baroreceptor activity undergoes fairly rapid adaptation and plays a part only initially, the autonomic effects produced by movement from sitting to supine are sustained by influences associated with an increase in the central venous pool (Pump et al., 2001).

There are mechanisms in addition to the direct autonomic changes (those mediated by sympathetic and parasympathetic efferents) that promote the maintenance of blood pressure in different postural conditions. In the standing position, rhythmic contractions of leg muscles (the skeletal muscle pump) enhance venous return, and thus reduce the decrease in the central venous pool. Furthermore, adopting a more upright posture induces changes in plasma concentrations of hormones (including renin, angiotensin, aldosterone, vasopressin and atrial natriuretic factor). These changes function to retain blood volume by decreasing the amount of water excreted by the kidneys and are particularly important in coping with a prolonged upright posture. Though acting slower than the more direct autonomic effects (heart rate etc.), postural differences in hormone levels are also a product of changes in baroreceptor activity and can be considered as part of the baroreflex (e.g., for vasopressin see Pump et al., 1999).

Extra-homeostatic effects of baroreceptor activity

It has been known for many years that baroreceptor activity has extra-homeostatic effects, which Rau and Elbert (2001) define as "effects that are not related primarily to the baroreceptor reflex and therefore are not closely connected to the homeostasis of blood pressure" (p.188). Koch (as cited in Rau & Elbert) observed in 1932 that, in addition to cardiovascular effects, carotid baroreceptor stimulation induced sleep in dogs. This is consistent with other studies in animals, which have generally indicated

that baroreceptor activity reduces cortical arousal (reviewed by Lacey, 1967; Vaitl & Gruppe, 1991). In humans, initial investigations into extra-homeostatic effects of baroreceptor activity sought changes in arousal or sensorimotor performance that were time-locked to the cardiac cycle (due to changes in arterial stretch across the cycle: minimal during diastole and maximal during systole) (Lacey).

There is electrophysiological evidence that the cardiac cycle is associated with changes in cortical arousal or excitability that are consistent with an inhibitory effect of baroreceptor activity. Walker and Sandman (1982) found that visual evoked responses were smaller during systole than during diastole at a right occipital site. Furthermore, a shift to lower frequency activity in the EEG was reported to occur at the vertex during the cycle's period of maximal baroreceptor activity (Koriath & Lindholm, 1986); this particular study also found that the reaction time for deciding whether a digit was odd or even was not affected by the cardiac cycle. Conversely, others have found sensorimotor measures to vary in association with the cardiac cycle. For example, Sandman, McCanne, Kaiser and Diamond (1977) reported that visual perception was relatively enhanced when stimuli were presented during the P-wave of the cardiac cycle (when arterial stretch is close to the minimum of the cycle); however, this relationship was found for only one of three stimulus presentation rates. On the whole, findings relating the cardiac cycle to reaction time and sensory perception have been mixed; consequently the technique has not yielded clear-cut conclusions regarding the effects of baroreceptor activity on sensorimotor performance (see Vaitl & Gruppe, 1991).

Rather than relying on the cardiac cycle for changes in baroreceptor activity, other studies have actively stimulated carotid baroreceptors via negative pressure (suction) in a cuff placed around the neck (this increases carotid sinus stretch). Elbert, Rockstroh,

Lutzenberger, Kessler and Pietrowsky (1988) found that there was less slow brain potential negativity (that develops in anticipation of an imperative stimulus) during neck suction than during neck pressure (which deactivates carotid baroreceptors by decreasing carotid sinus stretch). This is the typical finding of neck cuff studies, and indicates a reduction in cortical arousal/excitability during carotid baroreceptor stimulation (see Rau & Elbert, 2001). This conclusion is further supported by Mini, Rau, Montoya, Palomba and Birbaumer (1995), who in addition to finding lower cortical negativity, reported smaller skin conductance responses to painful stimuli delivered during neck suction, compared with neck pressure (skin conductance is accepted as an indicator of central nervous system arousal, e.g., Barry et al., 2004).

As well as effects on cortical arousal, Mini et al. (1995) observed that baroreceptor stimulation diminished sensitivity to pain; a similar effect was reported for hypertensives by Elbert et al. (1988). In fact, numerous studies have demonstrated that neck suction reduces sensitivity to pain (reviewed by Rau & Elbert, 2001). Consistent with this, sensitivity to pain has been found to be lower during systole than during diastole (Edwards, McIntyre, Carroll, Ring & Martin, 2002). Furthermore, D'Antono, Ditto, Sita and Miller (2000) reported that increasing venous return (by raising the legs while in the supine position), and thus enhancing cardiopulmonary baroreceptor activity, produced a slight increase in pain tolerance in participants with high systolic blood pressure. Supporting the findings from human studies, animal data has also demonstrated that both arterial and cardiopulmonary baroreceptor activity reduces sensitivity to painful stimuli (see Randich & Maixner, 1984).

Both animal and human studies have shown that the stimulation of baroreceptors leads to a reduction in cortical arousal; sensitivity to painful stimuli is also affected by

baroreceptor activity. These phenomena are not necessary components of the baroreflex, that is, they do not directly participate in blood pressure maintenance, and are therefore considered extra-homeostatic effects of baroreceptor activity. The similarities between human and animal findings indicate that animal models are useful for understanding the physiological processes underlying the extra-homeostatic effects of baroreceptor activity in humans.

The effects of posture on cortical arousal

Posture influences the distribution of blood throughout the body, and thereby influences baroreceptor activity; thus, it could be expected that the effects on cortical arousal and sensitivity to pain shown to be associated with baroreceptor activity will also be associated with particular postures. In support of this, Shimoda and Ikuta (2000) observed that for participants to perceive an electrical stimulus applied to the skin, a higher current was needed in the horizontal position as compared to 70° head-up tilt (electrical stimulation activates nerve fibres normally associated with pain). However, rather than sensitivity to sensory or painful stimuli, it is cortical arousal that has been the focus of most postural modulation studies (though not all of these have given consideration to potential baroreceptor mechanisms).

EEG measures have been used to study the effects of posture on cortical arousal, with effects reported for all of the traditional frequency bands (alpha, beta, theta, delta). Ivanova (1988) observed that tilting the upper body to 70° upright from supine increased EEG power recorded from sites across the scalp, both in the alpha and beta bands; this result was thought to reflect an increase in cerebral metabolic rate in the more upright condition. Also compared with a supine condition, whole body head-up tilt to 40° was found by Cole (1989) to be associated with more beta band activity at left

and right central recording sites; other spectral bands or scalp sites were not investigated. The greater beta band activity is likely to reflect higher cortical arousal in the more upright position, especially given the additional observation of longer sleep onset latencies in participants when tilted (which, in turn, is consistent with anecdotal reports that when tired it is easier to remain awake in a more upright posture, e.g., Bonnet, 2000).

Other studies have found effects of posture on the EEG in different spectral bands. Vaitl and Gruppe (1990, 1992) compared EEG activity obtained while participants were tilted upright, to 30° or 45°, with while they were either supine or tilted 6° head-down (head-down tilt increases the central venous pool, with 6° often used to simulate weightlessness as at this angle body fluids are fairly evenly distributed, see Vaitl, Gruppe, Stark & Possel, 1996). Low frequency EEG activity, particularly in the theta band at an occipital site (1990) and bilaterally at both frontal and parietal sites (1992), was found to be higher in the supine or head down position than in the upright tilt conditions; tilt angle did not affect power in any other spectral band. Similar postural differences in EEG were also observed for small differences in tilt angle, 6° head-down and 6° head-up, however these effects were relatively slow to develop and thought to be associated more with the prolonged nature of the conditions (23 hours were spent in each) rather than acute effects of posture (Vaitl et al., 1996).

Increases in theta power at scalp sites that overlap with, or are in proximity to, those in the Vaitl and Gruppe (1990, 1992) studies have been found to be related to decreases in arousal. For example, theta power measured at bilateral central and occipital sites was directly related to subjective measures of sleepiness (Lafrance & Dumont, 2000), and increases in theta power (at the vertex and midway between midline parietal and

occipital sites) corresponded with decreased performance on a vigilance task (Makeig & Jung, 1996). Thus, differences in theta power between postural conditions suggest that a less upright posture is associated with less cortical arousal. This is supported by Caldwell, Prazinko and Hall (2000), who reported that increases in theta (and delta) band activity (at the vertex and both frontal and parietal midline sites) associated with sleep deprivation were attenuated by standing (from the seated position); a similar effect of posture was found for the theta band in sleep-deprived participants performing a vigilance task (Caldwell, Prazinko & Caldwell, 2003).

Though effects of posture on the EEG have been found at sites across the scalp, there are differences in the particular spectral bands for which effects have been observed. This could, at least in part, be due to differences in whether participants had their eyes open (Caldwell et al., 2003; Vaitl & Gruppe, 1990, 1992) or closed (Cole, 1989; Ivanova, 1988) when recordings were made. It has been long known (e.g., Cram, Kohlenberg & Singer, 1977) that the state of the eyes affects EEG recordings, with eyes-open and eyes-closed conditions forming part of a routine clinical EEG examination (Gevins, 1987). Given this, it is not surprising that Caldwell et al. (2000) found that the effects of posture on the EEG were influenced by whether participants' eyes were open or closed (e.g., differences in theta activity between standing and sitting were greater when compared in the eyes closed condition). Further studies are required if interactive effects of posture and eye state on EEG recordings are to be clarified.

In addition to EEG studies, evoked potential responses have been used to investigate the effects of posture on the central nervous system. Wei, Yan and Guan (1992) compared evoked responses during the performance of cognitive tasks in both a 45° head-up tilt condition and a head-down tilt condition (either 10° or 15°); the results supported an

inhibition of brain function during head-down tilt. This conclusion was reinforced by the group's follow up studies, in which the amplitude of evoked responses to target stimuli of a visual attention task were lower during 10° head-down tilt compared with 20° head-up tilt (Wei et al., 1995, 1998).

Both EEG and evoked potential studies indicate that cortical arousal is relatively reduced in a less upright posture. For EEG data, the most relevant findings were in the theta spectral band. In addition to subjective indicators of reduced arousal, theta band activity (measured along the midline and at bilateral frontal, temporal and occipital sites) has been found to be directly related to reaction time, with both measures increasing over time on a vigilance task (Paus et al., 1997). It would therefore be expected that a less upright posture is associated with longer reaction times; indeed, there is some evidence to support this.

Vercruyssen and Simonton (1994) have summarised a series of studies in which the effect of postural manipulations on simple and choice visual reaction times were investigated: standing and sitting (Vercruyssen, Cann & Hancock, 1989; Vercruyssen et al., 1988); standing, sitting and supine (Cann, 1990; Woods, Vercruyssen & Birren, 1993). Postural effects on reaction time interacted with the many independent variables (pertaining to both test and participant characteristics) of these studies. However, each study found reaction times to be fastest while standing in at least a subgroup of participants. In only one situation, and in the context of a complex interaction, was the reverse effect observed; reaction times were faster while supine than while standing for elderly subjects performing a four choice task with both degraded stimuli and low stimulus-response compatibility (Cann). This is likely to be a detrimental result of the relatively high amount of attention that elderly adults require to maintain an upright

posture, as demonstrated by studies investigating the role of cognitive resources in maintaining balance (either while static or walking, Woollacott & Shumway-Cook, 2002).

Some dual balance and cognitive task studies have compared reaction times obtained in the seated and standing positions. Lajoie, Teasdale, Bard and Fleury (1993) found verbal reaction times to an auditory stimulus to be faster when participants were sitting compared with when they were standing; however, with only six subjects, and therefore the potential for sampling error, this result should be treated cautiously. Indeed, Teasdale, Bard, LaRue and Fleury (1993) found no differences in reaction time between sitting and standing. In a further study, while reaction times to an auditory stimulus seem to have been faster for elderly adults when seated, it is not clear whether there was any difference between standing and sitting for young adults (Marsh & Geel, 2000). At the least, the results of these studies ostensibly suggest that reaction time is not faster while standing than while seated. However, unlike the studies in which a more upright posture was generally associated with faster reaction times (Vercruyssen & Simonton, 1994), in the dual-task studies the participants' primary task was to maintain their balance as stable as they could; the reaction time task was treated as secondary to this. The additional cognitive requirements of consciously focussing on posture while standing (cf. seated) could result in a slowing of reaction time in this posture that reduces effects associated with increased arousal. Indeed, in a study outlined earlier (Caldwell et al., 2003), and in which balance requirements were not stressed, the development of slower reaction times (and increased lapses) on a vigilance task during sleep deprivation was more prominent when participants were seated than when they were standing.

Experimental evidence suggests that reaction time is typically faster while standing than while supine or seated, provided that task performance is not secondary to consciously focussing on maintaining balance while standing. Findings for naturalistic postures are extended by Vaitl et al. (1996), who found that auditory reaction times during 6° head-up tilt were faster than those during 6° head-down tilt. Given the inverse relationship between arousal and reaction time (Paus et al., 1997), the difference in reaction times between different postural conditions suggests that there is greater cortical arousal in a more upright posture. This is consistent with the conclusions drawn from studies in which the effects of posture on EEG recordings and evoked potential responses were investigated.

Posture affects the distribution of blood throughout the body, and consequently influences baroreceptor activity. Therefore, given that baroreceptor activity modulates cortical arousal, the demonstrated effects of posture on cortical arousal could be mediated by baroreceptor activity; there is experimental evidence to support this idea. Lower body compression, or the application of positive pressure to the legs, displaces blood towards the upper body and thereby increases baroreceptor activity. Cole (1989) found that the EEG changes produced by tilting participants towards upright from supine were attenuated by the application of positive pressure to the legs. Consistent with this, Vaitl and Gruppe (1990) observed that applying lower body compression to head-up tilted participants produced a small increase in the amount of theta power in the EEG. However, in contrast, compression while supine decreased theta power, ostensibly suggesting an increase in cortical arousal with baroreceptor stimulation. Steps were taken to minimise any pain or discomfort associated with the lower body pressure suit; nevertheless, it is possible that the experience of lower body compression may be psychologically arousing and thus reduce the effects of baroreceptor mediated cortical

inhibition (this may be more apparent when arousal is already low, i.e., in the supine position).

The locus coeruleus and the noradrenergic arousal system

Cortical arousal can be modulated by many neurotransmitter systems, including the noradrenergic, dopaminergic, serotoninergic and cholinergic (Robbins, 1997). At the core of the noradrenergic system is the locus coeruleus, a nucleus in the pons that is the origin of nearly all noradrenaline in the central nervous system, and which has extensive connections to both subcortical and cortical regions (Moore & Bloom, 1979). Some of these connections facilitate a role for the locus coeruleus in cardiovascular regulation (for a review see Philippu, 1988). More important here are the cortical and behavioural effects of locus coeruleus activity.

In anaesthetised rats, electrical stimulation of the locus coeruleus has been shown to be associated with an increased release of noradrenaline in the medial prefrontal cortex (Florin-Lechner, Druhan, Aston-Jones & Valentino, 1996). Also in anaesthetised rats, Berridge and Foote (1991) demonstrated that chemical stimulation of the locus coeruleus induced cortical activation, as shown by a change in frontal cortex EEG recordings from low to high frequency activity. Furthermore, Curtis, Lechner, Pavcovich and Valentino (1997) showed that both noradrenaline release and EEG activation (a reduction in the power of low frequency activity) increased in the prefrontal cortex of anaesthetised rats when locus coeruleus discharge rates were elevated by chemical stimulation.

In cats, direct recordings from the locus coeruleus have shown that the activity of this nucleus varies in a direct relationship with cortical arousal levels (as indicated by

behavioural state and verified by EEG measures): locus coeruleus activity was higher during waking than during sleep (Hobson, McCarley & Wyzinski, 1975) and higher during periods of active waking than quiet waking (Rasmussen, Morilak & Jacobs, 1986). Similar findings were reported for both rats and monkeys by Foote, Aston-Jones and Bloom (1980) and have been supported by additional studies in each of these animals; rats (Aston-Jones & Bloom, 1981a, b); monkeys (e.g., Grant, Aston-Jones & Redmond, 1988).

Evidence directly linking locus coeruleus activity to cortical and behavioural effects in humans is more difficult to obtain than in animals. However, there are a few reports concerning electrical stimulation of the locus coeruleus in patients with intractable epilepsy or spasticity; stimulating electrodes were implanted with the intent of alleviating these medical conditions. Brain noradrenaline turnover is associated with spillover of the noradrenaline metabolite 3-methoxy-4-hydroxyphenethyleneglycol (MHPG) into the jugular vein (Maas, Hattox, Landis & Roth, 1977). Libet and Gleason (1994) reported that electrical stimulation of the locus coeruleus (in two patients) induced an increase in the concentration of MHPG in blood from the jugular vein, thus indicating an increase in central noradrenaline turnover. In a patient with epilepsy, Faber and Vladyka (1983) observed a shift in the EEG (recorded from seven sites) from predominantly theta band activity (with periods of delta and epileptic activity) to alpha activity with electrical stimulation of the locus coeruleus. Kaitin et al. (1986) reported that, in a patient with spasticity, nights on which the locus coeruleus was electrically stimulated were associated with frequent arousals from sleep and more time awake than on nights in which stimulation did not occur.

The findings of locus coeruleus stimulation studies in humans (though limited to a small

number of observations) are consistent with those conducted in cats, rats and monkeys; together they indicate that locus coeruleus activity is directly associated with cortical noradrenaline turnover and both EEG and behavioural indicators of cortical arousal. Accordingly, the locus coeruleus can be seen as the core nucleus of a noradrenergic arousal system (Berridge & Waterhouse, 2003).

Baroreceptor activity modulates the noradrenergic arousal system

There have been numerous investigations into the effects of baroreceptor activity on the locus coeruleus; initially this was in relation to baroreflex-modulated changes in autonomic activity, though other broader effects (cortical noradrenaline turnover, EEG activity and behaviour) have received considerable attention. There are anatomical bases by which baroreceptor activity may affect the noradrenergic arousal system; this includes a connection from the nucleus tractus solitarius (NTS; which, as described earlier, is the primary target for baroreceptor afferents) to the locus coeruleus via the nucleus paragigantocellularis (Van Bockstaele & Aston-Jones, 1995). A monosynaptic connection from the NTS to the locus coeruleus has also been demonstrated in rats (Van Bockstaele, Peoples & Telegan, 1999), and in the suncus (a type of shrew, Ito & Seki, 1998). Baroreceptor activity has frequently been manipulated by controlling blood volume (and thereby the central venous pool); an increase in activity produced by creating a hypervolemic state with blood volume loading (injections of donor blood or substitute), or a decrease in activity by producing a hypovolemic/hypotensive state by blood withdrawal or administration of a systemic vasodilator, typically nitroprusside.

Blood volume loading has been shown to inhibit locus coeruleus firing rates in anaesthetised rats (Elam, Yao, Svensson & Thoren, 1984; Murase, Inui & Nosaka, 1994; Svensson & Thoren, 1979) and conscious cats (Morilak, Fornal & Jacobs, 1987).

This effect was both sensitive to very small changes in blood volume and long lasting (for at least the 10 to 15 minutes that recordings could be maintained, Elam et al.; Svensson & Thoren). Evidence for the involvement of baroreceptors in the modulation of locus coeruleus activity includes the findings that decreases in firing rates associated with blood volume loading were abolished by severing the vagus (Svensson & Thoren) and attenuated by denervation of the aortic depressor nerve (Murase et al.). Also, while blood pressure elevations (pharmacologically induced, e.g., with the systemic vasoconstrictor phenylephrine) inhibited locus coeruleus firing rates in rats with all but aortic baroreceptor afferents denervated, the inhibitory effect was lost once these were also transected (Murase et al.). Furthermore, direct stimulation of both the vagus (Takigawa & Mogenson, 1977) and aortic depressor nerve (Murase et al.) has been shown to inhibit the locus coeruleus. These findings suggest that cardiopulmonary, aortic and carotid baroreceptors all appear to modulate locus coeruleus activity (despite mixed results regarding their relative contributions, see Murase et al.). In contrast to the effects of hypervolemia, a long lasting increase in locus coeruleus firing rates has been observed during blood withdrawal in anaesthetised rats (Elam, Svensson & Thoren, 1985). Consistent with this, locus coeruleus activity has reported to be enhanced during inferior vena cava constriction (which reduces venous return) in anaesthetised cats (Ward, Lefcourt & Gann, 1980), by carotid artery occlusion (which reduces carotid sinus pressure, Murase et al.), and during nitroprusside administration in both anaesthetised (Murase et al.; Valentino & Wehby, 1988) and conscious rats (Curtis, Drolet & Valentino, 1993).

Singewald & Philippu (1993) found (in anaesthetised cats) that increases in locus coeruleus firing rates during nitroprusside administration were highly correlated with noradrenaline release within the locus coeruleus. Because of this finding, there being an

increase in noradrenaline release in the region of the locus coeruleus during the administration of nitroprusside in both anaesthetised (Kawahara, Kawahara & Westerink, 1999; Singewald, Kaehler & Philippu, 1999) and conscious (Kawahara et al.) rats further supports an increase in locus coeruleus activity when baroreceptor activity is decreased. Conversely, an increase in blood pressure (produced by phenylephrine) reduced the release of noradrenaline within the locus coeruleus; this effect was both abolished by transection of the vagus and aortic depressor nerve, and replicated by electrical stimulation of these same nerves (Schneider, Singewald & Philippu, 1995). Changes in noradrenaline turnover during blood volume/pressure manipulations have been demonstrated not only within the locus coeruleus, but also in cortical regions innervated by this nucleus. Persson and Svensson (1981) found that blood volume load and blood withdrawal were associated with decreases and increases in noradrenaline release respectively in the neocortex and cerebellum of rats. In addition to increased noradrenaline turnover in the locus coeruleus (see above), Kawahara et al. found that systemic nitroprusside administration increased the release of noradrenaline in the medial prefrontal cortex of both anaesthetised and conscious rats; a similar result was reported by Swiergiel, Palamarchouk, Smagin and Dunn (1998) in anaesthetised rats.

Cortical effects of blood volume/pressure manipulations have also been demonstrated with EEG data. Valentino, Page and Curtis (1991) found a temporal association between increases in locus coeruleus activity and high frequency EEG activity in the frontal cortex during nitroprusside administration in rats. In a further study, these effects were abolished by chemical inactivation of the locus coeruleus, suggesting its necessity for increased EEG activity during hypotensive stress (Page, Berridge, Foote & Valentino, 1993). The effects of systemic vasodilators on the EEG have also been

reported for humans receiving either sodium nitroprusside or trimetaphan (also spelt trimethaphan; vasodilation is produced by ganglionic blockade and may be augmented by the release of histamine, T. C. Westfall, 1990) to induce controlled hypotension while anaesthetised for surgery (Thomas, Cole, Etherington, Prior & Stefansson, 1985). Group increases in EEG activity associated with reductions in blood pressure were not reported; in fact, EEG activity decreased once mean blood pressure fell to 57 mmHg. However, this cannot be interpreted as a failure of locus coeruleus activity to increase in response to hypotensive stress in humans because propranolol was administered to prevent tachycardia. The central beta-adrenoceptor antagonism produced by propranolol has been shown to block the excitatory effects of locus coeruleus activity on the EEG (Berridge & Foote, 1991). More consistent with the animal research, the spillover of noradrenaline and noradrenaline metabolites into the internal jugular vein has been found to rise during both trimethaphan and adrenaline (also causing systemic vasodilation) administration in humans, with the locus coeruleus cited as a likely source of this increase (Lambert et al., 1998).

There is compelling evidence that baroreceptor activity modulates the noradrenergic arousal system. An increase in baroreceptor activity (e.g., produced by blood volume loading) decreases locus coeruleus firing rates; accordingly, cortical noradrenaline turnover is also diminished. Conversely, a decrease in baroreceptor activity (e.g., produced by systemic vasodilation) increases locus coeruleus activity, cortical noradrenaline turnover and EEG indicators of cortical arousal. Persson and Svensson (1981) and Svensson and Thoren (1979) have proposed that the central effects of a decrease in baroreceptor activity may function as an alerting mechanism in response to blood loss.

Behavioural phenomena modulated by the noradrenergic arousal system: effects of baroreceptor activity

In addition to the changes in locus coeruleus activity, cortical noradrenaline turnover and EEG indicators of arousal, there is evidence that behavioural phenomena influenced by the noradrenergic arousal system are modulated by baroreceptor activity. Persson and Svensson (1981) observed that the locomotor behaviour of rats was decreased by blood volume load and increased by blood withdrawal: These changes were reported to correspond with previous research into the effects of cortical noradrenaline on locomotor behaviour.

Central noradrenergic function can be altered by drugs acting at alpha$_2$-adrenoceptors; these are located pre-synaptically on noradrenergic nerve terminals and their stimulation inhibits the release of noradrenaline (Fleming & Robinson, 1990). Alpha$_2$-adrenoceptors are also present post-synaptically within the locus coeruleus (Lee, Rosin & Van Bockstaele, 1998). It has been shown in rats that the alpha$_2$-adrenoceptor agonist clonidine decreases both locus coeruleus firing rates (Svensson, Bunney & Aghajanian, 1975) and cortical noradrenergic activity (Astier et al., 2003; Pudovkina, Kawahara, de Vries & Westerink, 2001). Consistent with this inhibitory effect on the central noradrenergic arousal system, alpha$_2$-adrenoceptor agonists are known to produce sedation (i.e., reduce arousal) in humans (e.g., Hall, Uhrich, Barney, Arain & Ebert, 2000; Hall, Uhrich & Ebert, 2001). The inhibition of the locus coeruleus produced by clonidine is reversed by yohimbine, an alpha$_2$-adrenoceptor antagonist (Svensson & Usdin, 1978).

Both clonidine and yohimbine have been used to demonstrate that the amplitude of the acoustic startle eyeblink reflex is directly related to central noradrenergic activity in

humans: it is enhanced by yohimbine (Morgan et al., 1993) and reduced by clonidine (Kumari, Cotter, Corr, Gray & Checkley, 1996). Rau (cited in Rau & Elbert, 2001) reported that carotid baroreceptor stimulation (using a neck cuff) significantly reduced the amplitude of the acoustic startle eyeblink reflex. In addition, Commissaris and Davis (1983) found, in rats, that the systemic vasodilator hydralazine potentiated the response to acoustic startle (which was measured in terms of the cage displacement associated with whole body movement rather than an eyeblink reflex). These findings are consistent with changes in baroreceptor activity being associated with inverse changes in central noradrenaline turnover.

The amplitude of the acoustic startle eyeblink reflex is enhanced in situations where anxiety is elevated, as for example, during anticipation of receiving an electric shock (Grillon, Ameli, Woods, Merikangas & Davis, 1991). This is not surprising given that anxiety is associated with central noradrenaline turnover; for example, yohimbine can increase the subjective experience of anxiety in humans (e.g., Morgan et al., 1993). White and Depue (1999) have established that trait anxiety is related to central noradrenergic activity, with pupil diameter used as a measure of this: pupil diameter is increased by yohimbine and decreased by clonidine (Phillips, Szabadi & Bradshaw, 2000), and has also been shown to reflect locus coeruleus activity (Rajkowski, Kubiak & Aston-Jones, cited in Gilzenrat, Cohen, Rajkowski & Aston-Jones, 2003). Furthermore, in patients with the anxiety disorder post-traumatic stress syndrome, cerebrospinal levels of noradrenaline are higher than in controls, indicating a greater level of central noradrenergic activity (Geracioti et al., 2001). Given that cortical noradrenaline turnover is directly related to locus coeruleus activity, it could be expected that electrical stimulation of the locus coeruleus would increase feelings of anxiety. However, this was not found in three patients (with electrodes implanted to

treat either epilepsy or spasticity) tested by Libet and Gleason (1994); the authors interpreted their findings as evidence against a relationship between locus coeruleus activity and anxiety in humans. An effect of stimulation on the patients' medical conditions casts doubt on this conclusion. For example, in the patient with spasticity, locus coeruleus stimulation "was accompanied by an observable reduction in his spasticity, which otherwise produced muscle discomfort and even pain" (Libet & Gleason, p. 179): the potential for alleviation of such a severe medical condition to have dominated the patient's psychological state was not considered. Indeed, in monkeys, electrical stimulation of the locus coeruleus has been shown to produce behaviours indicative of anxiety (Redmond, Huang, Snyder & Maas, 1976). On the whole, there is a large body of evidence from both animal and human studies indicating that anxiety (e.g., Geracioti et al.; Morgan et al.; White & Depue; for reviews see Bremner, Krystal, Southwick & Charney, 1996a, b; Tanaka, Yoshida, Emoto & Ishii, 2000), and the related aversive state of psychological stress (for reviews see Stanford, 1995; Van Bockstaele, Bajic, Proudfit & Valentino, 2001), are intimately associated with, and/or facilitated by, increases in locus coeruleus activity and central noradrenaline turnover.

Given that anxiety and psychological stress are associated with increases in locus coeruleus activity and central noradrenaline turnover, which in turn are both inversely modulated by baroreceptor activity, it seems reasonable to think that anxiety and/or stress may be reduced by an increase in baroreceptor activity, and enhanced by a decrease in baroreceptor activity. Indeed, Svensson and Thoren (1979) have suggested that an increase in locus coeruleus activity in response to blood loss (via a decrease in baroreceptor activity) might underlie the apprehensiveness, restlessness and anxiety associated with hemorrhagic shock. However, beyond this comment, the potential for baroreceptor activity to affect anxiety via modulation of the locus coeruleus does not

appear to have been considered. Nevertheless, and without this theoretical construct, behavioural observations have led others to claim that baroreceptor activity reduces anxiety (Dworkin et al., 1994).

Dworkin, Filewich, Miller, Craigmyle and Pickering (1979) trained rats to avoid or terminate an aversive stimulus (electrical stimulation of the sensory nucleus of the trigeminal nerve) by running on a treadmill. Subsequently, in both stimulation and extinction (no stimulation) trials, rats ran less on the treadmill when their blood pressure was elevated by phenylephrine (vs. saline control); this effect was abolished in a group with denervated arterial baroreceptors. For the stimulation trials, these findings are consistent with baroreceptor activity reducing sensitivity to pain (an effect detailed earlier). For the extinction trials, there being less running when blood pressure was elevated in the absence of an aversive stimulus has been interpreted as indicating that anxiety was also reduced (Dworkin, 1988; Dworkin et al., 1994), though the reasoning behind this was not made explicit. A related experiment was reported by Szekely, Koo and Adam (1963), who conditioned rats to open a door leading to food upon presentation of a sound or light (positive conditioned stimulus). Carotid afferents were then destroyed unilaterally in one group. Subsequently, "neurosis" was induced in both deafferented and control rats by electrically shocking them when they fed, once a day for ten days. During this period, deafferented rats appeared to take longer than control rats to open the door to food in response to the positive conditioned stimulus (statistical analyses were not reported). It was concluded that the presence of carotid afferents was associated with the development of a less severe neurotic condition. Though these studies in rats are thought to support the notion that baroreceptor activity reduces anxiety, neither really seems to provide evidence of a clarity sufficient to justify this.

Other studies into the relationship between baroreceptor activity (or blood pressure) and negative affect have been conducted in relation to a theory that attempts to explain the development of hypertension in some people. Apart from specific medical conditions that promote hypertension (e.g., renal disease and endocrine disorders), the aetiology of hypertension in individual cases is generally not known, though is likely to include a combination of genetic and environmental factors, such as obesity, alcohol, salt, size at birth, and psychosocial stress (Braunwald & Williams, 1987; Isles, 2000). The theory of learned hypertension (also known as the baroreceptor reinforcement hypothesis) is an attempt to explain how one of these factors (stress) could lead to the development of hypertension in genetically predisposed people; the theory posits that a reduction in cortical arousal associated with an increase in blood pressure mitigates the psychological impact of exposure to stress, and that the rewarding nature of these blood pressure elevations may lead to the development of chronic hypertension in some cases (Dworkin 1988). There is some evidence that could be seen to be consistent with this idea: both Suter, Maire, Holtz and Vetter (1997) and Winkleby, Ragland and Syme (1988) have found an inverse association between resting blood pressure and self-reported stress. In a review of studies on the relationship between stressor exposure and blood pressure in hypertensives, Nyklicek, Vingerhoets and Van Heck (1996) clarified an influence of whether the measure of exposure used was objective or subjective, which typically yielded positive and negative associations respectively; altered perceptions of stressful stimuli brought about by central inhibiting effects of baroreceptor activity (supposedly increased in relation to the hypertensive state) was one of the factors suggested to account for the negative association between subjective measures of exposure to stress and blood pressure. Subsequently, the same group investigated whether sensitivity to acute psychological stressors was affected by resting blood pressure levels (Nyklicek, Vingerhoets & Van Heck, 2001). Both unmedicated

hypertensives and normotensive controls performed a number of tasks (e.g., mental arithmetic, watching films depicting stressful scenes) with ratings obtained for pre-stressor anxiety (self-reported tension) and post-stressor appraisals (how unpleasant, stressful etc. the task was). Hypertensive women reported less pre-stressor anxiety and less negative appraisals of the films than controls.

Consistent with the idea that high blood pressure may be associated with cortical dampening effects, the basal firing rate of neurons in the locus coeruleus has been found to be reduced in hypertensive rats compared with normotensive controls (Olpe et al., 1985). Though this could be seen to be consistent with an extra-homeostatic effect of elevated baroreceptor activity, there are reasons for thinking that baroreceptor activity is not elevated in hypertension. Baroreceptors have been found to be less sensitive than normal in people with hypertension, as demonstrated by a smaller than normal decrease in heart rate (baroreflex) in response to an increase in systolic blood pressure (e.g., Bristow, Honour, Pickering, Sleight & Smyth, 1969). Furthermore, baroreceptors are reset to maintain a higher blood pressure level in hypertension; to illustrate, the threshold for baroreceptor firing has been shown to be lowered in parallel with a lowering of blood pressure (during pharmacological treatment of hypertensive rats, Ichikawa et al., 1995). Thus, a hypertensive state cannot be used as an indicator of greater than normal baroreceptor activity, and therefore extra-homeostatic effects of baroreceptor activity cannot be assumed to be especially engaged in association with hypertension. In keeping with this conclusion, the subcortical noradrenaline turnover (measured from the internal jugular vein, and of which the locus coeruleus may be an important source) was found to be higher in patients with hypertension than in normotensive controls (Ferrier et al., 1993). Nevertheless, there is some evidence for inhibited cortical functioning in hypertensives, as demonstrated by delayed evoked

responses to auditory stimuli compared with normotensive controls (Tandon & Joon, 1997). However, decreased cortical arousal has also been found in people with tonically reduced blood pressure (i.e., hypotension), as demonstrated by slower reaction times and smaller brain potentials (Weisz, Schandry, Jacobs, Mialet & Duschek, 2002). These findings are the opposite of those that would be expected were blood pressure lowered by exogenous means (e.g., based on the production of higher cortical arousal during the administration of nitroprusside in rats, Valentino et al., 1991), and may be the result of having hypersensitive baroreceptors (Weisz et al.).

The potential interpretative complications of resting blood pressure levels and extra-homeostatic effects of baroreceptor activity were avoided in a study into aspects of the theory of leaned hypertension by Schweizer, Roth and Elbert (1991). In that study, self-reports of stress and anxiety were obtained before and after the performance of a mental arithmetic task by normotensive participants administered one of atenolol (a hydrophilic beta-blocker), metoprolol (a lipophilic beta-blocker) or placebo. The perceived stress response to mental arithmetic was greater in the atenolol condition than in either the metoprolol or placebo conditions; anxiety followed the same pattern though did not quite reach conventional levels of significance. Primarily due to differences in systolic blood pressure reactivity, pulse pressure increased during the task in the placebo group though not in either beta-blocker group. Assuming a direct relationship between pulse pressure and baroreceptor activity the authors concluded that the increased stress in the atenolol group was due to the drug dampening the task-associated rise in pulse pressure and thereby preventing the same level of baroreceptor stimulation as in the placebo condition; this peripheral effect was thought to be offset by central beta-blockade in the metoprolol group.

There is evidence that the magnitude of the acoustic startle response is inversely related to baroreceptor activity; given that startle is facilitated by central noradrenaline this is consistent with findings that an increase in baroreceptor activity reduces, and a decrease in baroreceptor activity enhances, activity of the noradrenergic arousal system. Because of their association with locus coeruleus activity and cortical noradrenaline turnover, it could be expected that baroreceptor activity reduces anxiety and psychological stress. While such an effect has been claimed, the evidence for this in animals is not clear, and the predominantly associative evidence from humans with hypertension is complicated by interpretative difficulties regarding the degree of baroreceptor activity associated with the condition. Nevertheless, the idea that baroreceptor activity decreases anxiety and psychological stress remains an important aspect to the theory of learned hypertension.

Posture, the noradrenergic arousal system and a global sympathetic system

In addition to the homeostatic maintenance of blood pressure by the baroreflex, baroreceptor activity is associated with extra-homeostatic effects (covered in detail earlier). One of these effects manifests as an inverse relationship between baroreceptor activity and cortical arousal; evidence suggests that this relationship may be modulated by the locus coeruleus, the nucleus at the core of a noradrenergic arousal system (Berridge & Waterhouse, 2003). Baroreceptor activity is decreased (e.g., Mohrman & Heller, 2003), and cortical arousal is increased (e.g., Cole, 1989), in a more upright posture: this is consistent with locus coeruleus (and therefore central noradrenergic) activity being greater in a more upright posture. The notion of a relationship between posture and locus coeruleus activity is not new: given that (in rats) only small changes in blood volume are needed to alter firing rates, it was previously suggested that the distribution of blood associated with reclining may inhibit the locus coeruleus (Elam et

al., 1985; Svensson, 1987) and thereby reduce arousal in that posture (Elam et al., 1984).

The parallel changes in central noradrenergic and peripheral sympathetic nervous system activity that occur in relation to changes in baroreceptor activity, and that are thought to occur in relation to changes in posture (e.g., Elam et al., 1984), also occur in a wide variety of other situations. For example, Elam, Svensson and Thoren (1986) have shown, in rats, that both noxious mechanical and thermal stimulation produce parallel increases in locus coeruleus and peripheral sympathetic activity (as measured in terms of blood pressure, heart rate and sympathetic nerve firing rates); in fact the correlation coefficient for the relationship between percentage changes in sympathetic nerve activity and locus coeruleus activity during thermal stimulation was 0.994. Co-variation between locus coeruleus activity and peripheral sympathetic nerve activity has also been found across a range of behavioural states, including active/quiet waking and slow wave/paradoxical sleep, in cats (Reiner, 1986).

The close relationship between central and peripheral sympathetic activity demonstrated in animals is supported by human experiments, in which central sympathetic activity (subcortical noradrenaline turnover) is determined from the spillover of noradrenaline and its metabolites into the internal jugular vein; as already mentioned, the locus coeruleus is thought to be an important source of this spillover (e.g., Ferrier et al., 1993; Lambert et al., 1998). Using this method, central sympathetic activity has been shown to be correlated with muscle sympathetic nerve activity, which in turn co-varies with heart rate and blood pressure (as determined for resting values across healthy participants, Lambert et al., 1997). Moreover, a positive correlation between subcortical noradrenaline turnover and each of cardiac (Esler et al., 2002) and whole body (Ferrier

et al.) noradrenaline spillover in healthy participants has been reported, while Lambert et al. (1995) found that a greater than normal level of cardiac noradrenaline spillover was accompanied by a greater than normal level of central noradrenaline spillover in patients with heart failure. Lastly, central noradrenergic activity has been shown to be reduced in line with a reduction in peripheral sympathetic activity produced by clonidine, to increase in parallel with muscle sympathetic nerve activity following systemic vasodilation induced by adrenaline (Lambert et al., 1998) and to be greater in patients with hypertension than in normotensive controls (Ferrier et al.).

Findings that central and peripheral sympathetic activity change in parallel, as evidenced in both animal and human studies, is in accordance with the idea that the locus coeruleus acts "as the cognitive limb of a *global* sympathetic system" (Aston-Jones, Valentino, Van Bockstaele & Meyerson, 1994, p. 35). Activation of this global sympathetic system can be seen to prepare an organism for interaction with the environment, or to enable it for coping with a stressor, as at the same time that peripheral sympathetic activity is increasing in readiness for physical activity, the central noradrenergic system is providing an appropriate psychological state, one consistent with heightened arousal and alertness (Aston-Jones et al.).

Applying the principle of a global sympathetic system to posture can be seen to support the idea of greater locus coeruleus activity when more upright (e.g., Elam et al., 1984), as this would be expected to occur in parallel with the increase in peripheral sympathetic activity that develops under these conditions (e.g., Morhman & Heller, 2003). While the peripheral changes work to maintain blood pressure in the face of gravitational challenge, a concomitant change in activity of the noradrenergic arousal system would appear to provide a psychological state appropriate for body posture:

alertness when upright and likely to be interacting with the environment. Conversely, a decrease in locus coeruleus activity when lying down would be expected to be accompanied by relative sedation, an effect that may facilitate relaxation or sleep in that posture. These ideas are consistent with reports detailed earlier; for example, demonstrations that cortical arousal is lower when supine than when tilted towards upright (e.g., Cole, 1989), it being easier to remain awake when in a more upright posture (Bonnet, 2000), and findings of both faster reaction times (Vercruyssen & Simonton, 1994) and improved vigilance (Caldwell et al. 2003) when standing (cf. seated and/or supine).

Evidence for parallel changes in central (locus coeruleus) and peripheral sympathetic activity supports the notion that there is greater locus coeruleus activity and noradrenergically mediated arousal in a more upright posture; this can be thought of as just one example in which a global sympathetic system operates.

Posture and processes modulated by the noradrenergic arousal system

It is thought that a more upright posture entails greater activity of the locus coeruleus and thus of the noradrenergic arousal system (e.g., Elam et al., 1984), an idea supported in principle by the operation of a global sympathetic system (Aston-Jones et al., 1994). In addition to the changes in gross arousal that have been demonstrated (e.g., Cole, 1989), this line of reasoning leads to the prediction that psychological processes modulated by central noradrenergic activity should be influenced by the degree to which body posture deviates from upright.

As outlined earlier, both the amplitude of the acoustic startle reflex (e.g., Morgan et al., 1993) and anxiety (e.g., Tanaka et al., 2000) are directly related to central noradrenergic

activity, thus it could be expected that these processes will also be affected by posture. Vaitl et al. (1996) compared the amplitude of acoustic startle eyeblink reflexes obtained in 6° head-down and 6° head-up tilt conditions. Measurements were taken in morning, afternoon and evening sessions (participants spent 23 hours in each tilt condition). The startle amplitude was found to be lower (though not significantly so) in the head-down condition than in the head-up condition for both the afternoon and evening sessions. However, for the morning session, the startle amplitude was greater in the head-down condition than in the head-up condition. While this finding does not concur with the notion of higher levels of central noradrenergic activity in a more upright posture, it should be interpreted cautiously given that it was not consistent with data from the afternoon and evening sessions.

Hennig et al. (2000) reported on an investigation into the effects of posture (standing, sitting and supine) on salivary cortisol levels. Participants completed questionnaires on emotional states for each posture to determine whether this factor could account for any postural differences in cortisol; questionnaires contained thirteen items, one of which measured the presence of anxiety on a zero to six scale. In the analyses, anxiety was the only item for which a main effect of postural condition was found. However, pairwise comparisons were not reported, and even so, the results would likely be uninformative; this is because anxiety was generally very low (the highest mean being 0.6), and rather than during each postural condition, it was reported that participants completed questionnaires while seated (i.e., after having completed the postural conditions). A similar situation occurred in a study by Garvin, Trine and Morgan (2001), in which participants underwent one of three relaxation techniques (quiet rest, hypnosis, autogenic relaxation) in both supine and seated sessions. All three techniques reduced state anxiety, with no difference in the size of this effect between the supine and seated

conditions; however, participants rated their anxiety five minutes after the postural condition had been completed, meaning that any effects of posture per se on state anxiety remain unclear.

The results regarding the effects of posture on both the acoustic startle eyeblink reflex and anxiety are somewhat ambiguous with regard to whether or not processes modulated by the noradrenergic arousal system are also modulated by posture. Another process affected by central noradrenergic activity is vigilance (or attention): Rajkowski, Kubiak, Ivanova and Aston-Jones (1998) have shown, in monkeys, that performance on a vigilance task increases in direct relation to tonic locus coeruleus activity (unless activity is so high as to be associated with a hyperactive behavioural state). Monkeys were trained to release a lever when presented with a target visual stimulus (and to not respond if the stimulus was non-target); they made fewer responses to target stimuli when tonic locus coeruleus activity was low (reflected behaviourally as drowsiness) than when at moderate levels. This is consistent with a general role for the locus coeruleus and central noradrenergic system in increasing both attention and the cortical responsiveness to sensory (environmental) stimuli (Berridge & Waterhouse, 2003). As discussed above, Caldwell et al. (2003) found that both reaction time and lapses on a vigilance task were reduced when sleep-deprived participants were standing compared with when they were seated. In fact, there being faster reaction times to sensory stimuli while standing than while seated or supine (as also discussed above, e.g., Vercruyssen & Simonton, 1994) could be considered as support for increased vigilance in a more upright posture, given that behavioural latencies to stimuli are an index of vigilance (Rajkowski, Kubiak & Aston-Jones, 1994). Findings that vigilance is improved when standing support that idea that psychological processes modulated by the noradrenergic arousal system are also modulated by posture.

Posture and higher-order psychological processes: investigations past and present

Studies were outlined above in relation to an argument for an effect of posture on psychological processes that are modulated by central noradrenergic activity. There are reports in addition to these that were conducted with the intent of investigating whether posture influences psychological processes. Under conditions of sensory deprivation, participants who were supine reported experiencing a greater number of hallucinations than those who were sitting (Morgan & Bakan, 1965). Furthermore, Berdach and Bakan (1967) saw psychotherapeutic implications in their finding that participants who were lying down related more childhood memories than those who were sitting. More closely aligned with the nature of studies performed in this thesis, Schulman and Shontz (1971) reported that participants who were sitting upright solved more convergent thinking problems (enclosed box problems from the Stanford-Binet Intelligence Scale) than participants who were either standing (with feet apart and hands on hips) or lying down; worst performance was by the participants who were lying down. Other aspects of cognition were also investigated by Schulman and Shontz: there were no differences between the groups of participants tested in different postures in terms of memory for words, or in scores on the "Uses for a brick" test, in which participants are asked to provide as many uses for a brick that they can think of.

While the effects of posture on some higher-order psychological processes have been investigated previously, this was done in the absence of a solid theoretical rationale. For example, Schulman and Shontz (1971) did not consider any physiological mechanisms for the differences in convergent thinking problem scores between the groups of participants tested in different postures, rather, they suggested that best performance in the seated participants may have been related to this posture being the one in which people normally take tests. In this thesis, the psychological processes used to investigate

the effects of posture were chosen with consideration of how their underlying neurophysiological mechanisms may be modulated by a postural manipulation.

An effect of posture has been shown for performance on a vigilance task (Caldwell et al., 2003), supporting the idea that behaviours modulated by central noradrenergic activity are also modulated by posture. Despite this, it remains a largely theoretical notion that psychological processes (higher-order psychological processes in particular) modulated by (or associated with) the noradrenergic arousal system are influenced by the degree to which body posture deviates from upright; this is addressed by two studies in this thesis. In one of these studies the phenomena investigated are psychological stress and anxiety; as explained, the development of both of these is related to central noradrenergic activity (e.g., anxiety: Tanaka et al., 2000; stress: Stanford, 1995). The postural conditions employed were standing and supine; these were chosen so as to produce conditions that differed considerably in their deviation from upright (and therefore were expected to differ considerably in relation to baroreceptor activity and the extra-homeostatic effects thereof). Participants were asked to provide subjective ratings of stress and anxiety before and after performing a stressful mental arithmetic task. However, in the first of the studies into the influence of posture on higher-order psychological processes it is an aspect of cognition shown to be affected by central noradrenergic activity that is of interest. As explained in more detail in Chapter 2, this pertains to the ability to solve anagram word puzzles, which were presented to participants in both standing and supine conditions.

CHAPTER 2

STUDY 1

Introduction

Noradrenergic modulation of anagram task performance

As outlined in Chapter 1, central noradrenergic activity increases cortical arousal (e.g., Curtis et al., 1997), which in turn is associated with faster reaction times (Paus et al., 1997). Along similar lines, locus coeruleus activity has been shown to be directly related to performance on a vigilance task (e.g., Rajkowski et al., 1998). However, in contrast to these effects, there is evidence that central noradrenergic activity impairs the ability to unscramble anagram word problems.

Beversdorf, Hughes, Steinberg, Lewis and Heilman (1999) administered propranolol, ephedrine (a sympathomimetic that acts indirectly by promoting noradrenaline release, see Stitzel & Robinson, 1990) and a placebo to 18 participants on three separate occasions. On each of these occasions the participants made attempts to solve three types of problem: number series, spatial rearrangement and anagrams. Initial analyses revealed no difference in solution latencies (or the number of problems correctly solved) between the three drug conditions for any problem type. However, to avoid a floor effect for the spatial and anagram problems, an analysis was conducted using only the data from participants with solution latencies below the group average. While not affecting the spatial task results, the mean anagram solution latency was less for the propranolol condition than for the ephedrine condition in the seven fastest participants (though there was no difference in the number of anagrams solved). Because both propranolol and ephedrine can cross the blood brain barrier (being lipophilic), the relative contributions of central and peripheral processes in modulating performance on

the anagram task were unclear. This was addressed in a second study (Beversdorf, White, Chever, Hughes & Bornstein, 2002) in which another 18 participants attempted to solve anagrams in three drug conditions: propranolol, nadolol (a hydrophilic beta-adrenoreceptor antagonist) and placebo. On average, anagrams were solved more rapidly in the propranolol condition than in the nadolol condition; there was no difference between either drug condition and placebo. Given that propranolol antagonises both central and peripheral beta-adrenergic receptors, while nadolol is limited to peripheral actions (being unable to cross the blood brain barrier) it was concluded that the cognitive processes used in solving anagrams are modulated by a central influence of the noradrenergic system; implying that endogenous central noradrenergic activity compromises the ability to solve anagrams.

Walker, Liston, Hobson and Stickgold (2002) have also investigated the effects of central aminergic (noradrenergic and dopaminergic) states on anagram task performance. Their study utilised findings indicating that both wakefulness and non-rapid eye movement (NREM) sleep are associated with greater levels of central aminergic activity than rapid eye movement (REM) sleep; 16 participants attempted to solve anagrams in an awake condition, and immediately after being awoken from both REM and NREM sleep (the neurophysiological conditions associated with the preceding sleep state were expected to persist during problem solving attempts). Participants solved a greater number of anagrams following awakening from REM sleep than from NREM sleep (though there was no difference in solution latencies between these conditions). However, there was no difference in either the number of problems solved, or anagram solution latencies, between the awake and REM sleep conditions. The authors considered neurophysiological differences other than central aminergic activity as likely to contribute to this outcome, which in addition to the potential for

non-specific effects associated with being awoken to perform a cognitive task, make the NREM condition the better to compare the REM condition with. Therefore, the difference in anagram task performance between REM and NREM conditions can be viewed as consistent with the idea that activity of the noradrenergic arousal system impairs the ability to solve anagrams.

There is another line of evidence to support the Beversdorf et al. (1999, 2002) and Walker et al. (2002) findings. As outlined in Chapter 1, there is an association between trait anxiety and central noradrenergic activity (White & Depue, 1999). A high level of trait anxiety (as measured by personality questionnaires) has been reported to be associated with a reduced ability to solve anagrams (cf. controls) by Zarontonello, Slaymaker, Johnson and Petzel (1984); a similar detrimental effect of trait anxiety has been found by others when anagrams did not represent words from a single conceptual category (Dey, 1978), and when attempts at solution were made in the presence of a confederate (Tomasini, 1973). Findings that trait anxiety is associated with reduced performance on an anagram task are consistent with the idea that central noradrenergic activity impairs the ability to solve anagrams.

Beversdorf et al. (1999, 2002) suggested that noradrenergic activity might impair the ability to solve anagrams via modulation of cognitive flexibility. There being less writing in the participants' test booklets for anagrams than for other problem types led Beversdorf et al. (1999) to conclude that cognitive flexibility (rather than trial and error or algorithmic strategies) was required to solve anagrams, an idea that the authors cited findings by Kounios and Smith (1995) and Smith and Kounios (1996) as being consistent with, and which they thought might involve cognitive flexibility facilitating the search of a network containing possible solutions. Noradrenaline reduces the signal-to-noise ratio of cortical neurons (Hasselmo, Linster, Patil, Ma & Cekic, 1997); it was suggested that this could compromise cognitive flexibility and thereby impair the ability

to solve anagrams (Beversdorf et al.).

Does posture modulate the ability to solve anagrams?

As reviewed in Chapter 1, there is evidence suggesting that noradrenergic arousal system activity increases as posture becomes more upright (e.g., Elam et al., 1984). In parallel with this, compared to supine and/or seated conditions, reaction times have been shown to be reduced (Vercruyssen & Simonton, 1994), and vigilance shown to be enhanced (during sleep deprivation, Caldwell et al., 2003), when participants are standing. However, rather than being facilitated, it has been demonstrated that the ability to solve anagrams is compromised by central noradrenergic activity (e.g., Beversdorf et al., 2002). Thus, unlike for reaction time and vigilance tasks, it could be expected that participants will exhibit better performance on an anagram task while supine than while standing; this hypothesis is tested in the current study.

Though some other problem types were used in the Beversdorf et al. (1999) study, anagrams were the only problems presented in both the Beversdorf et al. (2002) and Walker et al. (2002) experiments; this makes it uncertain whether cognitive processes specifically required for solving anagrams, or more general cognitive processes, were being manipulated. Walker et al. suggested that the similarities in anagram solution rates across their experimental conditions indicated that general cognitive processing speed was unaffected. However, solution latencies are an inappropriate control for more general cognitive effects given that this was the data type with which Beversdorf et al. reached similar conclusions to Walker et al. regarding the effects of noradrenergic (or aminergic) activity on anagram task performance. In the current study, both anagrams and mental arithmetic problems were presented in supine and standing conditions; mental arithmetic was included to determine whether any effects on cognition were particular to solving anagrams. Specifically, mental arithmetic was chosen as it

represents a problem type that utilises cognitive processes different to those needed for solving anagrams; for example, while solving anagrams may involve a search of a possible solution network (Beversdorf et al., 1999), mental arithmetic problem solutions are produced by a logical step-by-step process (Furst & Hitch, 2000).

Finding that the ability to solve anagrams is better when supine than when standing would support the idea that psychological processes modulated by the noradrenergic arousal system are also modulated by posture; this would expand upon existing knowledge regarding how peripheral bodily activity regulates psychological phenomena. Furthermore, an influence of posture on performance of the anagram task would help to clarify or support the neurophysiological processes involved in solving anagrams that have been previously proposed. This is likely to have application in understanding elements of creative thinking that share the processes used to solve anagrams.

Methods

Participants

Twenty undergraduate students (13 females) with a mean age of 18.9 years (range 18 to 24 years) participated in the study; all provided informed consent for the procedures, which were approved by the Australian National University Human Research Ethics Committee. Course credit was received for time spent.

Stimuli

A 32-item pool was developed for both anagrams and mental arithmetic problems (see Appendix A for item lists). Anagrams could be rearranged to form a five-letter word.

Five-letter anagrams were used in the study by Walker et al. (2002), while Beversdorf et al. (1999, 2002) presented participants with a mixture of five, six and seven-letter anagrams. Previous research has shown that anagram solution latencies are related to letter length: problems with four or less letters are typically solved fairly easily; in contrast, those with six or more letters are typically quite difficult to solve (Kaplan & Carvellas, 1968). In the current study, five-letter anagrams were chosen because a task of intermediate difficulty was thought likely to facilitate any difference in performance between the standing and supine conditions. Each anagram problem in the current study had a different letter order, with no more than one letter remaining in its original position and consecutive letters in the original word not ordered consecutively in the anagram. While an effort was made to develop anagram problems with only one solution, there was an unintended exception: "hsgot", for which the anticipated solution was "ghost" though the response "goths" was provided by three subjects (and accepted as an alternate solution). Mental arithmetic problems contained three operations (50% + – +, 50% – + –) with two-digit integers, presented on a single line followed by an equals sign (e.g., "46 - 21 + 13 - 16 = "); the solution was a two-digit integer. The mental arithmetic task was structured so as to emphasise sequential cognitive processing. An additional two problems of each type were used for practice trials. Stimuli were in black lower case Tahoma font (21mm high) against a white background, displayed on a monitor using Inquisit 1.33 (Millisecond Software, Seattle, WA).

Physiological measures

Autonomic activity was measured so as to demonstrate the magnitude of baroreflex-induced differences between supine and standing under resting conditions. Data was also recorded during problem solving periods in both postural conditions; this was in

light of previous research in which physiological reactivity to mental stress in different postural conditions has been investigated (e.g., Rusch, Shepherd, Webb & Vanhoutte, 1981). Measures of systolic blood pressure, diastolic blood pressure and heart rate were obtained at a rate of 1 Hz with a Finapres 2300 Blood Pressure Monitor (Ohmeda, Madison, WI). An appropriately sized Finapres cuff was placed on the middle finger of the non-preferred hand; for facilitating accurate blood pressure readings the cuff was maintained at heart level during recording (see Imholz, Wieling, van Montfrans & Wesseling, 1998). Skin conductance was sampled at 10 Hz using a μmhos 2701 (Bioderm, Morro Bay, CA); silver-silver/chloride electrodes, 8 mm in diameter and filled with a sodium chloride in glycerol electrolyte medium, were attached to the palmar surface of the index and fourth medial phalanges of the non-preferred hand. Respiration was monitored with a stretch-sensitive belt (Pro-Tech, Mukilteo, WA), worn around the lower rib cage, and sampled at 10 Hz; data was converted to breathing frequency traces off-line using Chart 4 (ADInstruments, Colorado Springs, CO). Physiological signals were digitised (if required) and recorded on a PC with Labview 6.1 (National Instruments, Austin, TX).

Procedure

For each participant, and separately for anagrams and mental arithmetic problems, the 32 pool items were randomly distributed into two 16-item blocks (one per postural condition); this was moderated so individual items appeared equally in both postural conditions across all participants.

As a standardising procedure for skin conductance recordings, participants washed their hands with soap and water (Venables & Christie, 1980). Attachment of the physiological measurement devices was followed by computer-presented explanations

of the problems and procedural instructions. Two practice trials of each problem type were then undertaken. A trial began when the participant pressed the button of a hand-held (in the preferred hand) response device after being prompted to do so on the monitor. Participants had been instructed to press the button again upon having a solution, at which point the screen went blank, and to then say their answer. The trial ended if there was no response within 45 s (this time limit was chosen because a previous study of five-letter anagram solution rates suggested that allowing any more time would have little effect on the probability of a solution being found, Kaplan & Carvellas, 1968).

Participants then adopted one of two postural conditions: standing or supine. In the standing condition a padded stand for the forearm and hand maintained the Finapres cuff at heart level. In the supine condition participants lay upon a mattress, with their head supported by pillows and the Finapres cuff propped to heart level with cushions. In both postural conditions the stimulus monitor was positioned 1.5 m directly ahead of the participant. Physiological recordings began; the participant then rested quietly for five minutes, with averages of physiological measures during the final minute of this period serving as baseline values. Participants were then presented with a 16-trial block of anagrams and a 16-trial block of mental arithmetic problems. Verbal responses from the participant were recorded by the experimenter, who was isolated behind a partition; no feedback was given regarding responses during the experiment. Physiological recording ceased and the participant rested for five minutes while seated; they then adopted the second postural condition and procedures were followed as per the first. The Finapres device was switched off in the rest period between conditions. Postural condition order was counterbalanced across all participants. The sequence in which participants received anagram and mental arithmetic problem blocks was the same in both postural

conditions and counterbalanced across postural condition order.

Data treatment and analysis

The maximum time of 45 s was recorded as the solution latency for problems to which a participant did not respond. Incorrect responses for anagrams, for example, "molde" instead of "model" for "edmlo", were excluded from the analyses; these were given for 6.4% of all anagrams presented, with a repeated-measures t-test revealing no difference between the standing (6.3%) and supine (6.6%) conditions, $t(19) = 0.18$, $p = .858$. Mean solution latencies for individual stimulus items were calculated (across both postural conditions). There was a range of mean solution latencies for mental arithmetic problems, 12.5 s to 24.3 s; for anagrams the range was much larger, 11.6 s (for "osien" = "noise") to 42.6 s (for "nodru" = "round"). Thus, though controlled for to some extent by the random allocation of items, it was possible that participants experienced discrepancies in problem block difficulty between postural conditions; this was addressed by transforming the solution latency data (along similar lines, Beversdorf et al. (1999, 2002) used transformed solution latency data to account for differences in problem set difficulty). For each participant, the transformed solution latency for a given item was calculated as the group mean solution latency for that item (across both postural conditions) subtracted from the participant's solution latency; these scores were then averaged within each block of trials. Thus, each participant had a transformed solution latency score for both anagrams and mental arithmetic in both postural conditions. To investigate whether posture affected performance on the anagram or mental arithmetic tasks, mean transformed solution latencies for the standing and supine conditions were compared within each problem type using repeated-measures t-tests. Similar t-tests were conducted using the number of problems correctly solved. Pearson correlation coefficients were calculated to determine the extent to which transformed

solution latencies for anagrams were related to transformed solution latencies for mental arithmetic problems; separate calculations were done for the standing and supine conditions.

To demonstrate the extent of baroreflex-associated changes in physiological activity between postural conditions, baseline values for standing and supine were compared using repeated-measures t-tests. Due to a pattern observed in the skin conductance data, repeated-measures t-tests were also conducted to identify if there was an increase in the value of any variable from the first to second postural condition baseline, regardless of postural order (i.e., whether the first condition was standing or supine).

For physiological measures during the performance of the mental arithmetic and anagram tasks, the mean values obtained for each trial in a block (of 16 trials) were averaged; this gave scores for both problem types in both standing and supine conditions. These scores were compared with the relevant baseline using repeated-measures t-tests to determine the extent of physiological reactivity associated with performing the mental arithmetic and anagram tasks.

It was also of interest to investigate whether anagrams and mental arithmetic problems were associated with different levels of reactivity on any of the physiological measures; analyses were conducted separately for the standing and supine conditions. Regression analyses were first performed to avoid any artefactual differences potentially stemming from unequal durations spent on the tasks. For example, it is possible that a decrease in heart rate reactivity over the time spent performing a mental task (Kelsey et al., 1999) could result in a longer task being associated with a lower mean heart rate; there is no certainty that this will be the case however, as for example, Ring et al. (1999) found stable heart rate reactivity over the duration of an eight minute cognitive task.

Nevertheless, for each participant, a latency difference score was calculated; this was their mean solution latency for mental arithmetic subtracted from their mean solution latency for anagrams. The latency difference score was entered as the independent variable into a linear regression equation; the dependent variable was a reactivity difference score, with the value for mental arithmetic subtracted from the value for anagrams. In not one analysis did the latency difference score predict differences in reactivity between anagrams and mental arithmetic (the largest F value calculated was for skin conductance while supine, $F(1, 18) = 2.72$, $p = .117$; see Appendix B, Table B1 for the other F ratios), meaning differences in task duration were not associated with any differences in reactivity. Subsequently, repeated-measures t-tests were used to compare the reactivity difference scores between anagrams and mental arithmetic.

Data are presented as mean (SD). All statistical tests were performed using SPSS 11.5 (SPSS Inc., Chicago, IL); an alpha of 0.05 or less was taken to indicate statistical significance. Alpha is calculated to three decimal places by SPSS: an output value of ".000" is stated here as $p < .001$.

Results

Anagram and mental arithmetic task performance

As illustrated in Figure 2.1, the participants solved anagrams an average of 3.1s more rapidly when supine than when standing, $t(19) = 2.94$, $p = .008$; there was no difference in the number of correct solutions provided, 8.3 and 9.1 for the standing and supine conditions respectively, $t(19) = 1.42$, $p = .173$. Mental arithmetic task performance was not affected by posture: there was no difference in solution latencies between standing and supine, $t(19) = 0.95$, $p = .353$ (see Fig. 2.1); likewise, there was no difference in the

number of correct responses given for the standing (14.1) and supine (13.5) conditions, $t(19) = 1.26$, $p = .225$.

Solution latencies for anagrams were not correlated with solution latencies for mental arithmetic in either the standing, $r(18) = 0.24$, $p = .309$, or supine, $r(18) = 0.15$, $p = .527$, conditions; indicating that a participant's ability to solve anagrams was not reflective of their ability to solve mental arithmetic problems.

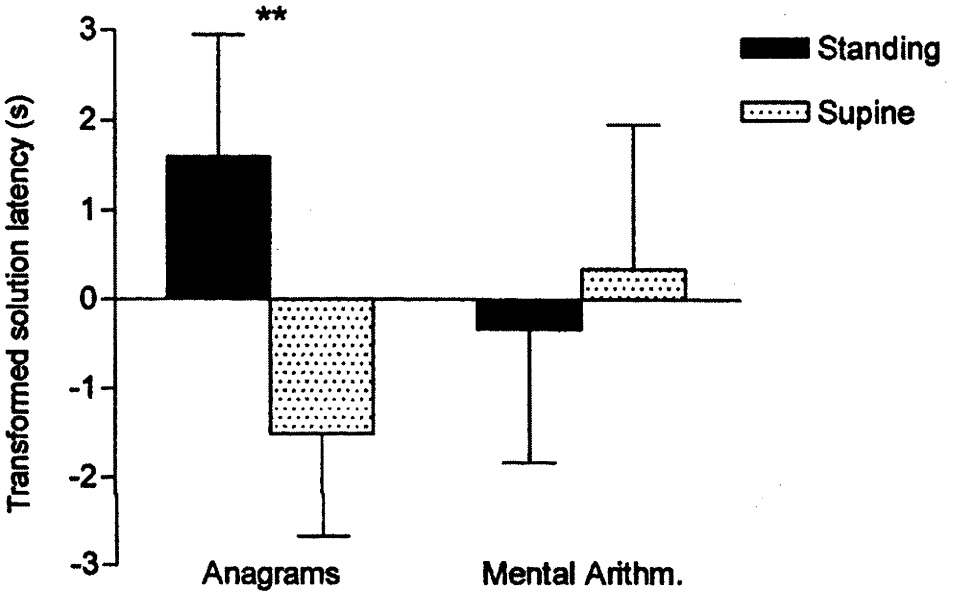

Figure 2.1. Mean (±SE) transformed solution latencies for anagram and mental arithmetic problems in the standing and supine conditions; negative values indicate shorter latencies. Anagrams were solved more rapidly in the supine condition than in the standing condition, **$p = .008$.

Physiological data

Standing and supine baseline values for all physiological measures are shown in Table 2.1. Baseline values for both diastolic blood pressure and heart rate were greater while standing than while supine, $t(19) = 4.74$, $p < .001$, and $t(19) = 7.41$, $p < .001$ respectively. Conversely, baseline respiratory rate was higher while supine than while

standing, $t(19) = 3.42$, $p = .003$. There was no difference in either systolic blood pressure, $t(19) = 1.45$, $p = .163$, or skin conductance, $t(19) = 0.21$, $p = .838$, between standing and supine at baseline. However, further analysis revealed that regardless of postural order, baseline skin conductance for the second posture, 13.6 (5.7) μmho, was greater than that of the first, 8.6 (4.1) μmho, $t(19) = 6.65$, $p < .001$ (see Fig. 2.2). A similar association between first and second postural conditions was not found for any of the other physiological measures (see Appendix B, Table B2 for these results).

Table 2.1

Mean (SD) values for all physiological measures

Standing	Baseline	Anagrams	Mental arithmetic
Systolic b.p. (mmHg)	128.6 (20.3)	132.8 (21.0)*	139.2 (22.8)***
Diastolic b.p. (mmHg)	81.8 (18.4)	84.9 (17.8)*	88.0 (18.8)***
Heart rate (beats/min)	91.1 (15.6)	91.5 (14.6)	92.7 (14.8)
Skin cond. (μmho)	11.1 (5.5)	12.4 (5.6)*	13.4 (6.2)***
Resp. rate (breaths/min)	15.4 (2.3)	17.0 (3.4)*	17.5 (2.8)**
Supine			
Systolic b.p. (mmHg)	123.0 (18.7)	127.6 (17.0)*	130.5 (15.2)**
Diastolic b.p. (mmHg)	69.1 (14.7)	74.1 (16.4)**	74.5 (14.4)***
Heart rate (beats/min)	71.1 (13.8)	72.3 (13.9)	75.0 (14.4)**
Skin cond. (μmho)	10.8 (5.4)	10.5 (5.3)	10.8 (5.2)
Resp. rate (breaths/min)	17.1 (3.0)	18.1 (3.3)*	18.4 (2.9)**

Note. Comparisons vs. baseline: df = 19, *p< .05, **p< .01, ***p< .001.

Physiological reactivity during the performance of anagram and mental arithmetic tasks

is shown in Table 2.1. Systolic blood pressure, diastolic blood pressure and respiratory rate increased above baseline during both mental arithmetic and anagrams for both standing and supine conditions. The only significant level of heart rate reactivity was for mental arithmetic while supine. Skin conductance increased during both problem types in the standing condition; there was no change while supine.

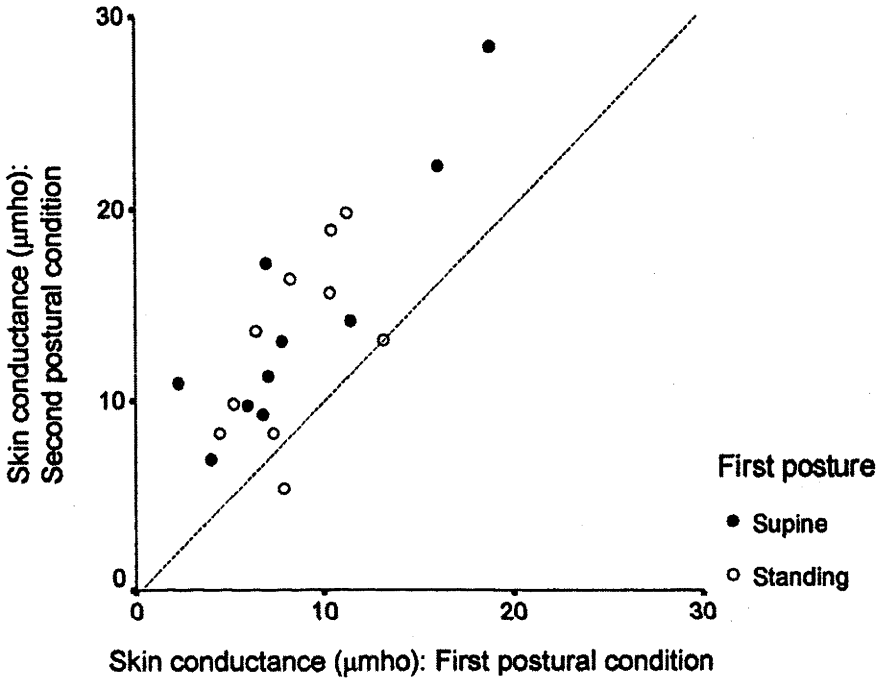

Figure 2.2. Baseline skin conductance of individual participants in their first and second postural condition (nature of the first postural condition is indicated); points above the dashed line represent greater skin conductance in the second condition.

Reporting of the analyses for postural differences in physiological reactivity is limited to mental arithmetic; this is because of the potential for extraneous effects on reactivity associated with differences in cognitive performance between standing and supine conditions for anagrams. Heart rate reactivity was greater in the supine condition than the standing condition, $t(19) = 2.47$, $p = .023$. Conversely, skin conductance increased more during mental arithmetic in the standing condition than in the supine condition, $t(19) = 3.10$, $p = .006$. There was no difference in reactivity between postures for

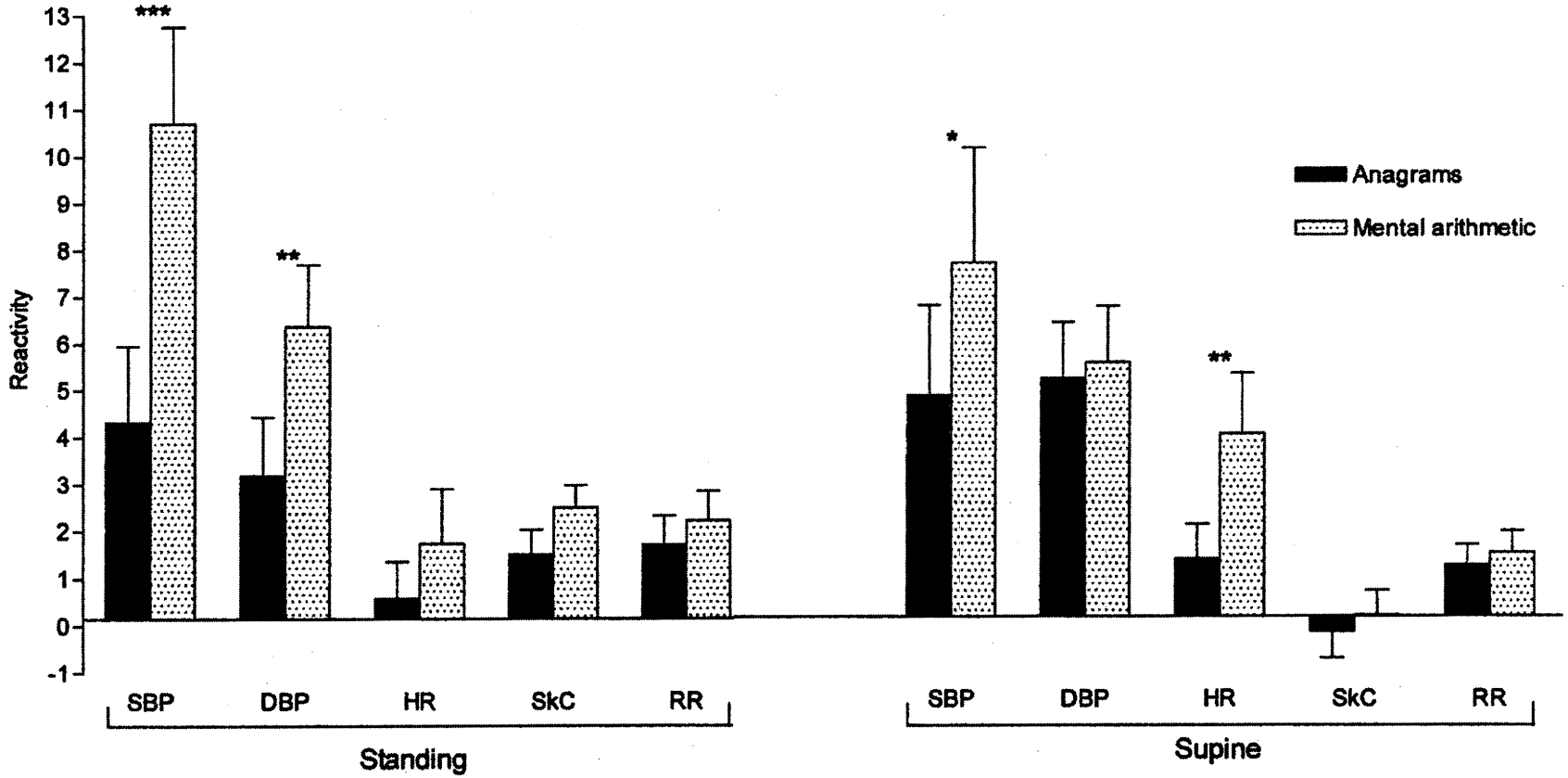

Figure 2.3. Mean (±SE) physiological reactivity (change from baseline) during anagram and mental arithmetic tasks in both standing and supine conditions. Units of measurement are: systolic blood pressure (SBP) and diastolic blood pressure (DBP): mmHg; heart rate (HR): beats/min; skin conductance (SkC): μmho; respiration rate (RR): breaths/min. Significant differences between anagrams and mental arithmetic: *$p < .05$, **$p < .01$, ***$p < .001$.

systolic blood pressure, $t(19) = 1.41$, $p = .176$, diastolic blood pressure, $t(19) = 0.65$, $p = .524$, or respiration rate, $t(19) = 1.16$, $p = .260$.

Differences in reactivity between mental arithmetic and anagrams are shown in Figure 2.3; where differences between the tasks were found, reactivity was greater for mental arithmetic than for anagrams. This included systolic blood pressure for both the standing, $t(19) = 5.40$, $p < .001$, and supine, $t(19) = 2.26$, $p = .036$, conditions. Diastolic blood pressure reactivity was also higher for mental arithmetic while standing, $t(19) = 3.55$, $p = .002$, though not while supine, $t(19) = 0.41$, $p = .685$. Increases in heart rate were greater during mental arithmetic than during anagrams in the supine condition but not the standing condition, $t(19) = 3.38$, $p = .003$, and $t(19) = 1.19$, $p = .248$, respectively. Differences in skin conductance reactivity did not reach conventional levels of statistical significance for the standing condition, $t(19) = 1.81$, $p = .086$; there was no effect for the supine condition, $t(19) = 0.96$, $p = .349$. No differences in reactivity were observed for respiration rate, $t(19) = 1.12$, $p = .277$, and $t(19) = 0.68$, $p = .508$, for standing and supine respectively.

Discussion

Posture influences the distribution of blood throughout the body. For example, when adopting a more upright posture blood pools in the lower body; this decreases the load on baroreceptors, which in turn leads to an increase in sympathetic nervous system activity that prevents mean blood pressure from falling (e.g., Mohrman & Heller, 2003). This homeostatic mechanism is demonstrated in the current study by resting (baseline) diastolic blood pressure and heart rate both being greater when participants were standing than when they were supine (as is often the case there was no difference in

resting systolic blood pressure between these postures). Changes in baroreceptor activity have effects beyond blood pressure maintenance; this includes changes in the activity of the locus coeruleus (the core nucleus of the noradrenergic arousal system), and consequently, changes in both cortical noradrenaline turnover (e.g., Persson & Svensson, 1981) and EEG indicators of arousal (e.g., Valentino et al., 1991). The relationship between baroreceptor activity and locus coeruleus firing rates is inverse in nature: decreases and increases in baroreceptor activity lead to increases and decreases in locus coeruleus firing rates respectively. Due to associated changes in baroreceptor activity, changes in posture are thought to modulate locus coeruleus activity; in particular, there being less locus coeruleus activity in a reclining posture (e.g., Elam et al., 1984). Support for this idea comes from considering postural differences in peripheral sympathetic nervous system activity in the context of a global sympathetic system that co-ordinates parallel changes in peripheral and central sympathetic activity (Aston-Jones et al., 1994).

The ability to solve anagrams has been shown to be facilitated under circumstances in which central noradrenergic influences are minimised (Beversdorf et al., 2002; Walker et al., 2002). In line with this, it was hypothesised in the current study that participants would exhibit better performance on an anagram task while supine than while standing; the results support this hypothesis, as anagrams were solved more rapidly when participants were supine than when they were standing (this was not the result of a practice effect as postural condition order was counterbalanced across all participants). In more general terms, this finding is consistent with the idea that processes modulated by the noradrenergic arousal system are also modulated by posture. Previous support for this idea comes from findings that performance on a vigilance task is related to locus coeruleus activity (in primates: Rajkowski et al., 1998) and that decrements in

performance on a vigilance task associated with sleep deprivation are less pronounced when participants are standing than when they are seated (Caldwell et al., 2003).

In the current study, a postural manipulation believed to affect central noradrenergic activity was shown to affect performance on a specific cognitive task. In a much broader psychological context, the relationship between posture and the noradrenergic arousal system activity may be reflected in the behavioural states typically associated with different postures. As indicated by EEG (e.g., Vaitl & Gruppe, 1990), evoked potential (e.g., Wei et al., 1992), sleep onset latency (Cole, 1989) and reaction time studies (e.g., Vercruyssen & Simonton, 1994), cortical arousal increases as posture becomes more vertical. This is consistent with standing being a bodily state likely to involve physical interaction with the environment and with lying down being conducive for rest.

The finding of the current study that participants solved anagrams more rapidly when they were supine than when they were standing provides an additional line of support for the notion that the ability to solve anagrams is impaired by central noradrenergic activity. Importantly, there was no effect of posture on the performance of a mental arithmetic task; this suggests that, rather than there being a more general effect of posture on cognitive processing, processes utilised specifically in solving anagrams were influenced by posture (and therefore potentially influenced by changes in central noradrenergic activity). This distinction was not demonstrated in the studies by Beversdorf et al. (2002) and Walker et al. (2002), in which participants were presented with anagrams only. Differences in the effect of posture on the performance of anagram and mental arithmetic tasks could be associated with differences in the cognitive processes required for generating solutions. There being no correlation between solution

latencies for the anagram task and solution latencies for the mental arithmetic task in either the standing or supine condition is consistent with the idea that these problem types have different cognitive processing requirements (that it would seem participants do not engage in to similar degrees). For mental arithmetic problems of the form used in the current study (i.e., with multiple operations), the cognitive processing requirements involve the application of logic in a step-by-step fashion (Furst & Hitch, 2000); the findings of the current study suggest that this sort of cognitive processing is unaffected by, or relatively robust to, changes in posture. This is supported by other studies in which performance on a mental arithmetic task was found (incidentally) to be similar between standing and seated conditions (Sherwood & Turner, 1993; Szabo, 1993; Turner & Sherwood, 1991).

Cognitive processes used in solving anagrams and their modulation by central noradrenergic activity

For a person to correctly solve an anagram they must access their memory store of words. In a cognitive sense, this lexical store can be represented as nodes in a network, with phonemically (and orthographically) similar nodes being interconnected; a network of semantically similar nodes is considered to exist in parallel to the lexical network. Cognitive nodes have a variable activity level, though they are evaluated (i.e., available to consciousness) only once an activation threshold is reached. A node can receive activation by conscious processing (e.g., by reading the word represented by the node), or by the spread of activation from other nodes to which it is connected. The spread of activation between nodes is the basis for the priming effect, where prior presentation of a word (the prime) facilitates processing of a semantically associated word (the target; e.g., seeing "bank" decreases the reaction time for identifying "money" as a real, rather than nonsense, word in a lexical decision task). In the absence of further processing, or

inputs from other nodes in the network, a node's activation decays with time towards its intrinsic level (see Collins & Loftus (1975) for an account of these ideas).

Subjective reports of attempts to solve anagrams have described periods involving concentration on particular letter groupings, syllables, or a single letter around which the others are placed in different arrangements (Hunter, 1959; Kaplan & Carvellas, 1968). Concentrating on a letter grouping or syllable is likely to raise the activity of nodes that contain this characteristic (this may involve multiple lexical networks e.g., orthographic for letter groupings and phonemic for syllables). If the solution is not found, that is, if the activation level of the node representing the solution has not reached threshold (because either the solution does not contain the letter grouping etc. or insufficient activation was produced) the participant may move to another strategy; this process may be repeated numerous times with different strategies. None of these strategies may in themselves raise the activation level of the node representing the solution word to threshold. However, any sub-threshold activation received by the node will accumulate over time and (provided it has not completely decayed in the interim) thus make following strategies more likely to lead to the solution being found; this idea is supported by the finding that unconsciously perceived, briefly presented visual hints can decrease anagram solution latencies (Bowden, 1997). It follows that the closer the initial (or intrinsic) activity of the relevant node is to threshold, the more rapidly threshold will be reached and the quicker the solution found.

Cognitive nodes (and therefore individual words) are represented in the brain by cell assemblies; these are groups of functionally connected neurons that may be dispersed throughout different cortical regions and/or hemispheres (Pulvermuller, 1996). Noradrenaline can increase the signal-to-noise ratio of cortical neurons (i.e., signals

become more salient above the level of background noise, which is thought to facilitate sensory processing). This effect of noradrenaline is achieved, in part, by inhibiting the spontaneous firing rates of cortical cells (i.e., reducing the background noise, Clark, Geffen & Geffen, 1987; Hasselmo et al., 1997). In fact, the capacity of noradrenaline to suppress the spontaneous activity of cortical neurons is well established (e.g., Ego-Stengel, Bringuier & Shulz, 2002), with a similar effect also produced via electrical stimulation of the locus coeruleus (Mantz, Milla, Glowinski & Thierry, 1988). These inhibitory effects may seem to be at odds with the role of the central noradrenergic system in enhancing cortical arousal (e.g., Berridge & Foote, 1991); indeed, the manner in which central noradrenaline modulates arousal remains to be determined, though may involve an action at specific cortical sites (Berridge & Waterhouse, 2003).

It has been proposed that a change in the signal-to-noise ratio of cortical neurons may underlie a detrimental effect of central noradrenergic activity on the ability to solve anagrams (Beversdorf et al., 1999, 2002). Such an effect could be because in reducing the spontaneous activity of cortical cells, noradrenaline reduces the activation level of many cell assemblies and thus the intrinsic activation level of many cognitive nodes. When the activation level of the node representing the solution to an anagram is reduced, more activation is required (and therefore more time is needed), before its threshold is reached. This means that the solution may effectively be more readily available to consciousness in the absence of noradrenergic activity. Other authors have discussed a similar effect in different terms, suggesting that a reduction in the signal-to-noise ratio of cortical cells constricts cognitive networks (Heilman, Nadeau & Beversdorf, 2003; Kischka et al., 1996). This is consistent with the report by Stickgold, Scott, Rittenhouse and Hobson (1999) that the priming effect for words with a weak semantic association (with the prime) was facilitated following awakening from REM

sleep, compared with awakening from NREM sleep (when there is a greater level of central noradrenergic activity). The idea that an increase in the signal-to-noise ratio of cortical cells leads to a constriction of cognitive networks is in agreement with an increase in the signal-to-noise ratio reducing the intrinsic activation level of the network's cognitive nodes, as under these circumstances the spread of activation from a node (representing a priming word) is less likely to activate connected nodes (representing target words associated with the prime).

Beversdorf et al. (1999, 2002) and Walker et al. (2002) have stated that cognitive flexibility is utilised when solving anagrams. Conceivably, this could involve limiting perseverance on an unsuccessful strategy (concentrating on a particular letter grouping etc.) and enabling a shift to another. Shifting between strategies is likely to be facilitated by pre-existing activation in cognitive nodes that are relevant to the new strategy. By suppressing the spontaneous activity of cell assemblies, noradrenaline could impair the ability to shift between strategies; in a more general sense this would be consistent with a noradrenergic impairment of cognitive flexibility (Beversdorf et al.).

Martindale (1995) has suggested that there are many more cognitive nodes simultaneously active, though at lower level of activation, in a state of defocused attention than in a state of focused attention. Thus, a reduction in the number of active (at least partially) cognitive nodes can be equated with a loss of defocused attention. Differences in the number of simultaneously active cell assemblies can be assessed by calculating the dimensional complexity of the EEG; this is a technique derived from chaos theory by which the number of independent processes contributing to the EEG trace can be estimated (Birbaumer, Lutzenberger, Schupp & Elbert, 1994; Lutzenberger, Preissl & Pulvermuller 1995). Dimensional complexity varies with attentional state,

being greater on a task of divided attention than a selective attention task (Molle et al., 1995). The dimensional complexity of the EEG in participants performing a selective attention task was found to be increased (cf. placebo) by the administration of an adrenocorticotropin fragment (peptides 4-10), suggesting that the peptide had a defocusing effect (Molle, Albrecht, Marshall, Fehm & Born, 1997). This effect could be associated with reduced activity of the locus coeruleus, as the same adrenocorticotropin fragment has been shown to suppress activity of the locus coeruleus in rats (Adams & Foote, 1988). Furthermore, the relationship between locus coeruleus activity and performance on a vigilance task in monkeys (previously outlined) indicates that best performance, and therefore focussed attention, requires a certain level of tonic activity (Rajkowski et al., 1998). In combination, the results of these disparate studies suggest that central noradrenergic activity is incompatible with defocused attention; this supports the idea that central noradrenergic activity causes a widespread reduction in the intrinsic activation level of cell assemblies.

Higher locus coeruleus activity is likely to disrupt the neurophysiological conditions that facilitate solving anagrams; which may be akin to disrupting the substrate for cognitive flexibility and/or defocused attention. This could involve noradrenergic suppression of the activation level of cell assemblies (groups of neurons) that are relevant to the solution, including that which directly represents the solution word; given the evidence suggesting that locus coeruleus activity is higher in a more upright posture, this suppression mechanism may explain the finding of the current study that anagrams were solved more rapidly while participants were supine than while they were standing. There would be further support for this explanation if future research were to demonstrate that the dimensional complexity of the EEG is greater when supine than when standing; this would indicate that there are more spontaneously active cell

assemblies in the supine position (which in turn would be consistent with less central noradrenergic activity).

An application of baroreflex effects to the findings of Beversdorf et al. (1999, 2002)

The findings of the current study indicate that changes in baroreceptor activity (produced by a postural manipulation) may be associated with changes in performance on an anagram task. A similar mechanism could help explain the Beversdorf et al. (1999, 2002) finding of no difference in anagram solution latencies between propranolol and placebo conditions. In keeping with the idea that central noradrenergic activity impairs the ability to solve anagrams, it could be expected that the blockade of central beta-adrenoceptors by propranolol would lead to faster solution latencies for anagrams (cf. placebo); Beversdorf et al. considered the absence of this effect to be a possible result of noradrenergic tone variability in the placebo condition.

While chronic beta-blocker therapy is useful in treating hypertension, it is often observed that acute administration has no effect on blood pressure; this is because a fall in cardiac output (due to beta-adrenoceptor blockade) may invoke a compensatory increase in total peripheral resistance (mediated primarily by alpha-adrenoceptors and thus not affected by beta-blockers) (Sproat & Lopez, 1991; Thomas et al., 1992). Because a decrease in baroreceptor activity leads to increases in both locus coeruleus firing rates (e.g., Elam et al., 1985) and cortical noradrenaline turnover (e.g., Persson & Svensson, 1981), the acute administration of a beta-blocker may evoke a mechanism that increases central noradrenergic activity (see Fig. 2.4).

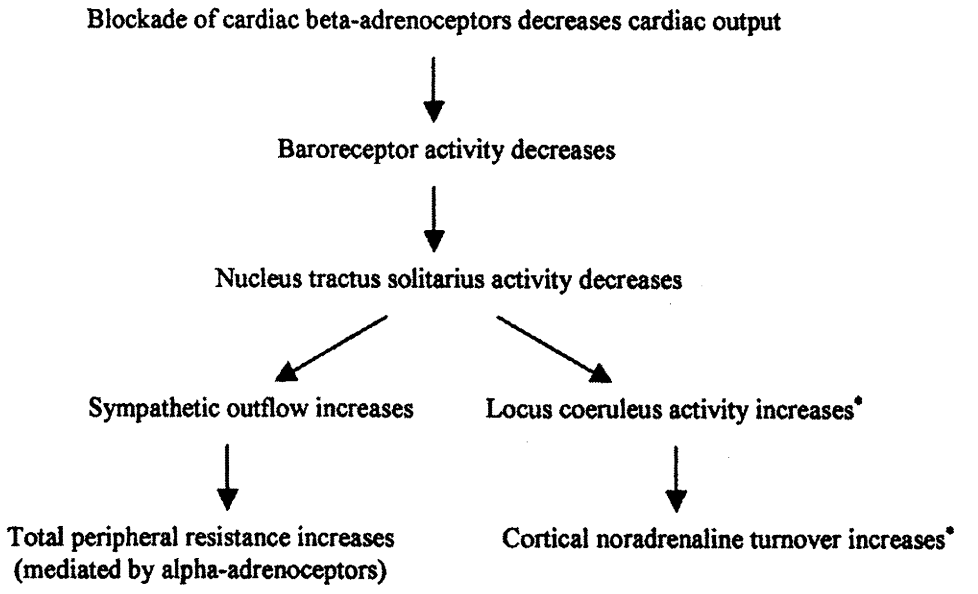

Figure 2.4. Flowchart detailing peripheral and central baroreflex responses to beta-blocker administration. *These effects are likely to be reduced when a centrally acting beta-blocker has been administered (given the finding that propranolol blocks the excitatory action of locus coeruleus activity on the EEG, Berridge & Foote, 1991).

Beversdorf et al. reported that, relative to placebo, heart rate was reduced by the acute administration of both propranolol (1999, 2002) and nadolol (2002); thus it is likely that there was a reduction in cardiac output and therefore possible that a baroreflex was invoked that increased total peripheral resistance in the beta-blocker conditions (this cannot be verified because measures other than heart rate were not reported by Beversdorf et al.). If a baroreflex was evoked, a concomitant increase in cortical noradrenaline turnover could have competed sufficiently with the effects of central beta-antagonism to prevent differences between the propranolol and placebo conditions from developing. An increase in cortical noradrenaline turnover in the nadolol condition would be unopposed by central beta-blockade (given that the drug does not cross the blood brain barrier), and thus could have facilitated the observed significant difference

in mean anagram solution latency with the propranolol condition by working to impair the cognitive processes used to solve anagrams. Consistent with this idea, though not statistically significant, the mean solution latency in the nadolol condition was almost two seconds greater than in the placebo condition. Given that there is thought to be a greater level of central noradrenergic activity in a more upright posture (e.g., Elam et al., 1984), this difference between nadolol and placebo conditions could be analogous to the finding of the current study that anagram solution latencies were lower in a supine condition than in a standing condition.

Creative insight: modulation by noradrenergic arousal and posture

Beversdorf et al. (1999) suggested that low levels of noradrenergic arousal should facilitate insight; this idea is expanded upon here. Insight refers to cognitive breakthroughs, often creative, that occur in a moment of sudden awareness; this phenomenon has been described as "the Eureka! or Aha! Experience" (Bowden, 1997, p. 545). Classic examples of insight in a scientific context are the experimental procedures by which Loewi discovered acetylcholine (Loewi, 1960) and Kekule's solving the structure of benzene (Japp, 1898); both of these events were reported to have occurred during sleep, when locus coeruleus (Aston-Jones & Bloom, 1981a) and central aminergic (see Walker et al., 2002) activity is low. A recent study provides an experimental analogue of these anecdotal accounts: Wagner, Gals, Halder, Verleger and Born (2004) found that sleep facilitated the discovery of a hidden abstract rule for solving a particular cognitive task.

Traditionally, the sudden awareness that characterises insight has been investigated in an experimental context with insight problems, such as the candle (Duncker, 1945) and nine-dot (Scheerer, 1963) problems. As with insight problems (Duncan, 1962), the

probability of the solution to an anagram being found is related logarithmically to the time spent trying (i.e., after a certain point, any more time spent trying to solve the problem will have little effect of the chances of success, Mayzner & Tresselt, 1958). Furthermore, both traditional insight problems and anagrams are associated with hindsight bias regarding problem difficulty, meaning that once the solution is made apparent the problem is rated as easier to solve than indicated by objective measures (Hom & Ciaramitaro, 2001). Most importantly, anagrams are frequently solved in a moment of insight, with the solution becoming known despite a lack of relevant conscious mental effort immediately preceding this sudden awareness (Metcalfe, 1986; Smith & Kounios, 1996). Though anagrams are not always solved in a moment of insight, there is ample evidence to warrant considering them as insight problems (Novick & Sherman, 2003). Thus, the result of the current study that anagrams were solved more rapidly under conditions believed to be associated with relatively low locus coeruleus activity (lying down) supports the claim by Beversdorf et al. (1999) that insight is facilitated when noradrenergic arousal is low.

In the context of a relationship between insight and creativity it could be suggested from the anagram results of the current study that creative thinking might be encouraged simply by adopting a less upright posture. Given that locus coeruleus activity and cortical arousal are thought to be reduced in a less upright posture (e.g., Elam et al, 1984), this idea is consistent with the proposed impairment of creative thought by locus coeruleus activity (Heilman et al., 2003). It is also in keeping with findings that creativity test performance is inversely related to cortical arousal. In one of the experiments to demonstrate this, white noise (which has been shown to increase locus coeruleus activity in both cats: Abercrombie & Jacobs (1987), and monkeys: Rajkowski et al., 1994) impaired performance on the remote associates test (though left scores on a

non-creative task unaffected, Martindale & Greenough, 1973). Furthermore, the ability to list alternate uses for common objects (brick, shoe, newspaper) was found to be directly related to the amount of EEG alpha power during performance of the task (Martindale & Hines, 1975). However, it has been previously reported that scores on the "Uses of a Brick" test (a brick was one of the objects in the alternate uses test of Martindale & Hines) were no different between groups who performed the task in one of supine, seated or standing conditions (Schulman & Shontz, 1971). This seems inconsistent with the results of the current study given the positive relationship between the number of five-letter anagrams solved and the number of uses that participants could think of for a coat-hanger reported by Gavurin (1975). The results of the current study, and a strong theoretical rationale, warrant further research to clarify whether posture modulates the ability to think creatively.

Non-noradrenergic differences between standing and supine

A more upright posture is associated with greater cortical arousal (e.g., Cole, 1989). Baroreceptor activity is reduced in a more upright posture, and because a reduction in baroreceptor activity has been shown to increase activity of the locus coeruleus and central noradrenaline turnover, it is likely that there is greater activity of the noradrenergic arousal system in a more upright posture (e.g., Elam et al., 1985). Nevertheless, as cortical arousal can be modulated by numerous neurotransmitter systems (Robbins, 1997), it is possible that neurotransmitters other than noradrenaline contribute to increased arousal in a more upright posture, and therefore could also be implicated in the poorer anagram task performance while standing compared with lying down found in the current study.

In addition to noradrenaline, Kawahara, Kawahara and Westerink (1999, 2002) found

that dopamine was released in the prefrontal cortex during nitroprusside induced hypotensive stress in rats; this effect was partially reduced by lesions of the locus coeruleus (Kawahara et al., 1999), possibly due to co-release of dopamine and noradrenaline from the terminals of this nucleus (Devoto, Flore, Pani & Gessa, 2001). Thus, given that (as with nitroprusside administration) baroreceptor activity is reduced in a more upright posture, it is possible that there is greater central dopaminergic activity when standing than when supine. L-Dopa is the immediate precursor of dopamine, and as such is administered to patients with Parkinson's disease (Rutledge, 1990). In addition, L-Dopa has been found to reduce the priming effect for target words with a weak semantic association with the prime, presumably by decreasing the spread of activation throughout the semantic network (Kischka et al., 1996). However, as explained by Kischka et al., L-Dopa is the precursor for both dopamine and noradrenaline, meaning a particular role for dopamine in modulating cognitive networks is unclear. Nevertheless, an effect of dopamine on priming (and therefore on cognitive node activation levels) would imply that dopamine could impair the ability to solve anagrams. Thus, given the possibility that cortical dopamine levels may increase with a more upright posture, an effect of dopamine on the relative impairment of anagram task performance while standing found in the current study cannot be excluded. However, it is interesting to note theories that dopamine facilitates cognitive flexibility (Ashby, Isen & Turken, 1999; Previc, 1999), which would appear to mitigate the potential for dopamine to impair the ability to solve anagrams (given that cognitive flexibility is thought to be used in solving anagrams, e.g., Beversdorf et al., 1999). Further studies are required if an effect of dopamine on anagram task performance is to be isolated and clarified.

The cerebral cortex is innervated by neurons from the cholinergic basal forebrain,

electrical stimulation of which leads to increases in both cortical acetylcholine turnover and EEG activation (in rats: Casamenti, Deffenu, Abbamondi & Pepeu, 1986). While there appears to be no direct evidence for baroreceptor activity modulating cortical acetylcholine turnover, there is an anatomical basis by which this could occur. The cholinergic basal forebrain receives inputs from baro-responsive nuclei, including both the nucleus tractus solitarius and locus coeruleus (as demonstrated in rats by Semba, Reiner, McGeer & Fibiger, 1988). Local application of noradrenaline has been shown to excite neurons in the cholinergic basal forebrain (in guinea pigs: Fort, Khateb, Pegna, Muhlethaler & Jones, 1995), leading to increased cortical arousal (as indicated by EEG activity in rats: Cape & Jones, 1998). Thus, via an increase in locus coeruleus activity, acetylcholine may (at least theoretically) facilitate the increase in cortical arousal associated with a more upright posture. Implications for the poorer anagram task performance while standing found in the current study are uncertain. However, cortical acetylcholine turnover is higher during REM sleep than during NREM sleep (as shown in cats by Jasper & Tessier, 1971); thus, the findings reported by Walker et al. (2002) that more anagrams were solved following awakening from REM sleep than from NREM sleep is inconsistent with a cholinergic impairment of the ability to solve anagrams.

A change in bodily posture may induce physiological effects on the brain other than alterations in neurotransmitter turnover. A more upright posture is typically associated with a reduction in cerebral blood flow (Levick, 2000), though Warkentin et al. (1992) reported no difference in mean hemispheric flow between 70° head-up tilt and supine conditions. However, Warkentin et al. did observe reduced flow in frontal regions of the cortex with upright tilt. Similarly, reduced frontal flow was found for standing (cf. supine) by Ouchi, Okada, Yoshikawa, Futatsubashi and Nobezawa (2001); this effect

was negatively correlated with age (i.e., there was less of a decrease in frontal blood flow during standing for the younger members of the group, though all participants were middle aged). Other studies have shown that the cerebral blood flow ratio (measured against cerebellar blood flow; which Ouchi et al. have shown to increase with standing) for both left and right frontal regions was not different between standing and supine in normal, middle-aged to elderly participants (Hayashida, Nishiooeda, Hirose, Ishida & Nishimura, 1996; Ohtani et al., 2003). The mixed nature of these findings mean the extent to which blood flow is reduced in frontal regions of the cortex when in a more upright posture is unclear.

Walker et al. (2002) found that performance on an anagram task was similar between conditions of normal waking and awakening from REM sleep; they suggested that the lack of an anticipated advantage for the REM sleep condition (due to less aminergic activity) could be due to this sleep state being characterised by a suppression of frontal brain regions, the dorsolateral prefrontal cortex in particular. In participants attempting to solve solvable anagrams (unsolvable anagrams were presented as a separate task), Schneider et al. (1996) found blood flow increases in some frontal regions (the midfrontal and inferior frontal; also see Chance, Nioka, Sadi & Li, 2003), though not in the dorsolateral prefrontal cortex; this suggests that the dorsolateral prefrontal cortex may not be critically involved in attempts to solve anagrams. Nevertheless, though the presence, or extent, of a reduction in frontal cortex blood flow during standing is uncertain (especially in young adults), it could be suggested that the development of this effect contributed to the anagram task performance being poorer while standing than while supine in the current study. However, consideration must be given to observations of increased blood flow in frontal regions during the performance of mental arithmetic (e.g., Kazui, Kitagaki & Mori, 2000; Rickard et al., 2000). In light of this, it is unclear

how any differences in resting cerebral blood flow between supine and standing would contribute to poorer performance on the anagram task while standing, though leave performance on the mental arithmetic task unaffected by posture.

Cognitive resources (including attention) are needed to maintain balance while standing; this is demonstrated by dual-task (cognitive and balance) study findings that cognitive task performance decreases when balance demands are increased (Woollacott & Shumway-Cook, 2002). However, the performance of a cognitive task can take precedence over balance control (e.g., bodily sway may increase); this is thought to occur when postural stability is relatively unimportant (i.e., when a loss of stability does not pose a physical risk, Shumway-Cook, Woollacott, Kerns & Baldwin, 1997). In the current study, participants stood with a normal stance, were not required to focus on their balance, and were partly supported by an arm rest; this implies that cognitive resource utilisation for maintaining balance (and that any discomfort related change in attentional focus) is likely to have been minimal and therefore unlikely to have been associated with the slower solution latencies for anagrams while standing than while supine.

Vestibular stimulation can affect cognitive processing; this is shown by studies in which caloric stimulation of the vestibular apparatus (via application of hot or cold water to the ear canal) alters activation of the contralateral hemisphere and thereby influences cognitive processes for which it is largely responsible (Bachtold et al., 2001). However, it is not known whether differences in bilateral vestibular (primarily otolith organ) activity between standing and supine influence cortical activity levels or cognition. Otolith organ stimulation affects cardiovascular activity, as demonstrated by changes in muscle sympathetic nerve activity in humans (Ray & Carter, 2003), though the

contribution to postural cardiovascular reflexes appears to be only during changes in posture (i.e., not while the new posture is maintained, Watenpaugh et al., 2002). Even so, it has been postulated that otolith organs modulate cardiovascular activity via the locus coeruleus (Lai, Tse, Shum, Yung & Chan, 2004). Accordingly, any vestibular contribution to the relatively poorer anagram task performance while standing in the current study may have occurred in synergy with a baroreceptor mediated increase in central noradrenergic activity.

The effects of baroreceptor activity on the ability to solve anagrams could be isolated from any influences of either vestibular activity or the allocation of cognitive resources for maintaining balance associated with a postural manipulation. This may be achieved by having participants attempt to solve anagrams during both positive and negative lower body pressure conditions (producing increases and decreases in baroreceptor activity respectively). By comparing positive and negative lower body pressure conditions, any potential contribution from extraneous psychological effects associated with the experience of lower body pressure manipulation would be minimised. A semi-tilted position (e.g., 45° head-up) would reduce the chances for a floor or ceiling effect associated with baroreceptor stimulation; for example, the effects of positive lower body pressure in a supine position might have less of an effect than anticipated given the level of pre-existing baroreceptor stimulation in that posture. Baroreceptor activity decreases locus coeruleus activity (e.g., Svensson & Thoren, 1979) and cortical noradrenaline turnover (e.g., Persson & Svensson, 1981). As the ability to solve anagrams is improved by conditions in which central noradrenergic activity is minimised (Beversdorf et al., 2002; Walker et al., 2002, and as suggested by the results of the current study), it would be expected that anagram task performance would be better during the application of positive lower body pressure than during the application of negative lower body

pressure.

Significant differences in anagram task performance: solution latencies or number of problems solved

In the current study, a significant difference in anagram solution latencies (though not the number of problems solved) was found between the different experimental conditions; the nature of this finding is consistent with Beversdorf et al. (1999, 2002) though not Walker et al. (2002), who reported differences in the number of anagrams solved (though not in mean solution latencies). There was potential in the current and Beversdorf et al. (as noted by the authors) studies for the number of problems solved to be less sensitive than solution latencies as a measure of differences in performance between experimental conditions; this is because possible discrepancies in problem set (or block) difficulty were addressed for solution latencies only (by using transformed solution latency data). In the Walker et al. study, this problem may have been offset to some degree by attempts to balance the difficulty of different problem sets. However, there were differences between studies in terms of the maximum time available in which to solve individual anagrams, 10 s (Walker et al.), 45 s (current study) and 120 s (Beversdorf et al.), which may have influenced whether task performance differences developed in the form of solution latencies or the number of problems solved. As the probability of solving an anagram is related logarithmically to the time spent trying (Mayzner & Tresselt, 1958), after a certain duration (once within the plateau of the probability of solution vs. time curve) any more time spent trying to solve the anagram will be of little use. Consequently, if the maximum time available for solution is near the plateau of the probability of solution versus time curve, all of the anagrams able to be solved by the participant (within a workable timeframe) are likely to be solved. Thus, a longer maximum solution time will favour the development of differences in anagram

task performance between experimental conditions in terms of solution latencies rather than the number of anagrams correctly solved.

Skin conductance and respiration rate: differences between standing and supine

Skin conductance is an indicator of both sympathetically mediated (Critchley, 2002) and cortical (e.g., Barry et al., 2004) arousal; thus it could be expected that skin conductance will be greater in a more upright posture, in parallel with changes in heart rate, diastolic blood pressure (both demonstrated in the current study) and cortical arousal (e.g., Cole, 1989). This is particularly so in considering the idea that locus coeruleus activity is greater while standing (e.g., Elam et al., 1984) in relation to the finding that skin conductance is reduced following destruction of the central noradrenergic system with 6-hydroxydopamine in cats (Yamamoto, Arai & Nakayama, 1990).

There is only limited empirical evidence for greater skin conductance in a more upright posture. Goldstein and Shapiro (1988) found that skin conductance increased in participants when they moved from sitting to standing (recordings only lasted 60 s however); though a statistical postural comparison was not reported, a similar result appears to have been found by Naliboff, Gilmore and Rosenthal (1993). Skin conductance has been measured previously in both standing and supine (with knees up) conditions, though differences between these were not investigated (Cohen, Swanson, Naliboff, Schandler & McArthur 1986; Collins, Cohen, Naliboff & Schandler, 1982). In the current study, there was no difference in baseline skin conductance between standing and supine. However, further analysis revealed that skin conductance increased from the first to second postural condition baseline irrespective of postural order. Skin conductance recordings can be elevated during recovery from stress (e.g., Jorgensen &

Zachariae, 2002; Kohler, Fricke, Ritz & Scherbaum, 1997), and this can be due to peripheral mechanisms rather than residual cortical arousal (Bundy & Mangan, 1979). In the current study, a rise in skin conductance across the course of the experimental session may underlie the increase in skin conductance from the first to second postural condition baseline. This effect could have been facilitated by a number of factors, including the performance of mental tasks in the first postural condition, an increase in conductive area caused by electrolyte spread (e.g., Lykken, 1970) during the incidental movement of electrodes when participants changed postures, and the use of a sodium chloride based electrolyte (which can produce exaggerated readings, Venables & Christie, 1980). Indeed, the mean values for skin conductance found in the current study are far greater than the 3 μmho norm published by Venables and Christie, for example the baseline skin conductance level in the supine condition was 11.1 μmho. Consequently, the skin conductance values obtained in the current study are unlikely to accurately reflect inherent postural differences and thus explain why they are not consistent with the idea that skin conductance will be greater while standing than while supine.

Participants breathed at a slightly more rapid rate when supine than when standing. A similar finding was reported by Stanley, Verotta, Craft, Siegel and Schwartz (1997), though Tulen, Boomsma and Man in 't Veld (1999) found no difference in respiratory rate between these same postural conditions. Despite differences in respiratory frequency, Stanley et al. postulated that minute ventilation (the volume of air entering the lungs in one minute) differed only minimally, if at all, between standing and supine due to a lesser tidal volume (the volume of air associated with a single breath) in the latter; this may be related to lung volume being compromised by abdominal contents pushing on the diaphragm (Bettinelli et al., 2002). Nevertheless, as the difference in

respiration rate between postural conditions is only small (17.1 breaths/min while supine and 15.4 breaths/min while standing), it is unlikely to be of substantial physiological significance.

The effects of posture on physiological reactivity

Mental arithmetic generally evoked increased activity in all the physiological variables measured, consistent with expectations (e.g., Kjellberg & Magnusson, 1979). However, there was no heart rate reactivity associated with mental arithmetic in the standing condition; nor was there any skin conductance reactivity associated with mental arithmetic in the supine condition.

A number of studies have investigated the effect of posture on physiological reactivity to mental tasks. However, only one of these appears to have made comparisons between standing and supine; this was using mental arithmetic and in only six participants (Rusch et al., 1981). Most studies of postural influences on physiological reactivity to cognitive tasks have compared standing and sitting, with a range of tasks having been used: mental arithmetic (Sherwood & Turner, 1993; Szabo, 1993; Turner & Sherwood, 1991), speech presentation (Cacioppo, Uchino & Bernston, 1994), role-played interpersonal conflict (Waldstein, Neumann & Merrill, 1998) and the colour word test (Tulen et al., 1999).

Two studies have reported an effect of posture on blood pressure reactivity. Rusch et al. (1981) found that mean arterial pressure increased more during mental arithmetic while supine than while standing. Turner and Sherwood (1991) reported greater systolic blood pressure reactivity for standing than for sitting; however, both Tulen et al. (1999) and Waldstein et al. (1998) found no difference between these postures. All three

standing/sitting studies found that posture did not affect diastolic blood pressure reactivity. With no difference in either systolic or diastolic blood pressure reactivity between standing and supine conditions, the findings of the current study are supportive of posture not affecting the magnitude of blood pressure reactivity to mental tasks.

There have been mixed findings regarding heart rate reactivity in the standing position, with some studies finding an increase in heart rate (Cacioppo et al., 1994; Rusch et al., 1981; Szabo, 1993) and others finding a decrease (Sherwood & Turner, 1993; Tulen et al., 1999; Waldstein et al., 1998); however, in all of these studies increased heart rate while in the less upright posture resulted in significantly greater reactivity than while standing. This effect, also present in the current study as greater heart reactivity in the supine condition than in the standing condition, may be due to a pre-existing reduction in parasympathetic withdrawal to the heart while standing (Cacioppo et al.; Tulen et al.; Waldstein et al.).

There has been less research concerning an influence of posture on respiration rate during cognitive tasks. Cacioppo et al. (1994) reported that respiration period (the duration between breaths) increased more in response to speech presentation in a standing condition than in a seated condition, though the effect was only small. Tulen et al. (1999) found no difference in respiration rate reactivity between standing and seated conditions; the larger postural difference in the current study was also found not to affect changes in respiration rate.

The effects of posture on skin conductance reactivity during mental tasks do not appear to have been addressed previously. Given that all other indicators of sympathetic activity increased during mental arithmetic while supine it is interesting that skin

conductance did not do likewise; this is especially so given that skin conductance increased, while heart rate did not, in standing participants. These postural differences are reflective of Fowles' (1980) predictions regarding the psychophysiological correlates of the behavioural approach and behavioural inhibition systems (these systems, and a fight/flight system, have more recently been discussed in terms of fundamental emotion systems, e.g. Gray, 1990). Fowles hypothesised that independent increases in skin conductance and heart rate are related to behavioural inhibition and approach respectively; thus, the results of the current study could be seen to suggest engagement of the behavioural approach system while supine and the behavioural inhibition system while standing during the performance of mental arithmetic.

Gray (1982) has proposed that the behavioural inhibition system is associated with the development of anxiety. Therefore, if this system was engaged in the standing condition of the current study there may also have been an anxiety response; this would be in keeping with an increase in anxiety in response to mental arithmetic (e.g., Schweizer et al., 1991). There is some evidence that behavioural approach system activation creates a predisposition to experience positive affect (Gable, Reis & Elliot, 2000). While it seems unlikely that performing mental arithmetic in the supine position would induce positive affect, an anxiety response may have been prevented in this posture. This is consistent with the idea outlined in Chapter 1 that baroreceptor activity (being greater in the supine condition) can reduce anxiety (Dworkin et al., 1994), and more generally, reduce the aversiveness of a stressful situation; an important aspect to the theory of learned hypertension (e.g., Dworkin, 1988). The idea that postural condition may be associated with effects on anxiety (and stress) is investigated and discussed further in Chapter 4.

It is possible that the rise in skin conductance attributed to the performance of mental

tasks (and associated with behavioural inhibition) while standing was in fact simply an effect of time. McGrady, Kern-Buell, Bush, Khuder and Grubb (2001) found a slight increase in skin conductance over time during 70° head-up tilt in resting patients (being evaluated for signs of syncope). The contribution of a simple time effect to the skin conductance readings for mental arithmetic while standing in the current study could have been ascertained by following the mental tasks with a recovery period; if the skin conductance effects were due to performing mental tasks, after their completion a decrease towards baseline levels would be expected. Alternatively, a rise in skin conductance during a control condition in which no cognitive task is performed would be indicative of a time effect (see Chapter 4).

Differences in physiological reactivity between anagrams and mental arithmetic

Where differences in physiological reactivity between mental arithmetic and anagrams were observed, reactivity was always greater for mental arithmetic: this was the case for systolic and diastolic blood pressure while standing and heart rate while supine, with skin conductance reactivity tending to be higher while standing. Larkin, Ciano-Federoff and Hammel (1998) have also measured cardiovascular reactivity to both anagram and mental arithmetic tasks in the same participants (while seated); consistent with the results of the current study, they found that across experimental conditions of social fear (high/low) and observation (alone/confederate), mean changes in heart rate and systolic blood pressure were greater for mental arithmetic than for anagrams. However, these differences could have been influenced by a social threat manipulation, which was high for mental arithmetic and low for anagrams.

Jausovec and Bakracevic (1995) measured the heart rate of participants during their attempts to solve a variety of problem types, including insight (the triangle and gardener

problems) and logical thinking (involving mathematical calculation). Average heart rate increases were found to be less for insight problems than for the logical thinking problems. Given that anagrams are a type of insight problem (Novick & Sherman, 2003), this is consistent with heart rate reactivity being lower for anagrams than for mental arithmetic in the current study.

In addition to the quantitative difference, there may be a qualitative difference in the physiological reactivity observed for anagrams and mental arithmetic. Different stressors can have different haemodynamic profiles. These can be primarily either a cardiac output or total peripheral resistance profile, as determined by a dominant contribution of one of these factors to the blood pressure reactivity associated with the stressor (e.g., Gregg, James, Matyas & Thorsteinsson, 1999). Mental arithmetic has been reported to have a cardiac output profile (e.g., Gregg et al., 1999), though a mixed cardiac output/peripheral resistance profile is also reported (e.g., Ring et al., 1999). In the current study, the increase in heart rate reactivity during mental arithmetic is suggestive of an increase in cardiac output, and therefore of a cardiac output (or mixed) haemodynamic profile. For anagrams however, blood pressure increased in the absence of any change in heart rate (and therefore likely in the absence of any change in cardiac output); this suggests that performance of the anagram task was associated with a largely peripheral resistance haemodynamic profile.

In the current study, it was demonstrated that posture can modulate higher-order psychological processes. Specifically, performance on an anagram task was found to be better in a supine condition than in a standing condition. This effect can be explained by central noradrenergic activity being greater in a more upright posture (as proposed by Elam et al., 1984), given that the ability to solve anagrams is impaired by central

noradrenergic activity (e.g., Beversdorf et al., 2002). The current study also found that the level of physiological reactivity during problem solving attempts was greater for mental arithmetic than for anagrams. This finding is interesting to consider in relation to the global sympathetic system, which as discussed in Chapter 1, co-ordinates parallel activity of the central (i.e., the locus coeruleus) and peripheral sympathetic nervous systems in preparing an organism for interacting with the environment or for enabling it to cope with stressors (Aston-Jones et al., 1994); parallel central and peripheral sympathetic changes occurring in relation to posture would be just one example of this system in operation. From this idea it follows that, as there was less sympathetic reactivity (e.g., skin conductance), there was also less locus coeruleus activity associated with performance of the anagram task than with performance of the mental arithmetic task. There being a relatively small change in locus coeruleus activity during attempts to solve anagrams could be related to the vulnerability of these attempts to impairment under conditions in which central noradrenergic activity is elevated. However, before going any further into the possible meaning of relatively low physiological activity during attempts to solve anagrams, it needs to be ensured that this finding is related to intrinsic characteristics of the task (e.g., processing demands) rather than arising through differences between anagrams and mental arithmetic that can be accounted for by secondary characteristics of these tasks.

Two salient secondary characteristics of cognitive tasks that may influence physiological reactivity are difficulty and the level of psychological stress associated with performance (described in detail in Chapter 3). Any contribution of these factors to the differences in reactivity between anagrams and mental arithmetic observed in the current study is investigated in Study 2. Participants were asked to provide ratings of difficulty and stress after performing both types of tasks (while seated); physiological

measures were recorded during the tasks. The ratings of stress provided by participants were also used to further investigate an aspect of the theory of learned hypertension outlined in Chapter 1, in which it is thought that a rise in blood pressure activates baroreceptors, in turn reducing the aversiveness of a stressful situation (Dworkin, 1988); there is currently little supporting evidence for this mechanism.

CHAPTER 3

STUDY 2

Introduction

In Chapter 1, it was outlined how changes in baroreceptor activity lead to changes in peripheral sympathetic nervous system activity; this reflects a homeostatic mechanism that maintains adequate blood pressure across postural conditions (e.g., Morhman & Heller, 2003). It is thought that differences in peripheral sympathetic activity between body postures are paralleled by differences in locus coeruleus (and therefore noradrenergic arousal system) activity (e.g., Elam et al., 1984); this was the basis for investigating the influence of posture on the ability to solve anagrams in Study 1 (given that this ability has been shown to be affected by central noradrenergic activity, e.g., Beversdorf et al., 2002). However, parallel changes in central and peripheral sympathetic activity occurring in relation to posture would be just one example of a global sympathetic system, under which peripheral and central changes are co-ordinated in response to stressors or in preparation for interacting with the environment (Aston-Jones et al., 1994).

In addition to finding that posture influenced the ability to solve anagrams (though not the ability to solve mental arithmetic problems), it was observed in Study 1 that autonomic reactivity (of sympathetic origin) during mental arithmetic was greater than during anagrams. In the context of a global sympathetic system this suggests that there was less central noradrenergic activity during attempts to solve anagrams; this is interesting because it could complement findings that the ability to solve anagrams is improved when central noradrenergic activity is relatively low. However, it is possible that the differences in physiological reactivity between mental arithmetic and anagrams

found in Study 1 are not due to intrinsic characteristics of the tasks employed in that study, but stem from secondary characteristics that co-varied with the tasks and that also affect autonomic reactivity. Reports are described here that illustrate how differences in either task difficulty or the perceived stress associated with performing a cognitive task can influence physiological reactivity; the implication being that differences in physiological reactivity between tasks can be a product of differences in task difficulty or perceived stress.

In a study by van Schijndel, de Mey and Naring (1984), participants were presented with three sets of anagrams that differed in difficulty, as determined by the percentage (30, 50, 100) of problems likely to be solved (on the basis of pilot testing and with a maximum of five seconds for each problem). Heart rate and blood pressure were both elevated during all three difficulty conditions. However, while heart rate reactivity was not influenced by task difficulty, systolic blood pressure reactivity was greater in the 50% condition than in the 30% and 100% conditions (diastolic blood pressure tended to show a similar effect). It was thought that the relatively low blood pressure reactivity for the 30% condition might have been due to participants finding the problems too difficult and disengaging from attempts to solve them.

Unlike van Schijndel et al. (1984), Carroll, Turner and Hellawell (1986) found that task difficulty did influence heart rate reactivity. Participants were presented with Raven's matrices and mental arithmetic problems, each having easy, hard and impossible conditions (that were reflected in subjective difficulty ratings). For both types of problem, heart rate reactivity was greater for the difficult and impossible conditions than for the easy condition; respiratory rate was also measured though was not affected by difficulty. In comparing the tasks it was found that the participants perceived

Raven's matrices to be more difficult than mental arithmetic; heart rate reactivity was also greater for Raven's matrices, though this was only found when comparing the easy conditions. Because differences in heart rate reactivity between the tasks were not found for the hard and impossible conditions, despite Raven's matrices being perceived as more difficult than mental arithmetic, it was concluded that rather than difficulty per se, it is the psychological impact of a task (i.e., the affective or stress response that the task generates), and how this interacts with difficulty, that influences physiological reactivity.

A report by Callister, Suwarno and Seals (1992) supports the idea that the psychological impact of a task (measured in terms of perceived stress) is a significant determinant of physiological reactivity. Participants performed both the colour word test (Stroop task) and mental arithmetic, each with six difficulty levels. Increases in difficulty were associated with increases in both muscle sympathetic nerve activity and perceived stress; blood pressure also increased with difficulty (across the first three or four difficulty levels before reaching a plateau). When analysed in relation to stress ratings both muscle sympathetic nerve activity and blood pressure showed a positive relationship. Heart rate increased during both tasks but was not influenced by either difficulty or perceived stress. These findings suggest that task difficulty influences perceived stress, which in turn is associated with physiological reactivity (at least in terms of muscle sympathetic nerve activity and blood pressure). However, more inherent task differences can also contribute to differences in physiological reactivity between tasks, as even at similar levels of perceived stress, blood pressure and heart rate were higher for the colour word test than for mental arithmetic.

Consistent with the Callister et al. (1992) findings, Lepore (1995) reported positive

correlations between both systolic and diastolic blood pressure, though not heart rate, and the perceived stress associated with giving a speech. However, these findings contrast with those of Freyschuss, Hjemdahl, Juhlin-Dannfelt and Linde (1988), who found that increases in perceived stress (from pre to post-task) associated with performing the colour word test were correlated positively with increases in cardiac output and negatively with decreases in systemic vascular resistance; there was no correlation between stress and reactivity as measured in terms of heart rate, stroke volume, systolic and diastolic blood pressures or plasma catecholamines. In a further study by Freyschuss et al. (1990) participants performed both the colour word test and a word identification task. Similar relationships to the 1988 study were found between the stress associated with performing the colour word test and physiological reactivity; in addition, there was a positive correlation between heart rate and perceived stress. There was less physiological reactivity associated with performing the word identification task than the colour word test, which could be accounted for by there also being less psychological stress associated with performing the word identification task.

The findings of studies investigating the effects of task difficulty or perceived stress on physiological reactivity illustrate that, as a general rule, as the difficulty or perceived stress of a cognitive task increases, so does the level of physiological reactivity (though the findings relating to particular measures are mixed). Consequently, differences between tasks in terms of either difficulty or perceived stress could account for differences in physiological reactivity. However, difficulty and stress are not the sole determinants of reactivity, with more inherent task characteristics also making an important contribution. Nevertheless, before concluding that differences in physiological reactivity between cognitive tasks are due to inherent differences, it is important to exclude the possibility that differences in difficulty or perceived stress are

responsible.

In Study 1, the anagram task was objectively more difficult than mental arithmetic, as across both supine and standing conditions less anagrams were responded to on average (maximum of 32) and the mean raw solution latency was longer (19.3 and 27.8 s compared with 31.4 and 18.2 s for mental arithmetic). Indeed, solution latency has previously been used to classify different tasks in terms of difficulty (Paus, Koski, Caramanos & Westbury, 1998). Thus, it would appear that the greater physiological reactivity found for mental arithmetic than for anagrams cannot be attributed to mental arithmetic being more difficult than anagrams. However, it is possible that objective measures of difficulty are not predictive of subjective difficulty; for example, participants may have pre-conceptions about the difficulty of mental arithmetic problems that are reflected in subjective difficulty ratings. Therefore, it remains possible that differences in the perceptions of either difficulty or psychological stress could account for the differences in reactivity between mental arithmetic and anagrams found in Study 1; one of the reasons for conducting Study 2 was to determine if this were indeed the case. Physiological activity was recorded while participants solved both problem types; they also gave ratings of how difficult and stressful they found the tasks. If differences in difficulty or perceived stress are excluded, it is more likely that differences in cognitive processing demands are responsible for differences in physiological reactivity between mental arithmetic and anagrams; if so, this could further understanding of the neurophysiological mechanisms involved in solving anagrams (which was facilitated by the results of Study 1).

Because heart rate did not change significantly from baseline when participants performed anagrams in Study 1 it is possible that the increase in blood pressure that did

occur was a result of increases in stroke volume and/or total peripheral resistance. If the primary contributor was peripheral resistance this would imply that the anagram task was associated with a peripheral resistance haemodynamic profile and thus indicate a qualitative difference in physiological reactivity between anagrams and mental arithmetic, given that mental arithmetic has either a pure cardiac output (e.g., Gregg et al., 1999), or a mixed cardiac output/peripheral resistance (e.g., Ring et al., 1999) haemodynamic profile. This would mean that in addition to differences in the cognitive processes required for solution, mental arithmetic and anagrams might be associated with different central mechanisms responsible for generating physiological reactivity. Possible differences in haemodynamic profiles between anagram and mental arithmetic tasks were investigated in Study 2 by measuring cardiac output and total peripheral resistance (in addition to the measures of systolic and diastolic blood pressure and heart rate used in Study 1).

Previous studies have produced mixed results regarding the relationship between perceived stress and physiological reactivity, as measured in relation to cardiovascular variables. Callister et al. (1990) found that blood pressure (though not heart rate) increased in proportion to the perceived stress associated with performing a cognitive task; similarly, Lepore (1995) reported positive correlations between perceived stress and systolic and diastolic blood pressure though not heart rate. In contrast, Freyschuss et al. (1988, 1990) found a positive correlation between cardiac variables (though not blood pressure) with perceived stress, while a further study reported no correlation between perceived stress and any of the cardiovascular variables that were measured (Maier, Waldstein & Synowski, 2003). The current study attempts to provide some clarification to this issue by determining the extent to which changes in physiological measures are related to the ratings of perceived stress associated with performing mental

arithmetic and anagram tasks.

The nature of the relationship between perceived stress and blood pressure reactivity is important for the theory of learned hypertension (Dworkin, 1988) described in Chapter 1. It has been reported that preventing the normal rise in blood pressure associated with performing mental arithmetic, by administering the peripherally acting beta-adrenoceptor blocker atenolol, resulted in greater than normal levels of psychological stress (Schweizer et al., 1991). This is consistent with the idea, as contained in the theory of learned hypertension, that an increase in blood pressure reduces the level of stress associated with a stressful situation. However, the Schweizer et al. result contrasts with the positive relationships between blood pressure reactivity and psychological stress that have been reported by Callister et al. (1990) and Lepore (1995), which do not appear to be consistent with the stress-reducing mechanism outlined in the theory of learned hypertension.

Methods

Participants

There were 28 female and 10 male participants in the study; their mean age was 21.7 years (the range was 18-36 years). The height and weight of participants were measured so that haemodynamic indices could be determined (see below): mean (±SE) height was 166.3 (±1.3) cm for the females and 179.2 (±3.5) cm for the males; mean (±SE) weight was 63.4 (±2.5) kg and 77.1 (±4.1) kg for the females and males respectively. All participants provided informed consent for the procedures, which were approved by the Australian National University Human Research Ethics Committee. Course credit was received for time spent.

Stimuli

There was a 32-item pool (and two practice items) for both mental arithmetic problems and anagrams; these were of the same form as used in Study 1 (see Appendix A for item lists). Stimuli were displayed on a monitor using Inquisit 1.33; they were in black lower case size 32 Tahoma font on a white background.

Physiological measures

Heart rate and blood pressure were measured using a Finapres device as per Study 1. For determination of stroke volume, cardiac output and total peripheral resistance, the raw blood pressure waveform from the Finapres was sampled at 200 Hz for off-line analysis using Beatfast, a component of Beatscope 1.1 (Finapres Medical Systems B.V., TNO TPD Biomedical Instrumentation, Amsterdam, The Netherlands). Beatfast uses the Modelflow method to compute stroke volume from an arterial pressure waveform (Jellema et al., 1999; Wesseling, Jansen, Settels & Schreuder, 1993); this is done by simulating a model of aortic input impedance, with aortic flow computed on a beat-to-beat basis. Two elements of the model, aortic characteristic impedance and windkessel compliance, are derived from known aortic pressure-area relations (their dependence upon pressure makes the model non-linear), which are related to age, sex, height and weight (participant characteristics which are entered into the model). Values for the third element, peripheral resistance, are dependent upon average flow; consequently (and with peripheral resistance changing only slowly with time) flow for a given beat is simulated using the value for peripheral resistance determined for the previous beat (with the first value assumed). Integration of flow during systole yields stroke volume, from which cardiac output and total peripheral resistance can be calculated.

Respiration rate and skin conductance were both obtained as per Study 1, the only

exception being the use of an electrode paste specifically designed for skin conductance recordings: Medsafe TD-246 (Med Associates Inc., St. Albans, VT). Automated brachial sphygmomanometry was conducted using a Dinamap Plus Vital Signs Monitor (Critikon Inc., Tampa, FL) (the accuracy of this device had been checked against manually obtained measurements of blood pressure). Physiological signals (and trial start/stop markers) were conditioned and recorded on a PC with Labview 6.1.

Procedure

In an attempt to reduce the variability in skin conductance found in Study 1, all procedures in the current study were conducted in a climate-controlled laboratory. This is because both temperature and humidity may affect skin conductance recordings (Venables & Christie, 1980); average (±SE) conditions were 23.4 (±0.1) °C and 35.1 (±0.7) % humidity. Participants were asked to refrain from caffeine, alcohol and exercise in the two hours preceding the experiment.

After participants had washed their hands with soap and water the physiological measurement devices were attached; the Finapres cuff was positioned at heart level by supporting the participant's forearm and hand with a pillow on the desk at which they were seated. This was followed by computer-presented explanations of the mental arithmetic and anagram problems and procedural instructions for the experiment. Two practice trials of each problem type were then undertaken. A trial began when the participant pressed the "Enter" key, after being prompted to do so on the monitor. Participants had been instructed to press "Enter" again upon having a solution, at which point the screen went blank, and to then say their answer. The trial ended if there was no response within 45 s. Verbal responses from the participant were recorded by the experimenter, who was isolated behind a partition. No feedback was given regarding

responses during the experiment. To minimise any physiological effects associated with vocalising a response there was a five second delay after a response (or absence of response) was recorded before the next trial began.

Following the completion of practice trials the Finapres blood pressure trace was monitored for five minutes to allow for stabilisation (Imholz et al., 1998). Because the Finapres device can be sensitive to small changes in how the finger cuff is applied (Jones, Kornberg, Roulson, Visram & Irwin, 1993) Finapres values were compared with a brachial sphygmomanometer measure of blood pressure; discrepancies were corrected as much as possible by adjusting the Finapres cuff (tightening/loosening or moving it slightly along the finger). The participant then rested for a further five minutes; the average value of physiological measures during the last two minutes of this period served as baseline. Magazines (National Geographic, Time, The Bulletin) were available to the participants during rest periods.

Participants were presented with a 16-trial block of anagrams and a 16-trial block of mental arithmetic problems (stimuli were randomly selected from the relevant item pool); the presentation order of anagram and mental arithmetic problem blocks was counterbalanced across participants (and within gender). There was a three-minute rest period between problem blocks.

Following completion of the final trial of each problem block, instructions on the monitor asked the participant to rate both how stressful and how difficult they found the task from 0 to 10, where 0 = "not at all", and 10 = "extremely"; responses were entered using the keyboard.

Data treatment and analysis

For all physiological measures, the mean levels for individual trials were averaged (weighted by trial solution latency) for both mental arithmetic and anagrams. Respiration rate was unable to be determined for one participant due to a particularly noisy recording. Absolute values for stroke volume, cardiac output and total peripheral resistance calculated using the Modelflow method are unlikely to be accurate unless calibrated against invasive techniques (Remmen et al., 2002). While changes in these measures from baseline are frequently reported in the literature in terms of absolute values, Remmen et al. suggest the use of relative change scores in overcoming potential inaccuracies associated with such analyses. Adopting this recommendation, stroke volume, cardiac output and total peripheral resistance were expressed as a percentage of the relevant baseline prior to analysis. To demonstrate physiological reactivity during the performance of mental arithmetic and anagrams, task data were compared with baseline values using repeated-measures t-tests.

Stress and difficulty ratings for mental arithmetic were unavailable for one participant due to technical problems. To determine whether the mental arithmetic and anagram tasks were perceived as having been stressful and/or difficult, one-sample t-tests were used to compare the mean stress and difficulty ratings for each task against zero. Differences between mental arithmetic and anagrams in terms of the number of problems attempted, correct solutions provided and mean solution latencies were investigated using repeated-measures t-tests.

The first step in determining whether there were differences between mental arithmetic and anagrams for either physiological or subjective measures was to perform regression analyses. As per Study 1, this approach was taken to see if unequal durations spent

performing the tasks (rather than more inherent task characteristics) accounted for any differences between them (the potential for task duration to affect physiological reactivity was discussed in Chapter 2); a less conservative than normal alpha of 0.1 was adopted for this test so that any contribution from unequal durations would not be missed (and a difference in either physiological or subjective measures between the tasks erroneously attributed to inherent task differences). The independent variable for each analysis was a latency difference score, calculated for each participant as the mean solution latency for mental arithmetic subtracted from the mean solution latency for anagrams. The dependent variable was a mean reactivity (task value minus baseline) or subjective rating difference score, with the value for mental arithmetic subtracted from the value for anagrams; for each of cardiac output, respiration rate and task difficulty ratings an outlying difference score (greater than three standard deviations from the group mean) was not included in the analysis. If the latency difference score predicted differences in physiological reactivity or subjective ratings between the tasks, the coefficient (B) for the constant of the regression equation (i.e., the predicted difference if the tasks were of a similar duration) was used as the measure of task differences. If the regression equation was not statistically significant (i.e., the latency difference score did not predict differences in physiological or subjective measures between the tasks), task differences were ascertained using a single-sample t-test to compare the reactivity or subjective measure difference score (the difference between the anagram and mental arithmetic tasks) with a value of zero.

Relationships between stress and difficulty ratings, and between each of these variables and physiological reactivity scores were investigated using partial correlations that controlled for solution latency (thus ensuring that any relationships identified were not simply the product of a variable task duration); these were conducted separately for the

mental arithmetic and anagram tasks. One participant had a heart rate reactivity score for mental arithmetic that was more than three standard deviations higher than the group mean; this was not included in the correlation analyses.

Data are presented as mean (SD). All statistical tests were performed using SPSS 11.5. An alpha of 0.05 or less was taken to indicate statistical significance (except for the regression analyses, as explained above). Alpha is calculated to three decimal places by SPSS: an output value of ".000" is stated here as $p < .001$.

Results

Task performance and subjective measures

The average number of mental arithmetic problems for which a solution was provided was 15.3 (1.5), with 12.7 (3.0) problems answered correctly. For anagrams these values were 9.5 (3.1) and 8.9 (3.4) respectively. Both the number of problems attempted and correct solutions provided were significantly higher for mental arithmetic than anagrams, $t(37) = 12.41$, $p < .001$ and $t(37) = 6.27$, $p < .001$ respectively. Furthermore, the mean solution latency for mental arithmetic problems, 19.7 (7.1) s, was less than the mean solution latency for anagrams, 27.4 (7.8) s, $t(37) = 7.75$, $p < .001$.

Difficulty ratings were 5.4 (1.8) and 7.0 (1.9) for mental arithmetic and anagrams respectively, with both tasks considered difficult (values significantly greater than zero, $p < .001$). Differences in task difficulty ratings were predicted by the latency difference score, $F(1, 34) = 28.68$, $p = .001$, meaning anagrams were rated as more difficult than mental arithmetic in direct proportion to the extra time spent in attempting to solve anagrams (see Fig. 3.1). However, even in controlling for this there was a trend for

anagrams to be rated as more difficult than mental arithmetic problems (B = 0.55, t = 2.14, p = .053).

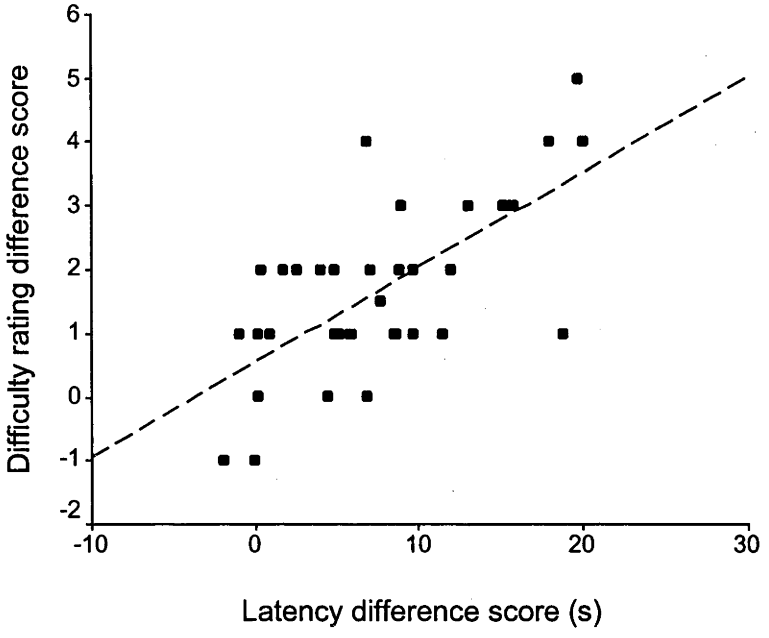

Figure 3.1. The relationship between (a) differences in difficulty ratings given to mental arithmetic and anagrams and (b) differences in mean solution latency for these tasks; the line of best fit is shown. Difference scores refer to anagrams minus mental arithmetic.

Mean ratings of task-related stress were 5.1 (2.2) for mental arithmetic and 5.7 (1.9) for anagrams; both tasks were perceived as stressful (the mean ratings for each were greater than zero, $p < .001$). In comparing stress ratings given for mental arithmetic and anagrams the latency difference score predicted differences in perceived stress, $F(1, 35) = 3.00$, $p = .092$; this indicates that, relative to mental arithmetic, the more time that was spent on the anagram task the greater was the stress rating provided (see Fig. 3.2). When this relationship was taken into account, there was no difference in perceived stress between mental arithmetic and anagrams (B = 0.00, t = -0.01, p = .993).

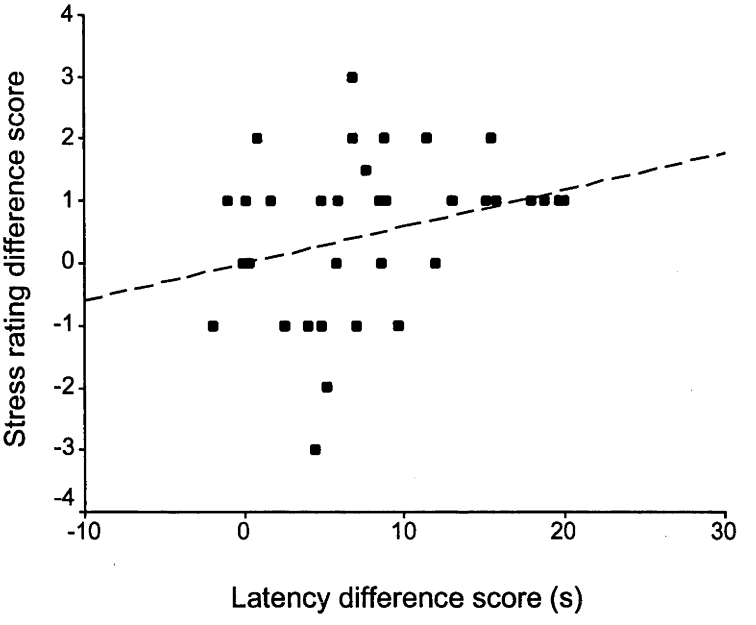

Figure 3.2. The relationship between (a) differences in stress ratings given to mental arithmetic and anagrams and (b) differences in mean solution latency for these tasks; the line of best fit is shown. Difference scores refer to anagrams minus mental arithmetic.

Physiological data

As shown in Table 3.1, systolic and diastolic blood pressure, heart rate, cardiac output, total peripheral resistance, skin conductance and respiration rate all increased above baseline levels during attempts to solve both mental arithmetic and anagram problems. Stroke volume was greater than at baseline during the performance of mental arithmetic; the difference in stroke volume between baseline and during anagrams did not attain statistical significance ($p = .085$).

Table 3.1

Mean (SD) values for all physiological measures

	Baseline	Mental arithmetic	Anagrams
Systolic b.p. (mmHg)	118.3 (14.3)	129.7 (16.4)***	125.5 (17.6)***
Diastolic b.p. (mmHg)	66.4 (9.1)	74.7 (10.6)***	72.0 (11.0)***
Heart rate (beats/min)	80.6 (12.0)	84.0 (13.2)***	82.4 (13.5)*
Skin cond. (μmho)	2.4 (1.5)	3.1 (1.8)***	2.9 (1.7)***
Resp. rate (breaths/min)	16.2 (3.1)	17.9 (3.37)***	17.4 (3.17)**
Stroke volume (%)	-	102.6 (8.0)*	102.0 (7.0)
Cardiac output (%)	-	107.2 (11.5)***	104.3 (9.8)*
Total periph. rest. (%)	-	105.8 (11.0)**	104.5 (9.2)**

Note. For stroke volume, cardiac output and total peripheral resistance, data shown is the percentage of the baseline value (which is indicated by a dash). Comparisons vs. baseline: df = 37 (except for respiration rate, df = 36), *p < .05, **p < .01, ***p < .001.

In comparing the physiological reactivity associated with mental arithmetic and anagrams, for only total peripheral resistance did the latency difference score predict task differences, $F(1, 36) = 3.78$, $p = .06$ (see Appendix B, Table B3 for all other F ratios), meaning that the more time spent on the anagram task, the greater the level of total peripheral resistance reactivity for anagrams relative to mental arithmetic (see Fig. 3.3). However, even in controlling for this, mental arithmetic was associated with a higher level of total peripheral resistance reactivity than anagrams, $B = -4.54$, $t = -2.16$, $p = .038$.

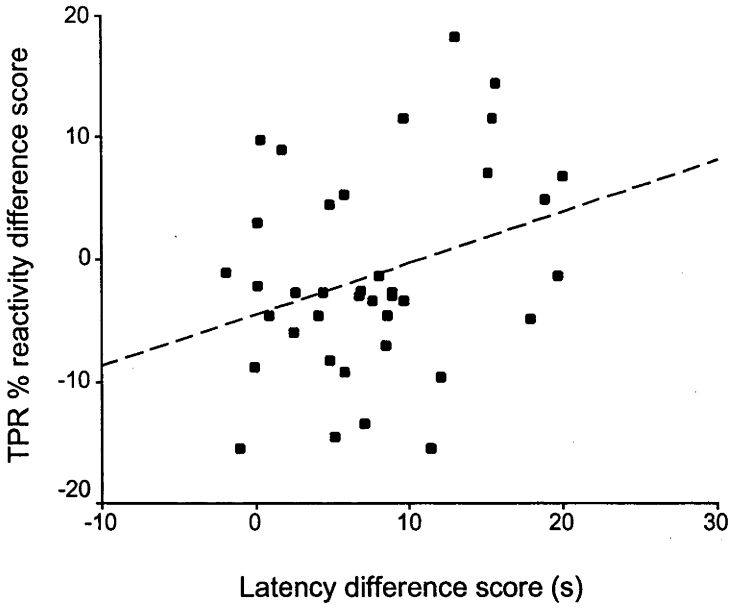

Figure 3.3. The relationship between (a) differences in total peripheral resistance (TPR) % reactivity during mental arithmetic and anagrams and (b) differences in mean solution latency for these tasks; the line of best fit is shown. Difference scores refer to anagrams minus mental arithmetic.

In addition to total peripheral resistance, and as shown in Figure 3.4, greater reactivity during mental arithmetic than during anagrams was found for systolic blood pressure, $t(37) = 4.03$, $p < .001$, diastolic blood pressure, $t(37) = 4.53$, $p < .001$, heart rate, $t(37) = 3.08$, $p = .004$, cardiac output, $t(36) = 2.11$, $p = .042$, and skin conductance, $t(37) = 3.67$, $p = .001$. There was a trend for respiration rate to increase more during mental arithmetic than during anagrams, $t(35) = 1.95$, $p = .059$, though no difference in stroke volume reactivity between the two tasks, $t(37) = 0.71$, $p = .483$.

Relationships between physiological reactivity and subjective ratings

The ratings of task difficulty given by participants were proportional to their ratings of the stress associated with task performance, as indicated by the partial correlation

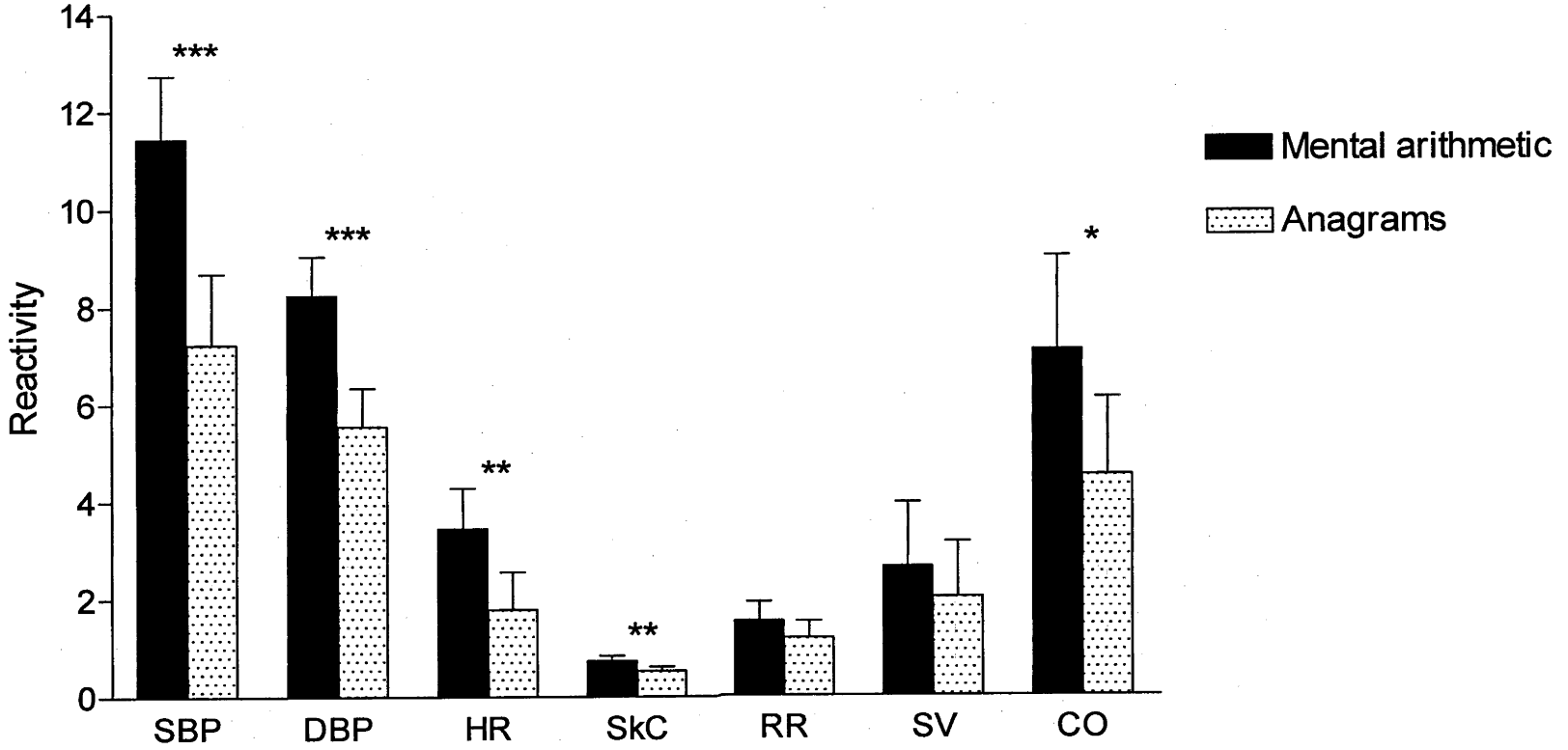

Figure 3.4. Mean (+SE) physiological reactivity (change from baseline) during anagram and mental arithmetic tasks. Units of measurement are: systolic blood pressure (SBP) and diastolic blood pressure (DBP): mmHg; heart rate (HR): beats/min; skin conductance (SkC): μmho; respiration rate (RR): breaths/min; stroke volume (SV) and cardiac output (CO); %. Significant differences between anagrams and mental arithmetic: *p < .05, **p < .01, ***p < .001.

results for anagrams $r(35) = 0.41$, $p = .013$, and mental arithmetic, $r(34) = 0.57$, $p < .001$.

Table 3.2

Partial correlation coefficients (controlling for solution latency) for relationships between subjective ratings and physiological reactivity scores

	Stress		Difficulty	
	Mental arith.	Anagrams	Mental arith.	Anagrams
Systolic b.p.	0.44**	0.26	0.19	-0.02
Diastolic b.p.	0.52**	0.45**	0.27	0.01
Heart rate	0.29	0.28	-0.03	-0.07
Skin cond.	0.07	-0.06	-0.09	-0.02
Resp. rate	0.13	0.07	0.09	-0.03
Stroke volume	0.10	-0.10	0.01	-0.03
Cardiac output	0.32	0.11	-0.01	-0.08
Total periph. rest.	-0.01	0.20	0.20	0.09

Note. For mental arithmetic df = 34, for anagrams df = 35 (for both tasks df is one less for respiration rate); **$p < .01$.

As shown in Table 3.2., increases in diastolic blood pressure varied in direct proportion to the ratings of stress given to both mental arithmetic and anagrams. A similar relationship existed between systolic blood pressure and stress ratings for mental arithmetic, though not for anagrams. There was a trend for changes in cardiac output to be related to ratings of stress for mental arithmetic ($p = .06$), though no relationship between these variables for anagrams. Relationships between heart rate reactivity and ratings of stress did not reach conventional levels of significance for either mental arithmetic ($p = .081$) or anagrams ($p = .088$). No relationships between reactivity scores

and stress ratings were found for stroke volume, total peripheral resistance, skin conductance or respiration rate. For both mental arithmetic and anagrams, there were no significant correlation coefficients to indicate a relationship between ratings of difficulty and reactivity scores for any physiological measure (see Table 3.2).

Discussion

In this study, physiological reactivity was measured during the performance of both mental arithmetic and anagram tasks. Participants were asked to provide ratings of difficulty and perceived stress for these tasks; this was done to determine whether either of these factors accounted for the greater level of physiological reactivity found for mental arithmetic than for anagrams in Study 1. Greater levels of physiological reactivity were also found for mental arithmetic problems than for anagrams in the current study. As mental arithmetic was not rated as more difficult than anagrams, and because the level of psychological stress associated with performing each of these tasks was similar, it is likely that intrinsic task characteristics are responsible for the observed differences in physiological reactivity. This was important to clarify because in the context of the global sympathetic system introduced in Chapter 1, relatively low intrinsic physiological reactivity (of sympathetic origin) could be taken to indicate relatively low central noradrenergic activity during attempts to solve anagrams; if so, this could be related to the finding of Study 1 that anagram task performance was better when participants were supine than when they were standing, given that locus coeruleus activity is hypothesised to be greater when standing (e.g., Elam et al., 1984).

As outlined in Chapter 1, the modulation of cortical arousal by posture (e.g., Cole, 1989) can be explained as an extra-homeostatic effect of baroreceptor activity (with

reason to believe that this involves modulation of central noradrenergic activity). Extra-homeostatic effects of baroreceptor activity are an important component of a theory of learned hypertension, which proposes that an increase in blood pressure activates baroreceptors, which in turn (via cortical effects) reduces the aversiveness of a stressful situation (Dworkin, 1988). However, this idea contrasts with previous reports that psychological stress and changes in blood pressure are directly related (e.g., Callister et al., 1990; Lepore, 1995); the same relationship was found in the current study, with there being a positive correlation between changes in blood pressure and the ratings of psychological stress provided for both mental arithmetic and anagram tasks (the implications of this for the theory of learned hypertension are discussed later).

Differences in physiological reactivity between mental arithmetic and anagrams

In general, there was greater physiological reactivity associated with performing mental arithmetic than with performing the anagram task. With regards to systolic and diastolic blood pressure and heart rate, this is consistent with the findings of Study 1 (depending upon postural condition). Larkin et al. (1998) also found greater heart rate and systolic blood pressure reactivity associated with mental arithmetic than with anagrams (though an effect of high social threat in the mental arithmetic condition cannot be excluded as contributing to the greater physiological reactivity). Unlike Study 1, skin conductance reactivity was also higher during mental arithmetic than during anagrams in the current study; the difference with Study 1 may be because of reduced variability in the skin conductance recordings in the current study (e.g., standard deviations of 6.2 μmho and 1.8 μmho associated with mental arithmetic in Study 1 and the current study respectively). Reduced variability can be attributed to a reduction in the development of peripheral recording artefacts, including those that may arise from an increase in conduction area (Lykken, 1970) caused by electrode slippage during movement (as

probably occurred when participants changed postures in Study 1); laboratory climate control and the use of a more appropriate electrode paste in the current study are also likely to have been beneficial. For example, a sodium chloride based electrolyte (as used in Study 1) may produce exaggerated readings (Venables & Christie, 1980); indeed, the values for skin conductance in the current study were far lower than those recorded in Study 1 (e.g., a baseline value of 2.4 μmho compared with 11.1 μmho for the supine condition baseline of Study 1), and are more in keeping with the mean normative values published by Venables and Christie. While there was a trend for respiration rate to increase more during mental arithmetic than during anagrams, the difference was only half a breath per minute; a similar difference was observed between mental arithmetic and anagrams in the standing condition of Study 1, however the smaller group size in that study meant that statistically the finding was of much less significance.

With regard to physiological measures that were not obtained in Study 1, there was a greater level of cardiac output reactivity associated with mental arithmetic than with anagrams; this was due largely to differences in heart rate, given that there was no difference in stroke volume reactivity between the two tasks. There was also greater total peripheral resistance reactivity associated with mental arithmetic than with anagrams. This result was found even after controlling for an effect of unequal task durations, which manifested as less total peripheral resistance reactivity for mental arithmetic relative to anagrams in direct proportion to the amount of time that a participant spent on the anagram task beyond the mental arithmetic task duration. A relationship between task duration and total peripheral resistance reactivity is consistent with the finding reported by Kelsey et al. (1999) that, though total peripheral resistance fell initially, it soon rose back to baseline and then continued to rise as more time was

spent performing a cognitive task.

Ratings of task difficulty and perceived stress

Previous research has shown that physiological reactivity may vary in relation to the difficulty of a cognitive task and/or the psychological stress associated with task performance; for example, Callister et al. (1992) found that blood pressure increased in proportion to both the difficulty of mental arithmetic problems and the ratings of stress that they generated. This implies that differences in reactivity between cognitive tasks could be due to concomitant differences in difficulty or perceived psychological stress.

Regression analysis showed that anagrams were rated as more difficult than mental arithmetic in direct proportion to the more time spent performing them. This effect of task duration on difficulty ratings is readily explained by task duration being an indicator to participants of their performance on the task (either directly, or because task duration was related to the number of problems that were solved, given that an unsolved problem remained displayed to participants for the maximum trial duration). A similar association with differences in task duration was observed for ratings of stress, however this relationship was substantially reduced in comparison to that for difficulty and may simply be due to there being a positive correlation between ratings for difficulty and ratings for stress (for both mental arithmetic and anagrams). The positive correlations between ratings for difficulty and stress are consistent with the notion that task difficulty is a determinant of the stress associated with performing a cognitive task (Callister et al., 1992).

When the effects of differences in task duration were accounted for, it was found that (a) participants rated the mental arithmetic and anagram tasks as similarly stressful, and

(b) there was a trend for ratings of difficulty to be greater for anagrams than for mental arithmetic. This latter finding is consistent with the objectively greater difficulty for anagrams than for mental arithmetic: there were a greater number of responses (including correct responses) given and a shorter mean solution latency for mental arithmetic problems than for anagrams. Cognitive tasks that are overly difficult may lead to participants disengaging from attempts to solve them and thus remove the impetus for physiological reactivity; this was thought to be the case for van Schijndel et al. (1984) finding lower blood pressure reactivity for an anagram set containing 30% solvable problems than for a set containing 50% solvable problems. With regard to the current study, it is unlikely that anagrams were so difficult that participants disengaged from trying to solve them, as responses were given on 59% of trials and subjective ratings of difficulty were only marginally higher than those given to mental arithmetic. Thus, because mental arithmetic was neither more difficult nor more stressful than anagrams, differences in task difficulty and/or perceived stress do not appear to be responsible for there being greater physiological reactivity during the performance of mental arithmetic than during the performance of the anagram task.

Differences in mental effort may underlie differences in physiological reactivity between mental arithmetic and anagrams

As described in Chapter 1, a global sympathetic system co-ordinates peripheral and central sympathetic activity in preparing an organism to interact with the environment or cope with stress (Aston-Jones et al., 1994). In this context, with the primary central sympathetic nucleus being the locus coeruleus (Moore & Bloom, 1979), greater cardiovascular and skin conductance reactivity could be taken to imply greater activity of the locus coeruleus during attempts to solve mental arithmetic problems than during attempts to solve anagrams. There being a relatively minimal role of locus coeruleus

activity in the neurophysiological (and cognitive) processes normally associated with performing an anagram task could be related to an impairment of the ability to solve anagrams when central noradrenergic activity is elevated. This has been suggested by experiments where central noradrenergic activity is manipulated: pharmacologically (Beversdorf et al., 2002), in relation to carry-over effects of sleep state (Walker et al., 2002) or by posture; as shown in Study 1 by there being faster solution latencies for anagrams when supine than when standing (with locus coeruleus activity hypothesised to be greater when standing, e.g., Elam et al., 1984). There are reasons for thinking that both the observed lower levels of physiological reactivity for anagrams (cf. mental arithmetic) in this study and in Study 1, and the inferred relatively low locus coeruleus activity, stem from there being less mental effort expended in attempts to solve anagrams. Discussion of mental effort in relation to peripheral activity follows, with discussion in relation to central activity (and implications of this) presented in a later section.

Brain scanning experiments have shown that during effortful mental arithmetic, blood flow in the anterior cingulate cortex increases, and does so in direct proportion to increases in blood pressure; changes in anterior cingulate blood flow and blood pressure during an effortless control task (e.g., simple counting) were both significantly less than during the effortful task (Critchley, Corfield, Chandler, Mathias & Dolan, 2000). Furthermore, patients with lesions of the anterior cingulate cortex exhibit reduced blood pressure and heart rate reactivity (but no difference in cognitive ability) when engaged in mental tasks (cf. controls, Critchley et al., 2003). These findings have led to the conclusion that the anterior cingulate cortex is involved with the generation of peripheral autonomic arousal in line with behavioural needs (e.g., in accordance with the amount of mental effort being expended, Critchley et al.). Consistent with a role in

generating physiological activity, electrically stimulating a particular region of the anterior cingulate has been shown to increase blood pressure in the rat (Burns & Wyss, 1985). Furthermore, the idea that activity of the anterior cingulate cortex is proportional to mental effort is supported by Raichle et al. (1994), who found that the anterior cingulate was one of the cortical regions in which blood flow decreased as the performance of a verbal task became more automated through practice.

The studies by Critchley et al. (2000, 2003) suggest that the physiological reactivity associated with performing a cognitive task may be reflected in the level of anterior cingulate cortex activity, which in turn is influenced by the amount of mental effort expended. In contrast to findings for mental arithmetic (e.g., Critchley et al., 2000), Schneider et al. (1996) reported that, though there were increases in some regions of the cortex, blood flow in the anterior cingulate did not change from baseline levels during the performance of an anagram task very similar to that used in the current study (solvable, five-letter problems, each with a maximum solution time of 45 s). Thus, there being less physiological reactivity for anagrams than for mental arithmetic in the current study and Study 1 could be due to there being less anterior cingulate cortex activity associated with anagrams. This would be consistent with there being less mental effort expended in attempts to solve anagrams than in attempts to solve mental arithmetic problems; there are at least two lines of evidence in support of this idea.

Performance on a mental arithmetic task has been shown to be reduced when a second task (e.g., a working memory task) is also performed (Furst & Hitch, 2000; Trbovich & LeFevre, 2003). However, while observing a performance decrement on an analytical task when a second task (counting auditory stimuli presented at varying intervals) was undertaken, Lavric, Forstmeier and Rippon (2000) found that performance of an insight

problem solving task (primarily the candle problem) was the same whether it was performed by itself or in conjunction with the second task. This indicates that insight problem performance is less affected than mental arithmetic performance by an increase in mental effort demands, suggesting that insight problems require less mental effort than mental arithmetic (given a finite mental effort capacity). Similarly, a relatively minimal amount of mental effort is likely to be expended in attempts to solve anagrams, given that they are a type of insight problem (Novick & Sherman, 2003, and as outlined in Chapter 2). Evidence for this extrapolation could be sought by conducting an experiment in which a secondary task is simultaneously performed during attempts to solve anagrams; it would be expected that the solution latency for anagrams performed alone would be no different (or less affected than those for mental arithmetic problems) to when the secondary task is also performed.

In addition to dual-task studies, subjective reports of the problem solving experience support the idea that there may be relatively little mental effort expended in attempts to solve anagrams. While participants have described periods of conscious mental effort (e.g., attempts to restructure the letters), they have also described periods of mental inactivity when trying to solve anagrams, as relayed by the statement "I didn't do anything but just looked at the letters waiting for the solution to come" (Hunter 1959, p. 205). Furthermore, reports by participants in the Hunter study indicated that it is during such periods of mental inactivity that the solution to an anagram often becomes known. This is consistent with other subjective reports of simply seeing the solution (which would appear to require minimal mental effort) and of approaching the anagram problem by just "looking for" a solution before trying any particular conscious strategy (Mendelsohn & O'Brien, 1974, p. 573).

In comparison to performing a mental arithmetic task, there is reason to think that less mental effort is expended when engaged in attempts to solve anagrams. Mental effort, via associated activity in the anterior cingulate cortex, has been shown to be involved in generating peripheral physiological reactivity. Accordingly, the findings of the current study and Study 1 that there were lower levels of physiological activity during anagram task performance than during mental arithmetic task performance could be because of relatively less mental effort being expended in attempts to solve anagrams.

Mental effort and subjective ratings

It might be expected that the amount of mental effort expended (reflected in activity of the anterior cingulate cortex) would be proportional to the perceived difficulty or stress associated with performing a cognitive task. Subsequently, there being less mental effort associated with anagrams than with mental arithmetic could be seen to be at odds with the trend for anagrams to be rated as more difficult than mental arithmetic and for the two tasks to be rated as similarly stressful. However, as noted by Dehaene, Kerszberg and Changeux (1998), "mental effort is clearly unrelated to objective measures of computational difficulty: we routinely perform vision and motor control tasks without awareness of the complex underlying information processing, whereas elementary tasks such as solving 37 – 9 call for our attention and conscious effort" (p. 14529). It is likely that cognitive factors contributed to the participants' ratings of difficulty and stress given to the tasks in the current study. Failure, either real or perceived, can generate psychological stress (Fisher, 1984); similarly, failure would be expected to be an important factor in perceptions regarding the difficulty of a cognitive task. Thus, given that, on average, 59% of anagrams were responded to (cf. 96% for mental arithmetic), perceptions of failure could have contributed to the ratings of stress and/or difficulty assigned to anagrams. Furthermore, as reported for anagrams, there are

periods of mental inactivity during attempts at finding a solution (Hunter, 1959); some of these periods could occur because the participant is unsure about how to solve the problem. This is a characteristic of insight problems, in which an impasse is often reached (Bowden, 1997), though not of mental arithmetic, where the approach required to solve the problem is more structured and readily apparent to the participant. It is likely that not knowing how to approach solving a problem will increase a participant's perception of its difficulty. Thus, cognitive factors may have influenced the ratings of difficulty and stress that participants assigned to anagrams (and mental arithmetic); this could have resulted in an enhancement of values for anagrams beyond those that might be expected on the basis of mental effort expenditure alone.

Conscious mental effort and the moment of anagram solution

A relatively low level of mental effort appears to be expended in attempts to solve anagrams; this is indicated by subjective reports (Hunter, 1959; Mendelsohn & O'Brien, 1974) and supported by the low levels of physiological reactivity associated with anagram task performance (cf. mental arithmetic) observed in the current study and Study 1. Furthermore, there is reason to think that somewhat paradoxically, solutions to anagrams may be favoured to arise in the absence of conscious mental effort.

As previously shown (in Study 1 and by Beversdorf et al., 1999, 2002; Walker et al., 2002) conditions in which central noradrenergic activity is minimised (or reduced) are associated with a relatively improved ability to solve anagrams; a mechanism to account for this was discussed in Chapter 2. In essence, centrally released noradrenaline may suppress the activation level of widespread cell assemblies; these are groups of neurons that represent cognitive nodes, discrete elements of cognition such as individual words. Attempts to solve an anagram are likely to be less effective when cell assemblies/cognitive nodes that contain solution relevant information are suppressed.

There are reasons for thinking that mental effort is associated with an increase in central noradrenergic activity. It has been shown across a range of tasks that mental effort is directly proportional to pupil diameter (Beatty, 1982), which in turn is known to reflect central noradrenergic activity (as shown with drugs that act via alpha$_2$-adrenoceptors, Phillips et al., 2000). The major source of noradrenaline in the central nervous system is the locus coeruleus (Moore & Bloom, 1979), and indeed, changes in pupil diameter during the performance of cognitive tasks are thought to reflect changes in locus coeruleus activity (Gilzenrat et al., 2003); this idea is supported by studies (described below) that have demonstrated the presence of projections from the cerebral cortex to the locus coeruleus.

Projections to the locus coeruleus have been demonstrated to arise from the medial prefrontal cortex in rats (Jodo, Chiang & Aston-Jones, 1998) and from the orbital prefrontal (Aston-Jones et al., 2002) and anterior cingulate cortices in monkeys (Rajkowski, Lu, Zhu, Cohen & Aston-Jones, 2000). Both electrical and chemical stimulation of the medial prefrontal cortex (corresponding to the dorsolateral prefrontal in primates) have been found to produce excitation of cells in the locus coeruleus (Jodo et al.). Projections to the locus coeruleus provide support for the notion that certain cortical regions are able to modulate locus coeruleus activity and thereby modulate behaviour (e.g., Rajkowski et al.); this would be expected to include behaviours that are particularly influenced by central noradrenergic activity.

In addition to direct modulation of the locus coeruleus, the anterior cingulate cortex could affect the turnover of noradrenaline in multiple cortical regions by an indirect mechanism. As revealed by brain scans in humans, there is a network of central nervous system structures involved in regulating intrinsic (i.e., in the absence of any imposed

change) alertness or arousal; structures included in this network are the anterior cingulate cortex, the thalamus and a region of the ponto-mesencephalic tegmentum that may contain the locus coeruleus (Paus et al., 1997; Sturm et al., 1999). Sturm et al. have proposed that in the absence of an external signal (e.g., a warning stimulus) intrinsic alertness may be regulated by signals from the anterior cingulate allowing noradrenergic activation originating in the ponto-mesencephalic tegmentum to pass through the thalamus and onto the cortex.

There is evidence indicating that activity within the anterior cingulate cortex can regulate central noradrenergic activity. Regardless of the mechanism (i.e., whether it involves an action at either the locus coeruleus or thalamus), the general principle is consistent with findings that anterior cingulate activity is directly proportional to cardiovascular activity (Critchley et al., 2000, 2003). The reason for this is that both the central (locus coeruleus) and peripheral (cardiovascular) outcomes of anterior cingulate activity dovetail within the context of the global sympathetic system, in which peripheral and central sympathetic activity is co-ordinated (Aston-Jones et al., 1994). Thus, via activity in the anterior cingulate, mental effort is likely to initiate activity of the global sympathetic system.

There are consequences that follow from the idea that mental effort, via the anterior cingulate, is associated with an increase in cortical noradrenaline turnover (possibly due to increased locus coeruleus activity). In many circumstances an elevation in central noradrenergic activity during mental effort would be a useful mechanism for regulating the neurophysiological conditions that facilitate behaviour, providing a basis for the focussed attention requirements of a vigilance task for example (Rajkowski et al., 1998). When the behaviour involves solving anagrams, an increase in central

noradrenergic activity is likely to suppress information that is not being focussed upon. In other words, the activation level of those cell assemblies/cognitive nodes that do not possess the characteristic (letter grouping etc.) of the current solution strategy will be suppressed. Ceasing to focus on a particular strategy entails a reduction in mental effort. This, in turn, is likely to be associated with a reduction in anterior cingulate cortex activity and thereby (via decreased activity of the locus coeruleus and/or the closing of thalamic gates) a reduction in cortical noradrenaline turnover. The subsequent disinhibition of widespread cell assemblies may raise the activation level of the cognitive node representing the solution word. When summed with any lingering activation (i.e., that which has accumulated during the previous attempts to find a solution), this may be sufficient to reach the activation threshold needed for the node's information to be made available to consciousness. Thus, somewhat paradoxically, the solution to an anagram may arise in the absence of conscious mental effort (except in cases where a solution strategy is directly successful). This idea gels with the distillation of anecdotal accounts by Beversdorf et al. (1999) concerning insight experiences, "where a difficult problem is repeatedly approached with effort, only to have the solution come later at a moment of rest" (p. 2767).

Along similar lines to the proposed basis for anagram solutions being favoured to arise when mental effort is low, Heilman et al. (2003) have suggested that relative inactivity of the anterior cingulate (and the dorsolateral prefrontal cortex) may reduce locus coeruleus firing rates, thereby facilitating the neurophysiological conditions necessary for creative thought. Because locus coeruleus activity is related to cortical arousal (e.g., Foote et al., 1980) this idea is consistent with demonstrations of an inverse relationship between creativity test performance and cortical arousal, as altered by white noise (Martindale & Hines, 1975) or measured in terms of EEG alpha power (Martindale,

1977). Furthermore, a low level of anterior cingulate cortex activity, and thereby a low level of locus coeruleus activity and/or cortical noradrenaline turnover, could account for the findings of lower cortical activation (e.g., more alpha power in the EEG spectrum) and higher dimensional complexity of the EEG (reflecting a greater number of active cell assemblies) during creative thinking than during analytical thinking (Molle et al., 1996; Molle, Marshall, Wolf, Fehm & Born, 1999).

Relationships between mental effort and solving anagrams (or in relation to creative thinking tasks) might be clarified using measures of pupil diameter. As discussed above, pupil diameter increases in line with the expenditure of mental effort (Beatty, 1982); a mechanism thought to be mediated by central noradrenergic activity (Gilzenrat et al., 2003). Furthermore, pupil diameter reflects mental effort with high temporal resolution (Beatty). Fluctuations in pupil diameter would be anticipated to occur during attempts to solve anagrams, reflecting the periods of both conscious mental effort (during which time solution strategies are consciously engaged) and relative inactivity that have been described in subjective reports of the solution process (Hunter, 1959). If solutions to anagrams are favoured to arise when mental effort is low, it could be expected that the moment of solution will occur during (or be immediately preceded by) a period in which pupil diameter is relatively small (i.e., when central noradrenergic activity is relatively low). However, it may be the case that only solutions participants report as occurring in a moment of insight are associated with periods of low conscious mental effort, given the idea that insight is facilitated when central noradrenergic activity and arousal are low (Beversdorf et al., 1999).

Haemodynamic profiles associated with performing mental arithmetic and anagram tasks

In Study 1, blood pressure increased during the performance of both the mental arithmetic and anagram tasks. However, while there was also an increase in heart rate associated with mental arithmetic, this was not the case for anagrams. This suggested that the haemodynamics responsible for the blood pressure changes might have differed between the two tasks; more specifically, that mental arithmetic was associated with a cardiac output (or mixed cardiac output/peripheral resistance) profile and the anagram task with a peripheral resistance profile.

In addition to the increases in systolic and diastolic blood pressure, heart rate, skin conductance and respiration rate that were also observed in Study 1, mental arithmetic was associated with elevations in stroke volume, cardiac output and total peripheral resistance. Some previous studies (e.g., Gregg et al., 1999) have found that mental arithmetic has a cardiac output haemodynamic profile while others have observed a mixed cardiac output/peripheral resistance profile (e.g., Ring et al., 1999). Kelsey et al. (1999) demonstrated that total peripheral resistance can fall initially (i.e., in the first minute) during the performance of a mental arithmetic task but then rise back to and beyond baseline levels as more time is spent on the task; thus, an effect of mental arithmetic on peripheral resistance (and therefore a mixed haemodynamic profile) is more likely to be found the longer the task duration. Indeed, participants in the Gregg et al. study (in which a change in peripheral resistance was not found) performed the mental arithmetic task for two and a half minutes; for Ring et al. (who did find a peripheral resistance effect) the task duration was eight minutes. In the current study, the time participants spent on the task (an average of nearly seven minutes) and the findings of increases in both cardiac output and total peripheral resistance are consistent

with the report by Ring et al. of a mixed haemodynamic profile for mental arithmetic.

Associated with the anagram task in the current study were increases in systolic and diastolic blood pressure, skin conductance and respiration rate (as per Study 1). There was also an increase in heart rate; though this was not found in Study 1 it has often been reported for anagrams (e.g., van Schijndel et al., 1984). While some other studies have exerted greater time pressure on the participants performing an anagram task (e.g., by presenting an anagram every five seconds), a factor which has been shown to be associated with increased heart rate reactivity (Wahlstrom, Hagberg, Johnson, Svensson & Rempel, 2002), the findings of the current study suggest that heart rate reactivity could have been expected for the supine postural condition of Study 1 (it being less likely for the standing condition given the pre-existing parasympathetic withdrawal from the heart, e.g., Cacioppo et al., 1994). However, half of the participants performing the anagram task while supine in Study 1 did so having already been exposed to the task while standing. Repeated exposure to a task (i.e., reduced novelty) has been shown to decrease heart rate reactivity (e.g., Kelsey et al., 1999); this may have prevented the development of statistically significant heart rate reactivity during performance of the anagram task in Study 1 (with a similar situation not arising for mental arithmetic due to the greater overall level of heart rate reactivity associated with that task). Though there was no change in stroke volume, attempts to solve anagrams were associated with increases in both cardiac output and total peripheral resistance; thus the haemodynamics underlying changes in blood pressure were essentially the same as those for mental arithmetic (i.e., a mixed cardiac output/peripheral resistance profile). Therefore, differences in physiological reactivity between mental arithmetic and anagram problems were simply quantitative in nature, with there being no distinction between the tasks in terms of the haemodynamic profile underlying task-associated increases in blood

pressure.

Relationships between physiological reactivity and perceived stress: possible mechanisms

The nature of a relationship between perceived stress and physiological reactivity during the performance of a cognitive task is unclear, due to the mixed findings of previous studies. For example, Callister et al. (1990) reported that blood pressure (though not heart rate) increased in line with increases in perceived stress. Lepore (1995) reported positive correlations between perceived stress and systolic and diastolic blood pressure, though not heart rate, while Freyschuss et al. (1988, 1990) found that both cardiac output and heart rate (though not blood pressure) co-varied with ratings of stress. In the current study, a positive correlation was found between perceived stress and diastolic blood pressure reactivity for both the anagram and mental arithmetic tasks. A similar, though weaker, relationship was found between perceived stress and systolic blood pressure for mental arithmetic only. There not being a similar relationship between systolic blood pressure and stress for anagrams could simply be due to a restricted range, given the smaller level of systolic blood pressure reactivity for this task (which, as described earlier, may be due to there being less mental effort expended in attempts to solve anagrams than in attempts to solve mental arithmetic, given a demonstrated relationship between mental effort and blood pressure reactivity, Critchley et al., 2000, 2003). In addition to the relationships between blood pressure and perceived stress, there was a trend for cardiac output and stress to be correlated (for mental arithmetic); the correlations between heart rate and stress did not reach conventional levels of significance. Unlike perceived stress, ratings of task difficulty were not correlated with changes in any of the physiological variables for either mental arithmetic or anagrams; this is consistent with the idea of Carroll et al. (1992) that the

psychological impact of a task (measured in the current study as perceived stress) has a stronger relationship with physiological reactivity than does task difficulty.

There are a number of possible reasons why psychological stress and blood pressure responses have been found to be associated in a positive manner. Such a relationship could simply be inferred from the presence of a global sympathetic system (Aston-Jones et al., 1994), given that stress is related to central noradrenergic activity (e.g., Van Bockstaele et al., 2001). However, underlying the associative nature of this relationship could be changes in regional brain activity, similar to the previously discussed relationship between mental effort and peripheral physiological activity. As with that relationship, one of the brain regions potentially involved in a relationship between mental stress and physiological activity is the anterior cingulate cortex.

As outlined earlier, the anterior cingulate cortex is thought to regulate peripheral physiological activity in line with behavioural state or needs (Critchley et al., 2000, 2003). In addition to mental effort, this appears to include psychological stress, as indicated by a positive correlation between blood flow in the anterior cingulate and subjective distress induced by social exclusion (created when a participant was no longer passed the ball by others in a virtual throwing game, Eisenberger, Lieberman & Williams, 2003). Furthermore, Liberzon et al. (1999) found that exposure to combat sounds (explosions, gunfire etc.) was associated with increases in anterior cingulate blood flow, peripheral sympathetic activity (heart rate and skin conductance) and subjective distress. As discussed previously, central noradrenergic activity is associated with aversive psychological states, including stress (e.g., Stanford, 1995). Thus, anterior cingulate cortex activity could facilitate psychological stress experiences via the projections from this region to the locus coeruleus (Rajkowski et al., 2000), or by enhancing the passage of ascending noradrenergic activity through the thalamus (Sturm

et al., 1999).

Negative affective states have been found to involve co-activation of the anterior cingulate cortex and the amygdala in humans (e.g., for fear, LaBar, Gatenby, Gore, LeDoux & Phelps, 1998; Pissiota et al., 2003). The amygdala has also been shown to be activated during emotional stress in animals (e.g., in restrained rats, Dayas, Buller & Day, 1999). Furthermore, activity in the amygdala is necessary for increases in blood pressure that accompany periods of emotional stress in the rat (Kubo et al., 2004). Therefore, activity in the amygdala may also be involved in the development of a relationship between perceived stress and blood pressure during the performance of a cognitive task.

The insular cortex is another brain region in which activity co-varies with peripheral physiological activity. Cameron and Minoshima (2002) measured regional brain activation that occurred in association with the administration of isoproterenol, a largely peripheral acting beta-adrenoceptor agonist. Isoproterenol raised heart rate considerably (from 68 beats/min to 122 beats/min) and induced significant levels of activity in the somatosensory, medial cingulate and insular cortices. Blood pressure was not measured in the Cameron and Minoshima study, however changes in both blood pressure and heart rate have been shown to co-vary with changes in activity of the insular cortex during both mental stress (Critchley et al., 2000) and physical manoeuvres (e.g., isometric handgrip, Critchley et al.; King, Menon, Hachinski & Cechetto, 1999). This co-variation could be (at least in part) due to peripheral feedback, with insular cortex activity thought to be influenced by peripheral afferents both directly and via other regions of the cortex that also receive peripheral afferents (Craig, 2003). Furthermore, the insular cortex has been shown to be activated during internally generated (e.g., via

recall) negative emotional states and is thought to be involved in generating emotional experiences that are associated with distressing cognitions or internal sensations (Reiman et al., 1997). A more general role for the insular cortex as an important region by which changes in the physiological state of the body can contribute to affective processes has also been proposed (Craig, 2003; Critchley, Wiens, Rotshtein, Ohman & Dolan, 2004). Understanding precisely how activity in the insular cortex representing bodily state contributes to subjective experiences will require further work; however, the insular cortex does send projections to the amygdala (as demonstrated in monkeys, Mufson, Mesulam & Pandya, 1981).

The correlation between changes in blood pressure and ratings of psychological stress found to be associated with the performance of both mental arithmetic and anagram tasks in the current study could have been a product of activity in any one (or a combination) of the anterior cingulate cortex, the amygdala and the insular cortex. With regards to the anterior cingulate and amygdala, the relationship may be due to a common origin for changes in blood pressure and feelings of stress. Regarding the insular cortex, feeling of psychological stress may develop in association with activity in this region that represents the peripheral physiological state. This idea is similar to the general concept proposed over a century ago by James (1884) and Lange (1885/1912), in which changes in bodily state are thought to have a crucial role in the production of emotions, an idea discussed more recently by Damasio (e.g., 2003), and which appears to have some support in the results of brain scanning experiments. Regardless of the mechanism(s) underlying the relationship between blood pressure and perceived stress found in the current study, the moderate values of the correlation coefficients (0.44 - 0.52) indicate that perceived stress is associated with factors other than blood pressure (e.g., cognitive factors, as noted above); the moderate coefficients

also fit with there being similar levels of perceived stress, though dissimilar levels of blood pressure reactivity, between different cognitive tasks (as found in the current study).

Relationships between physiological reactivity and perceived stress: comparisons with other studies

The positive correlations between perceived stress and changes in blood pressure observed in the current study are similar to the finding reported by Callister et al. (1992) that perceived stress increased in proportion to changes in both systolic and diastolic blood pressure during the performance of mental arithmetic and Stroop colour word test tasks; they are also consistent with the findings of Lepore (1995), who reported positive correlations between both systolic ($r = 0.23$) and diastolic ($r = 0.27$) blood pressure (though not heart rate) and the perceived stress associated with giving a speech (the coefficients found in the current study were higher than those reported by Lepore, possibly because of differences in either the tasks or stress measures used). However, as previously noted, other studies have produced different findings: Maier et al. (2003) reported that there were no correlations between perceived stress and a range of cardiovascular variables (including blood pressure) for a mental arithmetic task, though this could be because the task was not perceived as being very stressful (i.e., there was a restricted range). Freyschuss et al. did not find a correlation between perceived stress and blood pressure reactivity but did find relationships between stress and other measures, including cardiac output, vascular resistance (1988, 1990) and heart rate (1990). Heart rate and cardiac output reactivity were far greater in the Freyschuss et al. studies than in the current study (e.g., for heart rate, around 136% vs. 105% of baseline values). It could be argued that a relatively restricted range in the current study contributed to there being no correlations between cardiac variables and perceived stress

at conventional levels of statistical significance (though there was a trend for cardiac output and stress to be correlated the coefficient was considerably lower than that found for blood pressure, diastolic blood pressure in particular). However, a restricted range does not explain why Freyschuss et al. did not find a correlation between blood pressure and stress, with the level of blood pressure reactivity being fairly equivalent with that of the current study.

Factors directly associated with cardiac activity, including stroke volume, cardiac output and pulse pressure, provide somatosensory cues (i.e., pulsations in the periphery) that facilitate conscious awareness of heartbeat (O'Brien, Reid & Jones, 1998). Also, it is thought that the representation of heart rate in the insular cortex is available to conscious awareness; this is based on a finding that blood flow in the anterior cingulate cortex was correlated with performance on a heart beat detection task (Critchley et al., 2004). In contrast to heart rate, evidence suggests it is unlikely that people have any accurate and conscious awareness of changes in their blood pressure. For example, it has been reported that while participants were able to adjust their workload on an exercise bike to match heart rates achieved in earlier sessions, this was unable to be done when the task was to match blood pressure levels (Kollenbaum, Dahme & Kirchner, 1996). Furthermore, Fahrenberg, Franck, Baas and Jost (1995) found that there were no significant correlations between participants' estimations of their own blood pressure levels and multiple ambulatory measurements of their actual systolic or diastolic blood pressure.

Thus, while relationships between stress and blood pressure would appear to be a product of unconscious processes, those between stress and cardiac variables may be influenced by conscious awareness. This is because a conscious awareness of changes

in bodily state is likely to influence participants' perceptions of the stress associated with performing a task (perhaps owing to preconceptions regarding relationships between cardiac activity and stress) and as this awareness applies to cardiac variables, may facilitate the development of a positive relationship between perceived stress and cardiac output (and/or heart rate); a relationship between stress and vascular resistance (as found by Freyschuss et al., 1988, 1990) may then be a result of vascular resistance being mathematically derived from cardiac output. The chances of awareness of heart rate developing would be expected to be proportional to the magnitude of reactivity (e.g., conscious awareness of heart rate appears to be enhanced when heart rate is accelerated, as shown by the finding that elevations produced by exercise facilitated more accurate assessments of heart rate, Jones & Hollandsworth, 1981). It is possible that when changes in cardiac output are large, conscious awareness of these changes could interfere with, or over-ride, the development of an unconsciously generated relationship between blood pressure and stress; as cardiac reactivity was considerable in the Freyschuss et al. studies this could explain why they found correlations between perceived stress and both cardiac output and heart rate, though not between perceived stress and blood pressure (as was the case in the current study).

How does a positive relationship between blood pressure and stress fit with the idea that baroreceptor activity reduces stress?

As discussed in Chapter 1, information regarding blood pressure (and volume) status is gathered by baroreceptors, located in the aortic arch, carotid sinuses and cardiopulmonary regions, and relayed to the central nervous system by way of the nucleus tractus solitarius. Perturbations in blood pressure (and volume) alter baroreceptor activity and evoke a homeostatic mechanism that works to maintain a fairly constant blood pressure level by regulating peripheral autonomic activity (e.g.,

Mohrman & Heller, 2003). In addition, there is evidence for extra-homeostatic (i.e., not involved in blood pressure regulation) effects of baroreceptor activity, including modulation of cortical arousal. For example, carotid baroreceptor stimulation has been found to induce sleep in dogs (Koch, cited in Rau & Elbert, 2001) and to be associated with less cortical negativity in humans (in whom stimulation can be produced by negative pressure in a neck cuff, Elbert et al., 1988). As an extension of these findings it is thought that an increase in blood pressure, by virtue of baroreceptor stimulation, can reduce feelings of psychological stress (Schweizer et al., 1991). This is an important mechanism in Dworkin's (e.g., 1988) theory of learned hypertension, which proposes that the cortical dampening and reduced aversiveness that are thought to occur when blood pressure rises in a stressful situation facilitate, by a process of reinforcement, a chronic increase in blood pressure. However, a rise in blood pressure being associated with a decrease in psychological stress would appear to be inconsistent with the positive relationship between changes in blood pressure and perceived stress associated with the performance of cognitive tasks found in the current and other studies (Callister et al., 1992; Lepore, 1995). There is also an inconsistency with the idea of parallel changes in peripheral and central sympathetic activity that occur in relation to the global sympathetic system (Aston-Jones et al., 1994). In this context, a rise in peripheral sympathetic activity (including that leading to an increase in blood pressure) would be expected to be concomitant with an increase in stress (which in itself is associated with increased locus coeruleus activity, e.g., Van Bockstaele et al., 2001).

For blood pressure to rise when a cognitive task is performed, a change in blood pressure must go unopposed by a compensatory baroreflex. This situation can occur because either the sensitivity of the baroreflex is reduced, or the pressure level that the baroreflex works to maintain (the operating point of the baroreflex) is raised; it is the

latter mechanism which seems to be the most important, given findings that baroreflex sensitivity is not reduced during mental stress (Fauvel et al., 2000). Similarly, an elevation in the operating point of the baroreflex occurs during physical exercise; which is thought to be achieved by a centrally mediated reduction in nucleus tractus solitarius activity for any given level of baroreceptor signalling (DiCarlo & Bishop, 2001).

Effectively, when the operating point of the baroreflex is reset, the new blood pressure level does not invoke compensatory cardiovascular mechanisms that would be invoked were the same load on baroreceptors produced by exogenous means that do not involve the operating point of the baroreflex being reset (e.g., neck cuff stimulation). It could be expected that the same might apply to the extra-homeostatic effects of baroreceptor activity; meaning for example that the higher blood pressure level will not have the inhibitory effect on the cortex that it would have in the absence of operating point resetting. In keeping with this idea, there is some EEG evidence that cortical arousal increases (rather than decreases) during exercise: Kubitz and Mott (1996) reported that alpha activity decreased and beta activity increased when participants rode on an exercise bike (though blood pressure was not reported and other factors could contribute to cortical arousal, e.g., muscular or respiratory activity). Indeed, it seems counter-intuitive that an elevation in blood pressure associated with performing a cognitive task would lead to a reduction in cortical arousal, especially when the task itself is likely to be cortically arousing; there is some evidence to confuse this point however.

Schweizer et al. (1991) found that the psychological stress associated with performing a mental arithmetic task was exacerbated by a peripherally acting beta-blocker (atenolol). It was thought that this effect was because atenolol prevented the usual task-related rise in systolic blood pressure, thereby preventing an increase in baroreceptor activity and an

associated cortical dampening action. However, performance on the task was not reported, leaving open the possibility that an effect of atenolol on the ability to perform the task could have contributed to the higher stress rating. Also, as previously discussed in Chapter 2, the acute administration of a beta-blocker may (theoretically) lead to an increase in cortical noradrenaline turnover; this could occur in association with a baroreflex that increases total peripheral resistance to maintain blood pressure following a decrease in cardiac output (due to beta-blockade of cardiac receptors). Indeed, in the Schweizer et al. study, atenolol significantly reduced heart rate though did not alter baseline blood pressure (cf. placebo), indicating that an increase in total peripheral resistance is likely to have occurred. Given the relationship between psychological stress and central noradrenergic activity (e.g., Stanford, 1995), an associated extra-homeostatic increase in central noradrenergic activity could possibly have facilitated the higher stress ratings associated with atenolol administration; a related effect was outlined in Chapter 2, in which it was suggested that, via an increase in central noradrenergic activity, peripheral beta-adrenoceptor blockade may lead to an impaired ability to solve anagrams (in relation to Beversdorf et al., 1999, 2002).

In another study, Foster and Harrison (2004) measured the blood pressure of participants who had been asked to recall an event in which they had been angry. The changes in blood pressure produced by this task were found to be negatively correlated with changes in the amplitude of EEG alpha band activity recorded at widespread recording sites. This is consistent with an increase in cortical arousal being associated with an increase in blood pressure during the performance of a cognitive task. However, a negative correlation was found between changes in blood pressure and changes in low beta (13-21 Hz) magnitude at numerous sites (including central, parietal and occipital); the data from this EEG band could be used to argue that an increase in blood pressure

was associated with a decrease in cortical arousal. Previously, Foster and Harrison (2002) had found that changes in low beta magnitude were directly related to participants' ratings for the intensity of the recollected memory of anger. Taken together, these studies could be seen to suggest that the intensity of an emotional experience (in this case anger) may be inversely related to changes in blood pressure, which in turn could be viewed as analogous to the idea that an increase in blood pressure can reduce feelings of psychological stress (Schweizer et al., 1991), and in a more general sense, reduce the aversiveness of a stressful situation (Dworkin, 1988).

It remains unclear whether or not extra-homeostatic effects of baroreceptor activity are altered in situations where the operating point of the baroreflex is reset so that blood pressure is maintained at a higher than normal level, as for example during the performance of mental tasks. In relation to this, the positive correlations found between blood pressure and perceived stress in the current and Lepore (1995) studies would appear to be in contrast to the idea that an elevation in blood pressure (via baroreceptor activity) decreases psychological stress (Schweizer et al., 1991). Regardless, it has been suggested that it is only in susceptible (e.g., genetically) people that hypertension develops because of a reinforcing cortical dampening effect accompanying an increase in blood pressure during a stressful situation (Dworkin; Rau & Elbert, 2001). If this is so, it is possible that there is a dissociation between the blood pressure maintenance and extra-homeostatic effects of baroreceptor activity in these cases. This idea would be supported if it were to be found that people (assessed when clinically normotensive) who exhibit either a relatively small increase or a decrease in cortical arousal during cognitive (or physical) tasks are at greater than normal risk of developing hypertension.

A study to investigate the relationship between cortical arousal during stressful tasks

and the risk of developing hypertension could be conducted along similar lines to one reported by Rau and Elbert (2001), in which the inhibitory effect of carotid baroreceptor stimulation (manipulated using the neck cuff method) on the pain produced by electrical stimulation was assessed in 120 people. This strength of this effect was positively correlated with the change in blood pressure that occurred over a two-year period, which was primarily a result of an effect in the subgroup of participants who reported experiencing high daily stress. While this is consistent with the idea that susceptibility to the cortical dampening effects of baroreceptor activity, combined with repeated exposure to stress, may result in hypertension, it does not show (or address) the basic mechanism postulated to account for this: that a rise in blood pressure (or baroreceptor activity) associated with a stressful situation reduces the level of psychological stress or aversiveness experienced.

Findings that blood pressure reactivity and psychological stress are positively related do not detract from the idea that baroreceptor activity has the potential to reduce feelings of stress. This relationship may emerge under conditions in which changes in baroreceptor activity are imposed without the operating point of the baroreflex being reset. Underlying this reasoning are findings that increases in baroreceptor activity produced by exogenous means lead to reductions in cortical arousal; for example, stimulation of carotid baroreceptors by neck suction has been shown to reduce slow brain potential negativity (Elbert et al., 1988). As stress occurs concomitantly with central noradrenergic activity (e.g., Stanford, 1995), the idea that a baroreceptor-mediated reduction in cortical arousal is associated with a reduction in psychological stress is reinforced by findings that an increase in baroreceptor activity results in a decrease in cortical noradrenaline turnover (e.g., as shown using blood volume loading in rats by Persson & Svensson, 1981). The potential for baroreceptor activity to modulate feelings

of stress is investigated in Study 3 (Chapter 4). Ratings of perceived stress (and the related negative affective state of anxiety) were obtained from participants who performed a stressful mental arithmetic task in both standing and supine conditions; as per Study 1, these were considered to be associated with relatively low and high baroreceptor (particularly cardiopulmonary) activity respectively.

CHAPTER 4

STUDY 3

Introduction

As body posture becomes more upright (from supine to sitting to standing, or in terms of the degree of upwards tilt from horizontal), the level of cortical arousal typically increases; this is demonstrated by both electrophysiological and behavioural evidence. For example, there is less EEG theta band activity (Vaitl & Gruppe, 1990) and larger evoked potentials to visual stimuli (Wei et al., 1992) when participants are tilted towards upright than when supine or tilted head down. Consistent with these findings, reaction times have been found to be faster when standing than when seated or supine (Vercruyssen & Simonton, 1994).

The effects of posture on cortical arousal may be a product of changes in baroreceptor activity. In a more upright posture, blood is drawn by gravity from the upper parts of the body towards the lower parts. Consequently, the load on baroreceptors in major arteries and cardiopulmonary regions is reduced, this decreases baroreceptor activity and in turn leads to an increase in the level of peripheral sympathetic activity that prevents blood pressure from being compromised (e.g., Mohrman & Heller, 2003). In addition, changes in baroreceptor activity have been shown to have extra-homeostatic effects (effects that are not directly associated with blood pressure maintenance); these include modulation of cortical arousal. In particular, an increase in baroreceptor activity has been demonstrated to reduce cortical arousal in both animals and humans (see Vaitl & Gruppe, 1991).

In addition to the demonstrated changes in cortical arousal, it is believed that

baroreceptor activity reduces anxiety (Dworkin et al., 1994). Furthermore, it is thought that, via enhanced baroreceptor activity, an increase in blood pressure reduces psychological stress (Schweizer et al., 1991); a potential problem with this idea was raised in Chapter 3, in which it was reported that the perceived stress associated with performing a cognitive task was positively correlated with changes in blood pressure (Study 2, this was also reported by Lepore, 1995). A positive relationship between blood pressure reactivity and psychological stress could involve the operating point of the baroreflex being reset (to allow blood pressure to rise when performing a cognitive task), though it is unclear whether or not the extra-homeostatic effects of baroreceptor activity (e.g., changes in cortical arousal) are also affected by operating point resetting. Regardless, this does not directly relate to the idea that an imposed increase in baroreceptor activity, that in itself does not involve operating point resetting, may decrease state anxiety and stress.

Though not featured in previous proposals that baroreceptor activity influences anxiety and stress, changes in cortical arousal associated with changes in baroreceptor activity may operate via the locus coeruleus (the core nucleus of a noradrenergic arousal system, Berridge & Waterhouse, 2003). As shown in animals, experimental manipulations that decrease the activity of baroreceptors enhance locus coeruleus firing rates (e.g., Elam et al., 1985), which in turn leads to increases in both cortical noradrenaline turnover (e.g., Persson & Svensson, 1981) and high frequency EEG activity (e.g., Page et al., 1993). Baroreceptor-mediated changes in cortical noradrenaline turnover are important to consider in relation to the idea that baroreceptor activity may modulate psychological stress and anxiety, because both stress (e.g., Stanford, 1995) and anxiety (e.g., Tanaka et al., 2000) are known to be associated with, and/or facilitated by, central noradrenergic activity.

The facts that baroreceptor activity affects the locus coeruleus, and that baroreceptor activity changes with posture, suggest that locus coeruleus activity is altered by posture (e.g., Elam et al., 1984). This conclusion may also be reached by considering that the locus coeruleus is the central component of a global sympathetic system; this system co-ordinates parallel peripheral and central sympathetic activity in preparing an organism to interact with the environment or cope with stress (Aston-Jones et al., 1994). Thus, increased locus coeruleus activity when standing could be expected on the basis of there being greater peripheral sympathetic activity in that posture (e.g., Mohrman & Heller, 2003).

Consistent with the idea that locus coeruleus activity is greater when in a more upright posture, there is evidence that psychological processes or behaviours regulated by central noradrenergic activity are also regulated by posture. Performance on a vigilance task has been shown in monkeys to be facilitated when locus coeruleus activity increases from low levels (Rajkowski et al., 1998), and to be better in sleep-deprived humans when standing than when seated (Caldwell et al., 2003). Furthermore, the ability to solve anagrams is facilitated under conditions in which central noradrenergic activity is low (Beversdorf et al., 1999, 2002; Walker et al., 2002); in Study 1 it was found that performance on an anagram task was better when participants were supine than when they were standing. Given the relationship between both psychological stress and anxiety and central noradrenergic activity, it could be expected that a cognitive task will generate less stress and anxiety when performed in a less upright posture (i.e., when baroreceptor activity is increased and activity of the noradrenergic arousal system thought to be reduced). This idea is investigated in the current study, in which participants were asked to give ratings of perceived stress and anxiety immediately after performing a stressful mental arithmetic task in both standing and supine conditions.

The persistence of any effects was investigated by obtaining similar ratings ten minutes after the task had been completed. Finding that subjective ratings of stress and/or anxiety were lower in the supine condition than in the standing condition would give further support to the idea that psychological phenomena regulated by (or associated with) the noradrenergic arousal system are modulated by posture. Such a result would also be in keeping with the idea that baroreceptor activity decreases psychological stress and anxiety, an important component of Dworkin's (1988) theory of learned hypertension, though for which there is currently little evidence.

In Study 1 there was a hint that the anxiety normally associated with performing mental arithmetic (e.g., Schweizer et al., 1991) was greater when participants were standing than when they were supine. This is because skin conductance (though not heart rate) increased in the standing condition, while heart rate (though not skin conductance) increased in the supine condition, during mental arithmetic. A rise in skin conductance in the absence of a positive change in heart rate has been suggested to imply activation of the behavioural inhibition system (Fowles, 1980), which in turn is associated with the production of anxiety (e.g., Gray, 1982). However, it is not clear whether the rise in skin conductance in the standing condition of Study 1 resulted directly from performing mental arithmetic or was simply a time-related increase (similar to that found by McGrady et al. (2001) for upright tilted patients). There are two ways of determining the degree to which an increase over time could have contributed to the finding that skin conductance was elevated during mental arithmetic while standing. One of these is to obtain measurements during a recovery period that follows the task, to see if there is a return in skin conductance towards baseline levels. Another is to have a control period in which the same postural condition is adopted though in which a task is not performed. Both of these approaches were undertaken in the current study; the

information that may be revealed was considered to be important not only for skin conductance, but for all of the physiological and subjective measures. This is because there may be instability or changes in other physiological measures that occur over time associated with particular postures (e.g., Jacob et al. (1998) found that after adopting a standing posture systolic blood pressure decreased during the first 7.5 minutes before slowly returning to initial levels over the remainder of a 60 minute session) and because there is virtually nothing known about the effects of posture per se on psychological ratings; anxiety ratings have been obtained for different postural conditions (supine, seated and standing) previously, however the results of this study did not yield any clear effects (Hennig et al., 2000).

It was anticipated in Study 1 that skin conductance would be higher under resting conditions in the standing condition than in the supine condition. This is because there is more sympathetic activity (e.g., as demonstrated by greater diastolic blood pressure) and a higher level of cortical arousal (e.g., as suggested by faster reaction times, Vercruyssen & Simonton, 1994) when standing, and because skin conductance is an indicator of both sympathetically mediated (Critchley, 2002) and cortical (e.g., Barry et al., 2004) arousal. There was no difference in resting skin conductance found between the standing and supine conditions in Study 1, however this finding could have been influenced by methodological problems (including inappropriate electrode paste and electrode slippage during changes in posture); these were addressed in the current study and differences in resting skin conductance between standing and supine again investigated. Given that skin conductance responses are reliant upon an intact central noradrenergic system (as shown in cats by Yamamoto et al., 1990), finding that skin conductance is higher when standing would be an additional piece of evidence in support of there being greater noradrenergically-mediated cortical arousal in a more

upright posture (e.g., Elam et al., 1984).

The effects of posture on physiological reactivity to mental stress were also investigated in the current study. In Study 1, while there were similar levels of blood pressure reactivity to mental arithmetic in the standing and supine postures, there was heart rate reactivity only in the latter. Because changes in blood pressure are a result of changes in cardiac output (the product of heart rate and stroke volume) and/or total peripheral resistance (determined by the degree of vasoconstriction in peripheral vessels) this suggests that there was relatively greater stroke volume and/or total peripheral resistance reactivity when standing than when supine. Rusch et al. (1981) found that vascular resistance in the forearm decreased less in response to (contralateral) isometric handgrip when standing than when supine; also in that study however, forearm vascular resistance decreased to a similar extent when mental arithmetic was performed in both standing and supine postures. The implications from the Rusch et al. study for total peripheral resistance are unclear, given that responses in the forearm may not be indicative of changes in other vascular beds; for example, it was also found that vascular resistance in the calf was unchanged during the performance of mental arithmetic (and another mental test) in the supine position (calf vascular resistance was not measured while standing). Differences in the total peripheral resistance, stroke volume and cardiac output responses to mental stress between standing and supine conditions were investigated in the current study. Understanding any differences in the haemodynamics underlying changes in blood pressure may have relevance for understanding any differences in psychological stress or anxiety associated with task performance in the standing and supine conditions.

Methods

Participants

Sixteen female and four male healthy volunteers, with a mean age of 20.6 years (the age range was 18 to 33 years), provided data for the study. Mean (±SE) height and weight were 165.0 (±1.4) cm and 58.0 (±2.4) kg for the females, and 179.6 (±2.8) cm and 80.8 (±5.9) kg for the males. Because state anxiety responses to laboratory stressors may be related to trait anxiety, participants were screened with the trait form (Y) of the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983). The mean STAI score for included participants was 37.0 (the range was 27 to 54); data from one participant with a trait anxiety score of 73 was replaced, as this score was greater than three standard deviations from the mean of relevant Australian normative data (Creamer, Foran & Bell, 1995). Partial data from an additional two participants who developed pre-syncopal symptoms while standing (and who reported having not eaten on the day of testing) were also not included. Participants provided informed consent before the study, which was approved by the Australian National University Human Research Ethics committee. Course credit was received for time spent.

Physiological measures

Measures of systolic and diastolic blood pressure, heart rate, skin conductance, respiration rate, stroke volume, cardiac output and total peripheral resistance were obtained in the same manner as for Study 2.

Mental arithmetic task

Participants performed a mental arithmetic task based on one demonstrated to induce both physiological and subjective indicators of stress (Schweizer et al., 1991). Instructions were to alternatively subtract thirteen and add five from a starting number

for two minutes as quickly and accurately as possible, voicing the response after each mathematical operation. An incorrect response resulted in being asked to begin again from the starting number, which was either 800 or 850 (counterbalanced across postural conditions) and not revealed until the task was to begin. A microphone was placed before the participant just prior to performing the task and removed immediately afterwards; performance was audio-recorded to enable accurate determination of responses. Participants practiced the task prior to experimental procedures beginning, starting from 660, until able to provide, without interruption, the first six responses in the sequence (647, 652, 639, 644, 631, 636).

Subjective measures

Participants were asked to provide subjective ratings of state anxiety and stress during the experiment, on each occasion being presented with written statements to which they were instructed to respond by circling an appropriate value. The statements were "How anxious you are feeling right now, that is, at this moment" and "How stressed you are feeling right now, that is, at this moment". Beneath each statement were the numbers 0 to 10 (on a single line), with the anchor point statements "Not at all anxious (or stressed)" and "Extremely anxious (or stressed)" above the 0 and 10 respectively. These self-report measures were followed by the six statements comprising the short state form of the STAI (as developed by Marteau and Bekker (1992) from the state form of the STAI): "I feel calm", "I am tense", "I feel upset", "I am relaxed", "I feel content" and "I am worried". Instructions are to respond on a 4-point scale, 1 ("Not at all"), 2 ("Somewhat"), 3 ("Moderately so") or 4 ("Very much so") with regard to "How you feel right now, that is, at this moment" (from which the structure for the self-reported anxiety and stress statements was taken). An additional statement was included on occasions immediately following the mental arithmetic task: "How difficult the mental

task is" responded to on an 11-point scale from 0, "Not at all difficult", to10, "Extremely difficult". All statements to be responded to on a given occasion (i.e., anxiety, stress, short form of the STAI, and difficulty if applicable) were included on a single sheet of paper, referred to here as a subjective measures sheet.

It is not uncommon for multiple measures of state anxiety to be obtained (e.g. Wilhelm & Roth, 1998) and as it is not always the case that similar information is provided (e.g., Man et al., 2003) it seems particularly prudent to use multiple measures under conditions where state anxiety responses have received very little attention. The short form of the STAI has been shown to produce similar scores to the long form in samples with both normal and elevated state anxiety (Marteau & Bekker, 1992) though is more practical when fairly rapid or repeated anxiety measures are required; scores for the three anxiety-absent statements are reversed, with higher score totals representing higher state anxiety. The short form of the STAI has been used in a large number of studies and in a variety of situations, for example, to evaluate the success of strategies for reducing anxiety in dental patients (Dailey, Humphris & Lennon, 2002), and to measure the level of anxiety associated with performing a laboratory-based mental arithmetic task (Uchino, Holt-Lunstad, Uno & Flinders, 2001).

A simple 0-10 self-report scale has also proven to be a useful tool for rapidly assessing state anxiety; scores are highly correlated with Spielberger state anxiety scores (Benotsch, Lutgendorf, Watson, Fick & Lang, 2000; Houtman & Bakker, 1989) and with anxiety scores from the Hospital Anxiety and Depression Scale (Lampic, von Essen, Peterson, Larsson & Sjoden, 1996). A 0-10 scale has been used to measure anxiety in conditions ranging from imminent open-sea diving (Baddeley & Idzikowski, 1985) to follow-up medical visits (Nordin, Glimelius, Pahlman & Sjoden, 1996).

The use of a numerical self-report scale for psychological stress has been validated against measures of cortisol, a key indicator of the stress response (Miller & O'Callaghan, 2002). Salivary cortisol concentrations have been shown to be strongly related to self-reported stress prior to an academic examination (Ng, Koh & Chia, 2003); in fact, a numerical scale was found to have greater accuracy than questionnaire based assessments with regard to salivary cortisol levels (Pruessner, Hellhammer, Pruessner & Lupien, 2003). A 0-10 stress scale was previously used by Schweizer et al. (1991) to measure the level of psychological stress associated with performing a mental arithmetic task essentially the same as that used in the current study.

Procedure

The procedures were conducted in an air-conditioned laboratory; average (±SE) conditions were 23.0 (±0.1) °C and 40.9 (±0.9) % humidity.

Each participant attended two sessions; these were separated (not including test days) by an average of 4.5 days (the range was one to nine days). The mental arithmetic task was performed in one session (experimental) and not the other (control session); the order of experimental and control sessions was counterbalanced across participants. Both sessions contained a supine and a standing condition, with the order of these counterbalanced across all participants (though the same for a given participant in each session). One each of the four male participants experienced the four possible session/condition order combinations.

Similar procedural stages were followed for both the experimental and control sessions. After participants had cleaned their hands with soap and water the Finapres cuff and respiration belt were attached. The mental arithmetic task was explained and practiced

(if an experimental session). Participants read an outline of the procedures and associated timing for that session. Included in the outline were instructions to try and stay still and quiet while resting and to remain awake with their eyes open at all times; closed circuit television monitoring (camera positioned for a lateral view of the participant's face) enabled verification of compliance. During the procedures participants were separated from the experimenter and data collection computers by a partition.

Participants rested while seated for five minutes before either standing or adopting the supine position. In the standing condition, participants stood before a customised lectern (used for positioning additional apparatus); a padded stand for the forearm and hand was used to maintain the Finapres cuff at heart level. In the supine condition participants lay upon a mattress, their head supported by pillows and the Finapres cuff propped to heart level with cushions; the lectern was turned on its side and placed over (i.e., not contacting) the participant's waist (serving essentially as a low table).

Skin conductance electrodes were attached (only done once the participant was standing or supine to prevent the conductive paste spreading during movement from the seated position) and the Finapres started. The timeline of procedures that followed is shown in Figure 4.1. Participants rested for eight minutes; the first six minutes of this period were to allow for the participant to adjust to the new posture and for stabilisation of the Finapres signal (Imholz et al., 1998); the average value of physiological measures during the last two minutes of this eight-minute period served as baseline. Participants completed a subjective measures sheet; a sheet (on a support which facilitated use both when standing and when supine) and pencil were placed before the participant only when required and collected immediately after completion, the experimenter returned

behind the partition while the statements were responded to. Two further minutes were spent resting (this period gave the experimenter time to prepare for the task phase). Participants then performed the mental arithmetic task (experimental session) or rested (control session, and which was labelled P2) for two minutes, with a second subjective measures sheet completed immediately afterwards. Physiological data were averaged over the time spent performing the task or the corresponding control session period (i.e., P2). A further ten minutes were spent resting; physiological data were averaged within each of the first and second five minutes of this period (these recovery phases were labelled P3 and P4 respectively). A third subjective measures sheet was completed before the skin conductance electrodes were removed (and residual paste cleaned from the fingers). These procedures, starting with five minutes of seated rest, were then repeated using the alternate posture. After both the standing and supine conditions were concluded the Finapres and respiration belt were removed and the trait form of the STAI completed (if the second session).

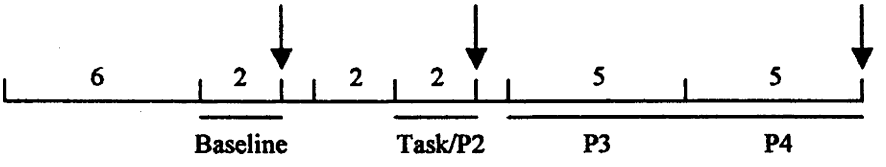

Figure 4.1. Timeline of procedures beginning once the participant was standing or supine; duration (in minutes) of stages and physiological measurement phases are shown; arrows indicate the completion of a subjective measures sheet.

Data treatment and analysis

For the mental arithmetic task, repeated-measures t-tests were used to compare the number of responses provided, errors made and task difficulty ratings between the

standing and supine conditions.

Experimental session blood pressure readings (and thus derived haemodynamic measures) were unavailable for two participants due to difficulties with the Finapres cuff (heart rate data from these sessions appeared to be normal and is included in the analyses). As per Study 2, stroke volume, cardiac output and total peripheral resistance were expressed as a percentage of the relevant baseline. Due to the vocal requirements, respiration rate was not analysed during the task (or corresponding control condition period, i.e., P2). STAI short form and self-reported anxiety data from a subject who had outlying scores (more than three standard deviations from the mean) for most baseline and recovery phases were not included in the analyses.

Differences between standing and supine control condition baselines were investigated. For systolic and diastolic blood pressure, heart rate, skin conductance and stress ratings this was done using repeated-measures t-tests. Anxiety rating comparisons were conducted using multivariate ANOVA (with both STAI short form and self-reported anxiety as measures) and univariate follow up analyses; this approach was taken for all analyses of anxiety ratings. For stroke volume, cardiac output and total peripheral resistance the standing value was expressed as a percentage of the supine value and analysed with a single sample t-test (test value = 100).

Analyses of the effects of mental arithmetic on physiological activity and subjective responses were conducted with the primary aim of investigating if changes from baseline developed during the task or recovery in either posture, and whether the magnitude of any changes differed between postures. To remove any changes associated purely with time, transformed data sets were produced for each posture by subtracting

control session values from task session values for corresponding measurement periods: for the physiological measures the measurement periods were baseline, task/P2, recovery phases P3 and P4; for the subjective measures the measurement periods were baseline, task/P2 and after recovery (see Fig. 4.1 for a timeline of the measurement periods). Each set of values was then adjusted by the amount required to zero the baseline. Comparisons were made between baseline and each later measurement period for each posture and between postures for each post-baseline measurement period. For each set of comparisons, either with a baseline or between postures, to control for the increased risk of type I errors with multiple comparisons p values were corrected using the Holm-Sidak step-down procedure (see Ludbrook, 1998). Briefly, p values for a set of m tests are ranked in ascending order and calculated as $p'_a = 1-(1-p_a)^{m-a+1}$, where a is the rank order of p (if required, the illogical situation of $p'a_{+1} < p'_a$ is prevented by assigning $p'_{a+1} = p'_a$).

Analyses were also conducted to investigate the stability of both the physiological and subjective measures during the control session using baseline versus later measurement period comparisons. Similar comparisons were conducted on the raw experimental session data to investigate whether the removal of control session data (as per the main analyses) had any effect on the analytical outcomes. The Holm-Sidak step-down procedure was applied within each set of comparisons.

Data are presented as mean (SD). All statistical tests were performed using SPSS 11.5. An alpha of 0.05 or less was taken to indicate statistical significance. Alpha is calculated to three decimal places by SPSS: an output value of ".000" is stated here as $p < .001$.

Results

Mental arithmetic task performance and perceived difficulty

The mean number of responses provided and errors made during the mental arithmetic task were 27.0 (2.6) and 1.6 (0.4) respectively in the standing condition and 25.0 (2.7) and 1.9 (0.4) respectively in the supine condition. There was a trend for more responses to be provided while standing than while supine, $t(19) = 1.98$, $p = .062$, though no difference in the number of errors made between postural conditions, $t(19) = 0.73$, $p = .474$. There was no difference in the mean difficulty ratings given for the standing, 5.3 (0.5), and supine, 5.1 (0.6), conditions, $t(19) = 0.48$, $p = .634$.

Ratings of stress and anxiety: raw data

Anxiety

The means of all anxiety ratings are displayed in Table 4.1. Control session baseline anxiety did not differ between the standing and supine conditions: multivariate (with both STAI short form and self-reported anxiety as measures), $F(2, 17) = 2.09$; STAI short form, $F(1, 18) = 2.37$; self-reported anxiety, $F(1, 18) = 0.16$ ($p > .05$ for all comparisons). For the following analyses see Appendix B, Table B4 for F-ratios. Anxiety did not differ from baseline at any later stage in the control session in either posture. In the standing condition of the task session, the difference in anxiety (multivariate) from baseline following the performance of mental arithmetic did not reach conventional levels of significance ($p = .076$); STAI short form anxiety increased significantly, though self-reported anxiety did not. Anxiety ratings provided after the recovery period in the standing condition were not different from those provided at baseline. In the supine condition, anxiety (multivariate) was elevated above baseline immediately after the mental arithmetic task, as were both STAI short form and self-reported anxiety; anxiety had returned to baseline levels after the recovery period.

Table 4.1

Mean (SD) values for subjective ratings

		Anxiety		Stress
		STAI short form	Self-reported (0-10 Scale)	Self-reported (0-10 Scale)
Standing				
Control	Base.	10.2 (2.3)	0.8 (0.8)	0.9 (0.9)
	P2	10.1 (2.5)	1.0 (0.9)	0.7 (0.9)
	Recov.	10.6 (2.8)	1.1 (1.2)	0.9 (1.3)
Task	Base.	11.7 (2.0)	2.2 (1.8)	1.4 (1.5)
	Task	13.3 (2.5)*	3.2 (2.8)	2.9 (2.3)**
	Recov.	10.8 (1.5)	1.7 (1.4)	0.8 (1.1)**
Supine				
Control	Base.	9.2 (3.3)	1.0 (1.1)	1.0 (1.4)
	P2	9.3 (3.1)	0.9 (1.1)	0.8 (0.9)
	Recov.	8.9 (2.8)	1.0 (1.2)	0.9 (1.3)
Task	Base.	9.3 (2.3)	1.2 (1.3)	1.1 (1.7)
	Task	12.5 (3.4)**	3.1 (2.3)**	3.5 (2.7)**
	Recov.	9.4 (2.5)	1.2 (1.3)	0.9 (1.4)

Note. Univariate comparisons vs. baseline: df = (1, 18), *p < .05, **p < .01.

Stress

The means of all stress ratings are displayed in Table 4.1. There was no difference in stress ratings between the standing and supine control session baselines, $t(19) = 0.15$, $p = .883$. Control session stress ratings were no different from baseline levels after P2 or after the recovery period in either posture. Stress ratings were elevated following mental

arithmetic in the task session for both the standing and supine conditions; after the recovery period, stress ratings fell below baseline in the standing condition, while returning to baseline levels in the supine condition (see Appendix B, Table B5 for t-ratios).

Ratings of stress and anxiety: transformed data

Anxiety

Transformed anxiety rating data and significant effects are depicted in Figure 4.2. In the standing condition, anxiety (multivariate) was elevated above baseline following the task, $F(2, 17) = 3.88$, $p = .041$; STAI short form anxiety was significantly higher, $F(1, 18) = 7.46$, $p = .014$; the increase in self-reported anxiety did not reach conventional levels of statistical significance ($p = .073$). After the recovery period in the standing condition, anxiety ratings were lower than at baseline: multivariate, $F(2, 17) = 8.96$, $p = .004$; STAI short form, $F(1, 18) = 10.26$, $p = .010$; self-reported, $F(1, 18) = 7.23$, $p = .030$. In an attempt to locate the source of this result, comparisons were conducted between the raw task and control session data both at baseline and after recovery. Baseline anxiety was higher in the task session than the control session: multivariate, $F(2, 17) = 8.60$, $p = 0.006$; STAI short form, $F(1, 18) = 8.98$, $p = 0.016$; self-reported, $F(1, 18) = 13.43$, $p = 0.004$; there was no difference between task and control conditions after the recovery phase (multivariate, $F(2, 17) = 1.40$; STAI short form, $F(1, 18) = 0.09$; self-reported, $F(1, 18) = 2.36$; $p > .05$ for all comparisons). These findings indicate that for the standing condition, ratings of anxiety prior to performing the mental arithmetic task were greater than at the equivalent time in the control session (in which the task was not performed).

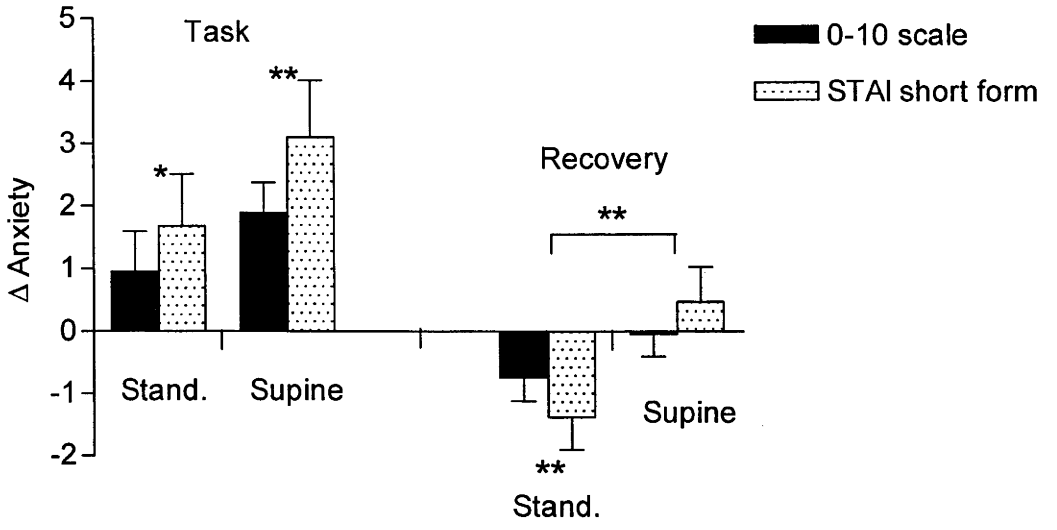

Figure 4.2. Mean (±SE) change in self-reported (0-10 scale) and STAI short form anxiety ratings from baseline, both immediately after mental arithmetic and following a recovery period, in the standing and supine conditions. Values represent transformed (task session – control session) data. Significant differences in anxiety (multivariate) from baseline, and between standing and supine: *$p < .05$, **$p < .01$.

In the supine condition, state anxiety was elevated after performing the mental arithmetic task: multivariate, $F(2, 17) = 12.36$, $p < .002$; STAI short form, $F(1, 18) = 26.47$, $p < .002$; self-reported, $F(1, 18) = 30.12$, $p < .002$. Anxiety had returned to baseline levels after the recovery phase: multivariate, $F(2, 17) = 0.26$; STAI short form, $F(1, 18) = 0.23$; self-reported, $F(1, 18) = 0.04$ ($p > .05$ for all comparisons). The increase in anxiety following mental arithmetic was not statistically different between the standing and supine conditions: multivariate, $F(2, 17) = 1.36$; STAI short form, $F(1, 18) = 2.29$; self-reported, $F(1, 18) = 2.07$ ($p > .05$ for all comparisons). However, the decrease in anxiety from baseline in the standing condition and the increase (non-significant) in the supine condition resulted in a significant difference in transformed anxiety scores between postures following the recovery period (multivariate, $F(2, 17) =$

9.48, p = .004; STAI short form, F(1, 18) = 10.81, p = .008; self-reported, F(1, 18) = 1.43, p > .05). This finding is consistent with the elevated anxiety observed prior to the task in the standing condition not developing in the supine condition.

Stress

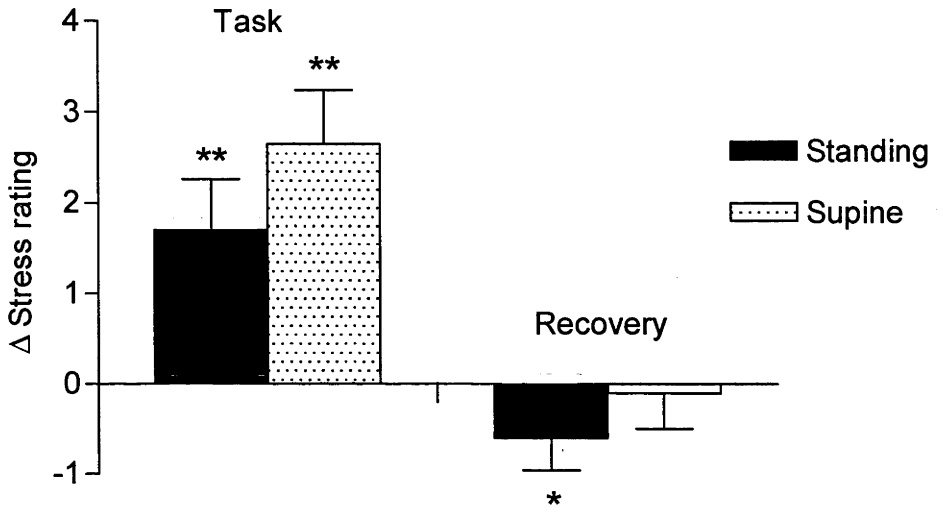

Figure 4.3. Mean (±SE) change in stress ratings (from baseline) after mental arithmetic, both immediately and following a recovery period, in standing and supine conditions. Values represent transformed (task session – control session) data. Significant differences from baseline: *p < .05, **p < .01.

Transformed stress rating data and significant effects are depicted in Figure 4.3. Stress ratings were greater than at baseline immediately after mental arithmetic in both the standing, t(19) = 4.20, p < .003, and supine, t(19) = 4.42, p < .003, conditions, with a trend for the increase while supine to be greater than that while standing, t(19) = 2.30, p = .065. After the recovery period, self-reported stress had returned to baseline levels in the supine condition, t(19) = 0.28, p = .785, and fallen below baseline in the standing condition, t(19) = 2.57, p = .019; the mean difference between postures at this time-

point was not significant, $t(19) = 1.63$, $p = .120$. Subsidiary analyses indicated that the fall in stress ratings after recovery while standing was not the product of a difference between control and task session raw data solely at baseline, $t(19) = 1.52$, $p = .144$, or after recovery, $t(19) = 0.28$, $p = .785$.

Physiological data: raw

Control session baselines

Expressed as a percentage of the control session supine condition baseline, control session standing condition baseline values were: stroke volume, 69.2 (8.6) %; cardiac output, 89.7 (12.3) %; total peripheral resistance, 124.9 (22.3) %. Baseline values for all of the other physiological measures are presented in Tables 4.2 (standing condition) and 4.3 (supine condition). Physiological variables that were higher during the control session baseline while standing than while supine include heart rate, $t(19) = 8.09$, $p < .001$, diastolic blood pressure, $t(19) = 5.30$, $p < .001$, skin conductance, $t(19) = 5.31$, $p < .001$, and total peripheral resistance, $t(19) = 5.01$, $p < .001$. Measures higher while supine than while standing were stroke volume, $t(19) = 15.91$, $p < .001$, and cardiac output, $t(19) = 3.75$, $p = .001$. There were no postural differences in either systolic blood pressure, $t(19) = 0.03$, $p = .980$, or respiration rate, $t(19) = 1.37$, $p = .186$.

Control session

For the following data and analyses, means and significant differences are shown in Tables 4.2 (standing) and 4.3 (supine); t–ratios are presented in Appendix B, Tables B6 (standing) and B7 (supine). In each of P2, P3 and P4, diastolic blood pressure and skin conductance were elevated above baseline in both the standing and supine conditions, and total peripheral resistance was elevated above baseline in the supine condition. In the standing condition, total peripheral resistance was elevated at P3 and P4, with the value at P2 not quite reaching statistical significance ($p = .070$). Systolic blood pressure

Table 4.2

Mean (SD) standing condition values for all physiological measures

	Control session				Task session			
	Base.	P2	P3	P4	Base.	Task	P3	P4
Systolic b.p. (mmHg)	119.9 (18.6)	122.3 (16.8)	122.8 (14.8)	121.4 (14.2)	118.1 (15.3)	138.9** (23.5)	120.8 (17.3)	118.9 (15.0)
Diastolic b.p. (mmHg)	72.4 (11.3)	74.3* (9.8)	75.0* (9.4)	75.1* (9.0)	71.9 (10.6)	84.9** (16.8)	74.9 (13.9)	75.4 (13.9)
Heart rate (beats/min)	94.0 (14.4)	93.7 (15.0)	92.8 (14.0)	94.0 (14.5)	96.6 (15.3)	102.5 (18.8)	96.7 (18.2)	96.4 (15.7)
Skin cond. (μmho)	2.9 (1.6)	3.6* (1.7)	4.1* (1.8)	4.3* (2.0)	2.7 (1.6)	5.2** (2.3)	4.3** (2.0)	4.1** (2.4)
Resp. rate (breaths/min)	18.6 (3.2)	-	19.0 (3.3)	18.9 (2.3)	18.9 (2.3)	-	19.7* (2.9)	19.8* (2.7)
Stroke volume (%)	-	100.4 (6.9)	100.6 (7.2)	98.7 (6.2)	-	102.0 (12.9)	98.8 (9.4)	96.2 (11.6)
Cardiac output (%)	-	99.9 (6.0)	99.0 (5.6)	98.4 (6.1)	-	108.0 (14.5)	98.5 (6.8)	95.9 (9.3)
Total periph. rest. (%)	-	102.8 (6.4)	104.9* (7.8)	105.4* (8.5)	-	111.0* (17.5)	105.6 (13.8)	109.6 (20.5)

Note. Comparisons vs. baseline: df = 19 (except in the task session for systolic and diastolic blood pressure, stroke volume, cardiac output and total peripheral resistance, where df = 17), *p < .05, **p < .01.

Table 4.3

Mean (SD) supine condition values for all physiological measures

	Control session				Task session			
	Base.	P2	P3	P4	Base.	Task	P3	P4
Systolic b.p. (mmHg)	120.0 (15.3)	121.8 (16.2)	123.4* (14.9)	124.9* (16.5)	118.7 (13.2)	131.0** (16.7)	124.8* (13.3)	124.2* (13.9)
Diastolic b.p. (mmHg)	61.8 (9.5)	63.4* (9.6)	64.0* (8.4)	64.7* (9.2)	62.1 (8.6)	71.2** (9.7)	64.9* (6.9)	64.9 (7.6)
Heart rate (beats/min)	72.3 (10.2)	71.3 (10.4)	72.4 (10.2)	71.6 (9.9)	74.9 (12.0)	89.0** (15.9)	64.9 (6.9)	64.9 (7.6)
Skin cond. (μmho)	1.2 (0.8)	1.6* (1.0)	2.1* (1.4)	1.9* (1.3)	1.5 (1.2)	4.1* (2.0)	2.8* (1.3)	2.1* (1.3)
Resp. rate (breaths/min)	17.7 (3.1)	-	17.5 (3.2)	17.5 (3.0)	18.2 (3.9)	-	18.5 (4.3)	18.5 (4.0)
Stroke volume (%)	-	98.7 (3.1)	99.8 (4.2)	99.9 (4.9)	-	98.8 (6.7)	101.9 (4.7)	99.9 (5.8)
Cardiac output (%)	-	97.6 (4.7)	99.8 (4.5)	98.9 (3.9)	-	117.0** (13.8)	102.1 (9.1)	99.9 (10.8)
Total periph. rest. (%)	-	104.6* (7.1)	103.8* (5.7)	105.6* (7.2)	-	98.8 (12.0)	103.3 (9.3)	105.9 (14.6)

Note. Comparisons vs. baseline: df = 19 (except in the task session for systolic and diastolic blood pressure, stroke volume, cardiac output and total peripheral resistance, where df = 17), *p < .05, **p < .01.

was greater than at baseline during both P3 and P4 in the supine condition, with an elevation during P2 bordering on significance ($p = .054$). There was no change from baseline during any phase for systolic blood pressure in the standing condition, nor was there a change for heart rate, stroke volume, cardiac output and respiration rate in either postural condition.

Task session

As for the control session, raw task session data and analyses are presented in Tables 4.2 and 4.3, and Tables B6 and B7, respectively. For the task, P3 and P4, skin conductance was greater than baseline in both the standing (see Table 4.2) and supine (see Table 4.3) conditions. Also, during the task there were increases in both systolic and diastolic blood pressure in both postural conditions. Heart rate and cardiac output increased during the task in the supine condition, though not in the standing condition. In contrast, total peripheral resistance increased during the task in the standing condition only. In the supine condition, systolic blood pressure was greater than baseline for both P3 and P4, while diastolic blood pressure was greater for P3 only. For both P3 and P4 in the standing condition, increases were seen for respiration rate, with a trend for diastolic blood pressure to also be greater ($p = .067$). Stroke volume did not change from baseline during the task, P3 or P4 in either postural condition.

Physiological data: transformed

As shown in Table 4.4, physiological measures (transformed) that increased during the mental arithmetic task for both the standing and supine conditions were systolic blood pressure: $t(17) = 5.57$, $p < .003$ and $t(17) = 3.85$, $p = .003$ respectively; diastolic blood pressure: $t(17) = 4.98$, $p < .003$ and $t(17) = 4.91$, $p < .003$ respectively; skin conductance: $t(19) = 4.44$, $p < .003$ and $t(19) = 5.58$, $p < .003$ respectively. Heart rate

Table 4.4

Mean (SD) values for transformed (task session – control session) physiological measures data adjusted for a zero baseline

	Standing condition			Supine condition		
	Task	P3	P4	Task	P3	P4
Systolic b.p. (mmHg)	18.8** (19.6)	-0.4 (12.3)	-1.1 (13.9)	10.9** (15.2)	3.4 (11.6)	1.2 (13.9)
Diastolic b.p. (mmHg)	10.9** (13.8)	0.3 (11.5)	0.6 (12.2)	7.7** (11.7)	0.8 (10.0)	0.1 (10.1)
Heart rate (beats/min)	6.2 (14.5)	1.3 (11.4)	-0.1 (9.4)	15.1** (13.2)	0.1 (9.1)	0.4 (9.5)
Skin cond. (μmho)	1.8** (2.4)	0.4* (1.5)	0.1 (1.6)	2.2** (1.9)	0.4 (1.1)	0.0 (0.9)
Resp. rate (breaths/min)	-	0.4 (3.7)	0.1 (2.6)	-	0.5 (3.3)	0.5 (3.2)
Stroke volume (%)	2.0 (14.0)	-1.7 (9.2)	-2.4 (11.9)	0.5 (6.2)	2.6 (5.4)	0.1 (9.8)
Cardiac output (%)	9.0 (15.2)	-0.4 (8.2)	-2.4 (10.9)	19.8** (15.7)	2.6 (8.5)	1.2 (12.6)
Total periph. rest. (%)	7.2 (18.6)	0.1 (14.4)	3.6 (22.1)	-5.9 (15.5)	-0.4 (10.6)	0.5 (16.3)

Note. Comparisons vs. baseline: df = 17 (except for skin conductance, heart and respiration rates, where df = 19), *p < .05, **p < .01

and cardiac output also increased during the task in the supine condition, $t(19) = 6.42$, $p < .003$ and $t(17) = 5.36$, $p < .003$ respectively; in the standing condition there was only a trend ($p = .067$) for cardiac output to increase. Skin conductance was elevated above baseline at P3 in the standing condition, $t(19) = 2.44$, $p = .049$, and was the only instance in which any of the measures were greater than baseline during either of the recovery phases (P3 or P4) in either postural condition. In comparing the changes for the standing and supine conditions, there was a greater increase in heart rate for the latter, $t(19) = 3.77$, $p = .003$; the difference in cardiac output reactivity was not significant at the .05 level ($p = .073$). The difference between the opposing changes in total peripheral resistance for standing (increase) and supine (decrease) also did not reach conventional levels of statistical significance. ($p = .076$). There was no difference between postures during the task for the other measures, nor was there a difference between postures at either P3 or P4 for any of the measures. For t-ratios not in the text, see Appendix B, Table B8 for comparisons versus baseline and Table B9 for comparisons between the standing and supine conditions.

Discussion

In the current study, the influence of posture on the psychological stress and anxiety generated by a cognitive task was investigated by asking participants to give ratings of these subjective states in association with performing mental arithmetic in both standing and supine conditions. State anxiety was assessed with two measurement tools, the short form of the STAI and a 0-10 self-report scale; both of these are valid measures of state anxiety, as indicated by their scores being highly (and positively) correlated with scores obtained using the well established traditional state form of the STAI (short form: Marteau & Bekker, 1992; 0-10 scale: Benotsch et al., 2000). A 0-10 self-report scale

was also used to measure psychological stress; the use of a simple numerical scale has been validated against salivary cortisol levels, a key physiological marker of the stress response (e.g., Pruessner et al., 2003). Given that a less upright posture is thought to be associated with less central noradrenergic activity (e.g., Elam et al., 1984), and given the established relationships between central noradrenergic activity and both psychological stress (e.g., Stanford, 1995) and anxiety (e.g., Tanaka et al., 2000), it was anticipated that ratings of stress and anxiety would be lower when participants were supine than when they were standing. This would also be in keeping with the idea that the cortical dampening effect of baroreceptor activity (greater while supine) decreases both anxiety (Dworkin et al., 1994) and psychological stress (Schweizer et al., 1991).

The influence of posture on the stress and anxiety associated with performing mental arithmetic

Analyses of the transformed subjective rating data indicated that immediately after performing the mental arithmetic task both the stress and state anxiety of participants were increased above pre-task baseline levels, in both the standing and supine conditions; thus, the task successfully induced a degree of negative affect (as previously shown by Schweizer et al., 1991). In comparing the postures there was a trend for stress to increase more in the supine condition; although there was no statistical difference in anxiety between standing and supine, state anxiety was elevated on both the self-report (0-10) scale and the short form of the STAI in the supine condition but elevated only on the latter in the standing condition (the self-report scale increase did not reach conventional levels of significance). Following the recovery period, ratings of stress and anxiety had fallen below baseline levels in the standing condition though were no different to baseline levels in the supine condition; stemming from this, relative to baseline there was less anxiety (though not stress) in the standing condition at this time-

point. Thus, the findings of this study do not support the idea of there being less stress and anxiety generated by performing a stressful task when supine than when standing. Furthermore, the results would appear to be inconsistent with the idea that increased baroreceptor activity (i.e., in the supine condition relative to the standing condition) decreases anxiety (Dworkin et al., 1994) and stress (Schweizer et al.). However, owing to unanticipated reasons, it is possible that reduced arousal prior to the mental arithmetic task while supine led to there being some evidence for a greater level of negative affect after task performance in that condition.

It has been proposed that the lower cortical arousal associated with a less upright posture may be a product of reduced central noradrenergic activity (e.g., Elam et al., 1984). An analogous situation to this may be achieved by the administration of centrally acting alpha$_2$-adrenoceptor agonists. Alpha$_2$-adrenoceptor agonists stimulate presynaptic adrenoreceptors and thereby decrease the release of noradrenaline from nerve terminals (Fleming & Robertson, 1990), an effect that makes alpha$_2$-adrenoceptor agonists useful for treating hypertension (D. P. Westfall, 1990). Centrally acting alpha$_2$-adrenoceptor agonists, such as clonidine (e.g., Hall et al., 2001) and dexmedototamine (e.g., Hall et al., 2000), produce sedation (i.e., reduce arousal) in humans, as measured by subjective ratings, objective behavioural ratings and the bispectral index (derived from processed EEG data and which indicates the state of wakefulness). However, the effects on arousal produced by alpha$_2$-adrenoceptor agonists are easily overcome. To illustrate this point, under resting conditions the bispectral index is reduced by alpha$_2$-adrenoceptor agonists, however when participants are asked to perform a cognitive task (and before actually beginning the task) the bispectral index increases to placebo condition levels. Even though alpha$_2$-adrenoceptor agonists may not prevent a normal level of arousal from being achieved in readiness for a task, they may result in performance on the task being

impaired (e.g., on a digit symbol substitution test, Hall et al.).

In keeping with the results of Hall et al. (2000, 2001), Clark et al. (1987) found that an alpha$_2$-adrenoceptor agonist impaired performance on a cognitive task. In particular, participants were slower to respond and identified less targets in aurally presented word lists after being administered clonidine (cf. placebo). It was concluded by the authors that a greater amount of effort must be expended if the sedative effect of clonidine is to be overcome and normal performance on a cognitive task to be achieved. Indeed, normal performance on a cognitive task after being administered clonidine can be achieved, with the sedative effects overcome either by an intrinsic mechanism (e.g., as shown for a rapid visual information processing task by Coull, Frith, Dolan, Frackowiak & Grasby, 1997) or by the presence of white noise (e.g., as shown for an attentional task by Smith & Nutt, 1996).[2] In both of these situations, normal levels of task performance have been found to be associated with a greater than normal increase in activity (blood flow) in the thalamus; this stems from there being less thalamic activity in the clonidine condition than in a placebo condition when participants were simply resting (Coull et al., 1997; Coull, Jones, Egan, Frith & Maze, 2004). Portas et al. (1998) had participants perform a test of attention either when arousal was reduced by sleep deprivation or in a normal or high (caffeine) arousal condition; as with Coull et al., they found that normal task performance in a low arousal condition was associated with greater than normal increases in thalamic blood flow. Based on this finding, and consistent with spontaneous reports by participants of fatigue and strong effort, Portas et al. suggested that the increased thalamic activity in the low arousal condition may reflect a compensatory mechanism that is "related to the subjective experience of greater mental effort" (p.8988).

[2] The effects of clonidine on cognitive performance may also be dose-dependent: Arnsten, Cai and Goldman-Rakic (1988) found that the performance of aged monkeys on a memory task was impaired by a low dose, and improved by a higher dose, of clonidine.

In the current study, though there was a trend for more responses to be provided for the mental arithmetic task in the standing condition this is likely to have been facilitated by the slightly higher mean number of errors while supine (despite there being no statistically significant difference in the number of errors made between the postural conditions); this is because an error by the participant resulted in them being asked to begin the mental arithmetic task again from the starting number, with each of these interruptions reducing the available time for providing responses. Therefore, it is unlikely that there was a true difference in performance on the task between postural conditions (in terms of either the number of errors made or responses provided). Given this, and accepting that there was lower central noradrenergic activity and/or arousal under resting conditions while supine than while standing (consistent with an effect of posture on the EEG, e.g., Cole (1989), and supported by the finding that skin conductance was lower in the supine position, as detailed below), it is likely that a greater amount of mental effort was expended by participants when performing the mental arithmetic task in the supine condition than in the standing condition. Compensatory mental effort of this kind is thought to have affective costs (in terms of negative affect in particular, Hockey, 1997). Thus, the expenditure of compensatory mental effort in the supine condition may have been, in itself, associated with feelings of stress and anxiety; this could have prevented the anticipated development of lower levels of stress and anxiety in the supine condition (cf. standing condition).

Because the ratings of subjective task difficulty were similar for the standing and supine conditions it might be argued that the amount of mental effort expended to complete the mental arithmetic task was not greater when supine. However, as explained in Chapter 3, mental effort and difficulty are not necessarily related (e.g., Dehaene et al., 1998) and therefore ratings of task difficulty cannot be taken as indicative of mental effort. This

may be particularly so in this case because the mental effort needed to perform a cognitive task is thought to be separate from the compensatory mental effort needed to overcome a situational state (e.g., of low arousal) to achieve normal levels of task performance (Mulder, 1986). To better measure differences in mental effort between standing and supine conditions subjective rating scales other than task difficulty could be used; these might include simple numerical ratings of perceived effort, or the NASA-TLX (Task Load Index) rating scale, which in addition to a measure of overall workload has subscales reflecting mental demand and effort (Hart & Staveland, 1988). Alternatively, objective measures known to co-vary with mental effort could be used, such as pupil diameter (Beatty, 1982) or blood glucose levels (Fairclough & Houston, 2004); these are likely to be more appropriate measures than cardiovascular reactivity, which, though also demonstrated to vary with mental effort (as discussed in Chapter 3 with reference to the performance of cognitive tasks, e.g., Critchley et al., 2000), may be dependent upon, or affected by, body posture. Also important in this regard is the difference between the mental effort used to perform cognitive tasks and the compensatory mental effort expended to overcome low arousal (Mulder), for which pupil diameter has been shown to be a useful measure (in relation to the compensatory mental effort needed to overcome physical fatigue, Deijen, Heemstra & Orlebeke, 1995).

In addition to an effect of compensatory mental effort, differences in physiological reactivity could have augmented ratings of stress and anxiety associated with performing the mental arithmetic task in the supine condition. Task-associated increases in heart rate were greater in the supine condition than in the standing condition. As discussed in Chapter 3, changes in cardiac activity may be consciously perceived, by the presence of peripheral somatic sensations (O'Brien et al., 1998) and/or via

representative activity in the insular cortex (Critchley et al., 2004). This awareness has the potential to influence ratings of one's psychological state and therefore may have contributed to the ratings provided in the supine condition.

The use of active and passive tasks to investigate changes in negative affect associated with baroreceptor activity

The results of the current study indicate that the level of psychological stress and anxiety immediately and ten minutes after performing a stressful cognitive task were not less when the task was performed while supine than while standing; in fact there was some evidence to suggest that the experienced stress and anxiety were greater in the supine condition. However, in testing whether stress and anxiety responses to a stressor are different between conditions in which baseline arousal may differ, the use of a task requiring active engagement by the participant (such as the mental arithmetic task used in the current study) may be inappropriate; this is because of the potential for effects on psychological state associated with the expenditure of compensatory mental effort in overcoming low arousal (and being able to perform the task at a normal level).

Other relevant reports have used tasks that are more passive (in terms of participant engagement). Nyklicek et al. (2001) found that hypertensive females gave less negative appraisals than normotensive controls for films depicting stressful scenes; a similarly strong effect was not found for other types of tasks, including mental arithmetic. In their study of the effects of posture on salivary cortisol, Hennig et al. (2000) aimed to reduce any effects associated with boredom by presenting participants with slides containing neutral and aversive content; the participants were asked to rate these slides on dimensions of arousal and affectivity. It was reported that different postures (supine, seated and standing) did not result in different ratings in aversiveness, though details and data for this aspect of the study were not presented. Thus, it is not clear whether the

slides could be considered adequately stressful for testing the idea that a passive stressor may induce less psychological stress in a less upright posture. Even so, given that cortical arousal is reduced under conditions in which baroreceptor activity is increased, it could be argued that lower ratings of psychological stress or other negative affective states may simply be a result of less attention being paid to the stimuli.

There being less attention in a condition of relatively low arousal (a less upright posture) is indicated by faster reaction times (Vercruyssen & Simonton, 1994) and improved vigilance (Caldwell et al., 2003) when standing (cf. supine and/or seated). The tasks used in these studies required participants to respond to the appearance of visual stimuli, and are likely to have been associated with relatively low levels of participant engagement; at least in comparison to the mental arithmetic task used in the current study, in which the requirement for participants to self-generate responses is likely to have facilitated task engagement and the development of compensatory mental effort. There is some evidence that compensatory mental effort can be expended so that attentional task performance is not compromised by a low level of arousal (at baseline); however, task characteristics are likely to be important in determining whether this mechanism develops. For example, Portas et al. (1998) found that sleep deprivation did not significantly impair performance on an attentional task (cf. higher arousal conditions), however the task lasted only 32 s and there were flashing dots continually on the stimulus screen. In comparison, the vigilance task used by Caldwell et al. (2003), and for which performance was better when participants were standing than when they were seated, lasted for 10 minutes and the interval between stimuli was relatively long (between 1 and 10 s).

In assessing affective responses to passive stimuli, the possibility that there is less attention given to the stimuli in a condition of low arousal may be a problem that is

difficult to control without evoking compensatory mental effort. This suggests that passive tasks are also an inappropriate tool (in addition to tasks requiring active engagement) for investigating the effects of baroreceptor activity on stress and anxiety. There is some hope for a suitable method however, as revealed in the current study by analyses of the subjective ratings provided prior to performing the mental arithmetic task.

The influence of posture on anxiety and stress before the mental arithmetic task

In the control session, neither stress nor anxiety ratings changed significantly from baseline at any time, suggesting that the prolonged nature of the postural conditions was not associated with changes in negative affect. Furthermore, removing the control session values from corresponding task session values (i.e., producing transformed data) did not have a large impact on the results concerning the effects of performing the mental arithmetic task on stress and anxiety; there was a similar pattern of increases (from baseline) in stress and anxiety ratings provided immediately after performing the task in both the raw task session and transformed data sets (though there was a lesser anxiety rating effect for the standing condition in the former). However, analysis of the transformed data produced an interesting result that did not emerge from the initial analysis of the raw task session data. After the period of recovery following mental arithmetic, transformed anxiety ratings were less than those provided at baseline in the standing condition; this was different from the supine condition, in which there was no change in transformed anxiety ratings from baseline at this time-point. Subsidiary analyses of the raw data revealed that, compared to the control session, anxiety was elevated prior to performing the mental arithmetic task (i.e., at baseline in the task session) when standing. This was not the case for the supine condition, suggesting the development of anticipatory anxiety in the standing condition only. A similar pattern to

the anxiety data was found for ratings of stress, however these effects were not as strong and did not reach the levels required for statistical significance.

It was anticipated that anxiety may be greater in the standing condition because of central noradrenergic activity both being greater in a more upright posture (e.g., Elam et al., 1984) and being associated with the production of anxiety (e.g., Tanaka et al., 2000). However, an increase in central noradrenaline turnover in itself might not be sufficient for producing anxiety, with a situational context needed for this to develop. This is consistent with the idea that "a relatively undifferentiated visceral activity and afference may prime affective reactivity generally, with the specific nature of the response determined by environmental cues" (Berntson, Sarter & Cacioppo, 1998, p. 240). The need for a situational context would explain why, even though standing may induce a neurophysiological substrate supporting the development of anxiety (i.e., an increase in cortical noradrenergic activity), there was no difference in anxiety between the standing and supine conditions in the control session (in which participants were simply resting). However, the anticipation of performing the mental arithmetic task is likely to have been a sufficient context for inducing anxiety, with a conducive neurophysiological substrate in the standing condition facilitating the development of anticipatory anxiety.

The finding of the current study that anticipatory anxiety developed in the standing condition, though not in the supine condition, indicates that a change in posture is sufficient to alter psychological state, at least in terms of negative affect. As standing is associated with relatively less baroreceptor activity, the presence of anticipatory anxiety in this condition, though not when supine, could be seen as support for the idea that baroreceptor activity reduces anxiety (Dworkin et al., 1994) and for the theory of leaned

hypertension (Dworkin, 1988), in which a reduction in negative affect associated with baroreceptor activity is a key concept. A role for baroreceptor activity in the modulation of anticipatory psychological states has been suggested previously, given the finding that hypertensive females reported less pre-stressor anxiety than normotensive controls prior to a range of tasks (Nyklicek et al., 2001). However, as previously discussed (in Chapter 1), the use of hypertensives to draw conclusions regarding the effects of baroreceptor activity may not be appropriate, as elevated resting blood pressure does not necessarily imply elevated baroreceptor activity (e.g., Weisz et al., 2002).

In future studies investigating the effects of baroreceptor activity (or any manipulation that alters cortical arousal at baseline) on psychological stress and anxiety, it may be necessary to consider the potential problems associated with compensatory mental effort and tasks that actively engage the participant (such as mental arithmetic); also, there may be differences in attention associated with more passive stressors (such as aversive films or slides). The development of negative affect during a period of anticipation prior to a stressor may be the most appropriate means of identifying differences in psychological state between conditions in which baroreceptor activity and/or cortical arousal differs.

Non-noradrenergic mechanisms for increased anxiety when standing

While there may have been an effect of central noradrenaline acting directly on structures that promote anxiety (e.g., the amygdala, Tanaka et al., 2000) in the standing condition (as a flow-on effect of relatively low baroreceptor activity), there are other mechanisms that could also have been involved. These include neurotransmitter systems other than the noradrenergic; for example, rather than acting directly on relevant cortical structures, Berntson et al. (1998) have suggested that central noradrenaline may

modulate anxiety via effects on the cholinergic system. Furthermore, the possibility that afferent feedback representing the elevations in either diastolic blood pressure or heart rate during standing helped prime an anxiety response cannot be excluded; this is similar in nature to the ideas expressed by James (1884) and Lange (1885/1912), and more recently by Damasio (e.g., 2003). Also, because of overlap between the neurological systems for balance control and anxiety, it is possible that vestibular information may contribute to the development of anxiety, at least when the information concerns body motion (Balaban, 2002); the implications for any relationship between vestibular activity, anxiety and static body posture are unclear.

It might be considered possible that the muscular activity involved with maintaining a standing posture may have contributed to the anticipatory anxiety found associated with that postural condition. A number of studies have shown that techniques producing muscle relaxation reduce reported levels of state anxiety (e.g., Lamb & Strand, 1980; Pawlow & Jones, 2002; Rasid & Parish, 1998). It is probably because of a perceived relationship between lying down and muscular relaxation that this posture has been used in previous experiments with the intent of invoking a relaxation response (e.g., Green & Green, 1987). However, in the current study, there being no difference in state anxiety between standing and supine in the control session suggests that muscle activity did not have a significant effect on state anxiety levels. Furthermore, if muscle activity associated with standing was having an effect on anxiety it may be expected that, with prolonged activity, state anxiety would have increased across the duration for which this posture was maintained; however, there was no difference in state anxiety ratings between baseline and at the end of the condition in the control session. Nevertheless, an effect of muscle activity on priming the anticipatory anxiety response in the standing condition of the current study cannot be excluded. The extent to which differences in

baroreceptor activity contributed to the postural differences in anticipatory anxiety found in the current study could be investigated in the absence of any contribution from muscular activity; achieved by using lower body positive and negative pressure conditions rather than a postural manipulation.

Lying down is associated with a decrease in core body temperature; this is the result of a baroreflex-mediated vasodilation of skin blood vessels (which helps to maintain blood pressure at a similar level to more upright postures) leading to heat being lost from the skin surface (Tikuisis & Ducharme, 1996), and is thought to be a mechanism that facilitates the onset of sleep (Krauchi, Cajochen & Wirz-Justice, 1997). In addition, changes in body temperature (heating or cooling) have been related to changes in affective state: Ebbecke (as cited in Krauchi et al.) found that an increase in core body temperature was associated with "a feeling of alertness and a refreshed state", while a decrease produced "a feeling of relaxation, comfort and tiredness" (p. 138). Others have postulated that an increase in core body temperature may be associated with the reduction in state anxiety that is frequently observed to develop after physical exercise (Morgan & O'Connor, 1988). Consistent with this idea, both a hot shower (Raglin & Morgan, 1985) and a sauna (Kuusinen & Heinonen, 1972) have been shown to reduce state anxiety levels. However, Koltyn & Morgan (1997) found that while the state anxiety of participants decreased after underwater (in a pool) swimming in a bathing suit, state anxiety increased when this was done in identical conditions though while wearing a wet suit; body temperature increased in the wet suit condition only. These studies have not clarified if and how changes in body temperature affect anxiety, and therefore it is not known whether differences in body temperature between standing and supine conditions could be associated with different state anxiety effects. The existence of any such effects may be able to be investigated by accurately maintaining body

temperature constant between postural conditions; achieved with the use of a suit lined with tubes through which temperature controlled water is circulated (e.g., Shastry, Minson, Wilson, Dietz & Joyner, 2000).

Increased anxiety in relation to other psychological processes when standing

A similar postural anxiety effect to that found in the current study may have developed in Study 1 of this thesis, in which participants were found to solve anagrams more rapidly when supine than when standing (and in which subjective psychological states were not measured); this effect was accounted for by the likelihood of greater central noradrenergic activity when standing (e.g., Elam et al., 1984) and findings suggesting that central noradrenergic activity impairs the ability to solve anagrams (e.g., Beversdorf et al., 2002). As anxiety has been reported to be associated with a reduced ability to solve anagrams (e.g., Zarontonello et al., 1984), it might be thought that anxiety contributed to the finding of poorer anagram task performance when standing in Study 1. However, rather than an effect of anxiety on the ability to solve anagrams in addition to that associated with the postulated greater level of central noradrenergic activity when standing, it is likely that both poorer anagram task performance and an anticipatory anxiety effect were a result of this (largely hypothesised) elevated activity.

In keeping with the idea of greater central noradrenergic activity when standing (e.g., Elam et al., 1984), the influences of posture on higher-order psychological processes demonstrated in the current study and Study 1 could be considered as products of a global sympathetic system; this system co-ordinates central and peripheral sympathetic activity in preparing an organism for interacting with the environment or to cope with stress (Aston-Jones et al., 1994). In overcoming the effects of gravity on the cardiovascular system, standing is associated with a greater level of peripheral sympathetic nervous system activity than less upright postures (e.g., Mohrman &

Heller, 2003). Complementing this physiological condition is a psychological state characterised by heightened arousal and improved vigilance (e.g., as shown in sleep-deprived participants by Caldwell et al., 2003), effects that are consistent with elevated locus coeruleus activity (e.g., Rajkowski et al., 1998). The demonstrated predisposition to developing anxiety and reduced ability to solve anagrams when standing may reflect this same mechanism, though in terms of costs rather than benefits, for example, improved vigilance may be at the expense of an impairment of the cognitive processes that are utilised when solving anagrams.

A note on stress rating magnitudes in Studies 2, 3 and Schweizer et al. (1991)

It is of interest to note that participants' ratings of perceived stress for the mental arithmetic task in the current study (means of 2.9 and 3.5 for standing and supine conditions respectively) were lower than for the mental arithmetic task in Study 2, in which the mean rating was 5.1, and which given the lower levels of physiological reactivity, would appear to have been less stressful. This difference could be due to differences in the length of the task (longer for Study 2) or the fact that, while ratings were given after the task had been completed in both studies, participants in Study 2 were asked to rate how stressful they found the task, whereas in the current study participants were asked to rate how stressful they felt at a particular moment in time; relief at the task being over could have reduced the ratings to some degree. The ratings of perceived stress were also slightly lower in the current study than those found for a similar task in the placebo condition of Schweizer et al. (1991) (the mean for which was approximately 4.2). In the current study the participant conducted the task separated by a partition from the experimenter, if this were not the case in the Schweizer et al. study (it was not reported to be) it may have enhanced feelings of stress during the task due to a social mechanism (i.e., caused by performing the task under the direct observation of the experimenter).

Posture and baseline physiology

Cardiovascular measurements during the control session baseline differed between standing and supine in line with well established findings (e.g., Mohrman & Heller, 2003): heart rate, diastolic blood pressure and total peripheral resistance were higher while standing than while supine; stroke volume and cardiac output were higher while supine than while standing; there was no difference in systolic blood pressure between postures.

In the control session, baseline skin conductance was found to be higher while standing than while supine. This differs from the finding of Study 1, in which there was no difference in skin conductance between standing and supine. It was also the case that the values (and associated variance) obtained for skin conductance were much lower in the current study and more in keeping with published norms (Venables & Christie, 1980); this may have been facilitated by the methodology used in Study 1 having been improved upon in the current study, with a more appropriate electrode paste used (cf. a sodium chloride based product, Venables & Christie) and the electrodes removed and reapplied to avoid slippage (and the spread of paste leading to an increase in conductive area, Lykken, 1970) during changes in posture. There being greater skin conductance in the standing condition than in the supine condition is consistent with skin conductance being an index of cortical arousal (e.g., Barry et al., 2004), and with cortical arousal having been shown to be higher in a more upright posture (e.g., Cole, 1989). Furthermore, the skin conductance data supports that idea that arousal in a more upright posture is associated with higher central noradrenergic activity, given that destruction of the central noradrenergic system has been shown to reduce skin conductance in cats (Yamamoto et al., 1990). As with differences in cardiovascular variables, postural differences in skin conductance may be a direct result of changes in baroreceptor

activity (i.e., a baroreflex). It has been found that (in anaesthetised cats) an increase in carotid sinus pressure, and therefore an increase in carotid baroreceptor activity, reduced sweat gland activity in the paw (as measured by skin potential, Horwitz & Kaufman, 1979). Similar results have been found in humans, with smaller skin conductance responses (to painful stimuli) during the stimulation of carotid baroreceptors (produced via neck suction) reported by Mini et al. (1995). However, it is changes in the central venous pool, and therefore changes in cardiopulmonary baroreceptor activity rather than carotid baroreceptor activity, that are more likely to be maintained throughout the duration of a postural condition (Pump et al., 2001). A role for cardiopulmonary baroreceptors in modulating skin conductance could be investigated, with cardiopulmonary baroreceptor activity manipulated by either raising the legs in the supine position (as per D'Antono et al., 2000) or by applying lower body positive pressure (20mmHg of lower body negative pressure unloads cardiopulmonary baroreceptors, e.g., Hamer, Boutcher & Boutcher, 2003).

Unlike Study 1, resting respiration rate was not greater in the supine condition than in the standing condition. There are mixed findings also from previous studies, with Stanley et al. (1997) finding a higher respiration rate in participants who were supine compared with standing; in contrast, Tulen et al. (1999) found no difference in respiration rate between these postures. If, as suggested in Study 1, a more rapid respiration rate in the supine condition is due to lung volume being compromised by abdominal contents pushing on the diaphragm (Bettinelli et al., 2002), it may be that differences in the mean weight (or body mass index) of participants between groups are involved; with a faster respiration rate in the supine position possibly more likely to develop in a heavier group. Consistent with this idea, compared to the Stanley et al. study (in whom faster respiration rates were found in the supine condition) participants

in the current study (in which there was no difference between postures) were of similar height, though weighed on average around 5.5 kg less. Comparison with the other relevant studies (Study 1 and Tulen et al.) cannot be made because height and weight measurements were either not obtained or not reported.

Physiological activity associated with mental arithmetic and recovery

Both systolic and diastolic blood pressure increased during the mental arithmetic task; as per Study 1 there was no difference in the magnitude of these increases between the standing and supine conditions. Rusch et al. (1981) found that mean arterial pressure rose less during a mental arithmetic task while standing than while supine, however there were only six participants and when a different task (isometric handgrip) was used, no difference between postural conditions was found. Unlike Study 1, heart rate increased in the standing as well as the supine condition; the difference likely to be due either to the task actively engaging the participants to a greater degree in the current study (as indicated by greater levels of reactivity for the other physiological measures) or by an additional increase in heart rate associated with the vocalisation of responses (Brown, Szabo & Seraganian, 1988). More importantly, and consistent with the finding in Study 1, heart rate increased more during mental arithmetic while supine than while standing; other studies have reported that heart rate reactivity is less when standing than when seated, and concluded that this may be because of pre-existing sympathetic withdrawal from the heart while standing (e.g., Cacioppo et al., 1994). The larger increase in heart rate was the basis for there being a trend for cardiac output to increase more while supine, given that there was no difference in stroke volume reactivity between the postural conditions. Others have reported effects of mental stress on stroke volume and cardiac output reactivity for standing and seated postures, though the results have been mixed. For example, both an increase (Sherwood & Turner, 1993) and a

decrease (Waldstein et al., 1998) in stroke volume have been reported to be associated with task performance while standing.

With regards to total peripheral resistance, no significant differences from baseline during the mental arithmetic task were observed for either the standing or supine conditions. Nevertheless, though not quite reaching statistical significance, the between task comparison was consistent with opposing changes in total peripheral resistance, an increase while standing and a decrease while supine. In keeping with this trend, both Sherwood and Turner (1993) and Waldstein et al. (1998) found that total peripheral resistance increased during task performance more in a standing condition than in a seated condition. There is evidence suggesting that differences in total peripheral resistance reactivity between standing and other postures may stem from reduced activity of cardiopulmonary baroreceptors when standing, as lower body negative pressure (which reduces the load on cardiopulmonary baroreceptors) has been shown to reduce the suppression in muscle sympathetic nerve activity produced by carotid baroreceptor stimulation (Ichinose et al., 2004) and to attenuate the decrease in forearm vascular resistance associated with performing the Stroop task (Hamer et al., 2003).

In the current study, skin conductance was found to be higher during the mental arithmetic task than at baseline in both the standing and supine conditions, though the magnitude of the increase was no different between postures. This is different to the finding of Study 1, in which a rise in skin conductance during task performance was found for the standing condition only. However, it was thought possible that this may have been due to a simple time effect rather than being a direct product of performing the task. A task effect was evident in the current study because (a) the rise in skin conductance was found after having removed changes associated with the control

session and (b) there was a decrease in skin conductance towards baseline levels during recovery from the task. Nevertheless, a time effect was also observed, with skin conductance increasing gradually over time in both the standing and supine conditions of the control session. This is unlikely to reflect an increase in arousal, rather, it is consistent with a report by Blank and Finesinger (1946) that skin resistance (the inverse of skin conductance) may decrease over time due to hydration of the outermost layer of the skin by the electrode paste. A gradual increase in skin conductance may also be due to the concentration of sodium chloride on the skin rising with time since the hands were last washed (Carrie & Heemeyer, as cited in Venables & Christie, 1980). Skin conductance appeared in the current study to increase at a faster rate while standing (i.e., from baseline to the final measurement phase, P4, in the control session it had increased 1.4 μmho while standing and only 0.7 μmho while supine): therefore it is possible that the increase in skin conductance attributed to the performance of cognitive tasks while standing in Study 1 was simply an effect of time. The more definitive task-associated increase in skin conductance found in the current study may be because, compared with Study 1, the task was a stronger stimulus for generating physiological reactivity (as shown by much larger increases on the other measures).

With regards to the recovery period after the task, the only physiological measure to not have returned to baseline by the first of the recovery measurement phases (P3) was skin conductance while standing. Even so, there was no difference in the change in skin conductance at this time-point from baseline between the standing and supine conditions. Previous studies have reported that skin conductance can remain elevated for some time after performing a cognitive task; however, unlike in the current study, a simple effect of time cannot be excluded as being responsible (e.g., Jorgensen & Zachariae, 2002). Regardless, elevated skin conductance readings in recovery could be

due to peripheral mechanisms rather than psychological arousal (Bundy & Mangan, 1979). Though no other physiological measures were elevated in either of the recovery phases (P3 or P4), a different conclusion would have been reached were the subject of analysis the raw task session data rather than transformed data (control session values subtracted from corresponding task session values): a number of physiological measures were elevated during recovery in the raw task session data that were not found to be elevated using the transformed data set. Elevated measures during the recovery phases in the raw task session data can be attributed to simple increases over time, as these also occurred in the control session; this applies to skin conductance in both postures (only for P4), respiration rate while standing, and systolic and diastolic blood pressure while supine. The means for diastolic blood pressure in the standing condition and total peripheral resistance in both postural conditions were also elevated during recovery in the task session to a similar or greater degree above baseline as in the control session; however, unlike in the control session, the larger variances in the task session prevented these elevations from being statistically significant. These findings demonstrate that erroneous conclusions concerning elevated physiological activity in recovery could be reached if there are underlying changes in the variables that are not taken into account.

Control session physiological data: drifting blood pressure and peripheral resistance

Possible reasons for why skin conductance may increase over time were discussed above. With regard to cardiovascular measures, some change over time during prolonged standing could be expected as a response to a continual decrease in plasma volume (at least for around the first 20 minutes or so), which may be due to increased capillary pressure in the legs (and arms) leading to the loss of fluid from blood vessels into the interstitial space (Hagan, Diaz & Horvath, 1978). Thus, continual and gradual

compensatory increases in total peripheral resistance and diastolic blood pressure (as found in the current study) might appear to be reasonably associated with prolonged standing. Consistent with this, Hellebrandt and Brogdon Franseen (1943) reported a gradual consistent rise in diastolic blood pressure with prolonged standing, however a stable diastolic blood pressure appears to be the more typical finding (e.g., Jacob et al, 1998). Regardless, in the current study, gradual increases in diastolic blood pressure and total peripheral resistance over time were observed also for the supine position (there was an additional gradual rise in systolic blood pressure in this position), indicating that these changes were not specific to standing.

It is possible that the gradual rise in blood pressure over time observed in the current study may be a recording artefact, one that is a product of the Finapres device. Ristuccia, Grossman, Watkins and Lown (1997) have shown that there is an upward drift in blood pressure readings obtained with a Finapres. In their study, three baseline periods of ten minutes duration were recorded; after the first and second of these, tasks that elevated blood pressure were performed (including paced breathing, mental arithmetic and a speech stressor). Blood pressure readings at the end of the second baseline period had not returned to those observed for the first; at the end of the third baseline period this discrepancy had increased even further (e.g., the difference in systolic blood pressure was 26 mmHg). The increase in blood pressure readings was not simply the result of slow recovery: the authors had found previously that using the same protocol, though with blood pressure measured from the arm (rather than with a Finapres), the difference between first and third baseline values for systolic blood pressure was only 6 mmHg. Furthermore, removing the finger from the Finapres cuff and exercising it reduced the problem, as did switching to a cuff on another finger (using a Portapres device: a portable version of the Finapres with cuffs for adjacent

fingers that can be alternated between). Uncertain accuracy of Finapres blood pressure readings following a perturbation in blood pressure was also reported by Birch and Morris (2003), who found that after a fall in pressure (produced by releasing occlusive thigh cuffs), readings from a Finapres remained lower than those obtained with an alternate, wrist-based measurement device (a Colin radial artery tonometer).

The Finapres determines blood pressure via continual adjustment of the cuff pressure so that finger arterial volume (measured via plethysmography) is maintained constant. The set-point volume is determined by the device's inbuilt calibration system (called "Physiocal" by the manufacturers) when the blood pressure measurement first begins, and then in periods of self-adjustment that occur every 70 heartbeats (or more frequently if necessary); this periodic checking of the set-point is designed to correct for changes in the vascular state of the finger (Wesseling, de Wit, van der Hoeven, van Goudoever & Settels, 1995). Nevertheless, Finapres blood pressure readings are affected by vasoconstriction in the finger, as shown by plethysmographic measurements in fingers adjacent to the cuff (Wesseling et al., 1985). It is also thought that venous engorgement (Jones, Brown, Roulson, Smith & Chan, 1992) or changes in the volume of interstitial fluid underneath the cuff (Ristuccia et al., 1997) may lead to inaccuracies in Finapres readings; this would be the case if such changes are not recognised by the Physiocal system.

In contrast to the findings of the current study, others have reported that blood pressure measured in healthy participants using a Finapres device is essentially stable during 20 minutes of supine rest (Mehagnoul-Schipper, van Kraaij & Jansen, 2000) and around 17 minutes of rest while standing (Ludwig, Vernikos, Wade & Convertino, 2001). However, in those studies, participants simply rested; they did not perform any tasks.

While participants mostly rested in the control session of the current study, they did complete a subjective measures sheet in between certain phases (i.e., between baseline and P2, and between P2 and the later phases). As a general observation, there were increases in skin conductance associated with the completion of a subjective measures sheet. This could be taken to indicate that the sympathetic tone of the fingers was altered during these stages; this is likely to include an alteration in vascular state (which could be verified using additional measures, e.g., plethysmography). It is therefore possible that perturbations in the sympathetic tone of the finger blood vessels are implicated in the upwards drift in blood pressure that was observed in the current study but not in other studies, in which participants were not exposed to any conditions that may produce such perturbations (Ludwig et al.; Mehagnoul-Schipper et al.). Because total peripheral resistance was derived from the blood pressure recordings, an artefactual upward drift in blood pressure would imply that the upward drift in total peripheral resistance observed in the current study control session was also artefactual (Ristuccia et al., 1997).

The problem of an upward drift in Finapres blood pressure readings does not appear to have been fully recognised in the literature, and can cast doubts upon the true source of findings that blood pressure is greater than baseline following an intervening task (the same applies to total peripheral resistance derived from the blood pressure trace). To illustrate, Mezzacappa, Kelsey, Katkin and Sloan (2001) reported that blood pressure, as measured by a Finapres, had not returned to baseline levels four minutes after participants performed a five minute long Stroop task, and appears to have increased even further four minutes after a second task (mental arithmetic) was performed. Though the findings were very similar in nature to those reported by Ristuccia et al. (1997), the possibility of a recording artefact was not explicitly addressed. The authors

did report that the Physiocal self-adjustment procedure of the Finapres device was enabled at the end of each measurement phase (it was disabled otherwise). However, as discussed, the Physiocal system probably does not compensate for all the changes (vascular and non-vascular) that may develop in the cuff finger. Thus, in the Mezzacappa et al. study, it is unclear to what extent the elevated Finapres blood pressure readings in recovery were a true reflection of changes in blood pressure or merely a recording artefact.

In conclusion, if using a Finapres device to measure blood pressure (and/or to derive total peripheral resistance), it may be prudent to include a control condition, particularly if the experimental protocol exposes the participants to any sort of physiologically arousing situation; this need not be a stressful cognitive task, the act of providing subjective ratings is probably sufficient. Using a control condition (in which tasks are not performed as per the experimental condition) to track any changes that develop over time, due either to recording artefacts or intrinsic physiological mechanisms, and removing these from the experimental condition may help to avoid spurious findings of elevated physiological activity during a period of recovery following a cognitive task. Alternatively, a device other than the Finapres could be used; with the ability to switch between fingers, the Portapres appears to enable more accurate readings (Ristuccia et al., 1997), yet still provides the continuous blood pressure trace that permits derivation of values for stroke volume, cardiac output and total peripheral resistance without the need for invasive procedures.

CHAPTER 5

CONCLUSION

This thesis investigated inter-relationships between the physical/physiological state of the body and psychological phenomena. Two studies were conducted to identify whether there was an influence of posture (by comparing standing and lying down) on either cognitive (the ability to solve anagrams) or affective (anxiety and psychological stress) processes. In another study, the effects of performing different cognitive tasks (anagrams and mental arithmetic) on physiological activity were determined; the relationship of this activity to the psychological stress generated by the tasks was also established.

Summary of theoretical background

Due to gravity causing blood to pool in the lower body, a more upright posture is associated with reduced baroreceptor activity (particularly cardiopulmonary baroreceptor activity when the posture is maintained, Pump et al., 2001); this promotes increased sympathetic activity that ensures an adequate level of blood pressure (a baroreflex), and thereby an adequate level of cerebral perfusion (e.g., Mohrman & Heller, 2003). There are also extra-homeostatic effects associated with baroreceptor activity levels, including an inverse modulation of cortical arousal. This means that a decrease in baroreceptor activity enhances cortical arousal, while an increase in baroreceptor activity inhibits cortical arousal; the latter of these effects is illustrated by there being less slow brain potential negativity during carotid baroreceptor stimulation (produced by neck suction, Elbert et al., 1988). Similar effects of baroreceptor activity on cortical arousal have been established in animals (see Vaitl & Gruppe, 1991), indicating the utility of animal models for understanding baroreceptor-mediated effects

in humans.

One of the neurotransmitter systems that regulates cortical arousal is the noradrenergic, the core nucleus of which is the locus coeruleus (Berridge & Waterhouse, 2003). Experiments in animals have shown that neurons in the locus coeruleus are stimulated by a fall in baroreceptor activity (e.g., Elam et al., 1985) and inhibited by an increase in baroreceptor activity (e.g., Elam et al., 1984). Given greater baroreceptor activity in a less upright posture, it has been proposed that locus coeruleus activity is decreased under these conditions (e.g., Elam et al., 1984). This conclusion could also be reached in considering a global sympathetic system that co-ordinates peripheral and central sympathetic activity in preparing an organism to interact with the environment or cope with stress (Aston-Jones et al., 1994); that is, greater central sympathetic (locus coeruleus) activity would be expected to occur in parallel with the increased peripheral sympathetic activity when more upright. Though hypothetical, the presence of greater locus coeruleus activity could account for findings of greater cortical arousal in a more upright posture (e.g., as indicated by less theta band activity in the EEG, Vaitl & Gruppe, 1990). Of particular interest in this thesis was the potential for psychological processes modulated by the central noradrenergic system to be modulated by posture. Previous support for this idea comes from findings that performance on a vigilance task is related to locus coeruleus activity (in monkeys: Rajkowski et al., 1998) and that decrements in performance on a vigilance task associated with sleep deprivation are less pronounced when participants are standing than when they are seated (Caldwell et al., 2003).

Study 1

Previous research has suggested that the ability to solve anagrams is improved when

central noradrenergic activity and arousal are low (Beversdorf et al., 1999, 2002; Walker et al., 2002). Given the postulated higher level of locus coeruleus activity and cortical noradrenaline turnover when standing (e.g., Elam et al., 1984), it was hypothesised that anagram solving performance would be better when supine than when standing. This was supported by the Study 1 finding that participants solved anagrams more rapidly when supine than when standing. Of note, this effect was not due to a generalised influence of posture on problem solving abilities, as performance on a mental arithmetic task was no different between the standing and supine conditions. Furthermore, it was found in Study 1 that a greater level of physiological reactivity developed during attempts to solve mental arithmetic problems than during attempts to solve anagrams. Postural differences in reactivity were also demonstrated, for example, heart rate increased during mental arithmetic while participants were supine, though not while they were standing.

Study 2

In Study 2 it was determined that the greater physiological reactivity for mental arithmetic than for anagrams found in Study 1 was not a result of differences between the tasks in terms of difficulty or psychological stress (as measured by subjective ratings). This was important to establish in the context of the global sympathetic system (Aston-Jones et al., 1994), under which peripheral sympathetic reactivity may give clues to changes in central noradrenergic activity during attempts to solve anagrams; thus the Study 2 finding complemented findings indicating that central noradrenergic activity impairs the ability to solve anagrams (e.g., Beversdorf et al., 2002 and including Study 1).

A theory of learned hypertension has been proposed by which a psychological stress-reducing effect of baroreceptor activity, thought to arise when blood pressure is

increased, reinforces the development of chronically elevated blood pressure (e.g., Dworkin, 1988). There is some evidence used to support the idea that a rise in blood pressure reduces psychological stress (Schweizer et al., 1991); however, other studies have found either no relationship (Freyschuss et al., 1990), or a positive relationship (Lepore, 1995), between blood pressure reactivity and subjectively reported psychological stress. Blood pressure reactivity was found in Study 2 to be significantly and positively correlated with psychological stress ratings for both the mental arithmetic and anagram tasks.

Study 3

Central noradrenergic activity is known to be associated with, and/or facilitate, the development of anxiety (e.g., Bremner et al., 1996a, b) and psychological stress (e.g., Van Bockstaele et al., 2001). Furthermore, though without recourse to this mechanism, under the auspices of the theory of learned hypertension it has been suggested that an increase in baroreceptor activity reduces not only psychological stress (as above) but also anxiety (Dworkin et al., 1994). As baroreceptor activity is increased, and locus coeruleus activity proposed to be reduced, when supine (cf. standing), it was thought likely that the anxiety and psychological stress associated with performing a cognitive task would be less when supine than when standing.

On one hand, the expected influence of posture on negative affect was demonstrated in Study 3: the subjective ratings provided by participants indicated the presence of anticipatory anxiety (prior to a mental arithmetic task) when standing though not when supine. On the other hand, neither the anxiety or psychological stress after the task were lower when participants were supine; in fact there was a trend for a greater level of negative affect to develop at this time-point when supine than when standing.

Salient physiological findings of Study 3 include greater resting skin conductance in participants when standing than when supine. In addition, the postural differences in physiological reactivity observed in Study 1 were complemented by those in Study 3: this includes greater heart rate reactivity when supine (cf. standing). Also identified were problems with using the Finapres device for determining blood pressure and derived haemodynamic information (e.g., total peripheral resistance) in the recovery period following a stressful task.

Mechanisms for a postural influence on psychological processes

Central noradrenergic activity is thought to impair the ability to solve anagrams (e.g., Beversdorf et al., 2002) and to contribute to the development of anxiety (e.g., Tanaka et al., 2000). Thus the findings of this thesis that standing was associated with poorer anagram task performance (cf. supine) and with the presence of anticipatory anxiety (not seen in the supine condition) are consistent with an increased level of central noradrenergic activity (arising from decreased baroreceptor activity) in a more upright posture (as hypothesised by Elam et al., 1984). Nevertheless, there are alternative mechanisms that could have potentially contributed to these results. Changes in the physiological state of the body associated with a more upright posture (e.g., increased heart rate and diastolic blood pressure) may induce activity in particular cortical regions via feedback from the periphery (e.g., Craig, 2003). The idea that peripheral feedback affects the generation of emotion has a long heritage (James, 1884; Lange, 1885/1912). The same idea is contained in a more recent theory (e.g., Damasio, 2003) which also suggests that, via their association with emotional states, representations of peripheral activity can affect cognitive processes; for example, a person's decisions may be guided by the valency of affective states (in essence "gut feelings") that occur when considering potential outcomes (Bechara, Damasio, Tranel & Damasio, 1997). In

addition, this thesis reports postural differences in the physiological reactivity associated with performing a cognitive task; for example, heart rate reactivity during mental arithmetic was higher when supine than when standing (Studies 1 and 3). Changes in cardiac activity may be consciously perceived (e.g., as peripheral somatic sensations, Obrien et al., 1998) and thereby influence psychological state; for example, people may feel their heart rate has increased and interpret this to mean that they are anxious or stressed (e.g., along the line of James, 1884).

There may be other effects associated with a postural manipulation that could potentially influence either cognitive or affective processes. To illustrate one of these, standing is associated with a higher core body temperature than lying down (due to heat loss from dilated blood vessels in the latter, Tikuisis & Ducharme, 1996). Furthermore, body temperature is thought to influence arousal (Krauchi et al., 1997) and state anxiety (Morgan & O'Connor, 1988), though mechanisms to account for these effects are not known.

The extent to which particular mechanisms contributed to the psychological effects associated with posture in this thesis remains to be determined; however, there is a sound theoretical rationale for believing postural differences in central noradrenergic activity (arising from differences in baroreceptor activity) played an important role (contributions from non-noradrenergic influences related to posture are less clear because the relationships of these effects to cognitive and affective processes are currently not well understood). The presence of greater central noradrenergic activity when standing is suggested by the Study 3 finding of a higher skin conductance level when standing than when supine, given that skin conductance responses have been shown to be reliant upon the central noradrenergic system (at least in cats, Yamamoto et

al., 1990). Postural differences in central noradrenergic activity may be able to be clarified in future studies with measures of pupil diameter, which is altered by drugs acting at central alpha$_2$-adrenoceptors (Phillips et al., 2000) and has been shown to reflect locus coeruleus activity (Rajkowski et al., cited in Gilzenrat et al., 2003).

The role of baroreceptor-mediated effects in the postural influences observed in this thesis could be more accurately determined by removing the postural factor; for example, baroreceptor activity could be manipulated using changes in lower body pressure (e.g., Hamer et al., 2003). Also, the postures investigated in this thesis, standing and supine, were chosen to represent extremes (of natural postures) so that differences in baroreceptor activity, and therefore differences in the extra-homeostatic effects of baroreceptor activity, would be marked; it remains to be determined if similar findings arise between different postural conditions (e.g., supine and sitting).

Implications for understanding the processes used to solve anagrams

Given the strong theoretical rationale for there being greater locus coeruleus activity when in a more upright posture (e.g., Elam et al., 1984), the Study 1 finding that participants solved anagrams more rapidly when supine than when standing is consistent with the idea that the ability to solve anagrams is impaired by central noradrenergic activity (e.g., Beversdorf et al., 2002). Central noradrenergic activity may impair the ability to solve anagrams by suppressing the activation of neuronal groups that represent individual words (by inhibiting the spontaneous activity of a group's cells, e.g., Mantz et al., 1988); attempts to solve an anagram are likely to be less effective when the neuronal group representing the solution is suppressed. Given this, it could be expected that the cognitive processes normally utilised in solving anagrams are not reliant upon central noradrenergic activity (unlike other forms of cognition, e.g.,

vigilance, Rajkowski et al., 1998). This idea is supported by the Study 2 finding of a relatively low level of peripheral physiological reactivity (including reactivity of sympathetic origin, e.g., skin conductance) associated with performing anagrams (cf. mental arithmetic). In the context of the global sympathetic system (Aston-Jones et al., 1994) this is consistent with there being a relatively low level of central noradrenergic activity (i.e., little change in central noradrenergic activity from resting levels) during attempts to solve anagrams.

There being a low level of physiological activity during attempts to solve anagrams could reflect low conscious mental effort expenditure; both physiological reactivity (e.g., blood pressure) and mental effort are proportional to activity in certain cortical regions, including the anterior cingulate (Critchley et al., 2000, 2003). Furthermore, demonstrated projections from the anterior cingulate to the locus coeruleus (Rajkowski et al., 2000) indicate that (via the anterior cingulate) mental effort may result in increased central noradrenergic activity (cortical modulation of central noradrenergic activity has also been suggested by Heilman et al., 2003, and Sturm et al., 1999). This suggests that, in keeping with the idea that central noradrenergic activity impairs the ability to solve anagrams (e.g., Beversdorf et al., 2002), solutions to anagrams may be favoured to arise in the absence of conscious mental effort.

The role of mental effort and central noradrenergic activity in solving anagrams could be determined with measures of pupil diameter, which is thought to reflect both mental effort (e.g., Beatty, 1982) and locus coeruleus activity (Rajkowski et al., cited in Gilzenrat et al., 2003). It might be expected that solutions to anagrams would be found when pupil diameter (and thus central noradrenergic activity) is low, at least for solutions that participants report to occur in a moment of insight (consistent with the

idea that insight is facilitated when central noradrenergic activity is minimal, Beversdorf et al., 1999). It is because anagrams are often solved in a moment of sudden awareness, and can thus be used to model the moment of insight classically associated with creative breakthroughs (e.g., Bowden, 1997), that makes understanding the role of central noradrenergic activity and mental effort in solving anagrams important in a broader cognitive context. For example, the Study 1 finding that posture influenced the ability to solve anagrams endorses the idea that posture may also influence insight and/or creative thinking.

Baroreceptor activity, negative affect and the theory of learned hypertension

Given that baroreceptor activity is greater in a less upright posture, the Study 3 finding that anticipatory anxiety developed while participants were standing, though not while they were supine, is consistent with the idea that baroreceptor activity can reduce anxiety. This is important in relation to the theory of learned hypertension, under which it has been proposed that baroreceptor activity reduces both anxiety (Dworkin et al., 1994) and, in a more general sense, the aversiveness of a stressful situation (Dworkin, 1988).

However, seemingly overlooked in the theory of learned hypertension, it is not straightforward to translate the effects of baroreceptor activity into effects of blood pressure. For example, in Study 2 (and as reported by Lepore, 1995) a positive correlation between blood pressure reactivity and self-reported stress was found. This relationship may be associated with a re-setting (to a higher blood pressure level) of the operating point of the baroreflex during mental stress (Fauvel et al., 2000), meaning that blood pressure per se can not be taken as an indicator of the likely strength of baroreceptor-mediated effects. This would appear to detract from the theory of learned

hypertension. However, it is currently unclear whether the modulation of cortical arousal and other extra-homeostatic effects of baroreceptor activity may occur in isolation to the homeostatic (blood pressure maintenance) effects. This is an important area for future research, as if it is the case that cortical arousal and/or negative affect are reduced by an elevation in blood pressure (at least in a sub-group of the population) it may indeed be a mechanism by which chronic hypertension could develop. Nevertheless, it must be remembered that there are many factors not addressed by the theory of learned hypertension (including obesity, alcohol, salt, and size at birth) that are likely to contribute to the development of hypertension (Isles, 2000).

Careful consideration must be given to the means by which negative affect is induced in future studies investigating a modulatory role of baroreceptor activity; this is because of there being low arousal in a high baroreceptor activity condition. When arousal is low, a task in which the participant is required to be actively engaged may invoke compensatory mental effort (required to overcome the state of low arousal) and thereby involve a greater level of negative affect (Hockey, 1997). Lower cortical arousal in the supine condition (at rest) could thus explain why negative affect after the mental arithmetic task was not reduced (cf. standing) in Study 3 (there was no difference in task performance between standing and supine conditions). There being lower cortical arousal in the supine condition was expected on the basis of EEG studies (e.g., Cole, 1989); these are augmented by the Study 3 finding that skin conductance was greater in participants when standing than when supine (given that skin conductance is an accepted indicator of cortical arousal levels, e.g., Barry et al., 2004).

As with engaging tasks, a task in which the participant is more passive (e.g., viewing affective slides) may also be problematical when assessing negative affective responses in a low arousal condition, as there may be insufficient attention devoted to the stimuli.

Given the Study 3 finding of postural differences in anticipatory anxiety, an aversive anticipatory state may be a more appropriate means of investigating differences in negative affect associated with differences in baroreceptor activity (or in fact between any conditions differing in arousal).

Further implications (posture and beyond)

Irrespective of the precise mechanisms underlying postural influences on psychological processes, there are implications from the findings of this thesis for any experiment in which procedures are conducted while participants are supine; which is typically the case during brain scanning studies. To illustrate, Chua, Krams, Toni, Passingham and Dolan (1999) reported that a distracter task shown in non-scan pilot studies to reduce the anxiety associated with receiving electric shocks, failed to have this anxiolytic effect in participants supine for PET scans. A modest increase in anxiety during the experimental phase was cited by Chua et al. as one of the reasons potentially responsible for this null result. Given that in Study 3 anticipatory anxiety was found to develop in the standing condition, though not in the supine condition, it may be that a larger anxiety effect would have been found in the Chua et al. study (and against which an anxiolytic effect of the distracter task could have arisen) had the experiment not required participants to be supine.

The psychological influences of posture found in Studies 1 and 3 of this thesis may have their origins in baroreceptor-mediated effects, thus there are potential implications for non-postural situations in which changes in baroreceptor activity occur. In the absence of significant gravity, blood is distributed more evenly throughout the body, leading to an increase in the central venous pool and thereby an increase in activity of cardiopulmonary (and maybe arterial) baroreceptors (Pump, Videbaek, Gabrielsen &

Norsk, 1999). Research into the physiological effects of low gravity is important for understanding the implications for health in such an environment (e.g., during space flight, Williams, 2003); this may be complemented by knowledge of how changes in baroreceptor activity influence psychological processes.

Concluding remark on the influence of posture on psychological processes

This thesis both extends and improves upon the limited number of previous studies that have found an effect of postural condition on higher-order psychological processes (e.g., in terms of performance on a convergent thinking task, Schulman & Shontz, 1971); it does this by demonstrating new postural influences in the context of a solid theoretical rationale. Two of the major findings support the notion that higher-order psychological processes modulated by central noradrenergic activity are also modulated by posture. This adds to a previous report that vigilance, known to be regulated by locus coeruleus activity (Rajkowski et al., 1998), is improved in sleep-deprived participants when standing (cf. seated, Caldwell et al., 2003). In making this addition, the findings of the thesis suggest a trade-off concerning the influence of posture on cognition; to illustrate, vigilance may be improved in a more upright posture at the expense of other cognitive processes, such as those utilised when solving anagrams (as per Study 1).

Finally, while it was demonstrated in this thesis that posture influences both the ability to solve anagrams and the development of anticipatory anxiety, these higher-order influences are likely to be corollaries of more basic effects. The most basic, and the most frequently demonstrated, is an effect of body posture on cortical arousal: a more upright posture is associated with a higher level of cortical arousal (e.g., Cole, 1989, and as suggested by skin conductance data in Study 3). This low-level psychological effect can be envisioned as facilitating the behaviours required of particular body postures; this includes alertness when upright and interacting with the environment, and relative

sedation when lying down for relaxation or sleep. Regardless of whether the modulation of higher-order processes is part of this facilitating effect, findings that commonplace differences in posture influence arousal, cognition, and affect highlight a fundamental role of the peripheral body in regulating psychological phenomena.

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APPENDIX A

STIMULI FOR STUDIES 1 AND 2

Mental arithmetic

"16 + 27 - 11 + 22 = " 54

"28 + 19 - 16 + 22 = " 53

"17 + 38 - 21 + 25 = " 59

"38 + 18 - 32 + 13 = " 37

"18 + 35 - 21 + 14 = " 46

"26 + 17 - 22 + 13 = " 34

"16 + 26 - 11 + 18 = " 49

"15 + 28 - 32 + 16 = " 27

"21 + 15 - 13 + 28 = " 51

"13 + 26 - 15 + 18 = " 42

"23 + 15 - 22 + 17 = " 33

"14 + 35 - 21 + 19 = " 47

"27 + 12 - 16 + 29 = " 52

"13 + 25 - 12 + 18 = " 44

"21 + 18 - 23 + 15 = " 31

"22 + 17 - 24 + 15 = " 30

"33 - 17 + 41 - 12 = " 45

"43 - 26 + 22 - 15 = " 24

"42 - 15 + 31 - 23 = " 35

"32 - 13 + 26 - 18 = " 27

"34 - 18 + 23 - 13 = " 26

"42 - 25 + 11 - 13 = " 15

"31 - 19 + 25 - 14 = " 23

"53 - 16 + 22 - 18 = " 41

"54 - 23 + 15 - 18 = " 28

"45 - 17 + 11 - 21 = " 18

"47 - 25 + 35 - 19 = " 38

"48 - 31 + 12 - 16 = " 13

"24 - 12 + 25 - 17 = " 20

"27 - 13 + 33 - 18 = " 29

"39 - 17 + 25 - 11 = " 36

"38 - 16 + 15 - 12 = " 25

Anagrams

"ruasg" sugar	"lnfai" final
"ahecb" beach	"ebrag" barge*
"edmlo" model	"gaiem" image
"gnita" giant	"csofu" focus
"tubhm" thumb	"aicbn" cabin
"tmlae" metal	"hmdui" humid
"ithgl" light	"nodru" round
"tomhn" month	"hsgot" ghost*
"wreta" water	"nmoev" venom
"hualg" laugh	"culne" uncle*
"iehtw" white	"crsaf" scarf*
"rciep" price	"epdsa" spade
"pcarm" cramp	"ndikr" drink*
"apytr" party*	"garhp" graph
"osien" noise	"onmwa" woman*
"ihrac" chair*	"isrht" shirt

*These anagrams were not included in Study 2; they were replaced with:

"neyrt" entry	"eclri" relic
"ohsac" chaos	"upmle" plume
"ortai" ratio	"ndukr" drunk
"proea" opera	"ynhea" hyena

APPENDIX B

STATISTICAL DATA NOT IN THE TEXT

Table B1

Study 1: F-ratios for regression analyses

	Standing	Supine
Systolic b.p.	2.05	2.66
Diastolic b.p.	0.02	0.04
Heart rate	1.66	0.00
Skin cond.	1.52	2.71
Resp.rate	0.54	1.88

Note. df = (1, 18).

Table B2

Study1: t-ratios for first to second posture comparisons

Systolic b.p.	0.51
Diastolic b.p.	0.30
Heart rate	1.25
Skin cond.	6.65***
Resp. rate	0.63

Note. df = 19. ***p < .001.

Table B3

Study 2: F-ratios for regression analyses

Systolic b.p.	0.18	Stroke volume	2.22
Diastolic b.p.	0.28	Cardiac output	2.12
Heart rate	1.41	Total periph. rest.	3.78[a]
Skin cond.	0.88	Difficulty	28.41***
Resp. rate	0.03	Stress	3.00[a]

Note. df = (1, 35) for cardiac output and stress, df = (1, 34) for difficulty and respiration rate, df = (1, 36) otherwise, ***p < .001, [a]p < .1.

Table B4

Study 3: F-ratios for raw anxiety data comparisons

	Task/P2			Recovery		
	Multivariate	Short form	Self-reported	Multivariate	Short form	Self-reported
Standing						
Control	0.65	0.10	1.00	0.80	1.03	1.51
Task	3.97	7.07*	4.68	3.86	3.04	4.10
Supine						
Control	0.20	0.19	0.09	0.20	0.27	0.00
Task	15.86**	18.98**	24.66**	0.46	0.89	0.04

Note. df = (2, 17) for multivariate and (1, 18) for univariate analyses, *p < .05, **p < .01.

Table B5

Study 3: t-ratios for raw stress data comparisons

	Task/P2	Recovery
Standing		
Control	1.07	0.00
Task	3.52**	4.66**
Supine		
Control	0.94	0.27
Task	3.27**	1.45

Note. df = 19, **p < .01.

Table B6

Study 3: t-ratios for raw standing condition physiological data comparisons versus baseline

	Control session			Task session		
	P2	P3	P4	Task	P3	P4
Systolic b.p.	1.32	1.37	0.63	6.60**	1.32	0.43
Diastolic b.p.	2.71*	2.70*	2.28*	6.60**	2.14	2.30
Heart rate	0.38	1.37	0.04	2.16	0.04	0.18
Skin cond.	4.15**	6.07**	6.91**	5.66**	7.57**	5.68**
Resp. rate	-	0.82	2.03	-	2.27*	2.50*
Stroke volume	0.26	0.38	0.96	0.65	0.55	1.37
Cardiac output	0.05	0.80	1.16	2.35	0.95	1.87
Total periph. rest.	1.92	2.83*	2.82*	2.67*	1.72	1.98

Note. df = 19 (except in the task session for systolic and diastolic blood pressure, stroke volume, cardiac output and total peripheral resistance, where df = 17), *p < .05, **p < .01.

Table B7

Study 3: t-ratios for raw supine condition physiological data comparisons versus baseline

	Control session			Task session		
	P2	P3	P4	Task	P3	P4
Systolic b.p.	2.05	2.42*	3.09*	4.07**	3.19*	2.18*
Diastolic b.p.	3.33*	2.81*	3.18*	5.90**	2.84*	1.68
Heart rate	1.36	0.24	0.99	6.85**	0.22	0.26
Skin cond.	3.04**	3.96**	3.67**	6.60**	6.75**	4.31**
Resp. rate	-	0.45	0.41	-	1.22	1.22
Stroke volume	1.83	0.23	0.10	0.77	1.76	0.08
Cardiac output	2.31	0.22	1.22	5.23**	1.00	0.05
Total periph. rest.	2.86*	2.97*	3.46**	0.43	1.49	1.70

Note. df = 19 (except in the task session for systolic and diastolic blood pressure, stroke volume, cardiac output and total peripheral resistance, where df = 17), *p < .05, **p < .01.

Table B8

Study 3: t-ratios for transformed physiological data comparisons versus baseline

	Stand			Supine		
	Task	P3	P4	Task	P3	P4
Systolic b.p.	5.57**	0.12	0.38	3.85**	1.75	0.51
Diastolic b.p.	4.98**	0.15	0.29	4.91**	0.62	0.08
Heart rate	2.30	0.96	0.17	6.42**	0.07	0.28
Skin cond.	4.44**	2.44**	0.40	5.58**	1.26	0.80
Resp. rate	-	0.95	0.22	-	1.12	1.06
Stroke volume	0.61	0.81	0.86	0.32	2.05	0.05
Cardiac output	2.50	0.21	0.95	5.36**	1.29	0.40
Total periph. rest.	1.66	0.04	0.70	1.61	0.14	0.14

Note. df = 17 (except for skin conductance, heart and respiration rates, where df = 19), **p < .01.

Table B9

Study 3: t-ratios for transformed physiological data comparisons between the standing and supine conditions

	Task	P3	P4
Systolic b.p.	2.01	0.99	0.61
Diastolic b.p.	1.31	0.22	0.15
Heart rate	3.77**	0.68	0.31
Skin cond.	0.98	0.09	0.25
Resp. rate	-	0.06	0.46
Stroke volume	0.48	1.98	0.76
Cardiac output	2.46	1.46	1.27
Total periph. rest.	2.45	0.13	0.51

Note. df = 17 (except for skin conductance, heart and respiration rates, where df = 19), **p < .01.