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

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The Neurogenic Hypothesis of RSI
John Quintner and Robert Elvey

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Commentaries:
Trevor Beswick and Annette Cursley
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INTRODUCTION

Bernardino Ramazzini (1713) is generally credited with writing the first comprehensive treatise on occupational health. In it he made several references to what we now call work-related neck and upper limb disorders. Although there is still no standard definition for these disorders, they include 'myalgia, periostinitis, tenosynovitis, carpal tunnel syndrome, thoracic outlet syndrome, Guyon's canal syndrome, hypothenar hammer syndrome, vibration induced white finger, game keeper's thumb, osteoarthritis of the CMC thumb joint, and fatigue. Numerous terms have been introduced to refer to these disorders and include: occupational cervicobrachial disorders, repetitive trauma disorders, cumulative trauma disorders, repetitive strain injuries and overuse syndrome' (Armstrong et al., 1988).

Since the 1700s interest in these disorders has fluctuated. The 1970s saw the beginnings of a renewed upsurge in research particularly in the field of ergonomics. Consequently there is an increasingly good understanding of the workplace factors, related both to biomechanics and work organisation, which are associated with these disorders. Clinical research has, however, lagged behind.

This deficit became critically important in Australia in the mid-1980s, when there was a marked increase in reporting of what were called repetition strain injuries or RSI. (By the late 1980s similar upsurges were also reported in the USA and UK.) For many workers the disorders produced severe hardship and this was compounded by the lack of understanding of underlying pathology and consequent difficulties with diagnosis and treatment. The disorders became responsible for a large portion of the payouts for workers' compensation and the associated economic cost was a major factor in sparking an intense debate about whether or not the disorders have an organic base. Protagonists in the debate played an important role in court cases to determine whether or not compensation should be awarded, and more importantly, in defending or discrediting those who claimed to be affected.

The underlying arguments were analysed by, among others, Brian Martin and myself (1988). Among the criticisms of what we called the standard view, namely that there is physical damage which is work-related, are that

* there are no objective signs on which diagnosis can be made,
* there is no underlying pathology,
* the symptoms do not make clinical sense,
* orthodox treatments, particularly rest, do not work, and
* there is no consistent relationship between symptoms and work.

The critics generally have not argued their cases systematically or in detail and the alternative explanations which they favour have even more problems than the standard view when examined with the same rigour. Nevertheless, the five points mentioned above need to be dealt with by any hypothesis proposing an organic basis for these disorders.
Introduction

The debate about whether or not these disorders have a physical underpinning has diverted attention and energy from another, and in my opinion more important, debate. If most of these disorders do have an organic basis (and even the critics would agree that some do), what is it? Most practitioners have some notions about the underlying pathology; some emphasise trigger points, others muscle fibre changes, others irreversible irritability of nerves and so on. In general these hypotheses have not been clearly expounded, let alone discussed, in an attempt to reach deeper understanding.

An important caveat needs to be made here. In many, and perhaps even most, individuals with work-related neck and upper limb disorders, a number of conditions with different underlying pathologies probably co-exist. The challenge is not only to establish clear organic bases for different conditions, along with diagnostic criteria and treatment strategies, but to define ways of identifying, in individual cases, co-existing conditions.

In late 1989 I conceived the idea of inviting some of the leading protagonists of different viewpoints to write detailed expositions of their hypotheses. These were to be circulated amongst a variety of people with different expertise, to encourage discussion from a number of perspectives. The protagonists were also to be invited to respond to these commentaries.

The following paper, *The Neurogenic Hypothesis of 'RSI*', by John Quintner and Robert Elvey is the first in this series. It provides an important overview of one explanation for these disorders and is an excellent starting paper for discussion. There are 11 commentaries, which highlight the most important points for debate and further work.

A number of other expositions are being prepared. Along with John Quintner and Robert Elvey, the protagonists are clinicians and undertaking this task on top of heavy caseloads. I am grateful to them and to the commentators for generously devoting time to this project. This series of papers will be important in furthering understanding of the pathology of these disorders, allowing those with 'RSI' to be diagnosed more accurately and treated with more success.

This is a working paper destined for publication, along with the other papers in this series, in a book. Further contributions to the debate, either commentaries on this paper or expositions of a particular hypothesis, are invited. Please contact me for details.

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THE NEUROGENIC HYPOTHESIS OF 'RSI'

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I. INTRODUCTION

The upsurge in cases of a syndrome known as repetition strain injury (RSI) or occupational overuse syndrome (OOS) in the 1980s highlighted lack of precision in the medical diagnosis of work-related neck and upper limb disorders. Exemplifying the prevailing ignorance, Ferguson stated that 'the majority of cases of repetition strain injury are not localised syndromes, but of a more diffuse disorder, apparently of muscles...and...little is known of its aetiology, pathogenesis and pathology...nor, if when established, why it appears to persist despite prolonged rest of the patient.'

We were impressed by the uniformity of presenting clinical features in many of our patients, from diverse occupations, who had been given (by others) a diagnosis of 'RSI' for compensation purposes. We were unable to detect recognisable upper limb musculoskeletal pathology in these patients. For reasons that will be explained in this discussion paper, we considered that the presence of a neurological disorder was a more likely explanation of their symptoms.

The stimulus to further investigate this possibility was the previous original research of one of the authors in the area of differential diagnosis of upper limb pain. The technique of physical examination which resulted from this research (the brachial plexus tension test) has proven valuable in the diagnosis of patients suffering from other conditions associated with widespread cervicobrachial pain.

In this discussion paper we first briefly outline the clinical features of the 'RSI' pain syndrome and then propose a hypothesis for their development. This is followed by a detailed description of the supporting evidence. We next discuss the differential diagnosis for this condition and conclude by outlining some of the implications of our hypothesis for treatment.

We also provide two attachments, the first of which outlines a systematic approach to the physical examination of upper limb tissues and the second deals with the concept, rationale and methodology of brachial plexus tension testing.

II. CLINICAL FEATURES OF 'RSI'

1. Common Symptoms
(a) pain—initially localised to one anatomical site, either proximal (neck, upper back, shoulder) or distal (hand, wrist, forearm or elbow)—subsequently becoming widespread in one or both arms, upper back, neck and head—pain descriptors include aching, burning, electrical, sometimes sharp and shooting—may follow course of major peripheral nerves of arm
(b) paraesthesiae* (includes dysesthesiae) e.g. tingling, pins and needles, numbness, weakness, heaviness, fullness, fatigue
(c) feeling of coldness of painful upper limb

* either induced by movement of the arm or occurring at rest
(d) sensation of swelling of the acral portion of the limb
(e) tenderness of tissues at site of pain
(f) cramping sensation of muscles in the affected arm
(g) painful limitation of movement
   [i] cervical spine
   [ii] shoulder
(h) varying degrees of psychological distress, pain amplification phenomena or frank psychiatric illness may be present.

2. Physical examination findings (based both upon the authors' observations and published studies)
(a) Tenderness on palpation of neural tissues related to the painful upper limb
   [i] in upper limb, felt along the course of nerves e.g. radial nerve anterior to lateral epicondyle when pain involves radial aspect of forearm; median nerve in the cubital fossa when pain involves ventral forearm; ulnar nerve in its groove behind medial epicondyle when pain involves medial aspect of forearm
   [ii] over the anterior aspect of ipsilateral lower cervical transverse process, corresponding to spinal nerve (anterior primary ramus) in the gutter of the transverse process. 33
(b) Provocative tests positive for upper limb symptoms
   [i] sustained cervical flexion/extension postures. 33
   [ii] brachial plexus tension test of Elvey 33
   [iii] free arm hanging test. 14
   [iv] forearm tension tests e.g. tethered median nerve stretch test,64 radial nerve stretch test. 24
   [v] false positive Finkelstein's test in radial sensory nerve entrapment. 24
   [vi] Phalen's test for carpal tunnel syndrome.87
(c) Painful (+/- limited) range of movement
   [i] cervical spine. 14
   [ii] active abduction/elevation of shoulder (with elbow extended) 14, 33
   [iii] shoulder capsulitis.14,119
(d) Antalgic posture of the arm - shoulder adduction and internal rotation, elbow flexion, wrist and finger flexion.
(e) Altered peripheral neural sensibility
   [i] hypoesthesia 14
   [ii] allodynia, hyperalgesia, hyperpathia syndrome.14
   [f] Vasomotor phenomena 14
      [i] coldness of painful extremity.79
      [ii] cyanosis or pallor of painful extremity.79
(g) Overt signs of reflex sympathetic dystrophy. 14

III. THE HYPOTHESIS OF CAUSATION OF 'RSI'

This hypothesis relates to the majority of patients who present, as described by Ferguson,37 with a diffuse pain syndrome of the upper limb(s), often accompanied by pain in the neck and upper back. For the purposes of this discussion paper, this syndrome will be referred to as 'RSI'. Clinically identifiable musculoskeletal pathology causing upper limb pain may coexist with 'RSI', but is usually insufficient to explain the full clinical picture. In addition, non-occupational causes of diffuse upper limb pain (e.g. other causes of cervical radiculopathy, brachial plexopathy and upper limb entrapment neuropathy) need to be differentiated from 'RSI'.

The hypothesis holds that:
1. The clinical features of 'RSI' (as outlined earlier) arise from irritable neural tissues related to the upper limb. These tissues exhibit the properties of increased mechanosensitivity and ectopic impulse formation. 97 Other pathophysiological mechanisms relevant to neuropathic pain 27, 38, 124 may be involved and are outlined in this discussion paper.

2. The sensori-neural tissues related to the painful arm have become irritable as a result of pathological changes induced by excessive mechanical tension and/or friction generated during manual work of a repetitive nature, usually performed with postural fixity of the head and neck. 14, 33, 88

3. The neural tissues predominantly affected by these forces are proximally situated (cervical spinal nerve, nerve root complex, brachial plexus) 14, 33; however, an identical clinical presentation (wide-spread neural pain) may result from entrapment of distal upper limb neural tissues. 71

IV. EVIDENCE IN SUPPORT OF THE 'NEUROGENIC' HYPOTHESIS

* hypoalgesia deleted; see commentary by Milton Cohen and response

** changed from (f) reflex sympathetic dystrophy; see commentary by Milton Cohen and response.
An understanding of the hypothesis requires a knowledge of anatomy and biomechanics of the neural elements of both the spine and the upper limb, together with related aspects of neurophysiology, neuropathology, clinical neurology and occupational medicine.

The evidence will be presented under the following headings:-
1. neuropathic pain
2. case studies of patients with 'RSI' and analogous conditions
3. human experimental studies
4. occupational health field studies
5. biomechanical studies of cervical and upper limb neural tissues
6. response of neural tissues to stretch, tension and friction
7. experimental studies of damaged spinal nerve root, dorsal root ganglion and peripheral neural tissues
8. the brachial plexus (upper limb) tension test of Elvey and other clinical tests of cervical spine and/or brachial plexus neural irritability.

1. Neuropathic Pain

First, the concept of referred pain needs to be briefly discussed. Kellog,58,59 using chemical injection of muscles, deep fascia, tendons, periosteum, and interspinous ligaments as the stimulus, was able to distinguish superficial (skin) pain from deep pain. He found that local pain arising from structures deep to the skin may be accompanied by referred pain. Referred pain was defined as pain falsely localised, and thus interpreted by the sufferer as arising from one deep tissue when, in fact, it had originated in another.58 Much of the pain arising from muscles and other soft tissues, bones and joints can be misinterpreted in this way and cause serious errors of medical diagnosis to be made.

Grieve47 has suggested the following classification of pain states commonly encountered in patients with musculoskeletal diseases:
1. local pain - pain perceived at the site of tissue damage
2. referred pain without root involvement - pain perceived in tissues which are not the site of tissue damage and whose afferent or efferent neurones are not involved in any way
3. referred pain of root (peripheral nerve) involvement - pain experienced in tissues which are not the site of primary tissue damage, but are generally innervated by neurones involved in the tissue damage.

Referred pain of non-root musculoskeletal origin has the following characteristics: ‘dull, aching, boring quality, difficult to describe; it lies deep; it radiates for considerable distances; the area outlined by the patient does not correspond to peripheral nerve distribution or spinal nerve root distribution. There may be accompanying feelings of numbness; feelings of heaviness; soreness of muscle (cramp); tenderness of muscle, and muscle spasm at times; tenderness of bony prominences; secretomotor and vasomotor changes (blanching, sweating).47

Referred pain of root involvement may be difficult to diagnose in the absence of accompanying neurological deficit.47 In an important hypothesis, Abusby and Fields3 proposed that pain due to peripheral nerve damage may be categorised as either dysesthetic pain or nerve trunk pain. Dysesthetic pain was described as burning, tingling, searing or raw, and was usually perceived in the cutaneous area innervated by the affected nerve. Nerve trunk pain was described as a deep aching pain which extended along the course of the nerve. The involved nerve was often found to be tender. Both types of pain may be present in a patient with nerve dysfunction.

More is becoming known about the persistent pain states associated with traumatised or irritated peripheral nerves.27,38,124 Fields38 refers to this pain as neuropathic. Neuropathic pain can be of extreme severity, can spread extensively and can be associated with muscle tenderness and cutaneous hypersensitivity in segmentally-related regions to the injured neural tissue. Onset of pain may be delayed following injury to nerve and may persist long after the original insult. Pain can be associated with abnormal or unfamiliar unpleasant sensations (dysesthesiae), frequently having a burning and/or electrical quality. A paroxysmal brief shooting or stabbing component is also described. Pain may be felt in a region of sensory deficit, and, within this region, mild stimuli may be painful (allodynia); there may be an increased response to normally painful stimuli (hyperalgasia). The phenomena of temporal summation, spread of pain and after-reaction with repetitive stimuli may also be elicited (hyperpathia).38

According to Devor27, ‘a body of data has begun to emerge that indicates previously unsuspected modes whereby nerve trauma and irritation could generate problematic pain states by actions at the level of the damaged nerve itself. Suggested mechanisms of neuropathic pain include ectopic impulse formation from a site of damage along the course of a nerve, loss of afferent inhibition,
ephaptic transmission, and sympathetic activation or facilitation of primary afferents. The dorsal root ganglion also becomes an ectopic generator when peripheral nerve is damaged. Sensitization of C polymodal nociceptors may explain hyperalgesia and other features of reflex sympathetic dystrophy which can accompany neuropathic pain.

One of the authors has argued on clinical grounds that the pain of 'RSI' resembles that described in brachial neuropathy. An obvious similarity can be seen between the behaviour of the pain of 'RSI' and neuropathic pain. In addition, the associated symptoms (paresthesiae etc.) of both conditions may be identical. Further clinical evidence in support of the 'neurogenic' hypothesis will be presented in the following section. The diagnostic relevance of brachial plexus tension testing will become obvious later in this discussion paper. However, it is important at this stage to point out that such testing may provide the major means of determining clinically whether or not there is a significant neural pathology underlying the upper limb pain of a particular patient.

2. Case Studies of 'RSI' and Occupational Cervicobrachial Disorder

1. The Australian experience of 'RSI'

In a study published in 1971, Ferguson reviewed medical certificates issued to process workers said to have suffered repetition injuries. This was the first Australian study to highlight the serious occupational health problems of female workers. On the one hand, he maintained that the ill-defined symptom complexes, affecting the majority of those studied, were 'probably most often simple muscle strains.' On the other hand, after noting that a simple muscle strain should recover in a week or two of removal from the source of strain, he speculated that brachial plexus or cervical nerve root pressure or traction may have been responsible for the severe, prolonged and widespread pain syndromes of some workers.

There were no other major studies until the 1980s. Taylor et al. studied 89 cases of 'process workers' arm', a syndrome which encompassed 'a range of musculo-tendinous lesions' of the upper limb and neck. Although 68% of their patients complained of numbness of the affected limb, muscle repetition injury (91%) and tenosynovitis (82%) were the commonest diagnoses made. Taylor et al. explained numbness as due to neural tissue compromised by swollen muscles. Their minimal criteria for the diagnosis of tenosynovitis and epicondylitis were inadequate tenderness by accepted orthopaedic criteria for these diagnoses. Their diagnostic category of muscle repetition injury suggested a failure to distinguish between true muscle injury and pain referred from elsewhere into muscle. It is likely that many of their patients were experiencing pain of referred neural origin.

Stone wrote of 100 patients with repetitive strain injuries who presented to him over a 13 week period. The more common components of repetitive strain injuries were 'tendinitis, tenosynovitis, peritendinitis, tenovaginitis, myositis or repetition muscle injury, epicondylitis, chronic muscle strain, ganglions and neuritis...reflex sympathetic dystrophy...and thoracic outlet syndrome.' Stone did not outline his criteria for the diagnosis of muscle strain or injury in his original paper; in a later paper he inferred that muscle belly tenderness indicated muscle injury and that tenderness over tendons denoted a pathological process involving either the tendon or its sheath. The possibility of muscle tenderness being associated with referred pain from either somatic or neural structures was not discussed by Stone.

Browne et al. proposed guidelines for both diagnosis and management of patients with occupational repetition strain injuries. The frequency of semi-objective physical findings such as local tenderness (in muscle, tendons, bone), pain on movement of joints, or on resisted contraction of muscles, and the reproduction of paresthesia and numbness by evocative measures, was emphasised. Objective features (of inflammation) were said to be unusual findings in chronic cases. Symptoms in these cases could arise from multiple sites (possibly caused by multiple pathologies). They attributed ongoing pain in the muscles of the neck and shoulders to the long-term effects of static muscle loading. Phenomena associated with referred pain were not considered by these authors, but their criteria for diagnosis are consistent with a neurogenic basis for the symptoms of many patients.

Fry studied 379 musicians with painful overuse syndrome of their upper limbs. He assumed that their pain was due to a pathological process involving their painful muscles, caused by overuse of these muscles. He based this opinion on tenderness found in muscle tissue at the site(s) of pain. Once established, upper limb pain tended to spread proximally and distally. Fry made but brief mention of more complex spinal pain in some
musicians. He later examined a larger group of musicians with upper limb and/or spinal pain. Upper limb pain was again attributed to overuse of the respective muscles whereas neck pain was attributed to asymmetrical loading (possibly of cervical musculature) in the sitting posture. His reported examination findings were said to have excluded nerve root lesions. Fry did not indicate how he was able to distinguish between pain arising from overused muscles and pain referred into the muscles from painful cervical spinal tissues. When Dennett and Fry reported minor and non-specific abnormalities in biopsy material from the first dorsal intersosseous muscle of patients in whom they had made a diagnosis of overuse syndrome, they concluded that these changes supported their hypothesis of overuse injury. But as Cyriax warned, anatomical diagnosis must precede pathological investigation: 'until the tissue at fault has been singled out, microscopy is out of place.'

Prominent involvement of the nervous system of female patients with ‘RSI’ was noted by Champion et al. Their evidence included a history of paraesthesiae to the fingers in 67%, and referral for neurophysiological investigations of 38% of the women. Clinical examination revealed evidence suggesting proximal neural irritability in 59% and distal neural irritability in 27%. They postulated irradiation of cervical spinal nerve roots and/or brachial plexus as an important neurogenic mechanism in the ‘RSI’ syndrome. This neural irritability presumably resulted from their exposure to friction generated during repetitive movements of the arm and neck, and from traction or tension, a consequence of both forward bending of the neck and drooping of the shoulder girdle.

Our own work also concluded that the upper limb symptoms of a group of severely affected patients diagnosed by others as ‘RSI’ arose from cervical or brachial plexus neural tissues. These symptoms were provoked by the brachial plexus tension test devised by Elvey. Symptoms in most patients were also reproduced during the sustained neck postures of forward flexion and/or backward extension, performed with the patient recumbent in the supine position. Abnormal tenderness of neural tissues on palpation over the gutters of the transverse processes of the related cervical levels on the side of the painful limb was elicited in most patients. We hypothesised that maintenance of the forward flexed or ‘poked forward’ head/neck postures during the performance of repetitive work may have been the major factor in the development of ‘RSI’ in our patients.

Miller and Topliss conducted a cross-sectional study of 229 consecutive patients referred with chronic upper limb pain which had been labelled 'repetitive strain injury' or 'overuse syndrome.' Two hundred (57%) of these patients did not fulfil criteria for a specific rheumatological diagnosis. However, as a group, their patients reported pain which had spread from an initial localised anatomical site to become widespread. Paraesthesiae were reported in the painful upper limb by 91%. Their conclusion that there was no evidence of physical injury in the majority of their patients was challenged in an editorial comment by Smythe on the grounds that a more careful examination of the cervical region on the side of the painful arm would have revealed unsuspected tenderness related to the anterior aspect of the lower transverse processes. Smythe deduced that ‘RSI’ in Australia was a syndrome of arm pain referred from the neck.

A population of 127 (62%) volunteers and 77 (38%) patients, all with pain in the upper limb or neck and 'a highly stylized and repetitive' occupation, were prospectively studied by Sikorski et al. Most of their subjects reported multiple areas of pain involvement and 122 (60%) reported a sensory disturbance of some form. They were able to make a diagnosis of a recognisable musculoskeletal disorder in 118 (58%) 'without resorting to vague clinical terminology such as fibromyalgia, fibrosis or regional pain syndrome.' Their clinical assessment was, in part, based on their subjective judgement as to whether arm symptoms were compatible with a radicular or peripheral nerve entrapment. Electrodiagnostic tests were performed on the 81 subjects thought on clinical assessment to have a nerve entrapment; they were positive in only 26. There were 86 (42%) subjects in whom a diagnosis could not be made. These subjects complained of poorly localised pain, diffuse tenderness, easy fatigability of upper limb muscles, and widespread upper limb weakness. Sikorski et al. discussed possible explanations: that they were not fit enough for the physical demands of their jobs, that they were suffering from a discrete physical disease or disorder that has so far resisted definition, that their problem was a psychosomatic disorder, that they were suffering from an iatrogenic disorder, or that their claims were fraudulent. They favoured the first of these, but offered no explanation for the persistence and severity of the reported symptoms of these patients. In many of their undiagnosed subjects, the symptoms appear compatible...
with our hypothesis but this cannot be fully assessed as the authors did not use an examination technique to assess the mechanosensitivity of neural tissues related to the upper limb.

In summary, studies in Australia of those suffering from work-related neck and upper limb conditions have shown that the commonest of these conditions is a syndrome, more frequent in females, characterised by widespread pain in one or both arms, in the upper back, neck and head, often accompanied by complaints of paraesthesiae, numbness, heaviness, weakness, or perceived swelling of the painful arm. Tenderness of painful muscles, tendons and/or ligaments was a prominent finding. This was the syndrome which became known as 'RSI'. The concept of repetition strain injury was originated based on the hypothesis that performance of manual work of a repetitive nature could injure the muscles involved. This type of muscle injury was said to result in persistent and widespread arm pain. The diagnostic dictum of Cyriax that 'it is never tenderness of a muscle, but pain elicited by the appropriate resisted movement that identifies a muscle lesion' was apparently forgotten. That upper limb symptoms may have been referred from structures 'at fault' within the neck or the brachial plexus of many patients was not discussed by the authors of most of the Australian studies, although the evidence they presented is compatible with this explanation.

2. Overseas experience of 'RSI'.

It is not feasible, within the constraints of this paper, to comprehensively review all overseas work related to these disorders. A recent review has been carried out by Wallace and Buckle. Some key studies from Japan and Sweden are reviewed here.

In the 1960s, the Japanese occupational health physicians became aware of pain syndromes related to the occupation of their keypunchers and other keyboard workers. Initially, tenosynovitis was the diagnosis used to explain forearm, wrist and hand symptoms. However, a growing awareness that pain was often more widespread into the shoulder, neck and head led to the use of broader anatomical diagnoses such as cervicobrachial syndrome and, eventually, 'occupational cervicobrachial disorder' (OCD). Components of the OCD symptom complex included varying degrees of pain and stiffness in the neck, shoulder, arm, hand and fingers, paraesthesiae, functional disturbance of the peripheral circulation (e.g., coldness), and weakness of the painful arm. Characteristic findings on physical examination were positive (symptom provoking) neurological tests (e.g., Adson's test), tenderness and/or enlargement of affected muscles, tenderness of nerves, (cervical) paravertebral tenderness, percussion pain over (cervical) spinous processes and decreased muscle power. Autonomic disturbances as well as mental symptoms were found in patients who were severely affected.

According to Jonsson et al., the clinical manifestations of OCD and 'RSI' (OOS) are identical, the emphasis in Australia being placed upon the repetitive nature of the work performed whereas Japanese and Scandinavian studies tended to highlight constrained working postures. For example, Kvarnström studied musculoskeletal disorders of the shoulder region in 112 workers employed by a large Swedish manufacturing company. They fulfilled the following criteria: shoulder symptoms as the dominant reason for inability to work, loss of more than 4 (continuous) weeks from work, shoulder pain and fatigue related to work and eased by rest and tenderness of shoulder girdle musculature and/or rotator cuff tendon insertions. Although Kvarnström grouped neck and shoulder conditions together under the OCD diagnosis, he did not distinguish between cervical conditions referring pain into the shoulder girdle and shoulder conditions causing local pain. The etiology which he favoured implied injury (microtrauma) to the cervical and/or shoulder girdle musculature due to their repetitive contractions or ischaemia resulting from their continuous static contraction involved in supporting the weight of the arm.

3. Human Experimental Studies

The field studies described above have highlighted the importance of work-related factors such as posture and repetition in causing these disorders. A number of laboratory studies have looked carefully at the postural side of causation and these are summarised here. In terms of the neurogenic hypothesis, posture seems to be the most important causal consideration.

Chaffin reported a study of five young healthy women who were using microscopes. He studied the head tilt angles of each woman during her use of the microscope. He aimed to determine the time taken to reach a state of fatigue, which he defined as the presence of continuous 'cramping' with deep 'hot' pain intermittently present, when the head was held
at specific degrees of tilt for 50 minute intervals, with a 10 minute rest between intervals. The results indicated that tilting the head forward more than 30 degrees greatly increased the neck extensor (sic) fatigue rates. He noted that an angle of 15 degrees produced no subjective sensations after 6 hours.

In an important series of experimental studies using asymptomatic volunteers, Harms-Ringdahl and Ekholm 49 'aimed to see if pain could be felt after maintained experimentally-controlled extreme positions of the lower neck, similar to common sitting work postures, and, if so, whether the extreme position and/or pain induced changes in muscular activity.' They found that an extreme forward-flexed position of the head and neck, if maintained, resulted in complaints of neck and upper back pain, within the first 2-15 minutes, in all volunteers. Within 16-57 minutes after onset of pain, all required relief of this position. The pain always started in the lower cervical/upper thoracic region and spread towards the head and shoulders. Three subjects felt pain in the upper limbs; another two experienced numbness in one or both arms. Pain passed off quickly on release of the position but returned within the next 24 hours and lasted for 1 to 4 days in 9 of the 10 subjects. Electromyography (EMG) of cervical musculature indicated a very low level of muscle activity until high levels of pain were reported. At this stage of the experiment, muscle activity, as judged by EMG, increased in response to the pain. Harms-Ringdahl and Ekholm 49 found a positive correlation between the magnitude of the load moment induced by the weight of the head and neck during provocation and the levels of average pain intensity and of pain intensity accumulated at the point when provocation was discontinued. Their studies suggested that pain brought on by an extreme forward position of the cervical spine is induced by strain on passive connective tissues rather than by muscle contractile activity. They also considered the possibility that pain could arise from mechanically-induced changes within the cervical nervous tissue and its meninges occasioned by the adaptation of these tissues to the increase in length of the cervical spinal canal which occurs in the forward flexed position. 9

Colombini et al. 16 studied the seated posture of 10 healthy volunteers. Using rather complex formulae, they calculated the approximate load on the lower portion of the cervical spine in six different seated postures. Their estimate was that the mean compressive load on the C5/6 intervertebral disc was likely to increase from 20-30kg with the head upright (e.g., looking at a VDU screen) to 30-40kg with the head bent forward (e.g., reading from notes flat on a desk). These loads are concentrated in a relatively small area of the spine and may be shared by other structures which include cervical spinal musculature, ligamentous structures and neural tissues. It is possible that some or all of the tissues which are exposed to loads of these magnitudes may become symptomatic.

Although not an experimental study, Levy 69 reported the case history of a man who had been tied up for 12 hours with his head forced into and held in extreme forward flexion. This man developed a partial motor and sensory loss (C5 - T1 on the right, C3-T1 on the left). Investigations did not reveal evidence of underlying pre-existing cervical disease. Wilder 128 discussed this case in relation to the mechanism of midcervical quadriplegia after posterior fossa operations carried out with the patient in the sitting position. His hypothesis of the mechanism of such neurological catastrophes, as well as the mechanism in Levy's case, was that such extreme positions of the neck were damaging to the cervical spinal cord due to the high tension that develops with biomechanically induced stretching of the cord. He postulated that spinal ischaemia developed due to mechanical interference with spinal cord vasculature.

In summary, the evidence from human experimental studies shows that both the upright and the forward flexed head/neck posture so commonly adopted when sedentary work is performed, can result in an adverse load falling on cervical connective and neural tissues. If maintained, these postures may lead to pain in the neck, head, upper back, shoulder and arm. The risk of spinal cord damage increases with the length of time an extreme adverse forward flexed posture is maintained. A conscious and mobile person develops intense pain within a relatively short time spent in the extreme forward flexed head/neck position and is compelled to release this position. A single exposure can result in this pain recurring, without further provocation, over the next few days. We are not suggesting that other cervical postures are not potentially harmful. However the above evidence strongly suggests that the forward flexed head/neck posture does lead to difficulties and this clearly ties in with the neurogenic hypothesis.

4. Occupational Health Field Studies

The field studies described below show that the postures found to be problematic in
laboratory studies are commonly found in the workplace.

Hunting et al.\textsuperscript{55} carried out a field study of 119 female accounting machine operators. They included a control group of 57 female shop assistants. Whilst working, the first group maintained a continuous sitting posture, the control group mostly stood. In both groups, reports of trunk complaints did not differ, with problems reported in the neck (30\%), shoulders (20\%) and in the back (50\%). However, there was a two-fold increase in upper limb complaints in the accounting machine operators. The researchers concluded that trunk complaints were not related to the specific working postures of those in either group, but that the upper limb complaints of the accounting machine operators were so related. Their right arms were more frequently symptomatic (pain, tiredness, cramp) than their left arms. Keyboard operations were thought to impose a special load on the right upper limb. After analysis of the dimensions of the workplace and the body postures of the operators, they concluded that increasing head/neck angles (mean value, 60 degrees) in forward bending, increasing elbow angles and lateral deviation of the right hand and wrist were unfavourable constrained postures in this work situation. The researchers assumed that all upper limb symptoms of the operators arose from upper limb tissues (particularly from muscles). Between 20-30\% of operators reported left hand-arm symptoms, despite the left hand being used only to turn over coupons and the left elbow being supported on the desk. No attempt was made to explain these symptoms. However, they did observe that, in order to read the source documents, the operators were obliged to turn their trunk and head to the left as well as to maintain a forward-bent head position. As discussed elsewhere in this paper, this neck posture could generate tension in right-sided lower cervical neural tissues, thus producing a state of neural irritability with referred pain into the right upper limb.

Kilborn et al.\textsuperscript{61} conducted a cross-sectional study of 96 female employees in the electronics industry. None had lost time from work or had sought medical attention for cervico-brachial disorders within the previous year. Postures and movements of the neck, shoulders and upper arms during work were evaluated from video-tape taken using a systematic video recording technique. Their medical evaluation utilised a standardised questionnaire and a physical examination of the neck, shoulder girdle and upper limbs which relied heavily on the finding of tenderness on palpation over a muscle belly in the anatomical region of pain as evidence of the presence of a physical disorder. Physical capacity for each worker was determined by estimating maximal static strength and static endurance. They could not demonstrate any relationship between the physical capacity of the individual worker and the presence of symptoms. Their results revealed that the number of flexing or abducting movements of the upper arm per hour were negatively related to neck, shoulder/neck angle and shoulder symptoms. Time spent in neck flexion, shoulder elevation, upper arm abduction or total time when the arm was active were all statistically related to symptoms from these regions. Headache appeared as a strong determinant for neck and neck-shoulder angle symptoms. Kilborn et al.\textsuperscript{61} emphasised the probable multifactorial aetiology of cervicobrachial symptoms related to work. Of considerable importance were the large inter-individual variations in working technique and posture demonstrated by the video technique.

This group of workers was examined again after intervals of 1 and 2 years.\textsuperscript{57} At the initial examination some workers had been found to have relatively severe disorders which had been diagnosed as tendinitis and/or myofascial syndromes. At the end of one year there was an increase in the proportion diagnosed as suffering from severe disorders. A further increase occurred at the end of two years. The results showed that 10-25\% of workers had moderate to severe symptoms related to the neck-upper back region at the end of the second year of observation. Ten per cent had moderate to severe symptoms in their arms. Poor working postures related to the neck and shoulders were clear indicators of risk of developing cervico-brachial disorders.

Laville\textsuperscript{66} made observations both under experimental conditions and at the workplace of people performing repetitive work (television assembly, sewing and keyboard/VDU operation). He found a direct relationship between work tasks requiring speed and precision and postural immobility of the neck and torso. He stated that ‘posture participates in activity, and is itself a means for performing the activity.’ He recommended attention to work stress factors as well as to factors of workplace design in order to relieve workers of neck, shoulder and back pains.

In summary, repetitive manual work performed in the sitting position is intrinsically associated with postural immobility of the head and the neck. Increasing head/neck angles in forward flexion adopted by many
sedentary workers are directly related to reporting of symptoms related to the head, neck, upper back, shoulders and arms. Symptoms may become persistent and disabling in a proportion of workers who perform sedentary manual repetitive work.51

5. Biomechanical Considerations Relevant to Neural Tissues

Biomechanical studies of neural tissue show how these tissues painlessly adapt to normal movement of the spine and limbs. They do so by alterations in their length and tension. As will be shown, certain postures of the neck, if maintained, may result in potentially harmful adverse tension. Repetitive movements of the upper limbs may also be damaging to particular peripheral nerves at specific anatomical sites of vulnerability.

1. Cervical neural tissues

O'Connell83 performed important anatomical studies when seeking to understand the physical signs of spinal meningeal irritation (rigidity of the neck, limited straight leg raise). He observed that 'when the head is fully flexed upon the trunk, a cephalad movement of the dura and spinal medulla occurs - the tension in the intradural nerve roots being increased.' He also noted that full flexion of the head produced an increase in tension in the intradural roots and extradural nerves throughout the spinal canal. He hypothesised that the physical signs of meningitis were the result of the development of reflex muscle spasm, as a protective mechanism for inflamed spinal meninges.

Breig9 showed that the spinal canal increased in length by 7 cm in the average adult from flexion to extension. Neural tissues were shown to adapt to this change in length by passive deformation, as the dura is firmly anchored at both its cranial and its caudal end. In neck extension, the cervical cord and dura undergo axial compression and hence, shortening and slackening. In cervical forward flexion the cervical dura is pulled taut, the cord becomes thinner in its anterior/posterior diameter and is lifted away from the dorsal aspect of the canal due to increase in axial tension. The axis cylinders of the spinal cord are folded when the neck is in extension and are straightened when it is in flexion. Lateral flexion of the cervical spine stretches the nerve roots on the contralateral convex side and slackens them on the ipsilateral side. Rotation to the right stretches the right dural sleeve and the right dorsal rootlets while slackening the ventral ones; on the left, the dural sleeve and dorsal rootlets are slackened, whereas the ventral rootlets are stretched.7

In a series of anatomical dissections, Reid92 confirmed the findings of O'Connell.83 He undertook further investigations of changes of movement, stretch and tension in spinal neural tissues when the head moved from extension into forward flexion. He estimated that up to 1.8 cm of movement of the spinal cord and its dural sac could take place. The greatest stretch occurred between C2 and T1. Another important finding was that during this movement, the tension exerted by the cervical cord and dura upon the anterior wall of the spinal canal reached maximum values of 30-40 lb. per square inch (about 250 kilopascals). The cervical nerve roots were also shown to move up and down, pivoted from the dorsal root ganglion. Reid also noted slight movement of the cervical nerve roots on cervical forward flexion. However, this movement was more marked during abduction of the shoulder or downward traction on the arm.

Adams and Logue,1 using wire and pin markers, performed radiological studies of the flexed and extended cervical spines of 10 cadavers. The dural sac moved both by shift and by unfolding of the dural segments under consideration. The extrathecal roots were found to be fixed in the lateral part of the intervertebral foramina, but to move up and down in the long axis of the spinal canal. Their findings confirmed those of Reid.92

Louis72 used 24 fresh cadavers to study the entire vertebral canal and its contents during relative displacement of the spine from hyperextension to hyperflexion. The cervical nerve roots were stretched and pulled taut during hyperflexion as well as during lateral hyperflexion to the opposite side. He found that, in adapting to the position of spinal hyperflexion, the cord segments and meningeal structures slide towards the most mobile vertebra in the cervical (C6) and lumbar (L4) regions, where the osteo-meningomediullar relations are constant. Stretch forces were mainly concentrated at the C6 and L4 myelomeres and the roots of the cauda equina distal to the 4th lumbar roots.

The evidence from these studies supports the hypothesis that a high physical tension may develop within the cervical spinal neural tissues of those who perform repetitive manual work which is accompanied by elements of cervical forward flexion, rotation and lateral flexion.
2. Peripheral neural tissues

A peripheral nerve, when cut, retracts approximately 10-20% of its length. This fact indicates that peripheral nerves are under some tension in vivo. A certain amount of resting tension appears necessary in a peripheral nerve to help it adapt to limb movements which diminish the length of its nerve bed. Biomechanical changes occur in a nerve as its bed elongates; it straightens, untwists and stretches. As shown by McLellan and Swash, the median nerve slides longitudinally in its bed when adapting to changes in length of the upper limb during movement. In this way, a local increase in nerve tension can be dispersed along the extent of the nerve.

Sunderland summarised the features which protect peripheral nerves from forces of stretch, tension, friction and compression generated during the normal use of a limb. These include their fascicular complexes, the loose framework of the bed which separates them from neighbouring structures, their undulating course in a limb, their innate tensile strength and elasticity, their 'cushioning' epineurium and, for the nerves of the brachial plexus, the tone of muscles which elevate the shoulder girdle and the first rib. Peripheral nerves are especially vulnerable under the following circumstances; where they cross the extensor aspect of a joint; where they pass over a rigid fibrous or ligamentous band; where they travel through fibrous, osseofibrous or osseous canals or tunnels; where they pass through two closely applied muscles, or through deep fascia to become a superficial cutaneous nerve.

The anterior primary rami of the lower cervical spinal nerves (C5, C6, C7) appear prone to entrapment as they pass through the gutters of their respective transverse processes. Peripheral nerves of the upper limb are vulnerable at a number of sites; the ulnar nerve in the cubital tunnel; the posterior interosseous nerve in the radial tunnel; the anterior interosseous branch of the median nerve between the two heads of the pronator teres muscle; the median nerve in the carpal tunnel. In the next two sections of this discussion paper, the pathoanatomy and pathophysiology of damaged neural tissues are considered.

6. Response of Neural Tissues to Stretch, Tension and Friction

Under experimental conditions, stretch has been shown to cause varying degrees of structural damage to blood vessels, nerve fibres and perineurium of peripheral nerves. Stretch has also been shown to impair the epineurial circulation, which, in turn, may compromise the intraneural microvascular flow leading to endoneurial anoxia and oedema formation. In the clinical situation, a diffuse inflammatory reaction (swelling, fibrosis) in the epineurium can ensue from chronic irritation of peripheral nerves in the anatomical situations, mentioned above, where they are vulnerable. Localised changes in myelin sheaths, often associated with intraneural fibrosis and axon degeneration have also been described. Axonal degeneration is usually only apparent in the presence of severe compression neuropathies.

Neural fibrosis caused by varying degrees of chronic mechanical irritation may be distributed in the tissues of the nerve bed and the superficial epineurium, in the interfascicular epineural connective tissues and perineurium and, following severe trauma or compression, intrafascicularly. Fibrosis (scar tissue) has potentially severe consequences as it imperils nerve fibres by constricting them, by impairing their blood supply and by forming adhesions at the injury site. Traction on the damaged nerve caused during limb movements now deforms a hyper-sensitive nociceptive focus and results in pain.

Pathoanatomical studies of spinal nerve roots have been confined to the lumbar region. The findings of these experimental studies may not be applicable to cervical nerve roots. Rydevik et al., after reviewing the responses of lumbar nerve roots to compression and tension, put forward a model to explain ongoing painful nerve root conditions. The blood vessels of the lumbosacral nerve roots appear to be well adapted to withstand temporary forces of tension. This is because they possess blood vessels with primary and secondary compensating coils, numerous cross-connecting and relatively large bore arteriovenous anastomoses and are bathed in cerebrospinal fluid which supplies nutrition to the root tissue. In experimental animal models, acutely applied compression forces appear capable of injuring nerve roots and dorsal root ganglia by causing an alteration in their microcirculation, intraneuronal ischaemia and oedema. In mice, both exudative and proliferative histological changes were found in sciatic nerves exposed to chronic mild irritation. A model of intermittent
mechanical irritation had been devised which was thought to simulate the clinical situation of loss of nerve root mobility due to lumbar disc protrusion.

Lindahl and Rexed\textsuperscript{70} reviewed previous reports of nerve root pathology found at autopsy of patients who had been known to suffer from sciatica. They were able to confirm that pathological changes of inflammation may be found in the majority of biopsy specimens taken from nerve roots at the time of the spinal operation on patients with sciatica of long standing.

In summary, there is evidence that the microcirculation of peripheral nerves, spinal nerve roots and the dorsal root ganglia can be seriously impeded by mechanical forces. Both acute and chronic mechanical irritation have been shown to cause varying degrees of damage to peripheral nerve and spinal nerve roots. Although the relevance of neuropathological findings derived from animal experimental studies to the clinical situation of spinal and peripheral nerve disorders in humans requires clarification, the evidence is supportive of our general thesis.

\section*{7. Experimental Studies of Damaged Spinal Nerve Root, Dorsal Root Ganglion and Peripheral Neural Tissues}

Howe et al.\textsuperscript{54} proposed a physiological basis for the radicular pain of nerve root compression. They observed that, in the cat, prolonged firing of axons ensued after mechanical stimulation, however slight, of chronically injured lumbar dorsal nerve roots. This mechanosensitiviy was not present in normal nerve roots. Compression of the dorsal root ganglion was also found to result in prolonged repetitive firing in sensory axons.

The dorsal root ganglion is a possible source of ectopic impulses contributing to the pain and paraesthesiae of nerve root injury and disease in humans. Wal1 and Devor\textsuperscript{125} showed that injury to the rat's sciatic nerve induced dorsal root ganglion cells to increase their tendency to discharge spontaneously and to bombard the spinal cord with ectopic sensory signals. They hypothesised that, in humans, pain felt in the leg on the straight leg raising test could be the consequence of tension transmitted to the dorsal root ganglia which are mechanically stressed by this manoeuvre.

Although the preceding studies are important in that they provide a possible neurophysiological basis for human neuropathic pain states, human experimental studies are necessary to confirm their hypotheses. With the use of microneurography, Nordin et al.\textsuperscript{82} demonstrated ectopic sensory impulse formation which corresponded to symptoms of pain and paraesthesiae in patients with different types of nerve disorder. Paraesthesiae in a patient with suspected thoracic outlet syndrome which were provoked by arm elevation were recorded microneurographically, as were symptoms provoked by straight leg raising in a patient with sciatica due to S1 nerve root fibrosis.

Electrophysiological responses were recorded at operation by Crawshaw et al.\textsuperscript{17} from nerve roots of 11 patients with chronic and complicated lumbar spinal radicular pain syndromes. They found an attenuation of evoked potential which possibly reflected abnormal sensitivity of either nerve root or dorsal root ganglion to mechanical disturbance.

In humans, a marked increase in the sensitivity of inflamed nerve roots has been demonstrated in the lumbar spine\textsuperscript{75,104} as well as in the cervical spine. Frykholm\textsuperscript{42} performed operations on the cervical spine for disc pathology using local anaesthesia. He found that both dorsal and ventral roots at the level of operation were extremely sensitive. When the dorsal root was touched, the patients immediately experienced arm pain in a dermatomal distribution. When the ventral root was touched, pain radiated into muscles of the upper limb which had been painful and tender prior to operation. The first type of pain was referred to as 'neuralgic', the second type as 'myalgic'. Myalgic pain, in contrast to neuralgic pain, did not conform to a dermatomal pattern. These two types of pain correspond, respectively, to the dysaesthetic and nerve trunk pains hypothesised by Asbury and Fields.\textsuperscript{3} Descriptions of upper limb pain in patients with 'RSI' provide evidence of both types of painful response.\textsuperscript{33} The clinical model provided by Frykholm\textsuperscript{42} strengthens the hypothesis of a cervical neural origin for the upper limb symptoms of many patients with 'RSI'.

\section*{8. The Brachial Plexus Tension Test of Elvey}

Elvey\textsuperscript{30,32} developed the brachial plexus tension test (BPTT) to assist the examiner in differentiating between painful local upper limb disorders and cervical/brachial plexus disorders referring pain along neural tissues into the upper limb. This test is described in more detail in Attachments 1 and 2.
In the cadaver, movement and tension of the brachial plexus and the cervical nerve roots with their investing sheaths and anatomically related dura occurred with specific movements of the upper limb.30,32 The position of the upper quarter that placed the cervical nerve roots under maximum tension combined gleno-humeral joint abduction to 110 degrees, and external rotation with the arm behind the coronal plane, with contralateral cervical flexion, shoulder girdle depression, elbow extension, forearm supination and wrist extension.45,95,101 The greatest movement took place in the C5 and C6 nerve roots, with lesser effect at C7.32

Kennally60 studied the responses to BPTT in 100 asymptomatic, healthy volunteers. Fifty were aged between 18-30 years, and fifty between 50-67 years. At end range of the BPTT, the responses most consistently evoked were a deep stretch or ache sensation in the cubital fossa, extending down the anterior and radial aspects of the forearm into the radial side of the hand, and a definite tingling sensation in the thumb and first three fingers. These responses have now been documented in studies of over 400 normal individuals.5,65,95 Techniques involving positioning of the contralateral upper limb55 and straight-leg-raising5 have been shown to alter the responses elicited on brachial plexus tension testing, thus providing additional support for the concept of the test.

Selvaratnam et al.100 carried out a clinical validation study of the BPTT. They used three groups of subjects. The first group consisted of patients who developed shoulder or upper arm symptoms which were likely to have arisen from the brachial plexus, damaged as a result of cardiac bypass surgery. The second group were sportspeople with shoulder or upper arm symptoms arising from injuries caused by throwing. The third group consisted of asymptomatic normal subjects. The BPTT was found to have discriminative validity, moderate to high intra-examiner reliability and could therefore be used in the clinical situation to discriminate between the presence or absence of a brachial plexus involvement in patients with upper limb symptoms.

The BPTT appears to be a useful test* in clinical situations where upper limb pain and paraesthesiae may** arise from dysfunctional neural tissues related to the symptomatic arm.89,90,103,123 There are other clinical tests said to assist in the diagnosis of putative brachial plexus or cervical neural irritability causing upper limb symptoms. Tests designed to provoke upper limb symptoms include the Adson manoeuvre,2 the Spurling-Scoville test,107 the three minute elevated arm stress test of Roos,94 the arm hyperabduction test,130 passive downward traction on the upper limb,14,117 and the exaggerated erect military posture.34 Tests which depend upon relief of upper limb symptoms are axial manual cervical traction127 and the shoulder 'abduction' test (strictly, the test depends on shoulder girdle elevation).23 Interpretation of these tests is based upon the subjective responses of the patient together with, for some tests,2,34,130 obliteration of the ipsilateral radial pulse. However, the frequent finding of pulse diminution when these tests are performed in the asymptomatic normal population makes them poor diagnostic tools for symptomatic neuro-vascular compression in the thoracic outlet.20,94

Glassenberg46 noted that the upper limb pain of some of his patients diagnosed as suffering from thoracic outlet syndrome was reproduced on neck movement (particularly hyperextension) or on Spurling's test, thus resembling the responses of patients with cervical radiculopathy. Roos94 claimed that a positive response to the three minute elevated arm stress test and shoulder bracing distinguished thoracic outlet syndrome from other conditions with similar symptoms, such as cervical disc disease and carpal tunnel syndrome. Spurling's test,107 axial manual cervical traction127 and the shoulder abduction test23 were all found to have high specificity but low sensitivity for radicular pain associated with nerve root compression due to cervical disc disease.122

The BPTT is unique amongst the above-mentioned tests in that both subjective and objective (range of movement) responses can be evaluated by the examiner. A positive test not only reproduces the patient's symptoms but the examiner can predictably alter these responses by adding or subtracting known sensitising manoeuvres.5,95 For a review of the tests used to assess adverse mechanical tension within the nervous system (BPTT, slump and response

* changed from 'highly sensitive' to 'a useful test'; see commentary by Milton Cohen and response

** 'may' inserted; see commentary by Milton Cohen and response
V. DIFFERENTIAL DIAGNOSIS OF THE PAIN SYNDROME 'RSI'

As indicated earlier, non-occupational causes of diffuse upper limb pain need to be differentiated from 'RSI'. Neural pathologies caused by mechanical factors (of possible relevance in an occupational setting) will be considered in this section. Musculoskeletal conditions will not be discussed as their diagnostic features are well established, and there is general agreement that they are uncommon in the majority of patients with 'RSI'.

1. Cervical Radiculopathy

Associated with cervical spondylosis

Mechanical irritation of the lower cervical nerve roots is the most common cause of brachial neuralgia. Cervical spondylosis and acute cervical disc herniation are the pathological entities which are usually associated with the development of cervical radiculopathy. An acute radiculopathy is usually the result of cervical intervertebral disc prolapse. The way in which the subacute and chronic cervical radiculopathies develop is not clear. Frykholm regarded cervical spondylosis as a predisposing factor for the development of nerve root symptoms. Nerve root sleeve fibrosis and osteophytic encroachment into the intervertebral foramina were changes which appeared to diminish the ability of the lower cervical nerve roots to adapt to stretching occasioned by forward flexion of the neck. Occupational factors, such as work necessitating prolonged or even intermittent neck hyperextension, hyperflexion or rotation, heavy labour or an atypical activity, may have a causal relationship in the development of symptoms.

The symptoms of subacute and chronic cervical radiculopathy associated with cervical spondylosis may be indistinguishable from those of the pain syndrome 'RSI'. It is generally accepted that radiological changes of cervical spondylosis may be found in totally asymptomatic individuals. However, Lawrence did find a relationship between cervical disc degeneration and reports of a past episode of neck-shoulder-brachial pain in both sexes, but only on those with moderate or severe disc space narrowing. More severe degrees of disc degeneration were found in those who had been engaged in heavy manual labour. In their review of cervical radiculopathy, Dillin et al. stated that the diagnostic criteria necessary to establish a diagnosis of cervical radiculopathy due to compressive pathology included a positive myelographic study and/or CT scan, a root distribution neurological deficit and radicular arm pain. According to these criteria, cervical spondylotic radiculopathy should be clearly distinguishable from the 'RSI' pain syndrome. If a worker presents with a cervicobrachial pain syndrome and radiological evidence of cervical spondylosis, it may be difficult to assess the relative importance of occupational factors, as opposed to the degenerative pathology affecting cervical nerve root tissues, in the genesis of the pain syndrome.

Apart from cervical spondylosis, other, less common, causes of cervical radiculopathy, such as spinal tumours, congenital spinal defects and various inflammatory spinal disorders, may need to be considered in the differential diagnosis.

2. Following cervical injury

After cervical injury, many patients report that their neck and referred upper limb symptoms are worsened by prolonged reading or writing, especially if performed in an unnatural or uncomfortable position. There are reports that upper limb symptoms may be delayed in onset for weeks, months or even years after a neck injury sustained in a motor vehicle accident. Nine of 13 patients with delayed onset of upper limb symptoms were in employment of a clerical nature at the time of onset of these symptoms. It may therefore be very difficult to assess the contribution of work-related factors to the development of a cervicobrachial pain syndrome in a previously neck-injured worker.

2. Thoracic Outlet Syndrome

Chronic injury to the neurovascular bundle in the thoracic outlet has been attributed to repetitive movements of the shoulder and arm. Diagnostic tests suggested by these authors include the Adson manoeuvre, the costoclavicular manoeuvre and the hyperabduction test. They recognised that these tests may cause pulse diminution in normal individuals; nerve conduction velocity studies and (in some cases) angiography were therefore recommended to confirm the clinical diagnosis. Enthusiasm for making the diagnosis of thoracic outlet syndrome has waned in recent years as the previously accepted diagnostic criteria (including electrodiagnosis) have not stood up to critical analysis. In the absence of signs of digital...
ischaemic insult, thoracic outlet syndrome appears extremely rare and difficult to diagnose.\(^{46}\) According to Elvey,\(^{32}\) a pathological process which involves the brachial plexus (e.g. Pancoast tumour), and thereby causes upper limb symptoms, may result in a positive response to BPTT. The absence of physical signs of cervical vertebral dysfunction would alert the examiner to search for pathology in the thoracic outlet (as well as for more distal upper limb pathology involving neural tissue).\(^{51}\)

### 3. Upper Limb Entrapment Neuropathies Associated with Wide-spread Pain

Pain associated with entrapment neuropathies is usually localised, but it may radiate to other sites, some at a considerable distance from the site of damage.\(^{110}\) This phenomenon has been noted in patients with the pain syndrome 'RSI'.\(^{14,79}\) It is also important to recognise the inability of clinical electrophysiological testing (nerve conduction and EMG) both to infer symptoms or neuropathic deficit, and also to infer pathological alteration of nerve fibres or interstitial pathology.\(^{29}\) Electrodiagnosis has not given positive information in most patients with 'RSI'.\(^{14,79,102}\)

#### 1. The median nerve

The clinical and electrodiagnostic features of carpal tunnel syndrome have been well described.\(^{50,87,109,110,118}\) Entrapment of the median nerve in the carpal tunnel may cause pain in the hand which can radiate up the arm to the shoulder, and sometimes to the neck.\(^{19,71,87}\) The component of proximal radiation of pain into the forearm can be reproduced in patients with chronic carpal tunnel syndrome by simultaneous extension of the supinated wrist and the distal interphalangeal joint of the index finger (the 'tethered' median nerve stress test).\(^{64}\) Overlap of symptoms between carpal tunnel syndrome and cervical radiculopathy can therefore occur, e.g. distal paraesthesiae and numbness, hand weakness and arm pain.\(^{71,85}\) Russell\(^{96}\) postulated that, in patients with cervical spondylosis, median nerve dysfunction at the level of the carpal tunnel may, at least in part, be biomechanically dependent upon changes in the elasticity of the sheaths of its related cervical nerve roots.

Dual lesions (carpal tunnel syndrome and cervical radiculopathy) have been described in some patients. Upton and McComas\(^{121}\) presented evidence that most patients with carpal tunnel syndromes or ulnar neuropathy at the elbow have evidence of cervical nerve root damage. They proposed a 'double-crush' hypothesis; that neural function is impaired because single axons, having been compressed in one region, become especially susceptible to damage at another site, due possibly to interference with axoplasmic flow.

In a study of patients' upper limb (neurological) pain syndromes, distal symptoms were usually dominant in those with isolated carpal tunnel syndrome, whereas proximal symptoms tended to be dominant in patients with cervical radiculopathy or double crush syndrome.\(^{85}\) Comparison of the physical findings in the three groups showed that thenar atrophy occurred only in the carpal tunnel group; but abnormal sensibility testing, Tinel's sign at the wrist and a positive Phalen's test were present in some patients from each group.

The median nerve may become entrapped between the two heads of the pronator teres muscle from excessive pronating movements of the forearm or damaged by fibrous bands in this region.\(^{35,110}\) This entrapment may cause pain on the volar surface of the forearm, reduced sensibility and paraesthesiae in the radial three and one half digits of the hand, weakness of grasp and clumsiness of hand movements. Pain elicited on resistance to elbow flexion, forearm pronation or the superficial flexor to the middle finger may help determine the exact location of nerve entrapment. Electrodiagnostic testing may also be helpful in localisation of the site of entrapment.\(^{110}\)

#### 2. The radial/posterior interosseous nerve

Radial tunnel syndrome can cause pain over the site of entrapment, usually where the posterior interosseous nerve enters the sharp fibrous arch of the supinator muscle or the tendinous margin of the extensor carpi radialis brevis.\(^{53}\) Tenderness over the site of entrapment and pain on resistance to forced supination are said to be important diagnostic tests in the clinical situation where the nerve damage has not resulted in paresis of finger extendors. In some patients there may be a correlation between cervical radiculopathy and compression of the radial nerve at the level of the elbow.\(^{85}\)

Entrapment of the superficial branch of the radial nerve in the forearm (between the tendons of brachioradialis and extensor carpi radialis longus) has become increasingly recognised as a possible cause of pain and sensory disturbance on the radial aspect of the lower forearm and wrist in those who perform repetitive pronation/supination movements of the forearm at work.\(^{24}\) In some patients with
this syndrome, pain was reported as radiating up the arm towards the shoulder. Physical examination findings of importance are said to include a positive Tinel's sign over the radial sensory nerve as it exits the deep fascia, a false-positive Finkelstein's test and a positive forearm hyperpronation provocative test. Nerve conduction studies may assist in the diagnosis of this entrapment syndrome.

3. The ulnar nerve

Entrapment of the ulnar nerve at the elbow produces a distinct clinical syndrome. The frequent association of ulnar neuropathy and cervical radiculopathy was noted by Upton and McComas. In the absence of acceptable criteria for the diagnosis of thoracic outlet syndrome, the possible association between entrapment of the ulnar nerve in the cubital tunnel and simultaneous compression of the C8-T1 roots in the thoracic outlet remains undetermined.

Although it is usually possible to accurately diagnose the specific entrapment neuropathies of the upper limb, some patients may present with symptoms which have spread beyond the territory of the entrapped nerve. In the presence of widespread neural pain and neural irritability (as found in 'RSI'), it may be then difficult to determine whether the primary site of neural dysfunction is proximal (cervical/brachial plexus) or distal within the upper limb. There may even have been multiple sites of neural entrapment at onset as a consequence of the transmission of excessive neural tissue tension over the course of the nerve.

VI. CONCLUSION

The body of evidence presented in this discussion paper provides support for each of the elements of the neurogenic hypothesis outlined earlier. The accepted pathogenic mechanisms of entrapment neuropathies may also explain the development of the 'RSI' pain syndrome. Entrapment may affect either the neural tissues in the cervical region, with ensuing distal spread of pain and sensori-neural irritability, or more distal neural tissues (e.g. median nerve in carpal tunnel) followed by proximal spread of pain and sensori-neural irritability. The end result of both processes may be indistinguishable.

'RSI' appears to be an example of 'pathophysiologica! pain. According to Devor, this is 'pain that occurs spontaneously, or in response to weak stimuli, due to pathophysiologica! abnormalities of neural excitability...it is a disease in its own right...a disease of membrane excitability regulation.'

VII. IMPLICATIONS FOR TREATMENT OF THE 'RSI' PAIN SYNDROME

1. To date, the lack of general agreement on the diagnostic criteria for the complex pain syndrome 'RSI' (OOS) explains the dearth of studies (in Australia and in other countries) of both its natural history and response to treatment. The limited information available indicates that those who have been more severely affected may take a considerable time to recover. According to Hosokawa, 36% of OCD patients recovered within 3 years and 64% recovered within 5 years. The average period for recovery was 4.6 years, but some never recovered to the extent of returning to work.

2. Many workers develop pain which is severe, widespread and unresponsive to treatment, whether pharmacological, psychological, physical or surgical. These patients may develop serious psychological disturbances. Their poor response to treatment may be explained by the hypothesis of this discussion paper, that the pain of 'RSI' is neurogenic. A recent study has confirmed the known paucity of effective treatment methods available for chronic nerve-damage (neuropathic) pain.

3. The prognosis for cervical nerve root syndromes due to occupation has not been determined. Twenty-nine of the 37 patients of Lishman and Russell, followed up for one year, were relieved of all symptoms. A good response to a period of enforced rest for the whole limb was noted in those patients where the 'painful neuropathy had spread to involve all levels of the brachial nerves.' These results cannot be extrapolated to those patients whose cervicobrachial pain syndrome appears to have developed due to work-related factors.

4. It is reasonable to postulate a stage of early reversible peripheral neural dysfunction related to occupation which may respond to rest of the limb with or without local corticosteroid injection therapy. Elvey noted that a cervical nerve root complex which had been the subject of an inflammatory response was likely to become oedematous and develop fibrous adhesions within its sheath. He hypothesised that this pathology may favourably respond to specific gentle
passive movement techniques which he described. Butler and Gifford emphasised that neural tissue dysfunction causing adverse mechanical tension could be an important contributor to a symptom complex in a limb or in the spine. They described principles and methods for mobilising components of the nervous system when treating symptoms and signs whose origins may derive from either biomechanical compromise (pathomechanical), or irritative (pathophysiological) conditions of the nervous system. These are concepts which will stimulate much-needed research into the area of treatment of 'RSI'.

5. The results of decompressive surgery for various peripheral nerve entrapment syndromes related to occupation appear to be unpredictable unless very careful criteria for diagnosis and operative intervention are followed. Failure of some patients to return to manual work of a repetitive nature, after adequate carpal tunnel decompression, has been documented. Poor results following carpal tunnel surgery have been attributed to failure to recognise double crush syndrome, thoracic outlet syndrome, cervical radiculopathy and diabetes mellitus.

6. Based on current knowledge and understanding of 'RSI', emphasis must be placed upon its prevention by identifying all possible risk factors which relate both to occupation and to the potentially affected worker. The increasingly sedentary nature of employment occurring throughout many industries exposes an increasing number of workers to the risk of developing 'RSI'.

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ADDENDUM TO ATTACHMENT I

ADDENDUM BY ROBERT ELVEY

Physiotherapists consider that it is necessary, in the physical examination of patients with neck/shoulder/arm pain syndromes such as 'RSI', to define the anatomical structure(s) which specifically relate to the source of symptoms. Only when this aim is achieved can a more balanced appreciation of the condition(s) presenting for treatment be obtained and a resolution of symptoms be attempted.

In many instances the physical examination needs to explore the possibilities of local pathology causing local symptoms or more distant pathology causing referral and/or projection of symptoms. To be complete, the examination must therefore include the neuro-musculo-skeletal system of the entire upper quarter and not be confined to tissues or structures beneath the area of pain.

In such an examination it is necessary to provoke or reproduce symptoms by selectively stressing the many tissues which may be symptomatic, either individually or in combination.

All tissues must be examined for 'normal' function in terms of their movement, length, extensibility, and strength, as well as their ability to withstand stress or tension and to conform to and comply with positional and postural changes.

For specific details of the physical examination of various structures the reader is referred to relevant texts.1-4 In this addendum, some important aspects of the examination of the upper limb are discussed.

Inspection

The entire upper quarter is observed and compared with the opposite side for any asymmetry, including general or local muscle atrophy, and for skin changes, swelling or effusion, colour changes and perspiration.

A general appreciation of active physiological movement is gained of the joints of all areas—cervical/thoracic spine, shoulder girdle, shoulder, elbow, wrist and digits.

Examination of Joints

All joints are individually and precisely examined for range of passive movement, end feel and feel throughout range in all physiological directions of movement. Accessory movement or joint play is examined in all possible anatomical directions. As an example, metacarpophalangeal and interphalangeal joints are examined for joint play in the sagittal and coronal plane, whereas rotary joint play is examined in the axial plane, as is the joint play in the form of distraction of the articular surfaces. In the wrist, joint play (gliding) occurs between the articular surfaces of the carpal bones and between the proximal and distal carpal joints. Examination of the wrist is then performed in the sagittal and coronal planes so that wrist rotation and distraction can be observed.

Articular ligaments and joint capsules are examined by stress provocation tests. Distraction of joint surfaces and over-pressure in directions of physiological movement will tension both these structures. Provocation strain in non-physiological movement directions will stress articular ligaments. The various responses to these tests, together with any response to palpation of articular ligaments is noted.

At times, articular ligaments need to be stressed in various positions of a joint. For example, the collateral ligaments of the elbow should be tested with adduction and abduction provocation strain in various degrees of elbow flexion, commencing at full extension through to full flexion. This can be done at the same time as joint play in the coronal plane is assessed. If, for example, adduction strain to the elbow results in an adverse response, further assessment with palpation is necessary. Care should be taken in making a differential assessment between involvement of the tissues of the radio-ulnar joint and the radio-humeral joint.

Examination of muscles, tendons and entheses

Muscles, tendons and musculotendinous attachments are tested by isometric contraction up to maximum provocation in mid-joint range; but if required, testing should be carried out with the joint in variable through-range positions.

The muscle/tendon complex must be tested for length and response at maximal extensibility; through-range length and response must also be noted.

Palpation (of muscle, tendon and enthesis) is used to further assess any adverse response.

Particular attention must be paid to the medial and lateral epicondyles of the elbow with examination by palpation of the related enthesis; the greater tuberosity of the humerus is examined in similar fashion. A combination of palpation and the addition of specific
muscle/tendon provocation may be useful, e.g. to differentiate between extensor carpi radialis longus and extensor carpi radialis brevis.

Any adverse extensibility or length of muscle/tendon complexes should be carefully assessed in order to make a careful distinction between an articulation being responsible as opposed to an actual loss of extensibility or length. Where a muscle/tendon complex spans multiple articulations, care must be exercised to ensure that all articulations are included in the examination so that the complex is tested fully through its maximum length.

**Neural tissues**

If full consideration is to be given to the possible role played by neural tissue as the pathoanatomical source of symptoms in 'RSI' (as outlined in the main text), neural tension tests should be part of the physical examination. This is necessary as primary cervical neural tissue pathology can result in widespread distal symptoms that may mimic local conditions. At the same time, primary peripheral neural conditions may result in proximal symptoms which may mimic a more proximally situated condition.

It is important that certain points related to nerve tension testing are clearly understood. They are but one aspect of a comprehensive physical examination. In the particular patient, the information obtained from these tests needs to be interpreted in the light of all the available clinical information.

Although commonly referred to as 'brachial plexus tension techniques for signs of adverse neural tension' or 'upper limb tension tests', the concept of testing includes an assessment of peripheral nerve trunks, cervical radicular nerves and nerve roots as well as the brachial plexus.

Depending upon the severity and sensitivity of a condition, it may become necessary to place neural tissue at maximum extensibility to provoke a symptomatic (clinically relevant) response. For example, in some patients with carpal tunnel syndrome, the whole extent of median neural tissue, from the hand, wrist and arm to the involvement of that nerve in the brachial plexus and cervical radicular tissue, needs to be placed under conditions of maximum extensibility in order to elicit a response that can be assessed in terms of the patient's presenting symptoms.

At times there appears to be insufficient stress placed on the median nerve when using Phalen's test of wrist flexion or using wrist extension alone. The response can differ to a surprising (and meaningful) extent when combinations of wrist extension, elbow extension, shoulder abduction/external rotation, shoulder girdle depression and cervical spine contralateral lateral flexion are used.

The same principle applies to the examination of other peripheral nerve trunks. The posterior interosseous nerve which may be involved in forearm symptoms mimicking lateral epicondylitis, and the ulnar nerve at the elbow, mimicking medial epicondylitis. The testing technique needs to be based on the particular anatomical relationships of these nerve trunks so that they too can be tested under conditions of maximum extensibility.

Other important aspects of neural tension testing include: consistent and and predictable reproduction of the signs; consistency with other findings during the physical examination; the ability of the examiner to analyse responses to the systematic imparting of tension to neural tissue without depending solely on the patient response; the ability of the examiner to make a judgement of neural tissue tension being adverse in comparison to 'normal responses'.

It is also important to make a careful examination of shoulder mobility, particularly in abduction/elevation. Conditions such as 'RSI' may result in some restriction of abduction/elevation in the coronal plane. When signs of adverse neural tension are a factor in the condition under examination, this restriction will be much greater when this movement is carried out with the shoulder girdle held in a degree of depression and the cervical spine is held in contralateral lateral flexion by the examiner.

Overall consideration should also be given to the fact that when neural tissue is sensitive to tension, due either to intraneural or extraneural pathology, it will result in protective (reflex) muscular contraction. It is this protective muscle activity which the therapist must be aware of in order to fully assess conditions such as 'RSI'. In the basic test technique for signs of adverse brachial plexus tension, the action of extending the elbow imparts tension to neural tissue, which, if irritable, invokes a nociceptor-activated reflex contraction of the elbow flexors. The experienced therapist using correct technique is able to detect this contraction and attribute it (correctly) to a protective response; it is distinguishable from the end-feel of physiologically lengthened muscle tissue.
REFERENCES


ATTACHMENT II

Taken from

Papers and Poster Abstracts
International Federation of Orthopaedic Manipulative Therapists
I.F.O.M.T. Congress 1988
Lady Mitchell Hall, University of Cambridge, 4 - 9 September 1988
The clinical relevance of signs of adverse brachial plexus tension

ROBERT L ELVEY

INTRODUCTION

The prevalence of upper quarter symptoms in conditions of the musculoskeletal system is considerable. In discussing one pain flacidity (1991) quotes the work of Lawrence (1986) when he states that: "...at any time during the month there is a considerable number of the adult population in the United Kingdom who are experiencing some discomfort in the neck or with or without extended arm pain, 25% of us can report an 'episode'. He also quoted the "Musculoskeletal Investigation" in which Elvey (1964) "...selected a group of volunteers whose symptoms were considered to be significant and in whose case the movement of the neck had been fixed to a greater extent. This estimate was modified to 51% in a further series that were expanded to include 1550 male workers".

Aspects of the prevalence of similar symptoms in the community are not available, it is well known in the clinical setting that patients with neck/shoulder symptoms or upper quarter symptoms constitute a major part of clinical manual therapy and sports practice. A calculation of those cases referred to the author in the first half of 1991 indicated a prevalence of 51% of the overall patient work load. The majority of these cases were straightforward, the diagnosis of which considerable attention had been given to a detailed examination in order to make a manual therapy diagnosis even when radiological evidence indicated cervical or shoulder degenerative disease. In other words, certainly in the author's experience, in conducting the clinical manual therapy practice there is a high prevalence of people within the community who suffer neck or arm symptoms. A diagnosis is most frequently made in the following conditions: the prevalence of the symptoms resulting from musculoskeletal pathology can still be assessed as possible in differential diagnostic techniques of physical testing.

In addition to the potential difficulty facing the manual therapist in differentiating between multiple causes, manual therapy examination will frequently reveal adverse signs at a number of levels of the nervous system. For example, in a patient with tension headache the neck is an area around which considerable attention has been given to a detailed examination in order to make a diagnosis of tension headache even when radiological evidence indicated cervical or shoulder degenerative disease. In other words, certainly in the author's experience, in conducting the clinical manual therapy practice there is a high prevalence of people within the community who suffer neck or arm symptoms. A diagnosis is most frequently made in the following conditions:

- The potential difficulty facing the manual therapist in differentiating between multiple causes, manual therapy examination will frequently reveal adverse signs at a number of levels of the nervous system. For example, in a patient with tension headache the neck is an area around which considerable attention has been given to a detailed examination in order to make a diagnosis of tension headache even when radiological evidence indicated cervical or shoulder degenerative disease.

- In addition to the potential difficulty facing the manual therapist in differentiating between multiple causes, manual therapy examination will frequently reveal adverse signs at a number of levels of the nervous system. For example, in a patient with tension headache the neck is an area around which considerable attention has been given to a detailed examination in order to make a diagnosis of tension headache even when radiological evidence indicated cervical or shoulder degenerative disease.

- In the cases conditions presenting upper quarter pain may well be exacerbated by dysfunction of the cervical motion segment, muscles and tendons to localised tenderness or trigger points both locally and proximately.

- In the cases it becomes obvious that successful manual therapy examination a treatment and management is going to depend upon detailed pain relief and skillful differential diagnosis as the anatomical site of the pathological condition or the anatomical site of the main contributing condition can be determined.

- In this and the examination must include all tissues which may cause referred upper quarter signs and must not be confined to any narrowness which may lead to the obvious point of view or philosophy on clinical neurological conditions.

- This paper is presented in order to draw attention to the possibility that the condition of the upper quarter any physiological dysfunction which may co-exist may result from abnormal neck movement and so the condition of the upper quarter is often accompanied by both clinical and neural signs and symptoms. Cervical neural tissue may be as implicated in the condition as the myofascial condition of the upper quarter or symptoms causing the symptoms or myofascial tendons to the symptoms.

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in the abducted position. There was a relative increase of 3.4 centimetres in length from the arm by the side position to the abducted position. This square to occur due to the geometry of the humeral head. As a result of this relative increase in distance the neurovascular tissue as it traverses across and infers to the glenohumeral joint has to accommodate to this abduction position. If the shoulder girdle elevates with the abduction movement the neurovascular tissue loses its relaxed undisturbed appearance, however if the shoulder girdle is fixed in order to prevent it elevating then the neurovascular tissue moves relative to the abduction movement and tension in it occurs. This tension is transferred to the nerve roots.

If the arm is flexed abduction at the glenohumeral joint with the shoulder girdle also fixed and the elbow is extended the neurovascular bundle behaves in the same manner as it does when the arm is abducted at the glenohumeral joint when the shoulder girdle is fixed. That is, tension is imparted to the related nervous nerve roots.

If the wrist is extended along with extension of the elbow even greater tension is imparted to the neurovascular bundle and the cervical nerve roots. The mechanics of this tension are a result of the peripheral nerve trunk dynamics in the arm. Wrist and elbow extension as can be observed circular tension in the median nerve. This tension is then transferred along the length of the median nerve to the brachial plexus. Similarly, the other peripheral nerve trunks will cause similar tensions. However it should be noted that different physiological positions of the glenohumeral joint and upper trapezius need to be considered when implicating the radial or ulnar nerves.

It becomes plainly evident on observing the biomechanics of neural tissue that it takes part in the dynamics of the musculoskeletal system and that it is a relative mobile tissue to all fascial and anatomical terminals in neural tension. In other words tension may be nerve root or peripheral nerve trunk has to simply with and contribute to, supraspinal function.

This fact makes it possible to use normal orthopaedic tests to stress the neural tissue of the brachial plexus in such a way whereby it can be objectively assessed in order to examine a condition affecting the upper quarter. This assessment involves differentiating the neural tissue from the articular tissue and the muscle tissue as it can be selectively examined within the musculoskeletal system of the upper quarter.

TEST CONCEPTS:

As a result of the cadaveric observations of the biomechanics of the brachial plexus and by careful assessment of patients presenting with peripheral neuropathies associated with cervical spinal pathology demonstrated previous studies have carried out which place tension on the cervical spinal nerve roots from C5 to T1 as a means of clinical assessment of the upper quarter. This clinical assessment can thus be likened to the S.R.I. or slump tests used in the assessment of low back and leg conditions.

Because of the multiple variations of positions the upper quarter can be placed to, tension can be varied and there can be variations applied to each test procedure. This allows for careful differentiation between neural tissue, articular tissue and muscle as having a role in the condition or if there may be a combination of structures involved.

In other words there isn't a standard brachial plexus tension test for the upper quarter but a concept existing which varies many individuals. This is important to realise at the outset for it answers the questions concerning what tissues are being stressed and it can separate in a differential manner anatomical structures.

For example, an abduction of the arm with the shoulder girdle fixed, stress will be imparted to the cervical nerve roots and the subclavian artery and vein. If this position is sustained and the cervical spine is laterally flexed to the affected side further stress is imparted to the cervical nerve roots but not to the subclavian artery or vein. The response of this manoeuvre can then be assessed in order to differentiate neural tissue from vascular tissue. However, the lateral flexion component causes tension within the subclavian muscle groups, in particular, and other muscle groups of the shoulder girdle. Further differentiation between muscles and neural tissue can thus be made by extending and abducting the arm, by flexing or extending the wrist or by varying the amount of abduction of the arm. These manoeuvres change the tension in the nerve roots but not in the muscles and hence the response can once again be assessed and a judgement made. These are just two simple examples of differential tests. The clinician with anatomical knowledge can work the patient through a variety of assessing tests such as these examples. See figures 1A and 1B showing movement of neural tissue at the thoracic inlet region with elbow extension but also showing that movement of the subclavian artery does not occur.

Kennedy (1966) demonstrated in 100 asymptomatic individuals that in this position the elbow can be fully extended. Kennedy's study has therefore given a basis for the extent of the neural tissue associated with the brachial plexus and upper limb.

Herein, in the objective assessment during the test technique when the elbow is extended in a normal individual the therapist should be able to move the elbow into extension freely with a 'hard end feel' and without a feeling of an attempt to elevate the shoulder girdle. In the asymptomatic individual the therapist may well feel an increasing 'elastic type feel' in the extension movement and at some point a terminal end range of available motion a sensation of attempted elevation of the shoulder girdle and an attempt of the movement well as the range of elbow extension which are the important aspects in an objective assessment. For this purpose it is essential for the therapist to have the theme lightly applied to the patients shoulder girdle and wrist/hand region as shown. In this manner as tension is applied to the neural tissue the therapist can 'sense' with the hands' any involuntary raising of the shoulder girdle and further of the shoulder's elevation in the test such a withdrawal reflex and similar to pelvic rotation and knee flexion during the examination of S.R.I. in a static condition.
In further assessing a condition and depending upon the severity, acuteness and irritability of the condition, wrist extension can be added. This component may be used as a ready differential indication as to whether or not the gleno-humeral joint is involved in a condition, or whether or not shoulder girdle muscle imbalance may be related to the condition. Many other examples can be given to differentiate conditions by varying the components of the procedure.

A similar affect to the basic test techniques above on the neural tissues of the upper limb can be gained by the position shown in Figure 2B. Here the peripheral nerve trunks are placed under tension due to the shoulder girdle fixation with the arm in abduction and external rotation of the gleno-humeral joint. As a result of tension being imparted to the brachial plexus peripherally from the peripheral trunks the central nerve roots can then be placed in tension by lateral flexion of the cervical spine to the contralateral side.

As before, the therapist's grip with both hands must be light and must enable a careful objective assessment of 'feel' through the movement of the contralateral side. This involves the same previous manoeuvre, i.e. shoulder girdle depression and lateral flexion of the cervical spine to the contralateral side. This position may be likened to a slump position when maximum tension is required through the cervical nerve and neural complex. In this position as can be done in other positions the different components of the test can be varied to gain differential information as may have been done using the slump manoeuvre, where different components of the slump position are changed to gain varying responses.

The position of this basic technique can also be varied in many ways so as further objective and subjective information can be gained in order to make differential judgments and a differential diagnosis. By flexing the elbow as shown in Figure 2B the peripheral nerve tension is released somewhat but not the tension which may be imparted to shoulder girdle muscles such as the trapezius group. Hence the response whether changed or unchanged gives more information. Further information may be gained by varying other components.

In all test techniques it is important to compare left side to right side, to compare against what has been established for 'normal neural tissue tension response' for the upper limb (Kenneally, 1986) and to correlate the responses to all other findings of the complete examination.

The test techniques are not 'positive' or 'negative' but are complimentary to a complete examination and in this context they may be said to be positive for a particular diagnosis providing the rest of the examination process indicates a diagnosis supported by the neural tissue tension techniques. The techniques indicate the presence or not of adverse neural tissue tension. They do not indicate a pathology or the site of pathology which may be the except of the adverse tension. For example carpal tunnel syndrome or a radicular syndrome may have the same effect on the same neural tissue. Only by a complete examination can a diagnosis and determination of anadotal site of pathology causing adverse neural tension be made. This is of utmost importance to the clinician when using these techniques.
DISCUSSION

A number of post graduate studies on topics concerned with adverse brachial plexus tension tests have been completed and the conclusions of some of these studies and their examination.

Some of these studies and their conclusions include:

1) Kennedy (1986). The "upper limb tension test" - the T.L.T. of the arm. The study showed that brachial plexus tension tests were clinically effective and that there was a "normal" examination for signs of nerve tension from the cervical spine to the hand accompanied by "normal" responses.

2) Selverston (1991). The "disembodied validity of the brachial plexus tension test". The results indicated that the test was a discriminating valid test and had a moderate to high intra-examiner reliability.

3) Rucker (1987). "The upper limb tension test - the effect of the position and movement of the contralateral arm". The results of this study indicated that in young, right hand dominant, asymptomatic subjects, tension was transmitted across the cervical spinal canal.

4) Holt (1987). The "measurement of tension changes in the cervical nerves". This bubble flow transducer was used to measure longitudinal tension and the results revealed that tension is transmitted through the cervical canal.

5) Bell (1986). An "investigation of responses to the brachial plexus tension test with leg position". A change in the response pattern of pain and radiation, indicating that neural tissue tension is transmitted through the vertebral canal when using the test techniques.

One study (Selverston 1987) on the intertherapist reliability of the test techniques showed a poor result; however, it becomes obvious from the preceding discussion that in testing for adverse brachial plexus tension tests, the equipment and components to the tests are such that it is a quite a difficult and almost impossible task to position the upper arm correctly for the same response to the test. This stress can be dramatized with just the slightest change in position and it is possible that the criterion of a critical change in tension is a critical obstacle to overcome in an intertherapist reliability study, thereby making the test unrepeatable.

Sunderland (1979) has stated that..."the 5th, 6th and 7th cervical nerve roots are firstly targeted to the fingers of their respective transverse processes. This means that the technique is sensitive and will be effective in testing for neural tissue tension...".

Several techniques of treatment have been described for use in the treatment of conditions accompanied by signs of adverse brachial plexus tension tests (Elvey 1986). In addition to the treatment techniques for signs of adverse or abnormal neural tissue tension, which may be a sensitive indicator of a condition, the response and reproduction of symptoms which are being sought.

By considering the biomechanics of the neural tissues of the upper quarter and by considering the anatomical relationships of these tissues with various processes, it is possible to develop techniques for examining for the mobility of the glenohumeral joint and shoulder mobility.

As a result of the comments above a study of importance to be considered is the technique of cervical nerve root conditions, thus linking the tests to pathological examination. It is carrying out such a study in the form of a controlled trial to determine if tests for signs of adverse brachial plexus tension are able to clinically indicate patients suffering from brachial plexus neck arm symptoms where they have a clinical rubrospinalis confirmed by radiological examinations. No study has linked such a study with linking techniques to cervical nerve root pathology thus giving acceptance in routine orthopaedic examination.

Some of these studies and their examination have been completed and the conclusions of some of these studies and their examination. It would also be considered that the tests can be used in a differential screening manner not only with regard to nerve root, neural tissue, articular tissue or muscle tissue but also to peripheral nerve tension.

In addition to peripheral nerve entrapments it has been the experience of the author that the upper cervical region can be assessed in a differential manner using the techniques and of importance space following the results of the above study. The feasibility of such a study can be seen clinically and by using the techniques described in the region of the cervical spine. Some of these studies and their examination have been completed and the conclusions of some of these studies and their examination. This would appear quite feasible as for the glenohumeral joint is concerned (Elvey 1984). A sensitive cervical nerve root could possibly alter the mobility of the glenohumeral joint and shoulder as a whole thus mimicking a true glenohumeral condition. Tests such as those for the determination of adverse peripheral nerve tension are therefore required in the differential examination to make a clear distinction.
FIGURE 3A
The arm has been abducted at the glenohumeral joint. The pin in the median nerve has moved peripherally.

FIGURE 3B
The arm has been abducted at the glenohumeral joint. The pin in the median nerve has moved peripherally.

FIGURE 4A
Lumbar spine with ruler inserted: T - transverse processes, S - spinous processes, D - dura, N - C5 nerve root, L - lumen. Arm by the side.

FIGURE 4B
The elbow has been extended with the arm abducted at the glenohumeral joint with the shoulder girdle fixed in some depression. It can be seen that there has been a relative increase in distance between the pins related to the neural tissue and the fixed scalene muscles but the distance between the subclavian artery and the scalene muscles has remained unchanged.

FIGURE 5A
Left thoracic inlet view from above.

FIGURE 5B
Neurovascular bundle at the level of the glenohumeral joint with the arm in abduction and external rotation. Note the tension in the neural tissue (white pin M - median nerve) over the index finger and over the humeral head - H.

1. Lateral cord of brachial plexus. The humeral head indicates how the neural tissue is "bowed" over it in the abducted externally rotated position.
In assessing the relationship between limited glenohumeral joint mobility and possible cervical nerve root pathology the patient abducts the arm as shown and range and symptoms are assessed with the shoulder girdle slightly flexed so it doesn't elevate as fully as it normally would.

A differential assessment can be made by adding cervical lateral flexion to the contralateral side plus shoulder girdle depression. Both components can be varied for detailed assessment.

The cervical spine is laterally flexed to the contralateral side thus fixing the cervical nerve roots and imparting tension to the brachial plexus. The arm is again abducted and the therapist lightly flexes the shoulder girdle in the same position as Figure A. The range of abduction and the response is assessed. If there is nerve root pathology the range of abduction will be decreased.

Slight of adrenalin brachial plexus tension can be helpful in the determination of the tissue which may be most dominant in dysfunction of the upper quarter. For example in assessing cervical rotation many extra-articular tissues are involved as well as the articular tissues. These include related muscles and neural tissues. When the neural tissue involves the cervical roots of the brachial plexus a differential test can be carried out by assessing full cervical rotation followed by instruction as shown in figures 9B and 9C.

The patients returns the head to the neutral position and the therapist abducts the arm at the glenohumeral joint and supports the shoulder girdle. Once again the patient rotates the cervical spine. The range is reassessed and if the internal central tissues involved the tension already imparted to it due to the arm position will cause a reduction in the available range of cervical rotation.
We welcome the opportunity to comment on this paper. We are familiar with the Brachial Plexus Tension Test (BPTT) and regularly use it in both assessment and treatment. The exact pathophysiology of 'RSI' is not currently known and we have no doubt that neurogenic factors form a part of this syndrome. They may well explain the actual generation of pain reported but we feel that there are other tissues involved or intermediate to the process of 'neurogenic' pain formation.

Comments are provided under each major section heading with the relevant paragraphs indicated by partial quotations.

I. INTRODUCTION

There appears to be an inconsistency between the quotation in paragraph 2 'we were unable to detect recognisable upper limb musculoskeletal pathology in these patients' and in Hypothesis paragraph 1, 'clinically identifiable musculoskeletal pathology causing upper limb pain may co-exist with RSI, but is usually insufficient to explain the full clinical picture.'

Clinically our own physical examination findings include palpable changes in muscle tissue consistency, including locally tender nodules, reduced muscle extensibility and reproduction of pain on passive stretch and strong active contraction. None of these signs are referred to in 'Physical Examination Findings', although in the Addendum (Attachment 1) 'Examination of Muscles, Tendons and Entheses', Elvey specifically includes palpation of muscles but does not refer to any findings.

There is a concern that readers of this paper, with experience in the palpable changes in muscle tissue regularly observed, would question the validity of a purely neurogenic concept.

It is felt that Quintner/Elvey need to acknowledge their physical examination findings related to muscle tissue alluded to in 'Introduction to Hypothesis' and attempt to discuss them in relation to the neurogenic hypothesis they propose before dismissing them.

It is felt that information contained within Attachment 1, Addendum, should form part of the main body of the paper as the clinical features found at examination need to be discussed in greater detail in order to arrive at the decision as to which particular tissue may be the causation of the RSI symptoms.

II. CLINICAL FEATURES

Under the heading 'Common Symptoms - (a) Pain', we disagree with the pain descriptions provided. Clinically, descriptions given are more closely aligned with those provided in 'IV. Evidence in support of ...', para 3 'Neuropathic Pain' as referring to non-root pain of musculoskeletal origin, i.e. dull, aching, deep etc. In addition the other commonly reported symptoms mentioned by Quintner and Elvey are also found in the same paragraph and again referring to pain of musculoskeletal origin.
IV. EVIDENCE IN SUPPORT OF THE 'NEUROGENIC' HYPOTHESIS

In the section on case studies, the assumption is made that the 'simple muscle strain' which did not resolve in a week or two, despite removal from the source of strain, must therefore indicate a neural origin for the symptoms. Many authors regularly refer to the fact that real rest is seldom achievable by the predominately female patient grouping affected by this condition. The leap from non resolving muscle strain to a neurogenic proposal is tenuous.

Quintner and Elvey quote case studies in which at least 4 authors (Taylor et al, Stone, Browne et al, Fry) argue for a somatic origin of RSI symptoms. Quintner and Elvey attempt to recruit those references in support of a neurogenic basis, without adequate argument.

In the paragraph regarding Quintner and Elvey's work (commencing 'Our own work has also concluded ...') it is suggested that the BPT provoked the full range of RSI symptoms. This has not been our clinical experience. The symptoms commonly reproduced are pain in the cubital fossa and distal hand, and the clinical sign of restricted elbow, wrist or finger extension. In early stage RSI the BPT is frequently negative. It is suggested that this paragraph be clarified to identify the exact symptoms reproduced by the test and postulated as being RSI related. In those RSI patients with positive brachial plexus tension signs, we have been able to eliminate those signs using a specific muscle stretch technique. This relieves only a portion of their overall RSI symptoms.

In the reference to Miller and Topliss, Smythe is reported as challenging the findings of no evidence of physical injury, stating that 'examination of the cervical region ... would have revealed unsuspected tenderness related to the anterior aspect of the lower transverse processes'. These regions have extensive muscle attachments and we would query the certainty with which anatomical structures can be differentiated as being either nerve or muscle. Smythe apparently does not state that the structures are definitely neural.

In Sikorski et al we would again comment that the specific symptoms compatible with Quintner and Elvey's hypothesis be identified.

The final assertion in this section that evidence presented by the quoted authors of the Australian studies is compatible with the neurogenic hypothesis is, we believe, insufficiently supported.

Under human experimental studies, in the case reported by Levy and Wilder it is stated that extreme forward flexion was forcibly maintained. We do not feel that this reference is relevant to the normal seated working posture. Our experience with ergonomic assessments indicates that cervical positioning involves primarily upper cervical flexion combined with a neutral or slightly extended position of the lower cervical region. Thus the levels of tension postulated are felt not to occur in the more common working environments.

Within the section Occupational Health Field Studies, in the Hunting et al study, Quintner and Elvey comment that no attempt was made to explain the left hand-arm symptoms. They postulate that the neck positioning necessary for the task could generate tension in right sided lower cervical neural tissues. This also does not explain the left hand symptoms.

The conclusion reached in this section is reasonable but does not actively support the neurogenic hypothesis.

In the section 'Biochemical Considerations Relevant to Neural Tissues', the reference to O'Connell involves a disease process (meningitis) and would not appear to be relevant to a work-induced injury.

With regard to the references in this section, particularly Breig and to the Summary, we would restate our previous comment under Human Experimental Studies that sustained hyperflexion is not a common seated work posture.

Under 'Experimental Studies of Damaged Spinal Nerve Root ...' in the reference Frykholm, detailed examination of this paragraph provides strong support for the production of muscle-specific pain possibly arising from the brachial plexus. The brachial plexus is usually formed by the
union of the ventral primary rami of nerves C5-C8 and T1. This point might usefully be expanded and highlighted further by the authors in support of the neurogenic concept.

V. DIFFERENTIAL DIAGNOSIS OF THE PAIN SYNDROME RSI

We would have to strongly disagree with the statement at the beginning of this section, that diagnostic features of musculo-skeletal conditions are uncommon in RSI patients. As stated earlier in our comments, we find clinical signs including palpable changes in muscle tissue, locally tender nodules, reduced muscle extensibility and reproduction of pain on passive stretch and strong active contraction. As previously noted in this paper, Taylor et al, Stone, Browne et al, and Fry argue for a somatic origin of RSI symptoms.

Quintner and Elvey appear to be differentially diagnosing cervical radiculopathy, thoracic outlet syndrome, and upper limb entrapment neuropathy as being distinct from RSI, thus giving RSI the status of a distinct condition in its own right. Most authors agree ‘RSI’ is an umbrella term and it is our contention that elements of the RSI syndrome can be attributable to a number of conditions, including those previously stated under Differential Diagnosis.

Referring again to the lack of recognition of musculo-skeletal involvement, the question arises in ‘3 Upper Limb Entrapment Neuropathies ....’ as to what is the mechanism that converts a previously asymptomatic muscle into an entrapping symptom-producing tissue.

CONCLUDING COMMENT

Elements of this paper are noted to have been reproduced in an article by Quintner (Aust. Journal of Physiotherapy, Vol. 36, no. 2, 1990) under the title ‘Stretch-Induced Cervicobrachial Pain Syndrome’. It is felt that Quintner and Elvey’s paper is relevant to a stretch-induced pathology. However, clinically RSI is not reported to involve such causation. Patients may have a neural component to their pain, but is the neural tissue the source of the pain?

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The syndrome referred to in the title “RSI” does not have a name which has achieved wide acceptance. For simplicity I shall refer to it as neck and arm pain arising in the workplace. The authors have presented circumstantial and logical support for a neurogenic theory to account for the symptoms, natural history and clinical observations of this group of disorders. The essence of their theory is that the chronic and diffuse neck and arm pain syndrome is an example of “pathophysiological” pain, in which the pain occurs spontaneously, or in response to weak stimuli, due to abnormalities of neural excitability. Thus it is proposed to be a disorder of membrane excitability regulation. The anatomical origin is suggested to be the neural tissues in the cervical region or more distal peripheral nerves.

The theory has considerable explanatory power. It can be applied to explain the evolution of the pain spread beyond the territory supplied by single nerves or single dermatomes, paraesthesiae, the dysaesthesiae, the sensory impairment, the hyperalgesia phenomena, the increased mechanosensitivity of peripheral nerves in the upper limb, the sympathetic and motor dysfunction, and finally the chronicity. The theory is consistent with current knowledge of the neurophysiology of peripheral nerve disorders including changes in central nervous system processing in neuropathic pain syndromes. Importantly the theory is testable, although the authors have not outlined the methods which could reasonably be applied. This has been a difficult issue.

The neurogenic theory explains a very important perplexing aspect of this group of chronic pain disorders, namely the widespread hyperalgesia with no obvious underlying pathology. Hyperalgesia refers in this context to the provocation of a painful response by a stimulus which is normally subthreshold, and excessively painful responses to increasingly painful...
stimuli. The hyperalgesia observed in neck and arm pain arising in the workplace is frequently interpreted as psychogenic, however it has the characteristics of secondary hyperalgesia and is the result of abnormal pain processing within the nervous system, a pathophysiological response. These characteristics include extensive spread; differing responses to various mechanical stimuli; persistence of pain, dysesthesiae or tingle; and a summation effect with increased response to repeated stimuli. Such secondary hyperalgesia is a consistent feature of neuropathic pain syndromes.

There are some difficulties with the proposed theory. The first problem, a really difficult one, is that there are no diagnostic criteria for neck and arm pain in the context of repetitive use ("RSI") which have achieved wide acceptance, certainly none empirically established and validated. This leads to a second fundamental problem, the establishment of reliability and validity of the brachial plexus tension test of Elvey. The sensitivity and specificity of this test for "RSI" cannot be established at present to the satisfaction of critical reviewers. It is not of course a test that one would anticipate to be specific for the neck and arm pain disorder arising from repetitive work. It should be tested for sensitivity, specificity and predictive value as a test of abnormal mechanosensitivity of the relevant neural structures. Nevertheless if it is interpreted correctly it can be a useful procedure in the same way that the straight leg raising/sciatic nerve stretch tests can be in the interpretation of neuropathic pain projected to the lower limbs.

There has been very little empirical research into the pathogenesis of diffuse neck and arm pain in the occupational context. Arroyo, Cohen and Champion (1991) have presented clinical psychophysical studies with an electrical testing method which confirmed reliable reporting by the patients and which support the interpretation that these conditions are hyperalgesic states and the hyperalgesia has the characteristics of secondary hyperalgesia. To date the studies have not determined whether this secondary hyperalgesic state is the result of primary neuropathic abnormality as proposed by Quintner and Elvey, the result of somatic pain inputs from the cervical spine or other musculoskeletal structures, or to what extent abnormal nociceptive processing within the central nervous system is relevant. Our data do not in any way refute the peripheral neural hyperexcitability theory which we also have discussed. Recent testing has provided initial support for polymodal C nociceptor fibre dysfunction in the affected limbs.

The paper by Quintner and Elvey is a well researched and presented account of the neurogenic theory and provides a stimulus for its testing. In presenting a theory, it is always important to indicate any weakness and points of refutation. Unfortunately the authors have not addressed those points.

REFERENCE
PART A. OVERVIEW

The authors have performed a useful service with a careful, detailed argument that the pain of ‘RSI’ is neuropathic. Their hypothesis, as stated succinctly in the conclusion is: ‘The accepted pathogenic mechanisms of entrapment neuropathies may also explain the development of the ‘RSI’ pain syndrome.’

This formulation has resulted from two sets of clinical observations in patients with the problem of diffuse upper limb pain:

(i) features of altered sensation (as well as pain)

(ii) results of a manoeuvre considered specifically to identify the brachial plexus as an anatomical site of symptoms.

These observations have led to two corresponding inferences:

(a) the pain is neuropathic

(b) entrapment (at the level of the brachial plexus, more proximally in the cervical spine, and possibly also distally) is the relevant pathophysiology.

(i) and (a) above have been carefully documented: Quintner and Elvey’s argument of clinical analogy with known neuropathological states is important, being the basis of the heuristic exercise undertaken by all those supporting neuropathic bases for the syndrome, including this commentator. However the evidence that entrapment neuropathy accounts for these patients cannot be admitted on epistemological grounds for the following reasons:

(1) The authors have failed to define which clinical syndrome they are addressing and frequently offer either known pathological entities (eg. shoulder capsulitis) or other heuristic constructs (eg. reflex sympathetic dystrophy, thoracic outlet syndrome) as clinical features or, later, even as differential diagnoses. Thus they have fallen into a tautological trap in which it is almost impossible to ‘diagnose’ ‘non-RSI’. I am also disturbed as to why they have limited the illness under consideration to an ‘occupational’ context only, there being no difference between arm pain acquired within or without the context of work.

(2) Although well-documented, the authors have not seized the clues to pathogenesis offered by the positive sensory phenomena elicitable in this clinical situation, in particular allodynia, hyperalgesia and hyperpathia, but also the abnormal vasomotor phenomena which they have interpreted narrowly. As such they have not been able to address the possibility that central nociceptive dysfunction may also contribute to these phenomena. Thus the whole issue of CNS plasticity has not been canvassed.

(3) The authors’ stated bias is towards entrapment of peripheral neural tissue as the primary ‘cause’. Notwithstanding the above comments, their explication does not satisfactorily account for the positive sensory phenomena, partly because very little attention has been paid to them in the neurological literature. It is a testable hypothesis that proximal peripheral neural tissue is the anatomical origin of symptoms: the proposal should have explained how that may lead to the observed phenomena and could have contained a strategy for testing the consequences of their proposals.

PART B. SPECIFIC COMMENTS, organised according to the major divisions of the paper.

II. CLINICAL FEATURES

1. Common Symptoms

(a) Almost by definition (see above) the pain should be diffuse in the arm, rather than following the course of major peripheral nerves. Pain restricted to such discrete anatomical distribution is more likely to be attributable to a definable structural lesion. Pain in the apparent distribution of more than one peripheral nerve is unlikely to be so caused and implies mechanisms other than ‘polyneuropathy’.

(g) Painful limitation of movement is not confined to the cervical spine and shoulder.
(h) 'Frank psychiatric illness' is too vague a term to be admitted into discussion of an ill-defined entity. At least accepted positive criteria for psychiatric illnesses do exist. Changes in affect are indeed common, in my experience almost all reactive.

2. Physical examination...

(a) I find the tenderness (or, more correctly, the hyperalgesia) to be diffusely elicitable and not readily localisable to neural tissue. The formulation presented harks back to their idea of pain following the courses of nerves.

(c) Shoulder ‘capsulitis’ is not admissible, being a confident diagnosis in itself. Painful limited shoulder movement is preferable; similar phenomena may be found distally.

(e) Hypoalgesia is foreign to my experience and indeed incompatible with hyperalgesia. These features are grouped to imply ‘altered peripheral neural sensibility’, thus begging the hypothesis; whether these features are peripheral or central is the fundamental question. Indeed these features of positive sensory alteration - hyperaesthesia to percussion and blunt pin, allodynia and hyperpathia - coexisting with hypoaesthesia to sharp pin and cotton wool are in my view the most important clues to pathophysiology. The authors should offer definitions of the terms used.

(f) The vasomotor phenomena cited do not convey the instability and frequent changes of affected limbs from cool/blue to hot/pink and back which may be noted, especially in response to usage or examination. ‘Reflex sympathetic dystrophy’ is itself a syndrome and should not be offered as a clinical finding.

The ‘hypothesis’ as formulated is clumsy. My reading is that the authors propose:
- it is a neuropathic syndrome
- discernable pathology exists in peripheral neural tissues of the upper limb
- the pathogenesis is entrapment/friction/ischaemia

I agree that the syndrome is neuropathic. The authors argue that it is a subtle complex entrapment polyneuropathy.

IV. EVIDENCE IN SUPPORT...

1. Neuropathic pain

This discussion is based on Grieve who appears to concentrate on ‘musculoskeletal diseases’ from which I infer tissue damage, a phenomenon not proven in ‘RSI’. Secondly ‘referred pain of root involvement’ is presented as a discrete category. I will accept that pain may be referred from peripheral nerves anywhere along their course, following Lindblom, but I contend that ‘referred pain of root involvement’ is impossible, not difficult, to diagnose. Confusion with the syndrome of reflex sympathetic dystrophy again is allowed to occur.

2. Case studies

This section reiterates

(i) description of a poorly-defined syndrome
(ii) assumptions regarding anatomical origin of symptoms based on ‘neurological’ phenomena
(iii) teleological assumptions regarding pathogenesis and illustrates the fundamental epistemological problem of definition.

5. Biomechanical considerations...

Evidence for potential neural entrapment is presented.

7. Experimental studies...

This section establishes only that there may be, in ‘RSI’ sufferers, neural distress.
8. Brachial plexus tension test
I continue to have great difficulty accepting the interpretations of the outcome of this manoeuvre - to the same extent of my difficulty with straight-leg-raising in the context of back/leg pain. The authors state, 'The BP2T appears to be highly sensitive in clinical situations where upper limb pain and paraesthesiae arise from dysfunctional neural tissues related to the symptomatic arm' (my italics). This illustrates that fallacy of reasoning in which the process which leads to a conclusion is itself held up as a justification of that conclusion. The sensitivity of a test is the probability that the test will be positive given the clinical disease or syndrome. If the BP2T is used to define the syndrome, it must be highly sensitive (in fact 100% so) but the argument is circular and does not confer validity. In this context the introduction of 'similar' phenomena elicited in other 'syndromes' such as 'thoracic outlet', 'cervical disc disease' and 'carpal tunnel' obfuscates the argument, by again failing to apply definitions.

V. DIFFERENTIAL DIAGNOSIS
This section is highly unsatisfactory, not only because Cyriax is accepted as the gold standard for 'musculoskeletal conditions' but also because it concentrates on entrapment neuropathies as 'alternative diagnoses' whilst I have been led so far to understand the authors themselves are proposing subtle entrapment.

Furthermore the distinction between occupational and non-occupational causes is artificial and implausible, whilst the discussion under 10) confuses somatic referred pain, radicular pain and pain referred from nerves themselves. Again tautologies abound.

VI. CONCLUSION and VII. IMPLICATIONS
The authors state: 'The accepted pathogenic mechanisms of entrapment neuropathies may also explain the development of the 'RSI' pain syndrome.' I feel that their choice of the entrapment neuropathy heuristic has been too restrictive and in fact incompatible with some of the 'implications' as listed, especially (5).

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This paper has the inherent attraction of suggesting that Repetitive Strain Injury (RSI) has a single neurogenic aetiology. William of Occam would have readily espoused such an idea though his followers, the nominalists, had little concern for whether their abstract concepts had a basis in reality. Herein lies the essence of the problem with this paper. RSI, or better, occupational over-use syndrome (OOS), has a myriad of presenting features from a wide variety of causes. However attractive a single aetiology might be, the merit of neatness is, unfortunately, outweighed by the complexity of reality.

Throughout their paper, Quintner & Elvey seem to base their hypothesis upon the patients they see—not an unreasonable primary stance—except that their patients bear little resemblance to those we see in the course of our clinical practice. Their patients would seem better described as 'end-stage' OOS. To illustrate the point: some time ago we both reviewed six individuals who were contemplating litigation against the poultry processing company for which they worked. These six cases epitomised the range of clinical presentations which may result from occupational exposures to repetitive movement jobs. At one end of the spectrum was an individual with symptoms restricted to abductor pollicis longus of the right hand who on examination exhibited tenderness and crepitus over the tendon sheath. The symptoms and signs resolved on removal from the workplace. At the other end of the spectrum was a person helped into the consulting room by solicitous relations. On examination she was effectively quadriplegic. Both patients had performed similar work but one had progressed to a state of invalidity. In both it was patently obvious that the exacerbating cause was excessive repetitive movements.
leading to tendonitis which, following prolonged pain and further discomfort had led in the extreme case, to postural modification, further pain and a progression of symptoms up to and including the shoulder girdle bilaterally. Clearly a case of end stage OOS.

Quintner & Elvey, however, propose that their patients commonly have tenderness over neural tissues and that the "majority" of patients present with a "diffuse pain syndrome of the upper limbs often accompanied by pain in the neck and upper back". This is not our experience. Perhaps it relates to the stage at which the clinician sees the patient but it also must require on the clinician's part the eliciting of a history of occupational exposure including a precise account of the work undertaken by the patient. When the latter is well characterised, and this should be supplemented by a visit to the workplace, then a mechanical aetiology is frequently the most plausible cause of the symptoms and signs. To postulate a neural hypothesis for all on the basis of only a proportion of the eligible patients makes no sense.

We have recently completed a research project on this subject (Thompson et al 1990) at eighteen workplaces and on subjects who are still at work. Clearly these study subjects are not incapacitated but they did show a surprising degree of pathology. Biomechanical assessment of the work including video recordings was supplemented by a structured physical examination to assess pain and mobility in a case control approach. Thoracic/neck joint dysfunction was noted in nearly half the study subjects despite the fact that most of the jobs involved repetitive movement of the upper limb distal to the elbow. Clearly postural factors as well as movement factors were involved in the resulting syndrome. The point we wish to make is that a careful and thorough review of workplace activities will often provide a logical explanation for the resulting clinical features and in addition, paves the way for preventive action by modification of the work practices.

Quintner & Elvey start at the wrong end of the clinical spectrum, take little note of actual workplace procedures and then proceed to postulate a neural hypothesis. Thereafter they endeavour, unsuccessfully in our view, to squeeze a whole range of real and perceived repetitive movement and postural disorders with a wide range of workplace exciting factors into a single causative hypothesis. They try, again rather unsuccessfully in our view, to rebut non-neural alternative hypotheses with a scant critique of the literature. By analogy it is frequently very difficult to diagnose specific aetiologies in renal disease if all one considers are patients in chronic renal failure with end stage kidneys.

The real problem with the neural hypothesis is that, as postulated, it appears to take us no further forward. It is not followed by any suggestions as how such an aetiology, if true, would propose a way forward to aid affected patients or prevent further cases from appearing. The hypothesis is thus sterile.

In short, the Quintner/Elvey paper has the attraction of postulating a single aetiological hypothesis for a potentially serious and widespread occupationally related disorder. It is not sustainable by careful clinical and epidemiological review of real patients who present with a wide range of symptom/sign complexes and in whom a workplace aetiology of a more mechanical nature can frequently be found if careful attention is paid to both the actual workplace activities and the natural history of the disorder. Such an approach, in our view, not only provides a more logical aetiology but also leads naturally to prospective studies and the opportunity to institute practical prevention - the ultimate test of an hypothesis and a basic tenet of good clinical epidemiological practice. Undoubtedly some OOS patients do have a neural component to their problems but the majority do not start that way whatever the final outcome may be.

REFERENCE

I have no major problems accepting Quintner and Elvey's clinical features and even physical examination of what they call "RSI". But when it comes to their hypothesis, I cannot hold with items 1. "irritable neural tissues" or 3. "those neural tissues are proximally situated".

I believe, from assessing all the patients referred to me, from all those physical examinations, special tests, and sifting through the voluminous and confused literature on the subject, that we are dealing mainly with cellular damage to overstressed muscle fibres and their associated tendons, sheaths, ligaments and neuromuscular junctions. There is accumulating evidence that "overuse" of unbalanced muscle groups leads to incoordinated single muscle fibre activity. The cycle of contraction, relaxation, rest period, regeneration (as cell metabolism both clears out waste products and regenerates new neurotransmitter substances) and contraction again of the single muscle fibre is disturbed. The muscle fibre may be called upon to enter another cycle before it has cleared out the waste products of the previous cycle, and then to have yet another and another cycle, and such activity will cause changes in the basic architecture of that cell's structure.

I believe that the apparent confusion in the physical signs seen by examining physicians occurs because they see the sufferers in different phases of this process of cell disorganisation. So I believe that the whole process starts at the cellular level, and as individual, and later groups of now damaged muscle cells fail to function normally the whole symptom complex begins.

But no-one can be dogmatic as to their own or anyone else's hypothesis until the evidence is. Quintner and Elvey argue along the line of neurogenic (proximal) pathophysiological pain. A detailed criticism of their paper which contains a great deal of research, is outside the scope of these comments, but I cannot agree with their hypothesis as it does not seem to begin at the right end. I repeatedly see patients where the symptoms begin with a peripheral pain in a muscle or muscle group that has been overstimulated over a prolonged period, and a syndrome commences which ends up in a complicated picture that resembles what Quintner and Elvey describe as a proximal neurogenic lesion, not the other way around.

Human tissues can only function physiologically within their evolutionary evolved limits. Overdoing those limits does lead to cellular damage. If the tissue is allowed to recover it usually can, providing the damage has not yet destroyed the cell function and capacity to recover. Continue the stress and overwork that cell and it will suffer, with consequential effects on cells in its vicinity, and so on. Eventually pain fibres are stimulated and so begins the history of another "RSI victim". Now prove it! Whatever the cause, we must be sympathetic to those with these problems.
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Repetition strain syndrome (RSI) is important because it causes a huge toll of suffering and is now the single most common cause of occupation-related absence from work. There are many barriers to the successful management of this condition (Table 1) and many discussions shed more heat than light. It is therefore essential to formulate concepts for management that are based on intellectually sound principles.

Table 1. Barriers to management of repetition injuries

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<td>1.</td>
<td>Poorly described in standard medical textbooks.</td>
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<td>2.</td>
<td>Interfaced between many specialities (neurology, orthopaedics, rheumatology etc).</td>
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<tr>
<td>3.</td>
<td>Unhappy patients who need special handling.</td>
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<td>4.</td>
<td>Doctors’ fear of involvement in provision of medicolegal reports.</td>
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<td>5.</td>
<td>Few physiotherapists trained to provide treatment (in UK).</td>
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<tr>
<td>6.</td>
<td>Time course prolonged.</td>
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<tr>
<td>7.</td>
<td>Time consuming - needs much liaison.</td>
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The authors advance the hypothesis that the clinical features of RSI arise from irritable neural tissues and that this irritation is the result of excessive tension or friction. The evidence that they advance supports the concept that mechanical pressure can cause a reduction in the pain threshold, that fixation of nerves occurs in some patients with RSI and that some patients with RSI have positive neural tension tests. Their experience that neural stretches are valuable as part of treatment is supported by my own observations in over 500 patients.1

Evidence against the hypothesis

My concern with the hypothesis as formulated is that it regards the neural irritability as prime in the pathogenesis of the condition. There is a considerable body of evidence that repetitive use of the limb that is excessive for that individual causes pain and tenderness in all tissues that have sensory innervation.23

An alternative explanation of the authors’ observations is that the initial mechanical damage is to muscular, ligamentous and other connective tissues and that the pain generated in these tissues causes a rigid carriage of the arm. This rigid carriage leads to spasm and shortening of the muscles and in turn to the formation of adhesions round the nerves.

Evidence of primary muscular, ligamentous and tendinous damage includes:

1. the clinical observation that in the early stages of the development of RSI, there is pain and tenderness in the muscles, ligaments and swelling of synovial sheaths independent of whether the neural stretch test later becomes positive. In addition, tenderness of muscles and their origins may persist after the neural stretch tests have become negative with treatment. These observations are in direct and important contrast to those of Quintner and Elvey who, in the second paragraph of the introduction to their paper state that they “were unable to detect recognisable upper limb musculoskeletal pathology in these patients”.

2. that there are well-established pathophysiological mechanisms associated with the initial events of exercise-induced muscular injury that do not invoke the involvement of the brachial plexus or any other parts of the nervous system.4 For example, it has been clearly shown that there is evidence of injury to skeletal muscle fibres with disruption of the normal myofilament structures in a proportion of the sarcomeres. In addition the release of
intramuscular proteins (e.g., creatinine kinase enzymes) into the plasma after unaccustomed exercise has been demonstrated. This release may coincide with the pain and swelling of muscles that is observed in the acute phase of some repetition injuries. The loss of intracellular calcium homeostasis could play a primary role in the propagation of exercise-induced muscular injury. In experimental models of muscle injury, elevated concentrations of calcium appear to cause release of muscle enzymes through activation of phospholipase A2 which in turn could promote formation of free oxygen radicals or detergent phospholipids and cause injury to the sarcolemma.

The role of dysethetic pain is misleadingly highlighted as causing RSI in the paper by Quintner and Elvey. Again, there is confusion here between cause and effect. It seems quite clear to me that what they describe is another complication of repetition injury namely sympathetically maintained pain (algodystrophy, reflex sympathetic dystrophy) that can follow any penetrating or non-penetrating injury to a limb.

**DIAGNOSTIC CRITERIA**

The description of the symptoms in the paper by Quintner and Elvey is broadly in agreement with my own experience. However, the list of physical signs is muddled. There is no specific mention of tenderness of muscles and their origins, of ligaments and of joint capsules which are the cardinal physical signs. Tenderness of neural tissues and provocative tests are positive in only a proportion of patients with RSI—usually in the later phases of established injury. When neural tension tests are positive on one side only, it is possible to measure a decrease in the neural arch-fingertip distance on the affected side. Altered neural sensibility can complicate RSI from either nerve tethering or reflex sympathetic dystrophy, but is not a primary feature of RSI. The most important physical sign of reflex sympathetic dystrophy that predicts response to sympathetic blockade is coldness of the affected limb. Distinction from Raynaud's phenomenon is by laser Doppler photoplethysmography and blood flow studies.

**TREATMENT METHODS**

Neural stretches are an important part of the treatment of RSI only if provocative tests are positive. Gentle mobilisation of the upper dorsal spine, frictions to the epicondyles and swollen connective tissues, laser and ultrasound are important modalities of treatment.

Electrotherapies should only be used to damp down reactions to treatment. Treatment may require prolonged attendance with the physiotherapist, often twice weekly for up to a year or more. Proven reflex sympathetic dystrophy should be treated with three intravenous guanethidine blocks before commencing physiotherapy.

**REFERENCES**


Essentially this paper is an attempt to explain a significant proportion of patients diagnosed as suffering from "RSI", as suffering from pathological change in cervical nerve roots/brachial plexus. There is a large bibliography, running to 130 papers and I consider that the arguments extracted from these scientific papers are highly selective and only in the narrowest of senses can these quotations be described as supportive of the authors' hypothesis. This is not in any way to suggest that the authors have misquoted other workers, but I have the distinct sense that small isolated items have been quoted, or picked out of various papers without taking heed of the general sense and conclusions of other accepted authorities.

Even the most casual reading of the literature on the Australian RSI epidemic will show that the symptoms claimed by patients cover an enormous range of modalities, although certainly pain is the predominant one and the general anatomical distribution is widespread throughout the upper limb, from the neck down to the finger tips. Numerous authors over the past decade have tried to provide a unified theory, or pathological process to explain this enormous multiplicity of symptoms in a very large patient population, although many people, including this reviewer, remain highly sceptical that there is any one pathological process present, and furthermore that in many situations, perhaps the majority, there is no physical pathological process present and that a considerable proportion of the patient's symptoms are of psychological origin, and even in the more extreme cases of conscious origin, that is based on malingering in relation to a continuing possible claim for compensation. I consider it naive of the present authors not to consider the very marked psychological factors in many patients who are complaining of pain without any overt physical abnormality, and studies such as Allen and Waddell's work on the low back pain problem, or Mendelson would illustrate this well.

Much play is made in this paper of Elvey's own clinical test, the brachial plexus tension test (BPTT), and an addendum to this paper describes the test in detail. I was not impressed by this test, but it must be appreciated that I am looking at the matter from the point of view of conventional medicine, and a formal background of training in orthopaedic surgery, and more specifically in brachial plexus surgery over the past 30 years. Mr Elvey is a manipulative therapist, and I am well aware that such therapists use a variety of tests, manipulations and the like which can be extremely effective in their patients, but are often not considered to have an established basis in conventional orthopaedic/scientific experimental or clinical practice. I have not the slightest doubt that Mr Elvey could reproduce pain and discomfort in a large number of patients who have symptoms of aches or pains in their upper limb, by putting the cervical spine and shoulder, and the inter-related brachial plexus and cervical nerve roots, on the stretch, as it were. I strongly suspect the same could be said of a large number of patients without any symptoms of pain in their upper limb or, for example, if a patient had an established carpal tunnel syndrome, then one might well increase the tingling in the fingers by stretching the brachial plexus...I am not quite sure where that takes one, but it certainly does not, in my opinion, imply that the patient has something pathological amiss with the cervical nerve roots or brachial plexus.

A considerable amount of scientific work is quoted to support the author's hypothesis that the nerves in the neck/brachial plexus are prone to pathological change when they are put on the stretch, as might occur in various working postures and the like. I am not at all impressed by the supportive papers which they quote...virtually all of them describe extremes of movement or posture, and from my own considerable experience of the brachial plexus clinically and at surgery I am well satisfied that the cervical nerve roots and brachial plexus as a whole, together with its peripheral branches, is well capable of moving easily to accommodate the vast majority of
naturally occurring movements and postures.

I fully accept that the cervical nerve roots will not move as easily as they used to once patients are in their middle or later years and have developed some mild cervical spondylosis, part of the natural ageing process. This is already well documented, as the authors point out, in the lumbar spine, and anyone, like myself, in his 50's, who lies in a peculiar posture for five or ten minutes, can end up with a troublesome ache in a somewhat ill-defined fashion, in the upper limb for some hours thereafter. I consider that this sort of ill defined aching in the upper limb is very common indeed, part of the ubiquitous slings and arrows of normal life, perhaps accentuated with advancing years, and is a perfectly reasonable explanation for the majority of aches and pains that patients attend their doctors with. I consider it fanciful sophistry to be producing arcane papers such as this brachial plexus concept in an attempt to rationalise everyday existence, and moreover imply fault in relation to what are generally considered as ordinary working positions and movements.

The clinical work which Quintner and Elvey adduce to support their own theory is described in a ridiculously naive sentence, "our own work...a group of severely affected patients diagnosed by others as RSI, arose from cervical or brachial plexus" - this sort of statement in a purported scientific paper is ridiculous.

In conclusion, if the authors wish to persuade the scientific community that they have a worthwhile theory to explain upper limb symptoms, then they must go about it in a very much more thorough fashion. There has been no attempt here to put forward an hypothesis and then describe an experiment to either prove or disprove the hypothesis and such work, particularly clinically, with a considerable and worthwhile body of patients and controls, and preferably with independent outside assessors, will be required before this concept can justify serious consideration.

REFERENCES


The paper presents a sound review of possible neurogenic mechanisms. No doubt these mechanisms do play an important role.

However, the paper seems to exclude that biochemical or morphological changes in the muscle tissue itself may cause muscle pain. I disagree with this concept. Most likely the etiology of RSI is rather diverse and its origin may be due to injury of nerves as well as of muscles and connective tissue including tendons and their sheaths.

Regarding pain due to intramuscular changes there are several lines of evidence:

First of all sensory axons originating as free nerve endings in the muscle have been identified and shown to respond to a large number of chemical stimuli (Stacey 1968, Mense & Schmidt 1974, Fock & Mense 1976, Kniffki et al.1978). This means that work-related intra-muscular changes, acute or delayed, may be mediated to the central nervous system as pain.

Secondly, a large number of work-related changes have been documented either due to mechanical overload of the muscle tissue (Fridén 1984, Newham 1983) or due to metabolic overload (for ref see Völlestad & Sejersted 1988, Sjøgaard 1990). It may take more than 24 hours to recover from such overload (Edwards et al. 1977). If sufficient time for recovery is not allowed processes of self destruction within the muscle are likely to occur (Jackson et al. 1984, Edwards 1988, Turner 1988) and noxious substances in the vicinity of the free nerve endings may well be a consequence due to leaky muscle membranes (Lovlin et al. 1987).

Finally, in patients with localized chronic work-related muscle pain in their trapezius muscles, muscle fibres with marked degenerative changes have been identified (Larsson et al. 1988). For further details see also Hagberg 1984.

REFERENCES


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My comments address some points I consider important rather than discussing the entire paper by Quintner and Elvey.

THE NEUROGENIC HYPOTHESIS

In addition to neurogenic mechanisms of work-related neck and upper limb disorders, muscular mechanisms are considered important. A common mechanism that could explain many of both neural and muscular symptoms is elevation of intramuscular pressure in compartment syndromes. This is a well documented condition in the lower extremity, but high pressures have been measured in the supraspinatus and infraspinatus muscles as well, and anecdotal cases of compartment syndrome have been reported in the forearm and hand.

THE BRACHIAL PLEXUS TENSION TEST (BPTT)

The neurogenic hypothesis of RSI largely relies on the BPTT developed by Mr Elvey. Important studies have been carried out on this test, e.g. on the findings among asymptomatic subjects of different ages, effect on the results of contralateral upper limb and leg positions, and reliability and validity of the test.

The problem with the test seems to be the reliability, especially interexaminer reliability. This is not much discussed in the text itself but in Attachment 2 by Mr Elvey. The results of the study of Zuluaga (1986, cited in Attachment 2) showed poor interexaminer reliability, probably due to the difficulties in the positioning of the structures of the neck, shoulder and upper limb. A similar result was obtained by myself following the test protocol described by Wells.
Interexaminer reliability is naturally dependent on the education and skill of the examiners. Although good results in one study do not guarantee that the reliability is acceptable in other hands, an acceptable level of interexaminer reliability has to be shown in a critically performed study, preferably carried out by others than those who have developed the test.

Another problem is that most studies on the BPTT have been published in the proceedings of meetings of manipulative therapist associations, which limits their accessibility. If the final goal of the authors is to make the BPTT generally used in orthopaedic practice, as mentioned in Attachment 2, the scientific evidence of the reliability and validity of the test should be published in peer-reviewed journals read by orthopaedic medicine doctors and surgeons. This would enhance co-operation between manual therapists and medical doctors and probably also result in a better outcome in the treatment of the patients.

TREATMENT OF RSI

Treatment directed towards the causes or aggravating factors is usually considered most effective in any disorder. Because a large number of cases of RSI are considered work-related, a very natural part of the treatment would be ergonomic improvements in the work-place. Changes in work organisation may be needed to make individual pacing of work possible, especially for work in static and constrained postures.

REFERENCES


The subdivision of pain origin into either physical or mental is outdated and dangerous. All pains with an overt physical cause have a mental component. On the other hand, pains with no physical lesions are rare and with characteristic properties not included in RSI (Merskey, 1989).

While therefore on the side of Drs. Quintner and Elvey, I believe they do their case a disservice by concentrating on peripheral nerve damage. They are correct that minor damage to peripheral nerve can produce the observed signs and symptoms in RSI. However, that should give objective histological signs which have been provided in other entrapment syndromes but which have not been shown in this condition. They have neglected the most likely alternative that soft tissue damage may set up hyperexcitable foci not only in the periphery but in the spinal cord. They correctly quote my work on nerve damage but fail to discuss the more recent work on the long term consequences of damage to deep soft tissue (Wall, 1989a&b).

REFERENCES


should be done with case control to avoid this error.

Where partly objective tests such as the demonstration of tenderness are used observer bias can be confounding. For instance in a recent study\(^2\) musculo-skeletal specialists examining cases they had diagnosed as having “myofascial pain syndrome” together with cases rheumatologists had diagnosed as having “fibromyalgia” mixed with a group of control cases under “blind” conditions, produced humbling results. Some alleged features of the “myofascial pain syndrome” were found as often in controls as in cases. The same method should be applied in “RSI”.

Indeed the same critical procedure could be applied to the brachial plexus tension test. The authors quote Keneally as producing positive tests in normals with this manoeuvre. The writer in addition to eliciting such “false positives” has also experienced “false negative” tests in otherwise typical RSI subjects. Further this test can be distressing to the point of threatening doctor-patient rapport and may produce continuing pain.

If, as an alternative hypothesis suggests, the pain and tenderness may arise in any arm muscles one cannot do the stretch test without compressing or stretching such muscles. This muscular hypothesis is that sustained muscle contraction leads to ischaemic pain in the affected muscle since only a fraction of full muscle power will obliterate capillaries within the muscle. This theory in addition to explaining main nerve entrapment syndromes can explain ischaemic paraesthesiae from smaller intramuscular nerves. This could explain the diffuse transient paraesthesiae so common in this complaint. Though there is now limited evidence for this possibility for “RSI”\(^3\) there is experimental evidence to support the ischaemic hypothesis in the closely related complaint, fibromyalgia\(^4\).

In describing the sites of tenderness the authors display their belief that the tenderness overlays main nerve branches and roots. Those who favour the muscle hypothesis would argue that the tenderness is in muscles and at sites of referred pain from muscles and so they may press at different points. Where you press and how hard you press can be important sources of bias.

Another practical difficulty is that electrophysiological and biochemical tests done in a laboratory remote from the work scene may not show transient reversible changes which may only be detectable in the real or simulated work scene.

The distinction between neural and muscular explanations of the symptoms may be unimportant since control of both requires improved posture (which implies muscular relaxation) and also frequent brief relaxation breaks\(^5\). Relaxation also helps control of nervous tension which, by increasing sustained muscle tension, can feed back to produce a self sustaining, escalating pain cycle. The writer is currently undertaking a pilot study of frequent brief relaxation (micro-pauses) using an instruction audio-tape and feels that the results are sufficiently encouraging to indicate a controlled trial.

Yet another hypothesis which attempts to explain the “RSI” syndrome on an anatomical basis considers tenosynovitis to be the basic lesion, indeed this is a prescribed compensatable occupational condition in Britain, though, as Barton\(^6\) points out, what evidence there is for this contention has been inappropriately extrapolated from a different condition, peritendonitis crepitans.

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Responses from Quintner and Elvey

PREAMBLE

From the commentaries of most discussants, it is clear that we did not deal adequately with the some of the clinical aspects of neural pain associated with peripheral neural dysfunction. We therefore provide the following summary:

1. Local pathology (e.g. entrapment) involving a peripheral nerve may cause pain felt at the site of pathology; pain may radiate from the site of pathology (see below) and can be accompanied by unfamiliar sensory phenomena e.g paraesthesiae.45-47 These are the symptoms of peripheral neural irritability; they may occur spontaneously but usually follow mechanical stimulation of the affected nerve.1,45-47 As an example, gentle tapping over the irritable nerve may elicit these symptoms (Tinel's sign).46 Palpation of nerve trunks for local tenderness is an important part of the examination of the peripheral nervous system.1 Tenderness found on palpation of a nerve at the site of local pathology may also be a function of neural irritability.2,46 Sensitised nociceptors in the sheath of the nerve (related to nervi nervorum) are thought to underly local nerve pain and tenderness.2,35,45 Pain projected to the innervation territory of the nerve is explained by activation of fibres within the nerve.45-47

2. Ideally, the neurological examination of the upper limb should include specific manoeuvres designed to selectively, as well as maximally, stress those nerve trunks which could be involved in the production of symptoms.1 Passive stretching of nerve trunks is an accepted means of reproducing a painful response in patients with lower limb pain.16 This is the biomechanical basis for the straight-knee-leg-raising test devised for the lower limb.16 In the discussion paper, we submit that the Brachial Plexus Tension Test of Elvey should be accepted as the analogous test for the upper limb.

3. In addition to the local pain felt on palpation or stretch of an irritable nerve trunk, pain may also be felt in deep tissues at a distance.46 According to Ochoa,47 the mechanism of this type of referred pain may be identical to that of the referred pain produced experimentally, through activation of muscle afferents in continuity with the irritable nerve trunk.

4. The initial localised pain caused by peripheral neural pathology may subsequently spread proximally as well as distally, following the course of the involved nerve.23,25,35,43,61 Possible explanations for this pain behaviour include; (a) sensitisation of peripheral nociceptors; (b) pathophysiology in primary afferents (abnormal discharges, either spontaneous or stimulus-induced, from the injured axon and from its dorsal root ganglion cell); (c) cross-talk (ephaptic transmission) between large and small (nociceptive) axons at regions of damage or demyelination; (d) pathophysiology of the dorsal horn causing changes in central processing of afferent signals (e.g. altered responses due to temporal dispersion of afferent impulses, spontaneous hyperactivity, unmasking of latent synapses, altered central connections).15,35,45,47

5. In those patients with widespread pain following local peripheral neural pathology, a painful response may be provoked by palpation at multiple sites along the course of the affected nerve.2,39 This phenomenon suggests that the entire nerve trunk is behaving as a sensitised nociceptor, generating ectopic impulses in response to minor mechanical stimuli.14

6. As with any pain of deep origin, neural pain can be referred into muscles which have a segmental relation to the injured neural tissue;46 these muscles become tender.22 This tenderness may be due to activation of muscle afferents which have been directly sensitised by impulses arising from the injured nerve.47 or indirectly sensitised through muscle spasm created by reflex activation of motoneurons from primary nociceptor spinal cord input.22
7. There is considerable interest in the pathophysiological mechanisms which underly the increase in nerve excitability whereby "damaged nerves can come to contribute actively to chronic pain both by injecting abnormal discharge into the nervous system and by amplifying and distorting naturally generated signals." Understanding these mechanisms may provide avenues for more effective treatment of pain of peripheral neural origin. Pain of local neural origin which becomes widespread (neuropathic pain) may represent an exaggerated response to injury, thus ensuring conditions of rest to allow for healing of that injured nerve. Unfortunately, this response may be self-perpetuating and cause disruption to the life of the patient.

1. RESPONSE TO MR BESWICK AND MS CURSLEY

I Introduction

In our original study, we reported finding tenderness to palpation of forearm muscles, tendons and entheses. We were unable to reproduce the dominant upper limb pain of these patients when we tested their tender muscles, and the muscles connected to the tender tendons or entheses, using contraction against manual resistance. In our opinion, tenderness of the aforementioned tissues is properly subsumed under the heading of mechanical allodynia (painful sensation to touch). With regard to pain produced on passive stretch of muscles, it is obviously impossible to be able to confine passive stretch to the muscles and not include neighbouring neural elements coursing axially in the upper limb.

In our original study, shoulder pain on upward movement of the arm, often accompanied by restriction of active range, was more difficult to interpret. In essence, if shoulder pain was reproduced on BPTT, and if the limited range of elevation was adversely influenced when performed with contralateral cervical flexion, these findings were thought to denote the presence of painful cervical/brachial plexus neural pathology limiting shoulder movement, rather than local shoulder pathology.

It has been recognised that tissues into which pain of neurogenic origin is referred may develop clinically relevant inflammatory changes. This reflex neurogenic mechanism of inflammation may explain conditions described in association with cervical radiculopathy; the stiff (frozen) shoulder, tennis elbow and the shoulder-hand syndrome. An association between syndromes of the cervical spine and accompanying upper limb tenosynovitis, tendonitis, tennis elbow and carpal tunnel syndrome was noted by Bland. A causal connection with the cervical spine disorder was presumed.

We agree that findings in muscle tissue should be acknowledged and discussed. These findings do not call into question the validity of a purely neurogenic concept. Muscle tenderness is a common accompaniment of referred pain. As outlined in the preamble, there are neurophysiological mechanisms which explain why muscle pain and tenderness can occur in the presence of primary neural dysfunction. One of the phenomena described by Beswick and Cursley (locally tender nodules) has long been recognised in association with underlying neural dysfunction.

II Clinical Features

It can be difficult to distinguish clinically between deep pain of somatic origin and deep pain of neural origin. This difficulty is implicit in the peripheral neural pain hypothesis of Asbury and Fields. A somatic origin is postulated for nerve trunk pain, whereas dysesthetic pain is thought to arise from dysfunction of the conducting portion of neural tissue.

IV. Evidence in support of the "neurogenic" hypothesis.

In the text of the discussion paper, we quoted the opinion of Ferguson who stated that a simple muscle strain should recover in a week or two...... etc. It was this (not unreasonable) speculation which lead Ferguson to consider the possibility of a neural origin of symptoms in those with persistent upper limb pain.
We affirm that the Australian studies quoted in the discussion paper contain many patients in whom the reported clinical findings suggest a neurological basis for their symptoms. It is not unreasonable to recruit the findings of Taylor et al. in support of the neurogenic hypothesis, as numbness in the affected limb was reported by 58% of their patients. In the study of Sikorski et al., 60% of patients reported a sensory disturbance of some form. They were able to distinguish neuralgic patterns (nerve or nerve root distribution) in 43% of their patients. We found it impossible to assess the possible diagnoses in Stone's 100 new patients, seen by him over a 13 week period, with repetitive strain injuries. Using the collective term repetitive strain injuries, Browne et al. focussed attention upon a search for identifiable musculoskeletal and neurological conditions affecting the upper limb. In addition, they indicated that the clinical picture could be complicated by "less clearly defined disorders such as thoracic outlet syndromes caused by compression of the neurovascular bundle at various levels and reflex sympathetic dystrophy syndrome following mild trauma, peripheral nerve injuries, painful disorders of the shoulder and surgery to the upper extremity."

In those with severe "RSI", a careful physical examination will usually enable the examiner to meaningfully provoke the dominant pain described by each patient. We do not regard the BPTT as being either positive or negative (see Attachment II). From the experimental studies to date, one can define a normal range for the test and also a normal response. In the clinical situation, one may determine whether a test response is a normal or abnormal one. A decision is then made whether the BPTT is clinically relevant. In patients who are seen early, there may well be localised distal upper limb neural irritability which may be diagnosed by using the specific nerve tension tests outlined in the discussion paper.

Smythe did suggest that the tenderness which he found was sited at the anterior aspect of intertransverse ligaments at C4-C5 and C5-C6. Our experience is that if these tender areas are gently palpated, particularly with the ipsilateral shoulder girdle depressed (brachial plexus under tension), tenderness may be accompanied by complaints of pain and/or paraesthesiae radiating into the upper limb. This phenomenon suggests that neural tissue is the source of tenderness.

The case report of Levy and the hypothesis of Wilder were used to emphasise that high tensions within the spinal canal, caused by the forward flexed head/neck posture, could prove harmful to neural tissue elements within the spinal canal. We have no means of recording in vivo tension within spinal canal neural elements during the performance of sedentary work of a repetitive nature requiring postural fixity of the head and neck.

As pointed out by Grandjean: "The posture of the head and neck is not easy to assess as seven joints determine the mobility of this part of the body. In fact, it is possible to combine a lordotic neck with a downward bent head, or a forward flexed neck with an upwards directed head." Forward bending neck postures were observed by Ferguson and Duncan in their study of keyboard design and operating posture of telegraphists. When keyboard operators are watching a VDU screen at or near eye level, poked forward head-neck postures are commonplace. A casual walk through any office or factory where process work is performed, will reveal that many workers habitually adopt poked-forward or forward-flexed head/neck postures.

The left sided upper limb symptoms of those in the study of Hunting et al. cannot be explained on the basis of dysfunction of upper limb tissues. The left arms of the operators appear to have been positioned in a strain-free manner, with elbow support. We postulate that symptoms were referred into the left upper limb from irritable neural elements on the ipsilateral side of the cervical spine. From the relevant biomechanical studies (see discussion paper), it is likely that these elements were also exposed to increased spinal neural tension generated within the contents of the spinal canal during the maintenance of constrained head/neck postures.

O'Connell's biomechanical spinal studies were carried out on cadavers, and the
results obtained were used by him to explain physical examination findings in patients with meningitis.

In our original study, we hypothesised that maintenance of the forward flexed or "poked forward" head/neck postures, both in and out of the work situation, was potentially harmful to cervical neural tissues. As mentioned above, we do not agree that sustained hyperflexion of the neck is an uncommon posture in seated subjects performing clerical or process work.

Referral of neural pain into muscles innervated by an irritable peripheral nerve is discussed in the preamble. Frykholm's work is difficult to interpret. The pain felt in the muscles of his patients undergoing operation may have been reflexly referred following his stimulation of inflamed dural sheaths of the respective ventral roots.

V. Differential diagnosis of the pain syndrome "RSI"

It is debatable whether the findings related to muscles (palpable changes in muscle tissues...etc.) do indicate a distinct musculoskeletal condition or whether they are phenomena associated with referred (somatic or neural) pain. Of the authors cited by Beswick and Cursley, only Dennett and Fry claimed an exclusively musculo­ligamentous origin of symptoms. The other authors allowed that RSI (repetition or repetitive strain injuries) embraced a multiplicity of pathological upper limb conditions, both somatic and neural. We believe, along with Ferguson, that discrete identifiable musculoskeletal conditions are uncommon findings in patients with the pain syndrome "RSI" (see response to Dr Cohen). In our opinion, "RSI" is a distinct clinical syndrome related to the occupational factors we have outlined.

Having defined the pain syndrome "RSI", it was then necessary to look at the differential diagnosis. Our hypothesis proposes that purely occupational factors can produce the pain states seen in cervical radiculopathy and the various entrapment neuropathies; these pain states being identical to those of various stages of the "RSI" pain syndrome. Thoracic outlet syndrome was discussed in order to point out the questionable validity of the physical findings said to indicate pathology in this anatomical region.

The neurogenic hypothesis does not require the presence of a change within muscle which then renders it capable of nerve entrapment (this subject is taken up again in our responses to Dr Wigley and Professor Sjøgaard). The anatomical sites where upper limb nerve trunks are vulnerable to damage have been long known. We propose that it is the combination of increased neural tension derived through maintenance of constrained head/neck postures together with friction from repetitive movements of the upper limb which adversely affect a nerve trunk at one or more of these anatomical sites. As will be mentioned elsewhere in our responses, it is possible that the physiological swelling occurring with normal usage of muscles within a closed compartment could also be a factor in nerve entrapment.

Concluding comment

Beswick and Cursely correctly point out that stretch-induced neural pathology has not been considered by most authorities as a possible cause of "RSI". Our views are obviously at variance with those of these authorities, as well as with those of Beswick and Cursely. We answer their last question in the affirmative.

2. RESPONSE TO DR CHAMPION

We accept Dr Champion's analysis of the weaknesses in the neurogenic hypothesis. On the other hand, as he points out, the hypothesis is testable and therefore, refutable. One important point of refutation is emphasised in the discussion paper: "that BPTT may provide the major means of determining clinically whether or not there is a significant neural pathology underlying the upper limb pain of a particular patient." It has not been logistically possible for us to undertake the stringently controlled studies necessary to determine whether the BPTT can be generally accepted as a reliable and valid means of assessing the mechanosensitivity of neural tissues related to the upper limb.

Another important point of refutation would be our presumption of pathology reflecting entrapment of ventral rami of the
lower cervical spinal nerves within the
gutters of the transverse processes. Our
hypothesis relies here upon the anatomical
descriptions of Sunderland58 (also see our
response to Dr Cohen).

3. RESPONSE TO DR WIGLEY

(i) Causation of "RSI" in most patients is
likely to be multifactorial. However, we
disagree with Dr Wigley's philosophy
that a search for a failure of "one part of
the anatomy" is not likely to lead to a
successful treatment and prevention of
"this malady." His own favoured
hypothesis contradicts this philosophy.
He holds that muscles used in the
performance of repetitive work have
become painful as the result of
ischaemia and subsequent lactic acid
accumulation.63 Norstrom44 advanced
the identical hypothesis to explain
writers' cramp.

(ii) Weakness: we agree that formal
investigation of this common complaint
is warranted. In our experience, and
judging from the reports of others,
objective evidence of weakness and/or
wasting of individual muscles are not
features of "RSI". Nor are they common
features of "brachial neuritis".25 Many
patients complain of feelings of
heaviness, tiredness and weakness of
the entire arm. "Dropping a cup" is a
common complaint and may be due to
the presence of pain, or to an
abnormality of sensory perception in
the hand holding the cup.

(iii) The term "regional pain syndrome" was
put forward by the Royal Australasian
College of Physicians, in place of "RSI",
in order to de-emphasise the injury
concept which had become enshrined
in Australia.8 Littlejohn34 claimed that
examination of the majority of those
suffering from the "regional pain
syndrome" associated with occupation
("RSI") revealed no evidence of
synovitis, tenosynovitis or neurological
abnormality.

It may be extremely difficult, on purely
clinical grounds, to distinguish between
a painful restriction of shoulder
movement which arises from primary
shoulder dysfunction and that which is
secondary to cervical pathology
referring pain into the shoulder28 (see
our response to Beswick and Cursley).

When one considers the lack of
agreement as to its underlying
pathology, tennis elbow is a complex
condition,29 There is evidence that
lateral humeral epicondylitis may be
associated with idiopathic carpal tunnel
syndrome, and that carpal tunnel
syndrome may be associated with
significant intervertebral disc
narrowing.41 Carpal tunnel syndrome is
a clearly definable entity only if one
accepts electrodiagnosis as the "gold
standard". There is a very poor
correlation between the responses to
the provocative tests recommended in
the orthopaedic literature, and
electrodiagnostically-confirmed
dysfunction of the nerve in the carpal
tunnel.12 We emphasise that the distal
pain associated with carpal tunnel
syndrome may, in some patients,
spread proximally within the upper
limb, possibly extending to the
shoulder girdle and neck. Their clinical
presentation would then coincide with
the "RSI" pain syndrome.

(iv) Many of our patients continue in
employment. For some, symptoms
remain relatively mild, whereas others
endure very severe pain. We agree that
population and/or workplace studies
should be done with case controls in
order to avoid possible confounding of
the underlying condition by secondary
and tertiary factors.

(v) The problem of observer bias in the
examination of patients with chronic
diffuse upper limb pain (variously
labelled as "RSI", regional pain
syndrome, fibromyalgia or myofascial
pain syndrome) needs to be addressed
urgently. We agree that the standard
orthopaedic examination may be
deficient (as found by Wolfe et al.). The
BPTT appears to be, in our hands, a
useful clinical examination technique
which needs to be evaluated by others
under "blind" conditions. It is not a test
which is positive or negative for a
particular condition; it is a test with
subjective and objective responses,
which both need to be interpreted in the light of the patient's symptoms and other examination findings (see Attachment II).

(vi) It is necessary to have assessed the extensibility of individual upper limb muscles and joints before administering the BPTT. The BPTT can be performed with the upper limb first positioned with the shoulder abducted to 110 degrees (or at the limit of pain free range) combined with external rotation and slight extension, and with the elbow, wrist and fingers in extension, the forearm in supination. The ipsilateral shoulder girdle is then passively depressed by the examiner and the cervical spine gently glided to the opposite side. Any pain produced within the arm by shoulder girdle depression and contralateral neck flexion cannot arise from within upper limb musculature. The findings from our original study if confirmed by others, refute the alternative hypothesis favoured by Dr Wigley.

(vii) As mentioned above, the muscle injury (ischaemia) hypothesis is not a new one. We agree that this hypothesis lacks sufficient empirical content and explanatory power in the pain syndrome "RSI". The structural changes in muscle described in the study by Dennett and Fry have not been accepted by others as evidence supportive of an "overuse syndrome" of muscle.

Simons makes the distinction between specific myofascial pain syndromes and the more general term fibromyalgia, which is applicable to the whole body. The former syndrome is characterised by the presence of trigger points whereas the latter syndrome requires the presence of an agreed number of tender points at predictable anatomical sites, together with non-specific symptoms such as sleep disturbance, fatigue etc. Sola postulated that hyperactive but subclinical trigger points exist in muscle as residual effects of previous injuries or degenerative changes and are "highly susceptible to increased activity at the time of a new assault upon the sympathetic nervous system." Onset of myofascial pain may then result from a variety of stress-inducing stimuli both physical and mental. The question of peripheral neural involvement in so-called myofascial pain syndromes needs to be addressed. According to Simons and Travell, taut bands associated with trigger points within muscles may entrap nerves. This mechanism is not described in the literature on entrapment neuropathies. The ischaemic paraesthesiae postulated by Dr Wigley, presumably due to entrapment of smaller intramuscular nerves, are not described by Simons and Travell in their latest publication. Simons has elsewhere discussed the differential diagnosis between referred pain of muscular origin and referred pain of neurological origin. He cites the presence of neurological deficits that match known peripheral nerve or root distribution, and the presence of electrodiagnostic abnormalities, as important markers of pain of neurological origin. These phenomena certainly do point towards a neural origin of pain, but their absence does not exclude such an origin.

Primary fibromyalgia cannot now be regarded as a discrete clinical entity. Surely the findings of Wolfe et al. (cited by Dr Wigley) have put the last nail in its coffin. Forslind et al. showed that this diagnosis could not be sustained in their patients at five year follow-up re-examination. In addition, muscle biopsy findings which were previously thought supportive of this condition have also been found to occur as frequently in normal controls.

(viii) The evidence that myofascial "trigger points" are situated within a taut band of muscle is largely anecdotal. It is equally possible that they may represent hyperalgesic neural tissues closely related to a particular muscle. Another possibility is that the taut band represents localised muscle spasm which is reflexly induced to protect adjacent irritable neural tissue. Deciding whether muscle or nerve underlying the examiner's finger is the tender structure causing pain may therefore be difficult.
There are some clues which are discussed in Attachment I, as well as in the preamble to our response. If nerve is involved in the generation of pain, it may be possible to reproduce that pain by indirectly placing the nerve under increased axial tension, using an anatomically distant manoeuvre. This is the basis of the BPTT.

(ix) In terms of prevention, we agree that the distinction between neural and muscular explanations may be unimportant. In terms of treatment and prognosis of the established pain syndrome, we consider the distinction to be of paramount importance. Muscle pain associated with exercise usually settles within a week or two in normal subjects and is reduced and eventually eliminated by repeated activity. Neural pain has a poorer prognosis and is more difficult to manage.

(x) The question of tenosynovitis as the basic lesion is an important one. In our experience it is an uncommon finding. Entrapment of the superficial radial nerve can be misdiagnosed as De Quervain's stenosing tenovaginitis, radial nerve entrapment syndromes can simulate tennis elbow and median neuritis in the carpal tunnel is often misdiagnosed as flexor tenosynovitis.

4. RESPONSE TO PROFESSORS HARRINGTON AND BACON

(i) The syndrome "RSI" (also known as OOS), as defined in the discussion paper, does not have a "myriad of presenting features from a wide variety of causes." The clinical features of the "RSI" syndrome are reasonably consistent; we acknowledge that the same syndrome may have different causes apart from those which are related to work. It is indeed appropriate to use Occam's razor (entities are not to be multiplied beyond necessity) in this situation.

(ii) We too have seen patients, with an initial diagnosis of radial wrist tendinitis, who have developed widespread pain in the ipsilateral arm extending to the shoulder and neck. Perhaps the correct initial diagnosis was entrapment of the superficial radial nerve, plus or minus the tendinitis of De Quervain's type.

(iii) Many of our patients with work-related upper limb conditions have had a mechanical (musculoskeletal) cause for their upper limb symptoms. They are not categorised by us as "RSI" or OOS. These diagnoses are not controversial. We deny postulating a neural basis for all with work-related upper limb pain.

(iv) We applaud the studies of subjects in their working environment undertaken by the research team at the University of Birmingham.

(v) We agree that non-neural alternative hypotheses do require critical evaluation. This task was beyond our original brief. To date, attempts to explain the clinical phenomena "RSI" on the basis of injury to, or dysfunction of, upper limb non-neural tissues have not, in our opinion, been impressive.

(vi) End-stage renal failure may result from a number of different renal pathological processes. However, no clinician would mistake end-stage renal failure for cardiac failure. In similar vein we plead for recognition of the neuropathic basis of Harrington and Bacon's end-stage "RSI" or, as we could perhaps call it, status neuropathicus. Just as we see patients with varying degrees of renal impairment short of renal failure, so may we expect to see patients with neural pain syndromes of lesser degrees of severity and complexity.

(vii) A better understanding of the neurogenic hypothesis may well take us a long way forward in terms of diagnosis and also in devising preventative strategies. Longitudinal studies will be necessary to determine both response to treatment and prognosis of patients with "RSI". Whether or not the hypothesis is sterile remains to be seen.

(viii) Harrington and Bacon acknowledge that some OOS patients do have a neural component to their problems. They then beg the question we are attempting to answer, by stating that "the majority do not start that way whatever the final outcome may be."
5. RESPONSE TO DR COHEN

Part A: Overview

Dr Cohen has stated our hypothesis extremely succinctly and we thank him for this. However, we do not claim that the BPTT specifically identifies the brachial plexus as the site of symptoms in "RSI" (see Attachment I).

We are grateful to Dr Cohen for pointing out our epistemological errors. In our discussion paper, Section II. Clinical Features 2. Physical Examination Findings..."shoulder capsulitis" should be changed to read "capsular pattern of restricted shoulder movement" and "reflex sympathetic dystrophy" should be a separate category (g) "overt signs of reflex sympathetic dystrophy". The other criticisms will be answered below.

(i) The terms of reference dictated that we were to primarily consider work-related neck and upper limb disorders. That "brachial neuropathy" can develop in those who are not engaged in remunerative work is not in dispute.

(ii) In the discussion paper, we do recognise the clues to pathogenesis offered by the findings of allosthenia, hyperalgesia and hyperpathia. We are aware of the important contribution that Dr Cohen and his team have made to this area of research and look forward to publication of their findings. The clues to the possible contribution of central nociceptive dysfunction and the issue of CNS plasticity have not been properly canvassed by us.

(iii) We are pleased to note an increasing interest in the subject of neural pain and other positive sensory phenomena. As is made obvious in the discussion paper, we are not convinced that proximal peripheral neural tissues are invariably the anatomical site of origin of initial symptoms.

Part B: Specific Comments

II. CLINICAL FEATURES

1. Common symptoms

(a) We stand by our statement that the widespread upper limb pain in "RSI" follows the course of one or more nerve trunks in the arm. It is our understanding that an irritative lesion involving a single spinal nerve (ventral root) can result in pain being referred along one or more of the major nerve trunks which contain sensory fibres contributing to that spinal nerve. Pain arising from an irritative lesion of a peripheral nerve tends to spread proximally, following the course of that nerve (see Preamble).

(g) Agreed

(h) Agreed

II. PHYSICAL EXAMINATION

(a) Our observation of tenderness which is localisable to peripheral nerve trunks requires confirmation from others. We find that gentle palpation over the course of the median, radial or ulnar nerve in the region of the elbow can produce a complaint of local pain which is accompanied by pain felt some distance away from the site of palpation.

(c) Agreed

(e) The reference to hypoalgesia was wrong and should be deleted. Champion et al. described minor sensory impairment within the distribution of paraesthesiae. The question as to whether the abnormal sensory phenomena are features of peripheral and/or central neural dysfunction cannot be resolved on the evidence available to us.

(f) Agreed

III. HYPOTHESIS

By clinically identifiable musculoskeletal pathology which may coexist with "RSI" we were referring to such conditions as lateral and medial epicondylitis, rotator cuff tendinitis and shoulder capsulitis. We agree that the "RSI" pain syndrome often occurs in the absence of any evidence of these musculoskeletal conditions. However, we note that Champion et al. reported musculoskeletal conditions coexisting with widespread neck and arm pain (with neurological features) in some of their female patients. In our response to Beswick...
and Cursley, we made the point that some authors have reported an association between disorders of the cervical spine and these common conditions.

IV. EVIDENCE IN SUPPORT

1. Neuropathic pain

Grieve was referring to the broad category of patients who present to physical therapists (and to rheumatologists) with persistent pain. He did not necessarily imply tissue damage was an accompaniment to their pain. In retrospect, the approach of Wall 62 would have been more helpful to our discussion. He stated that the cause of most intractable pain is either damage to deep tissues, to peripheral nerve, or to nerve root.

2. Case studies

Our attempts to draw meaningful conclusions from most of the case studies which we reviewed were seriously hampered by the difficulties outlined by Dr Cohen.

8. Brachial plexus tension test

We acknowledge the flaw in our reasoning. We wish to rephrase our statement as follows: "The BPTT appears to be a useful test in clinical situations where upper limb pain and paraesthesia may arise from dysfunctional neural tissues related to the symptomatic arm."

V. DIFFERENTIAL DIAGNOSIS (see response to Beswick and Cursley)

We felt obliged to include entrapment neuropathies in the differential diagnosis of "RSI" as most neurologists regard them as clear-cut entities, confirmed by electrodiagnosis, and separate from the "RSI" syndrome. One criticism of the neurogenic hypothesis has been the absence of confirmatory findings of neural dysfunction from conventional electrodiagnosis. The inability of EMG to predict the presence of both neuropathic pain and interstitial neural pathology has recently been emphasised. 17 The EMG examination is negative in approximately 30% of hands in which a clinical diagnosis of carpal tunnel syndrome is made. 56 It is therefore important for clinicians to understand that each of the upper limb entrapment neuropathies can be associated with widespread upper limb pain, in the absence of an abnormality on EMG examination.

In the context of workers' compensation systems in Australia, clinicians are required to distinguish pain syndromes caused (mainly) by occupational factors, from pain syndromes which may be identical, but caused by non-occupational factors. We accept that, with our current limited understanding of aetio-pathogenesis of these disorders, making this distinction can be a nigh impossible task.

The difficulty in making the distinction between somatic referred pain, radicular pain and pain referred from the nerves themselves is not helped by consulting the literature. In our defence, we refer to the recent study of patients with neck-arm pain by Dalton and Jull 11 in which the pain characteristics of area, region of greatest intensity, quality, depth and localisation did not allow a distinction to be made between pain of somatic and pain of neural origin.

VI. CONCLUSION and VII. IMPLICATIONS

The entrapment neuropathy heuristic still proves attractive to us as it provides a framework on which to base our physical examination and patient management. We agree that our emphasis on entrapment appears somewhat restrictive, but only if one does not consider the possibility of spinal nerve entrapment in the intervertebral foramen. As mentioned above, we have relied upon the work of Sunderland 58 who described the strong attachments binding the lower cervical spinal nerves to the transverse processes of the vertebrae. He made the point that this unique arrangement served to protect the more proximal cervical spinal nerves to the transverse processes of the vertebrae. We have since become aware of animal experimental work demonstrating the formation of a closed compartment syndrome in the dorsal root ganglion (DRG) of nerve roots subjected to mechanical compression. 49 It is an intriguing possibility that sustained high axial tension could reduce blood flow to the DRG of cervical spinal nerves and lead to the same pathology in humans, thus explaining persistent, widespread and radiating upper limb pain.
The failure of pain to resolve following adequate decompressive surgery at the site of entrapment is not incompatible with our hypothesis: surgical intervention may be too late to reverse a state of neural irritability and it may even add to the neural irritability. It is unlikely that a cervical spinal nerve (anterior primary ramus) entrapped within the gutter of its transverse process would be accessible to the surgeon's knife.

6. RESPONSE TO DR VIIKARI-JUNTURA

The neurogenic hypothesis. The references cited by Dr Viikari-Juntura do not provide evidence that upper limb and shoulder girdle compartment syndromes of muscle are common causes of pain in those who perform repetitive manual work. It is possible that elevated compartment pressures could be of importance in the pathogenesis of upper limb entrapment neuropathies and, if one accepts the neurogenic hypothesis, in that of "RSI".

The brachial plexus tension test. We are aware of Dr Viikari-Juntura's assessment of the earlier version of this test. This version was difficult to administer. It has been superseded by the test described in Attachment II. Although some studies have been carried out, the reliability and validity of the BPTT in different clinical situations have yet to be determined, as has interexaminer reliability. We endorse her plea for better understanding and cooperation between manual therapists and medical practitioners.

Treatment of RSI. Ergonomic improvements in the workplace can alleviate the symptoms of those who are able to remain in the workforce, but, on their own, have not been shown to be helpful in the rehabilitation of those with severe persistent pain.

7. RESPONSE TO MR SEMPLE

Mr Semple decries our attempt to provide a unified theory to explain this syndrome of diffuse pain extending from the neck to the fingertips because he does not believe that there is "any one pathological process present."

Who are the "other accepted authorities" to whom Mr Semple refers? In our review of the subject (which was not confined to the references listed in our paper), we found the literature on work-related neck and upper limb conditions to abound with unsupported assertions, some of which have acquired the status of medical dogma. The statement of Mr Semple that "a considerable proportion of the patient's symptoms are of psychological origin...etc." is in this category.3,30,36,37 We do not agree that the majority of patients who complain of pain have no overt physical abnormality. If one looks carefully, a physical abnormality can be found in most patients. The words of Taylor60 are particularly apt: "the average well-taught practitioner makes little use of his hands; exhibits indeed, marked distaste for manipulations; furthermore, that relatively few are equipped with tactile appreciation of morphology, minor differences in resistance, density, temperature, balance, position, tension, relaxation, rigidity, mobility, etc."

Mr Semple's opinion of the brachial plexus tension test (BPTT) in particular, and the scientific basis of manipulative therapy in general, does require a response. We believe that the extensive research which underpins the BPTT (as explicated in the discussion paper) stands up to critical analysis. Manipulative therapists in Australia have contributed to the scientific evaluation of techniques of physical examination used in orthopaedic medicine.31

That the cervical nerve roots, the brachial plexus and its peripheral branches easily accommodate the vast majority of naturally occurring movements and postures is self-evident. Our point is that there are cervical and/or upper limb postures adopted by some during the performance of repetitive manual work that lead to increased tension within some or all of these neural elements. This tension increase is concentrated at the well-known sites of potential neural entrapment.

We would be interested to hear of Mr Semple's more detailed explanation of the ill-defined aching in the upper limb which he claims are part of "the slings and arrows of normal life...etc." Do they have an
underlying physical basis or are they psychogenic? Having embarked on this relatively simple(?) exercise, he may then feel more charitably disposed towards our hypothesis which, of course, relates to those in whom these self-same symptoms appear but do not abate. It has been recently stated that "upper-extremity pain can be an enigma to physicians as it may be related to many different conditions." There is therefore an urgent need for others to critically examine our hypothesis.

8. RESPONSE TO PROFESSOR SJØGAARD

We do not doubt that muscles can be a source of pain following exercise. The subject of skeletal muscle pain and exercise has recently been reviewed. There is no evidence that the "RSI" pain syndrome is a form of delayed-onset muscle pain. Accordingly, Professor Sjøgaard is not on firm ground when she invokes injury to muscles (and other soft tissues) in the aetiology of "RSI".

The hypothesis of "self-perpetuating vicious circles of cellular damage in the pathogenesis of muscle pain" proposed by Edwards fails to explain the phenomenology of "RSI". At the clinical level, evidence of muscle destruction has not been detected in these patients. The muscle biopsy study conducted by Dennett and Fry did not provide evidence of such pathology.

9. RESPONSE TO DR PEARSON

Evidence against the hypothesis

Dr Pearson's support for a primary somatic origin of pain in patients with "RSI" requires examination. In our initial study, we examined a group of patients with chronic and widespread pain. Many of their upper limb tissues certainly were tender, but, as emphasised elsewhere in our responses to discussants, we do not agree with Dennett and Fry that tenderness is indicative of primary underlying pathology existing in the tender tissues.

In our experience, many patients with "RSI" seen at an early stage are found to have tenderness related to nerve trunks in the painful upper limb, and these nerve trunks are usually intolerant to stretch. Swelling of synovial sheaths is not a common finding.

The same model of acute muscle injury as an explanation for the pathogenesis of "RSI" has also been discussed by Dr Wigley. We do not doubt that the changes described occur, but they appear to be self-limiting and not to explain the clinical picture of "RSI". We are not clear just how spasm and shortening of muscles can lead to adhesions forming around nerves. The postulated mechanism is reminiscent of Gower's original concept of fibrositis. He visualised fibrositis as "an affection of the fibrous tissue; it may spread, and it spreads by continuity of this tissue." He described the symptoms of a condition which he called brachial (muscular) fibrositis. This condition could "spread to reach the nerve sheaths of the arm, and in them set up a secondary brachial neuritis with all its consequences, tenderness of the nerves, pain along their course and in their distribution, oedema of the hand, and even muscular wasting or impaired sensation." Dysesthetic pain was rightly included in our discussion of neuropathic pain as it appears to be a recognisable component of the pain of some patients with "RSI". There is no confusion between cause and effect. Whether or not their pain may be sympathetically-maintained is a separate issue which was mentioned but not dealt with in our discussion paper.

Diagnostic criteria

We again apologise for omitting tenderness of muscles and their origins, ligaments, and joint capsules from the list of physical findings. In our opinion, these are physical signs indicative of a state of mechanical allodynia associated with referred (neuropathic) pain.

Dr Pearson has not defined RSI in his discussion, but has highlighted the difficulties of definition in the accompanying paper authored by him, The Management of Upper Limb Disorders (Occupational Health Review, October/November 1990, pp 25-27). Using the definition of RSI contained in this paper (pain and loss of function in the upper limb associated with activities that involve repetitive movements), we would agree with Dr Pearson that tests of neural
irritability would be positive in only a proportion of patients with RSI (Repetition Strain Injuries). This does not invalidate our hypothesis for the pain syndrome "RSI", as defined in the discussion paper. We note that the role of primary peripheral neural involvement in work-related upper limb pain syndromes is becoming increasingly recognised.38

As Dr Cohen has already pointed out, the features of altered sensibility, both positive and negative, are the most important clues to pathophysiology of the "RSI" syndrome. We agree that clinical features of the reflex sympathetic dystrophy (RSD) syndrome may be found in patients with "RSI". Although coldness of the limb affected by RSD will be reversed by successful sympathetic blockade, the most important predictors of a favourable response appear to be correct diagnosis, early treatment, and complete sympathetic interruption of the limb, promptly followed by physical therapy.6

**Treatment Methods**

We have no experience of the use of intravenous guanethidine blocks; cervical sympathetic blockade has not been helpful in many of our patients. Neural stretches are not a new form of treatment in this context.60 All forms of treatment require urgent scientific evaluation. The efficacy of physiotherapy (or any other) treatment, administered on a twice weekly basis for up to a year or more, is likely to be challenged by those who are asked to foot the bill for such treatment.

The barriers to management of "RSI" are indeed daunting. Dr Pearson's paper in *Occupational Health Review* deals with these matters in more detail. Needless to say, our experience in Western Australia is no different from that of Dr Pearson in London. The biggest barrier to management is still our lack of understanding of the pathogenesis and pathophysiology of "RSI".

10. **RESPONSE TO MR OWEN**

We reiterate that our hypothesis refers to both distal as well as to proximal neural tissues as possible initial sites of origin of the "RSI" pain syndrome. We note that Mr Owen favours the hypothesis of cellular damage in non-neural soft tissues as explanatory of the clinical features of "RSI". We have dealt with this in our responses to other discussants who hold views similar to those of Mr Owen. Tissue damage of the type described by Mr Owen has not, as yet, been documented in patients with "RSI".

Distal (peripheral) origin of upper limb pain does require some discussion. In the context of upper limb peripheral neural dysfunction, such onset may result from irritability of either distal33 or proximal11,25 neural tissues.

If Mr Owen and other discussants still wish to pursue the possibility of a primary muscle injury leading to the "RSI" syndrome, then the hypothesis of sympathetic-dependent muscle pain advanced by Ochoa et al.45 could be explored.

11. **RESPONSE TO PROFESSOR WALL**

Professor Wall's response, although brief, proved most challenging. We did not set out to subdivide the origin of pain in patients with "RSI" into either physical or mental. The majority of patients with apparent work-related neck and upper limb pain whom we have examined, do appear to have a physical basis for their pain. By this we mean that we have been able to demonstrate a significant and reproducible physical abnormality. Our hypothesis proposes a peripheral neural origin for the pain of "RSI". As Dr Cohen has remarked, we are postulating subtle forms of entrapment neuropathy. Obtaining objective histological evidence to support this origin of pain has proven difficult. We are usually unable to provide the confirmatory evidence from standard electrodiagnostic testing that would enable us to recommend surgical intervention to confirm entrapment of upper limb neural tissues. As mentioned, there is no surgical approach to a presumed entrapment of spinal nerve in the gutter of its transverse process.

We are grateful to Professor Wall for pointing out the possibility that deep soft tissue damage may set up hyperexcitable foci within the spinal cord that can also trigger pain following input from normal
low-threshold afferents as well as from their inherent on-going activity. In the discussion paper, we drew on Devor's review of mechanisms of pain in patients with damaged peripheral nerves. Included in these possible mechanisms were abnormal central processing of afferent neural activity and abnormal neural discharges actually originating within the CNS.

Devor noted the "striking resemblance between the abnormal electrical behaviour of sensory axons in experimental models of nerve injury and the sensory symptoms that occur in various clinical pain syndromes." This resemblance has also attracted our attention. To date, damage to deep somatic structures has not been demonstrated in patients with "RSI". We are however open to the possibility of deep tissue(s), other than peripheral nerve, reacting to the physical forces generated during the performance of manual work of a repetitive nature. From the review of animal experimental evidence by McMahon and Koltzenburg, it appears that "peripheral injury to a variety of tissues can induce a slowly developing but maintained increase in the excitability of spinal sensory processes, in which dorsal horn cells show increased ongoing activity, increased responsiveness to peripheral stimuli and enlarged receptive field sizes."

CONCLUSION

We thank Dr Bammer for conceiving this project and giving us an opportunity to make known our hypothesis to those who are also grappling with these difficult problems of diagnosis and management. We also thank those who agreed to act as discussants. They have initiated the first "RSI" debate of a scientific nature. As one discussant remarked in private conversation, "it is good to be able to fire at a stationary target." We have endeavoured to provide that target and, where possible, to reply to criticisms of the neurogenic hypothesis, and to accept suggestions and alternative explanations. Obviously, the debate remains unresolved. We look forward to further dialogue from those interested in solving the serious problems of work-related neck and upper limb pain.

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