

The RSI syndrome in historical perspective

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Summary The pain syndrome repetition strain injury (RSI) has been variously interpreted as a psychogenic disorder, an overuse injury of upper limb musculature, and a state of peripheral neural irritability. A review of the history of work-related upper limb disorders was undertaken to ascertain whether RSI is a new medical phenomenon or an older syndrome in a new guise. In the mid-nineteenth century these disorders were known as either craft palsies or writer's and other occupational cramps. Notwithstanding clinical evidence suggesting that most were associated with peripheral neural or muscular dysfunction, a body of influential medical opinion considered them all to be disorders of the central nervous system, appropriately termed the occupation(al) neuroses. During the twentieth century, as discrete occupational upper limb nerve lesions were delineated and the spasmodic form of writer's cramp was recognized as a torsion dystonia, a unifying concept of aetiology for the occupational neuroses of the nineteenth century became untenable. The RSI syndrome of the 1980s can be identified from early case descriptions of both scrivener's palsy and the neuralgic variety of writer's cramp. Contemporary hypotheses proposed to explain RSI are remarkably similar to those proposed for the occupation(al) neuroses.

Introduction

During the 1980s large numbers of those in the clerical and process-work sectors of the Australian workforce reported onset of pain and other sensory symptoms in the upper limbs and/or neck. The various medical conditions diagnosed to explain these symptoms were grouped together as repetitive strain injuries (RSI).^{1, 2} There was general agreement that RSI included conditions with a known pathological basis, such as tenosynovitis, elbow epicondylitis, shoulder rotator cuff degeneration, and carpal tunnel syndrome. More difficult to understand were those syndromes characterized by poorly localized pain where the underlying pathology was difficult to define — for example, tension neck, cervical syndrome, and thoracic outlet syndrome³. The most commonly encountered RSI condition was a diffuse pain syndrome, termed repetitive (or repetition) strain injury (RSI).^{4–6}

RSI was more common in females and was characterized by pain which spread from an initial localized site to become diffuse in one or both arms, often accompanied by pain in the neck and/or upper back.⁶ Sensory symptoms, such as paraesthesiae, numbness, heaviness, weakness, and swelling, frequently accompanied the arm pain.^{4–6} Spasm of upper limb musculature did not appear to be a feature of RSI.^{2, 4–6} Objective signs of neurological deficit in the upper limb were usually absent.^{4–6} During a prolonged debate some authors postulated an underlying physical basis for these symptoms, whereas others put forward alternative models — psychological, psychiatric, or psychosocial (for a detailed discussion of the different hypotheses of RSI see Bammer and Martin,⁷ Mullaly and Grigg,⁸ Hall and Morrow,⁹ and Acutt¹⁰).

A large body of medical literature concerned with occupational upper limb disorders appeared in the last century.¹¹ These disorders were then variously referred to as: cramps (of specific occupations),¹² craft palsies,¹³ professional dyskinesiae (*sic*),¹⁴ professional neuroses,¹⁵ coordination occupation neuroses,¹⁶ fatigue neuroses,¹⁷ and occupation neuroses.¹⁷

The aims of this paper were, first, to ascertain whether RSI is a syndrome which has previously been described under one or more of these earlier terminologies, and second, to review the older theories of pathogenesis of these disorders

and to then compare them with those which were advanced in Australia during the 1980s as explanations for the RSI pain syndrome.

Writer's cramp

In a clinical lecture delivered at St Thomas's Hospital in 1864, Samuel Solly,^{18, 19} Senior Surgeon to the hospital, provided one of the earliest descriptions of a syndrome resembling RSI — severe and persistent arm pain accompanied by other sensory symptoms, and which could affect men whose occupations demanded that they write incessantly (they were known as scriveners). This condition, called scrivener's palsy, often resulted in the sufferer being unable to continue in his occupation. Symptoms included burning or aching pain spreading up the arm, pins and needles and/or numbness in the fingers, a feeling of fatigue in the arm, and a cold feeling in the arm. One of Solly's¹⁸ patients initially experienced cramp through the whole hand after excessive writing. Subsequently this man's writing difficulties were due not to cramp, but to severe pain in the hand and arm accompanied by tingling in the fingers. Cessation of writing occurred slowly in these patients and appeared to result from a combination of pain and fatigue. Solly¹⁹ postulated injury, through overwork, of a nervous centre which was to be found either in the spinal cord or in the cerebellum. In retrospect, the symptoms of scrivener's palsy described by Solly^{18, 19} can be recognized as neuralgic (pain in the distribution of a peripheral nerve, often accompanied by signs of nerve dysfunction).²⁰

According to the famous London physician, Sir John Russell Reynolds,¹³ scrivener's palsy was a synonym for writer's cramp. However, writer's cramp as described in his textbook of medicine was clearly different from the scrivener's palsy of Solly.^{18, 19} Abnormal sensations in the affected limb (pain, numbness, heaviness, coldness) and spinal pain were said to affect only a minority of cramp sufferers. The diagnosis of writer's cramp depended solely on the presence of involuntary spasmodic symptoms (cramp) 'when the attempt was made to execute special and complicated movements'. A 'disease pathologically similar' could be found in other occupational groups such as artists, musicians, seamstresses, smiths, and milkmaids.¹³ Professor Austin Flint,²¹ a prominent New York clinician,

categorized writer's cramp as a 'local spasmodic affection'; he saw no point in recognizing formal varieties of the condition.

The seminal research which led to better understanding of these disorders was performed by George Vivian Poore, Physician to University College Hospital, London.²² The importance of this contribution has been acknowledged by other neurologists.^{17, 23, 24} In his initial paper on the subject, Poore²⁵ described the treatment of a patient with the spasmodic form of writer's cramp, a 'curious sample of a most rare and difficult disorder'. By 1875 he had collected 42 patients whose principal symptom had been loss of writing power.²⁶ In over one-third, the failure of writing was 'undoubtedly due to fatigue of some of the writing muscles.' Cramp in the fatigued muscles, or sometimes in the muscles antagonistic to them, were phenomena observed infrequently. In the majority, there was 'mere impotence' without evidence of cramp or paralysis. Poore's clinical experience accorded with that of the French neurologist, Duchenne of Boulogne.²⁷ For example, in the case of Mlle R, a pianist of great talent, pain which was provoked by playing the piano was clearly neuralgic; she exhibited no evidence of upper limb muscle dysfunction.

Poore²² then analysed 75 cases of writer's cramp and impaired writing power. He recognized the possibility of differing causation and therefore classified the writing difficulties of his patients under six headings: paralytic, spasmodic, degenerative, neuritic or neuralgic, 'true' writer's cramp (the last two resembling each other very closely), and an anomalous group (locomotor ataxia, GPI, and so on). Those in the first group, with paralysis of muscles due to definite peripheral nerve lesions, were said to resemble writer's cramp least. In the second group obvious spasm was the cause of writing impairment. One of his patients, who had worked extremely hard as a writing clerk, experienced extensive spasm localized to the upper limb (focal dystonia). The other patients had evidence of underlying hemiplegia associated with their spasm. In the third group Poore²² observed that tremor of the upper limbs interfered with their writing and that often this was the first symptom of degenerative neurological disease.

The next two groups, which contained the majority of patients whose symptoms resembled those described by Solly,¹⁸ complained of neuralgic pain or fatigue pain after any attempt to use the arm, inability to find a comfortable position for the arm at night, numbness, and a feeling of cramping of the hand. Prominent findings in a typical case of 'true' writer's cramp described by Poore²² were tenderness along the radial (the proximal portion was then known as musculo-spiral) nerve; excessive irritability (as tested by faradism) of the radial nerve and the extensor muscles; difficulty in supinating the hand; pain over the distal radius on forcible flexion of the thumb; and hyperaesthesia on the back of the hand. Tenderness of one or more cords of the brachial plexus was noted in at least half of the patients with the neuralgic form of writer's cramp.

Ten years later Poore²⁸ presented 117 cases of impaired writing power, having excluded degenerative and paralytic cases; nerve tenderness (particularly of the median nerve), changes in faradic irritability (usually depressed) of some of the muscles, and tremor were the most important of his findings. Depressed electrical activity was considered symptomatic of muscle fatigue; nerve tenderness was also attributed to overexertion. Poore²⁸ believed that these patients were suffering from a peripheral neuromuscular disorder rather than from a more central neurological disturbance. When Poore²⁹ examined 21 pianists, with inability to play because of upper limb disability (usually pain), nerve trunk tenderness was again found to be present in many cases. In addition, he observed that slight stretching of the tender nerves was often extremely painful; he even devised

simple stretch (tension) tests specific for each of the median, radial, and ulnar nerves of the forearm.

Beard,³⁰ an American physician, agreed with the view of Poore²² that most cases of writer's cramp and allied disorders arose from local disease of upper limb nerves and muscles. From his own survey of 125 patients he noted that cramp was but one of many symptoms, that it might not be the most important symptom, and that in some patients it might not be present at all.

The occupation(al) neuroses

Clinical description of work-related upper limb disorders in the 1860s and 1870s was followed by concerted attempts at aetiological classification. Most nineteenth-century authors regarded the spasmodic, paralytic, and neuralgic syndromes described by Poore²² as different clinical manifestations of an underlying dysfunction within the central nervous system.^{13, 16, 17, 27} Their views gained ascendancy over those who favoured a peripheral (neuromuscular) origin for all symptoms.^{22, 30}

CENTRAL THEORIES

When he described two characteristic cases of the spasmodic form of writer's cramp, William Gowers,³¹ the great clinical neurologist, was well aware of the studies of Poore;^{22, 28} they were both on the staff of University College Hospital. Nevertheless, he made the surprising and far-reaching decision to follow the practice of German physicians, by referring to all upper limb disorders caused by occupational factors as 'occupation neuroses' (from the German *Beschäftigungs-neurosen*).¹⁷ According to Cassirer,³² a contemporary Berlin physician, 'the designation neurosis states at once that, according to our present knowledge, there is no underlying lesion of the nervous system'. This term was used by Gowers as a 'convenient designation for a group of maladies in which certain symptoms are excited by the attempt to perform some often-repeated muscular action, commonly one that is involved in the occupation of the sufferer.'¹⁷

Gowers¹⁷ distinguished motor (spasmodic) and sensory (neuralgic) varieties of occupation neurosis, which could be present singly or in combination. Those of 'distinctly "nervous" temperament, irritable, sensitive, bearing over-work and anxiety badly' were more frequently affected. Gowers¹⁷ formulated a unified but extremely complex hypothesis to explain the clinical phenomena of both varieties. He favoured a central cortical origin (deranged action in the centres concerned with the particular occupational act) as the source of the symptoms, although he recognized the possible additional importance of any local upper limb disease or injury.

Whereas Gowers¹⁷ denied a primary peripheral neural origin of symptoms, he tried to incorporate the observations of Poore²⁸ into his central hypothesis, by postulating that secondary pathological changes could develop in the sensory nerves as a result of 'pain that is purely central in origin' and in the motor nerves as a result of the centrally induced spasm. Reflex (pain) mechanisms were also thought to be responsible for secondary changes in the irritability and nutrition of the muscles of the affected upper limb. Although cramp could develop in those without predisposition, Gowers¹⁷ thought that anything which 'lowers the tone of the nervous system may doubtless act as a predisposing cause'. He singled out anxiety as deserving of special mention in this regard. He found it 'remarkable how many patients, at the time of onset of the affection, were enduring anxiety from family trouble, business worry, or weighty responsibilities. Note, however, that Reynolds,¹³ who had previously considered the influence of 'worry of mind and anxiety' on the development of writer's cramp, concluded that these psychological factors did not appear to be of great importance.

The German neurologist, Hermann Oppenheim,¹⁶ also espoused a central hypothesis to explain all occupation neuroses. Writer's cramp, the most common of these, was said to occur in different forms, either singly or in combination; spasmodic, tremulous, paralytic, and neuralgic. He doubted whether anatomical alterations would ever be found in patients with these disorders, and thought that 'continued emotion may cause it'. Oppenheim¹⁶ regarded writer's cramp as 'a purely functional disorder, an exhaustion neurosis, which has its seat in the centres for coordination, in the central apparatus which governs the associative acts of the muscles which are necessary in writing'. He found that his patients were 'rarely individuals with an intact nervous system, but most of them are of a neuropathic predisposition'.¹⁶ This was evident from the other conditions of a 'nervous' type found in his patients with occupation neuroses, for example, neurasthenia, hemicrania, neuralgia, epilepsy, stuttering, tabes, and agoraphobia. Cassirer,³² whose views coincided with those of Oppenheim,¹⁶ referred to the general experience that the majority of cases of writer's cramp developed from neurasthenia — a neuropathic disposition, either congenital or acquired.

PERIPHERAL THEORIES

In his comprehensive review of the literature Paul,²³ a Boston neurologist, was critical of the many authors who had proposed a central origin for the occupation neuroses: 'The symptom almost exclusively considered is cramp. The extensive symptomatic variations have received scant attention from the etiological standpoint'. He reviewed the symptoms recorded in the case records of 200 patients with 'occupation neurosis' who had attended the outpatient department of the Massachusetts General Hospital. Upper limb pain had been present in 177 cases; pain tended to be localized to muscles, joints, and regions near joints. Numbness had been recorded in 38 and weakness in 26; in no case had cramp been recorded. From these retrospective data, admittedly incomplete, Paul²³ claimed that the symptoms of all conditions subsumed as occupational neuroses could be explained by local injuries to muscles and/or to nerves running in muscles, tendons, fascia, and joints resulting from 'indefinitely repeated impacts and tensions of short duration'.

Norstrom³³ claimed that chronic myositic nodules within the muscles of the back of the neck were a frequent cause of headache. He then searched for indurated areas within the muscles of the head, forearm, and arm and found them in 34 of 47 cases of the spasmodic form of writer's cramp. He postulated that the cramp was secondary to chronic myositis, and treated the nodules with massage. Neurotic disposition, either pre-existing or developing after the onset of cramp, made the prognosis worse because the associated 'reflex irritability' was added to the local (muscle) condition. Noting that in some cases cramp could occur in the absence of these nodules, he thought that 'these may belong to that form of writer's cramp in which the real cause is probably a disturbance of the coordination of the movements.'

Monell³⁴ described a progression of symptoms in writer's cramp from an initial, reversible, state of acute muscle fatigue to a persistent fatigue stage with symptoms of 'pains, lameness, heaviness of the arm, and marked lack of power' which were relieved by rest but returned when work was resumed. The disease was seen by him as a state of chronic fatigue, wherein muscles damaged by toxic products of their own metabolism failed to regenerate during periods of rest.³⁵ He postulated that 'if overuse is continued day after day, the nutrition of the tissues does not keep pace with the waste of wear and rest becomes less and less effective.'³⁴

HETEROGENEITY

By the early twentieth century it had become obvious to

neurologists that the occupational neuroses comprised a group of truly heterogeneous disorders. This can be seen from the Society Proceedings of the 37th annual meeting of the American Neurological Association (1911), wherein is recorded the discussion of Paul's paper on this subject.²³ There was agreement among the neurologists present that the symptoms of many patients were of peripheral neural origin. Ramsay-Hunt³⁶ noted that 50–60 different types of occupation neuritis, from very different occupations, had been described. To avoid confusion, he recommended that these conditions should be kept separate from occupation neuroses of central aetiology (characterized by true cramp or spasm).

The heterogeneity of the symptoms of the occupational neuroses, as shown by Poore^{22, 28, 29} and Beard,³⁰ was also evident from the study conducted by Charles Dana, Professor of Nervous Diseases in Cornell University Medical College.³⁷ He presented his findings from 100 patients with symptoms (mainly upper limb) attributed to the work involved in their occupations and who were attending Cornell Clinic for Nervous Diseases. Most patients were writers, telegraphers, stenographers, typists, musicians, pressers, ironers, or tailors. Occupational neuralgias and neuritides were by far the commonest conditions, being encountered in more than 50% of these patients. A separate group of 23 patients suffered 'genuine' occupational cramps; symptomatic cramps accompanied brachialgia or neuritis in six patients.

Dana¹⁵ stated that 'neuritis is undoubtedly present in some forms of writer's cramp, so called' and 'if there is a great deal of pain in the arm, with tenderness along the course of nerves . . . if there are sensations of tingling, numbness, etc. . . . then the trouble is undoubtedly peripheral and due largely to an underlying neuritis.'

Occupational neuralgia and neuritis

Spontaneous pain has long been known to accompany inflammation of a sensory or mixed peripheral nerve.^{38–40} The following characteristics were attributed to neuralgic pain — persistence; paroxysmal (epileptiform); darting, shooting, tearing, or cutting; intensity varying from slight to atrocious; temporarily increased by external agents (movements, coughing, and so on); felt in the region supplied by the nerve involved; when intense, radiating to regions of other nerves; and changes in cutaneous sensibility in the distribution of the affected nerve (sometimes hyperaesthesia, sometimes anaesthesia).^{38–40} An inflamed sensory nerve was always found to be tender.³⁹ Local pain evoked on pressure over the nerve may travel in both the centripetal and centrifugal directions.³⁹

It has been accepted that signs of neurological deficit are not a prerequisite for the diagnosis of neuralgia.²⁰ Gowers⁴¹ emphasized the rarity of loss of sensation and significant muscular atrophy in cases of brachial neuritis. It is therefore surprising that he did not classify the sensory (neuralgic) form of writer's cramp as a form of brachial neuritis or brachial neuralgia. Windscheid,⁴² a contemporary German physician, had accepted this concept when he affirmed that: 'Neuralgia of the nerves of the cervical and brachial plexuses may be prevented by saving the arm from overstrain at various occupations; for instance smiths, locksmiths, file cutters, turners, tailors and telegraphists.' Perhaps Gowers's alleged dogmatism explains his reluctance to dismantle the central hypothesis of the occupational neuroses.⁴³

During the latter years of the nineteenth century, as mentioned, neurologists had identified a number of discrete peripheral nerve lesions of the upper limb caused by occupational factors.⁴⁴ They had also canvassed the possibility of the presence of incipient peripheral nerve lesions causing symptoms.⁴⁴ It is therefore likely, as suggested by Spaans,⁴⁴ that 'a number of conditions called "occupational neurosis"

in the older literature were based on lesions of peripheral nerves.' In fact, Oppenheim¹⁶ allowed that combinations of occupation neuroses with a neuritis might occur.

By 1912 four groups of patients with occupation neuritis had been recognized — those with repeated direct trauma to the nerves during their work; those exposed to poisonous substances, such as lead; those in whom fatigue of certain muscle groups had induced the nerve lesion (this group probably included the entrapment neuropathies); and those with neuritis due to non-occupational causes, such as alcohol and infectious diseases.⁴⁵

Although the sensory (neuralgic) form of 'occupational neurosis' was recognized by all authorities,^{12, 15-17, 21, 22, 24, 32} the absence of definite sensory or motor deficit in the affected limb prevented its inclusion in the category of occupation neuritis.³² Well in to the twentieth century this form of writer's cramp continued to be regarded by some experts as merely a variant of a functional disease, occupational neurosis, to which persons of a 'nervous' temperament were more liable.^{12, 24, 32, 46} The longevity of this viewpoint was probably attributable to the powerful influence of the opinions of Oppenheim¹⁶ and Gowers.¹⁷ Their opinions accorded with the nineteenth-century view that 'nervous diseases' were the consequence of the innate vulnerability of the human nervous system to the stresses and strains of civilization.⁴⁷ Occupational neuroses became explicable in these terms. A contrary view was held by Beard,³⁰ acknowledged as the authority on the neurasthenic diathesis.⁴⁷ Beard³⁰ believed that writer's cramp was primarily a peripheral neuromuscular disease which 'occurs mostly in those who are of strong, frequently of very strong, constitutions, and is quite rare in the nervous and delicate.'

Spasmodic form of writer's cramp

Understanding the pathogenesis of the spasmodic form of writer's cramp has also presented an enormous challenge to physicians. Pierre Janet,⁴⁸ a prominent French neurologist and psychiatrist, viewed the majority of these patients as psychasthenics. He brought into this single disease entity practically all the manifestations of the psychoneuroses other than the definitely hysterical. Included in this category were reactions characterized by phobias, compulsions, and anxiety.⁴⁹ Psychasthenia was thought to be the consequence of a general lowering of psychic energy (nervous exhaustion).⁴⁹

The following is Janet's description of one of his patients with the spasmodic form of writer's cramp: 'As soon as she tries to write, her fingers close spasmodically on the pen; they ride one upon the other; her whole arm becomes contracted. As a result of this, her hand rests too heavily upon the pen, digs it into the paper, so that she becomes unable to write legibly after the first two or three words.' Janet⁴⁸ noted that this woman 'was extremely well behaved and over-scrupulous, tried to be absolutely blameless in her conduct, with an overwhelming desire for orderliness and neatness . . . This overscrupulousness, this aspiration towards perfection, became localized (if I may use the term) towards the age of 20 upon one particular action, that of writing.'⁴⁸ When Janet's patient was promoted at work and given more responsibility, she was greatly troubled: 'Looking at her unfortunate handwriting, she was terrified at the thought that she would now have to write in public. And thenceforward her powers of writing were hopelessly impaired.' Efforts to teach her to write produced no benefits, and Janet⁴⁸ concluded that 'we shall obviously have to educate something more than the powers of extending the hand; and unfortunately it is not easy to ascertain with precision the nature of the tendency which has to be re-educated, and the technique of the required education.'

Janet's concept of this form of writer's cramp had obviously influenced Williams,⁵⁰ an American neurologist:

The term neurosis concerning occupational disabilities is a misnomer; for the disorders of occupation to which this name has been given are in reality psychodynamic inhibitions or disorders in the habitual series of co-ordinated associations gained by education in some art. A want of harmony in the controlling of the mechanism is the fault. Hence the disharmony is always psychological. This is easily proved by the fact that the neuromuscular apparatus which fails to perform a particular occupational act can quite well accomplish any other act, and this by means of the same muscles, nerves and brain areas.⁵⁰ Williams⁵⁰ dismissed all other theories previously put forward as explanations of 'professional dyskinesias attributed to neurosis' and attributed the lack of proper co-ordination to the influence of 'mental processes' preliminary to the occupational act. He concluded that measures to prevent fatigue and improve the general health of workers should be adopted, but were unlikely to prevent occupational neurosis 'if the ideational seeds are not prevented from germinating in the minds of workers'.⁵⁰

Millais Culpin,⁵¹ an English pioneer in medical industrial psychology, also saw an obvious link between occupational cramps and psychoneurotic affections: 'cramp may occur associated with severe anxiety, as one amongst other hysterical symptoms associated with an obsessional state, or as a pure conversion hysteria in subjects presenting no other symptoms whatever.'⁵¹ For Culpin^{51, 52} the infinite variety of cramp symptoms, their often capricious nature (some cramp subjects suffer only when under observation), and the occurrence of cramp in those whose occupation did not involve excessive writing, disproved the hypothesis that the condition was due to a disordered action of a part of the central nervous system.

In the more recent psychiatric literature, occupationally related spasm or cramp has been variously interpreted by different authors as a conversion reaction,⁵³ as a form of learning or conditioning in psychologically normal individuals,⁵⁴ and as a psychosomatic disorder occurring in obsessional and dependent individuals with unresolved conflicts in their lives.^{55, 56} Among neurologists there have also been differing opinions. Brain,⁵⁷ for example, favoured a primary psychogenic causation rather than the result of a state of fatigue of cortical ganglion cells or an organic disorder of basal ganglia. Other neurologists regarded the spasmodic form of writer's cramp as a localized dystonia.^{58, 59} Sheehy and Marsden⁵⁹ considered that any psychiatric disturbances present in these patients were either secondary to the condition or purely accidental.

By testing the H reflex, Nakashima *et al.*⁶⁰ demonstrated the first objective evidence of a disturbance of function in the upper limb musculature of patients with the spasmodic form of writer's cramp. Reciprocal inhibition in the forearm flexor muscles was found to be disturbed, resulting in co-contraction of agonist and antagonist muscles during the act of writing as well as overflow of contraction into remote muscles. Similar findings were noted in the dystonic arms of patients with symptomatic hemidystonia (due to structural brain lesions) and those with hemiparesis due to stroke. These workers hypothesized that basal ganglia dysfunction is present in the patients with occupational cramps producing a disorder of descending control of those spinal interneurons which mediate the group I presynaptic inhibition of afferent terminals in the spinal cord.⁶⁰

Historical perspective of RSI

NEUROGENIC HYPOTHESIS

The clinical features of RSI resemble closely those described in the neuralgic or true forms of writer's cramp.^{22, 28, 29} The symptoms described by patients with RSI⁴⁻⁶ are similar to those of patients with brachial neuritis.³⁹⁻⁴¹ It is therefore not surprising that RSI has been viewed by some

authors as a neurogenic pain syndrome.^{4, 5, 61} These authors postulated that the pain and other sensory symptoms arose from upper limb peripheral neural tissues, exhibiting increased mechano-sensitivity as a result of their exposure to excessive forces of friction and/or tension generated during the performance of repetitive manual work. This explanation had been advanced previously by Paul²³ for 'the occupation neuroses and so-called occupation neuritis, and occupation pain'.

MUSCLE OVERUSE HYPOTHESIS

Another interpretation of RSI symptomatology was advanced by Ferguson,⁶² who stated that 'the majority of cases of repetition strain injury are not localized 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.' Ferguson⁶³ had previously described the same disorder, then termed occupational myalgia, in a group of Australian telegraphists.

The muscle injury hypothesis of RSI received considerable support.⁶⁴⁻⁶⁸ Some authors commented on the resemblance of RSI to primary fibromyalgia syndrome.^{4, 6, 68} A hypothesis of muscle injury had previously been put forward for writer's cramp by Monell.^{34, 35} Paul,²³ although favouring a peripheral neural aetiology of upper limb pain in patients with writer's cramp, did suggest that forces generated by 'excessive physiological functioning' could also bring about myolitic (*sic*) changes which, presumably, could result in upper limb pain. As noted elsewhere in this paper, Norstrom³³ diagnosed and treated chronic myositic nodules in the upper limb musculature of many of his patients with the spasmodic form of writer's cramp.

PSYCHOGENIC/PSYCHOSOCIAL HYPOTHESES

On the other side of the debate, Lucire⁶⁹ maintained that the symptoms of 'RSI' were psychogenic. She saw the syndrome as containing elements of both somatization and conversion.⁷⁰ She argued strongly against RSI being a disease of muscles on the basis that 'there is no known organic disorder where a group of muscles become dysfunctional and painful for one intentional activity but not for another.'⁶⁹ Culpin⁵¹ had made a similar pronouncement when discussing the aetiology of the spasmodic forms of writer's and telegraphist's cramp. Lucire,⁶⁹ seemingly unaware of the obvious dissimilarity, drew an analogy between these conditions and RSI. She went even further and declared that 'its epidemiology, natural history and multimodal symptomatology are unknown in any organic disorder or injury.'⁷⁰

According to Lucire,⁶⁹ the 'idea that one has been, and still can be, "injured" actually created the neurosis.' Williams⁵⁰ put forward the identical argument in his paper on the spasmodic form of writer's cramp. The concept of RSI as a psychogenic illness was expanded by Lucire^{69, 70} and other authors, all adamant in their denial of an underlying physical injury.⁷¹⁻⁷⁵ They proposed a model of psychosocial causation of RSI. Using this model to explain the development of RSI in a particular individual, it was necessary to assume the presence of the following factors: (i) any stressful life situation which generates a conflict about working; (ii) the so-called everyday aches and pains (fatigue) during the performance of repetitive manual work; (iii) a belief strongly held that repetitive movements can injure upper limb tissues; (iv) reinforcement of this belief from fellow workers, trade union officials, politicians, the press, and other media; (v) medical diagnosis and certification of work-related injury in the absence of the accepted physical signs of injury; (vi) a workers' compensation insurance system easy of access; (vii) a favourable sociopolitical milieu for acceptance of RSI as a compensable condition.

Conclusion

It is certain that RSI is not a new medical condition. The different theories promoted to explain its development can be traced back into the last century. Despite a long-running debate in Australia over its diagnosis, pathogenesis, and nomenclature, only now are the full consequences of RSI to the individual sufferers being pieced together.⁷⁶ For many, RSI has meant prolonged and severe pain with ensuing physical disability, secondary anxiety and depression, disruption to personal relationships, curtailment of career prospects, and protracted arguments in a combative medico-legal environment.

The pain of RSI has proven extremely resistant to most modalities of treatment.^{2, 4, 61, 62} It is therefore appropriate, when considering the rival and apparently exclusive causal hypotheses of RSI, to pay heed to these words of Spaans:⁴⁴

Altogether the results are often not unsatisfactory during treatment . . . In the long run the results are very poor. This ought to be an extra stimulus not to give up searching for an organic lesion in all cases where an occupational neurosis seems to exist. That neurotic symptoms are found in people confronted with the impossibility of continuing their vocations is not surprising and does not signify that neurotic factors determine the nature of handicap.

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