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Review Article

Work related upper limb disorders

Michael Hutson ^{a,b,c}

^aThe Royal London Hospital for Integrated Medicine, Queen Square, London; ^bInternational Federation of Manual/Musculoskeletal Medicine, ^cBritish Institute of Musculoskeletal Medicine, UK

A B S T R A C T

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Neuropathic arm pain (NAP), commonly referred to as 'RSI', diffuse upper limb disorder or type II work related upper limb disorder (WRULD) is a regional pain syndrome affecting the upper limb(s), characterized by persistent pain and dysaesthesiae. It is commonly associated with long periods of keyboard use, though may also occur in workers engaged in other types of repetitive stereotyped activities of the hands. Adverse ergonomics is commonly present. Sleep pattern is usually disturbed, and depression, headaches, chronic fatigue, and frustration are frequently experienced. The pathogenesis of NAP is probably linked to overloading of sensorineural mechanisms responsible for pain production and perception. Clinical findings include muscle tension and hyperalgesia in the upper limbs and shoulder girdles, adverse neural dynamics, and frequently (and importantly) proximal dorsal spinal dysfunction. Other factors that are often associated with the development of neurosensitisation include premorbid psychological profile, environmental stresses, misattributions and beliefs, adverse posture and ergonomics, iatrogenesis, and litigation. Conventional investigations such as cervicodorsal spinal radiographs, nerve conduction studies, and MRI are negative. Clinical management is structured on reduction of provocative stresses and treatment of neuromusculoskeletal dysfunction. The differential diagnosis is discussed in this article.

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1. Introduction: The clinical picture

The regional pain syndrome, commonly referred to as neuropathic arm pain (NAP), 'RSI', or type II work related upper limb disorder (WRULD)¹ to distinguish it from those disorders in which there is demonstrable pathomorphology (type I WRULD), is characterized by persistent pain and dysaesthesiae in the upper limbs. Discomfort often extends diffusely from the hands, wrists and forearms to the shoulder, neck and upper back. In a minority of patients it is experienced in the contralateral upper limb. Usually the pain has a deep, burning, 'toothache' quality, sometimes accompanied

by a subjective sensation of swelling and by a variable degree of numbness and tingling in the hand and/or fingers. It is commonly associated with long periods of keyboard use at a computer, though may also present in workers engaged in other types of repetitive stereotyped activities of the hands. The symptoms are often precipitated by an intensive spell of keyboard activities or a change of work equipment, workstation, use of a laptop, or work/lifestyle issues.

Initially the symptoms resolve with rest but eventually become more persistent, intense, increasingly intrusive with respect to work and home activities, and refractory to either rest or conventional treatment. Sleep pattern is often disturbed, and depression, headaches, chronic fatigue, and

E-mail address: mahutson@aol.com.
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frustration are commonly experienced at this stage. The hypothesis with respect to the pathogenesis of NAP is that pathophysiological changes develop within the central nervous system, precipitated by repetitive or prolonged stresses applied to the soft tissues of the upper limb. Afferent stimulation at a consistently intense level may arise in the hands and fingers, but may also arise at the wrist, and proximally at the elbow, shoulder girdle or at the cervicodorsal spine. The overloaded sensorineural mechanisms for pain production and perception are augmented by sensitization of the wide dynamic range (WDR) neurones situated in the dorsal horns of the spinal cord. Pain amplification develops, resulting in allodynia (reduced threshold to painful stimuli), hyperalgesia (increased response to stimuli), wind-up (increased response to repeated stimulation), hyperpathia (prolonged response to stimuli), and the expansion of receptive fields (giving rise to relatively widespread symptoms).

Secondary hyperalgesia, manifesting as 'regional' muscle tenderness and sometimes terminal discomfort on joint movements, are present in the majority of patients. The proximal extensor and flexor forearm muscles are usually tender. The proximal scapular fixator muscles are also hyperalgesic. There are usually signs of segmental dysfunction in the cervical spine or proximal thoracic spine or both. Adverse neural tension is commonly present. It is noteworthy that although it is generally recognized that adverse neural tension may be associated with cervical spinal dysfunction, it is extremely common in this group of patients for proximal thoracic spinal dysfunction (between D3 and D5) to be present. It is hypothesized that in many patients poor work postures, associated with a proximal thoracic kyphosis and protracted shoulders, cause proximal thoracic spinal and cervical spinal dysfunction, and altered neurodynamics.

Other aetiological factors often play a role. These include psychosocial factors (such as premorbid psychological profile, environmental stresses, misattributions and beliefs, adverse posture and ergonomics, iatrogenesis, and litigation). Of these factors, iatrogenesis is often a powerful aggravating factor. Just when a patient sorely needs a knowledgeable medical practitioner in the early stages to deal with the described factors appropriately, help is not always at hand. Regrettably, the failure of the medical profession in general to recognize the early presence of neuropathic pain leads to misdiagnosis, inappropriate management, and an increasingly frustrated and despondent patient. It is not surprising that reactive depression often develops, sometimes compounded by the prescription for amitriptyline, useful in low dosages for pain relief, but without an appropriate explanation for the use of a tricyclic antidepressant, inculcating in the patient's mind the impression that the medical attendant believes 'it's all in the mind'.

Conventional investigations such as cervicodorsal spinal radiographs, nerve conduction studies, and MRI are negative. Occasionally nerve conduction studies may be equivocal for CTS, leading the unsuspecting orthopaedic surgeon to undertake carpal tunnel release. Surgery may also be undertaken for suspected epicondylalgia, though usually unsuccessful and a significant risk for aggravation of underlying neurosensitisation. NAP remains refractory to such ill-advised operative intervention.

2. Historical perspective

A grasp of the historical background to the evolution of contemporary medical views on the condition that has been referred to as RSI since the 1980s is instructive (Quintner, 1991). Bernardino Ramazzini, the founder of Occupational Medicine, described problems experienced by scribes (who wrote with a quill) in his treatise *De morbis artificum* in 1713. "Intense fatigue of the hand and whole arm" was noted by Ramazzini to be often associated with "the intense and incessant application of the mind" in work in which "the whole brain, its nerves and fibres, must be constantly on the stretch". Clearly the interaction between mental stress and musculotendinous strain was identified 300 years ago. When the steel nib replaced the goose-quill pen in the early decades of the 19th century, an outbreak of writer's cramp occurred in male clerks working in the British Civil Service, and described by Sir Charles Bell. In the late 1880s Vivian Poore of University College, London identified diverse upper limb overuse disorders, including "piano-failure" of which he wrote (in 1887) "... it seems very liable to occur in persons of delicate organisation who are in depressed health or who have been exposed to cold".² Sir William Gowers, neurologist at University College Hospital, London used the term "Occupational neurosis" for writer's cramp and similar conditions (using the word neurosis to indicate a disorder of peripheral nerves, not a psychological affliction). Much more recently, an "outbreak" of upper limb pain in data processing operatives, accounting machinists and typists was recorded in Australia in 1981/2 and subsequently labelled repetitive strain injury (RSI). Although it has been universally recognised that the mechanistic concept of repetitive strain injury is often inappropriate, the label 'RSI' has nevertheless remained in the public domain. Perhaps more disconcerting to some are the diverse views on its pathophysiology and aetiology within the medical profession.

It is my firm opinion that the regional pain syndrome RSI (known also by a variety of acronyms such as CTD, NSAP, OCBP, RCBP, WRULD) is not concordant with the biomedical model of disease or injury, first promulgated in the 19th Century as the paradigm of illness based on cellular pathology by Rudolf Virchow³ in 1858, and consolidated during the 20th Century by the structural fundamentalism of the vast majority of the medical profession including Rheumatologists and Orthopaedic Surgeons. There is no discernible tissue "injury" that can be detected by the investigative tools currently available to us, no agreed objective diagnostic criteria across the medical disciplines, hence the rejection by many physicians of the concept of a neuromusculoskeletal disorder to account for the diffuse symptoms and tenderness in the majority of patients with regional pain syndromes of this type.

As it is generally agreed that frequently there are associated psychological issues, albeit the symptoms described by patients are not typical of defined psychiatric conditions such as conversion states or somatoform disorders, a diagnosis of "psychogenic illness" following exclusion of organic pathology has often been applied in past years. The renowned psychiatrist Merskey⁴ inter alia has denounced such unwarranted disregard for the positive criteria of psychiatric illness; yet the inference to be drawn from the views of many "experts" in

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