

Work-related upper limb “overuse” syndromes: A review of historical descriptions and interpretations suggesting a psychogenic origin

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ABSTRACT

A previous review of historical descriptions and theories about the character and pathogenesis of writer’s cramp and other comparable chronic upper limb “overuse” work-related pain syndromes has indicated that somatic dysfunctions explain symptoms and findings. The first case studies and case series suggested that these conditions were caused by pathology affecting the peripheral nerves. The general perception gradually changed, however, with symptoms becoming attributed to central nervous system dysfunction and ultimately to represent a psychiatric condition. Work-related upper limb disorders remain diagnostically challenging to clinicians and there is still a tendency to see many patients’ pain as a psychiatric problem when a standard physical examination does not explain the condition. This article describes reports of writer’s cramp and comparable occupational upper limb “overuse” conditions that have occurred sporadically and epidemically, and reviews interpretations from the nineteenth century that relate symptoms to psychogenic conditions.

Keywords: Historical medicine, Nerve afflictions, Neurological examination, Occupational medicine, Upper limb disorders, Work-related disorders

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INTRODUCTION

A previous review of historical descriptions and interpretations of writer’s cramp and other work-related upper limb “overuse” conditions with a special emphasis on writer’s cramp has suggested that the workers’ symptoms and the findings on physical examination can be explained by peripheral nerve afflictions [1]. The initial interpretations by the ancient authors were that these ailments were peripheral and caused by afflictions of nerves or muscles although it was acknowledged that a psychic vulnerability could also play a role. Towards the end of the 19th century, this perception was gradually changed and the symptoms were assumed to represent dysfunctions in the central nervous system. At that time these conditions were understood as occupational “neuroses” – a term applied at the time when no somatic pathology could be identified. In the beginning of the 20th century these conditions were increasingly regarded as purely psychoneurotic.

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The various hypotheses can be seen against the diagnostic challenges in relation to upper limb work-related disorders that clinicians encountered. These difficulties were not only present previously but remain central to the scientific debate and clinical practice, e.g. in relation to the character of arm pain in computer operators. The majority of upper limb patients seen in an occupational context are regarded to be uncovered by consensus diagnostic case definitions [2] and cannot be diagnosed with a physical standard approach.

This article presents historical case reports and interpretations that attribute symptoms to the sphere of psychiatry, and compares the historical descriptions with current observations and assessments of upper limb pain conditions. The neurological examination of the peripheral nerves is particularly emphasized.

Occupational neuroses

Central nervous dysfunction in vulnerable/nervous workers

In 20th century, many experts perceived work-related “overuse” disorders such as writer’s cramp as functional conditions and termed them occupational “neuroses”, since no somatic pathology could be identified. Coined by William Cullen in 1769, the term “neurosis” referred to “disorders of sense and motion” caused by a “general affection of the nervous system”. Cullen used the term to describe various nervous disorders and symptoms that could not be explained physiologically. The meaning of the term was redefined by Jung and Freud in the early and middle twentieth century. It has continued to be used in psychology and philosophy while neurosis is no longer used as a psychiatric diagnosis.

Gowers, a prominent neurologist towards the end of 19th century, termed writer’s cramp and comparable work-related disorders as occupational neuroses understanding by this concept conditions, which according to the existing knowledge lack underlying lesions of the nervous system. He perceived symptoms to be caused by performing repetitive muscle actions, usually related to the patient’s work, and regarded excessive writing as the main causal factor because writer’s cramp was particularly prevalent among professional writers. He noted, however, that nervousness and anxiety predisposed for the condition, especially when it occurred in people who had not written much. He therefore believed that “a lowered tone” of the nervous system predisposed to the condition. He described most patients as nervous, irritable and sensitive with a psycho-neurotic personality showing signs and symptoms of pathological anxiety. The patients were frequently concerned about work and family issues, and heavy burdens of responsibility and problems relating to the work were other characteristic features [3]. This view was supported by Cassirer and Oppenheim [4, 5], who stated that writer’s cramp could

develop consequent to a dysfunctional nervous system such as with neurasthenia, a neuropathic predisposition, persistent emotions, a functional condition, or an exhaustion neurosis [5]. This understanding persisted, because it was in accordance with the general understanding at the time, namely that these diseases were consequences of a congenital vulnerability of the nervous system, which was influenced by the stress and strain of modern civilization [6]. The occupational “neuroses” were explained by somatic dysfunctions of the central nervous system, which nervous people were more prone to acquire than others [4, 7–9].

Psychological and psychiatric descriptions and interpretations of work-related “overuse” conditions

In 1925, Janet contributed to the debate by arguing that psychasthenic symptoms develop secondary to nervous exhaustion. Janet described a patient with perfectionistic traits who had spasmodic writer’s cramp and described the consequences of her promotion with allocation of greater responsibility [10]. Seeing clear links to psychoneurotic conditions with symptoms such as nervous exhaustion, phobia, obsessions, compulsions, hysteria and anxiety, Janet [10] and Culpin [11] emphasized that some patients had symptoms under observation only, and that symptoms could occur without excessive writing [11, 12]. Consequently, they concluded that writer’s cramp could not represent a disorder of the central nervous system. Williams who also rejected the previous theories of occupational “neuroses” [3] attributed the symptoms in writer’s cramp to psychodynamic inhibitions – a psychological disharmony in the control of the mechanism in the habitual series of coordinated associations obtained through learning. He emphasized that the neuromuscular system could be unable to perform a specific occupational action but was still able to perform other operations by using the same muscles, nerves and brain areas. Williams attributed the lack of coordination to the influence of mental processes on the work but also pointed out the importance of preventing fatigue and improving the workers’ overall health. He regarded prevention as impossible, however, unless the “growth of ideas in the worker’s mind” could be avoided [13].

The psychologist Lucire perceived chronic “overuse” upper limb disorders as of a purely psychogenic character [14] and found features of both somatization and conversion in these conditions [15]. She rejected the theory that they represented a disorder of muscles, since there is no condition in which muscles become dysfunctional and painful for one activity, but not for another. Excluding an underlying physical damage, Lucire suggested a psychosocial pathogenic model for its development [14–21].

The model assumed the presence of

1. A stressful life situation that may create a conflict in relation to work
2. So-called everyday aches and pain (fatigue) while performing repetitive manual work
3. A strong belief in the patient that repetitive movements can damage upper limb tissues
4. Confirmation of this view from colleagues, unions, politicians, the media, etc.
5. A medical diagnosis and certification of occupational injury, in spite of the absence of accepted physical signs of injury
6. Easy access to workers' compensation
7. An advantageous socio-political environment for the acceptance as a compensable disorder

Culpin reached the same conclusion about writer's cramp and telegraphist's cramp [11]. Drawing a parallel to these conditions, Lucire stated that the patients' multimodal symptoms with elements of somatization and conversion [14, 15], and the epidemiology and natural history of both conditions were unknown in any organic disorder [15]. According to Lucire and Williams, the very idea that one can and has been injured creates the neurosis [13, 14].

Hunter

In Hunter's textbook on occupational medicine published in 1975, "occupational cramp" was still the umbrella concept covering occupational "overuse" disorders. Hunter stated that the symptoms are triggered during the execution of a customary act involving complex, rapid and repetitive movements that are developed to perfection with a high degree of precision through training and experience. The disease develops when the demands exceed a certain level. The necessary coordination breaks down, and spasm, tremor, pain, weakness and loss of control occur in muscles that are accustomed to perform harmoniously the concerned act. Still, he regarded occupational cramps as psychoneuroses due to a psychological component of tension and nervousness that is connected with an attention to getting the job done in time and with the required quality, and because symptoms could develop with normal work when physical or psychological stress impairs the performance [9].

Although there was almost a century since the views of Gowers [3] were presented, Hunter supported his theories because no structural changes were described in the nervous system or the muscles, whereas psycho-neurotic symptoms could be identified by a careful and detailed clinical interview. Hunter described the various perceptions regarding the pathophysiology as alternating from physical exhaustion of muscles and nerves to a disorder of behavior, although he recognized that the causes could be multiple and of a both physical and mental character [9]. A diagnostic feature in Hunter's view was that the symptoms were limited to writing, and he emphasized the importance of separating writers' work-related conditions from other

painful and paralytic affections and from simple pain resulting from excessive writing.

He regarded the prognosis for established disease as poor if the patient was not completely released from writing, and if there was no other external cause than writing. Treatment should be immediate to be effective. A month relief could often alleviate most symptoms, but six months could be necessary. He recommended writing with the intact hand – and a typewriter in severe cases – and suggested the administration of sedatives, massage and exercises to relieve spasms and pain [9].

Newer editions of Hunter's textbook have moved away from the previous interpretation and stated that three quarter of work-related upper limb disorders cannot be diagnosed according to diagnostic consensus criteria [2].

More recent interpretations

Based on social and moral evaluations, many experts in upper limb disorders continue to view psychological issues, including compensation issues, as the main etiologic factors in unexplained upper limb pain.

Psychosocial interpretations continued throughout the twentieth century. More recent psychiatry interpreted occupational spasm or cramp as a psychiatric condition [22], a conversion reaction [23], and as a psychosomatic illness in obsessive and dependent individuals with previous unresolved conflicts [24, 25], but also as a form of learning or conditioning in mentally healthy subjects [26].

Lucire's [14, 15] and Culpin's [11] perceptions of work-related "overuse" upper limb disorders as psychogenic since muscles cannot become dysfunctional and painful for one activity, but not for another [16–21] were supported in Brains neurological textbook in which a primary psychogenic cause was emphasized rather than a cortical fatigue condition or an organic disorder of the basal ganglia [27].

Awerbuch summed up the general view when the Australian epidemic of repetition strain injury peaked in the early 1980s by stating that abnormal diagnosis behavior leads to abnormal illness behavior in the patient, and that this is invariably compounded by abnormal treatment behavior [28].

Arguments against regarding work-related "overuse" disorders as occupational "neuroses" or psychiatric conditions

The views of Gowers [3], Cassirer [4] and Oppenheim [5] were opposed by Beard who rather than an occupational "neurosis" regarded writer's cramp as a primary peripheral neuromuscular disease that would rather occur in people of a strong, often very strong constitution, and is quite rare in the nervous and fragile [29]. Paul argued that what was perceived as occupational "neuroses" was due to local damage to muscles and

nerves near tendons, fascia and joints resulting from repeated impacts and tensions of short duration [30]. Other neurologists perceive spasmodic writer's cramp as a localized dystonia [31, 32].

Merskey addressed the lack of positive criteria for diagnosing a psychiatric condition [33]. Sheehy and Marsden regarded any psychiatric disorder in these patients as either secondary to the condition or as a random occurrence [32].

Macfarlane et al. argued against a single uniform etiology [34] because the onset of arm pain could be related to work-related psychosocial exposures in addition to mechanical exposures, but also to other aspects of health and somatic symptoms. This statement supports the dominant hypotheses in 19th century, which argued that constitutionally vulnerable neurasthenics were prone to develop neuralgic upper limb symptoms that at the time were categorized as neurotic [35].

Authors such as Norstrom acknowledged that a neurotic disposition or reaction manner with established disease worsened the prognosis [36]. Oppenheim also conceded that a combination of occupational neuroses with neuritis might occur [5].

Occupational cervicobrachial disorder (OCD), repetition strain injury (RSI) and cumulative trauma disorder (CTD)

Occupational cervicobrachial disorder (OCD) was described in Japan as a condition with chronic fatigue and pain in the neck and upper limbs related to work tasks, which were characterized by repetitive work and possibly mental stress [37]. A similar condition, repetition strain injury (RSI), which developed epidemically in Australia, was particularly linked to office work [38–40]. Cumulative trauma disorder (CTD) was a similar construct in the USA [41]. The RSI, which became the common covering term in Anglo Saxon literature, was not regarded as a localized syndrome but of a more diffuse character, and apparently affecting muscles. There was, however, little understanding about the etiology, pathogenesis and pathology of this condition, and why once it has occurred, it seems to continue to exist despite prolonged relief of the patient [42].

While these designations could be convenient for the physicians, and for the patient who now became convinced about the disease and its cause, they were less useful for treatment and prevention. Furthermore, they incorporated tautological considerations about causation but without indicating any specific mechanisms, and said nothing about the pathology. The constructs of OCD, RSI and CTD have been criticized and regarded as caused by medical and social iatrogenesis, or again viewed as somatization, hysterical, depression or other psychiatric conditions. They have also been considered as conversion of indisposition meaning that everyday common symptoms that are prevalent among all workers lead to more severe and persistent complaints in people

who face difficulties in coping with their work or life due to limited personal resources. Authorities such as doctors and lawyers were alleged as responsible for supporting such a mechanism [43].

A muscular etiology for chronic "overuse" upper limb conditions was suggested by Ferguson, who described occupational myalgia in Australian telegraphists [44] and later in industrial workers [45] although he described neurotic personalities among the former but not the latter group of workers.

The muscle damage hypothesis for RSI got significant support [46–50], and remains the standard for many researchers and clinicians. Several authors have discussed the similarity with primary fibromyalgia syndrome [49, 51, 52] and with reflex sympathetic dystrophy, although RSI was suggested to represent a different disease entity, with vasodilation and decreased vasomotor response characteristic for the former [53].

Focal dystonia represents a cluster of symptoms that associates to the descriptions of upper limb "overuse" disorder. By testing the H-reflex Nakashima et al. in 1989 could objectively demonstrate upper limb dysfunction in patients with spasmodic writer's cramp with disturbed reciprocal inhibition in the forearm flexors during writing leading to agonist and antagonist muscle co-contraction [54].

A vascular basis for repetitive strain injury was studied by Pritchard who demonstrated a relatively contract radial artery in patients with diffuse forearm pain compared to controls, and that the artery does not dilate with use of the limb. The diffuse forearm pain was interpreted as related to a physiologic claudication in the working forearm muscles [55] with pain and paresthesia occurring with muscle exertion when the intra-compartmental pressure is increased. This will cause relative ischemia of the capillary supply of the peripheral nerves in the compartment [56]. Further elaboration on this theory has demonstrated that surgical decompression for forearm compartment can relieve pain in writer's cramp [57].

Contemporary researchers and clinicians [58] have supported the view of neurologists in the late nineteenth century that discrete upper limb peripheral nerve dysfunction were related to minor peripheral nerve lesions caused by occupational factors [59]. The concept of adverse nervous tension [60] and treatment modalities based on this concept [61] has addressed this issue.

DISCUSSION

The main arguments for the psychogenic character of writer's cramp and similar work-related "overuse" upper limb conditions are the following features:

Presence of nervous symptoms: A painful condition that does not respond well in treatment including conventional analgesics (that are not very helpful in neuropathic pain), and which pose a threat to the level of functioning and the work capacity, is likely to

put the patient in crisis. If work, earnings and providing for the family are challenged, a mental and even psychiatric reaction is to be expected although people differ in their vulnerability to work loads and in their thresholds for seeking medical care. A crisis is even more to be expected if the clinician fails to diagnose the condition.

Absence of physical findings: The examiner only finds what is looked for. Taking into account that the somatic symptoms are frequently pain of a neuropathic character, weakness and abnormal perception of sensation [1]. It would be relevant to apply a systematical neurological examination of the upper limbs. This examination should be sufficiently detailed and assess the strength in representative upper limb muscles, the sensation in homonymous territories, and the soreness of nerve trunks and the brachial plexus. It is my experience that this examination will reveal patterns of abnormalities that reflect nerve afflictions with specific locations and is therefore likely to explain the patient's symptoms [62].

Selective disability: The disability depends of the location of the pathology, which again depends on the location of the work-related strain. To me it is not surprising that reproducing the work-related strain will provoke the symptoms. That is why many people with work-related upper limb disorders need to change their occupation. To my experience this is indeed the case with peripheral nerve afflictions such as those related to intensive computer work [63].

Spread of symptoms, including collateral spread: Spread of symptoms from their primary location, e.g. to the opposite limb as noted by Gowers [3] and repeated by Hunter [9] was one of the arguments for refusing a somatic genesis. Still today this frequent phenomenon challenges clinicians. There are, however, many explanations for contralateral spread – so-called mirror-image pain. Firstly, a patient with unilateral symptoms may tend to compensate by using the contralateral limb, which may then be at risk. More importantly, mechanisms such as the activation of astrocytes and microglia in the contralateral dorsal spinal cord, activation of satellite glia and macrophages in the contralateral dorsal root ganglia, and sensitization of the peripheral nerve by neurotrophic factors resulting in enhanced nociceptor excitability are all mechanisms that may contribute to the spread of symptoms [64]. Hunter's advice to start to write with the healthy arm is not to recommend in this context [9].

Symptoms under observation only: This concern was only addressed by few authors [10, 11] and not brought up elsewhere by the authors cited in my previous study [1] and this review. I never noted my patients to behave differently when they were not under observation. On the other hand, clinicians may become biased when they find that they cannot explain the symptoms, and in this case it is not unusual to attribute the patient's complaints to a functional state, or malingering. Justification of this rationing would require statements to support it. The

patient, who experiences a clinician who cannot confirm the presence of a somatic disorder, may react by trying to appear convincing by demonstrating his difficulties to the clinician.

CONCLUSION

Upper limb patients constitute a major proportion of patients referred for assessment in occupational medicine. Some of them have clear diagnoses such as epicondylitis or rotator cuff tendinitis. The majority, however, do not fit into these categories. These patients are likely to be misdiagnosed and therefore mistreated. Regarding their complaints as psychogenic is not a feature of the past. Diagnosing workers with upper limb "overuse" pain conditions as malingerers, or interpreting their symptoms as so-called everyday aches and pain (fatigue) while performing repetitive manual work, is still a reality, which is likely to further exacerbate their condition with consequently increased physical disability, anxiety and depression.

The physical examination of these patients should not only focus on joints, tendons, and muscle soreness, but include a thorough physical assessment of the neurological qualities such as individual muscle strength, the presence of mechanical allodynia of nerve trunks and the brachial plexus, and sensory qualities at locations with homonymous innervation. This examination will permit diagnostic classification of almost all patients and provide improved guidance for management.

Author Contributions

Jørgen Riis Jepsen – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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REFERENCES

1. Jepsen JR. Work-related upper limb “overuse” syndromes: A review of historical descriptions and interpretations suggesting a somatic origin. *Edorium J Occup Environ Med* 2016;2:8–18.
2. Palmer K, Cooper C. Repeated movement and repeated trauma affecting the musculoskeletal disorders of the upper limbs. In: Baxter P, Adams P, Aw T, Cockcroft A, Harrington J eds. *Hunter’s Diseases of Occupations* 9ed. London: Arnold; 2000. p. 453–75.
3. Gowers WR. *A manual of diseases of the nervous system*, 3ed. London: J&A Churchill; 1899. p. 1892–93.
4. Gowers WR. *A Manual of diseases of the Nervous System*, 2ed. London: J&A Churchill; 1892. p. 710–30.
5. Oppenheim H. *Diseases of the Nervous System*. London: JB Lippincott Co; 1901.
6. Drinka GF. *The Birth of Neurosis: Myth, Malady, and the Victorians*, New York: Simon & Schuster; 1984.
7. Osler W, McCrae T. *The Principles and Practice of Medicine*, 10ed. New York: Appleton and Company; 1925.
8. Kinnear Wilson SA. *Neurology*, vol. 3, 2ed. London: Butterworth & Co. Ltd.; 1955.
9. Hunter D. *The diseases of occupations*, 5ed. London: Hodder & Stoughton; 1975.
10. Janet P. *Psychological Healing*. Vol 2. New York: Macmillan; 1925.
11. Culpin M. *Recent Advances in the Study of Psychoneuroses*. London: J&A Churchill; 1931.
12. Culpin M. *Mental Abnormality: facts and theories*. London: Hutchinson’s University Library; 1948.
13. Williams TA. Occupation neuroses; their true nature and treatment. *Medical Records* 1913;(83):464–67.
14. Lucire Y. Neurosis in the workplace. *Med J Aust* 1986 Oct 6;145(7):323–7.
15. Lucire Y. Social iatrogenesis of the Australian disease ‘RSI’. *Community Health Stud* 1988;12(2):146–50.
16. Brooks PM. Regional pain syndrome - the disease of the 80s. *Bulletin of the Postgraduate Committee in Medicine, University of Sydney* 1986;(42):55–9.
17. Brooks PM. Repetitive strain injury does not exist as a separate medical condition. *BMJ* 1999;307:1298.
18. Morgan RG. Repetition Strain Injuries. *Med J Aust* 1986;144:56.
19. Awerbuch M. Regional pain syndrome. *Med J Aust* 1987;147:59.
20. Ireland DC. Psychological and physical aspects of occupational arm pain. *J Hand Surg Br* 1988 Feb;13(1):5–10.
21. Bell DS. “Repetition strain injury”: an iatrogenic epidemic of simulated injury. *Med J Aust* 1989 Sep 4;151(5):280–4.
22. Kolb LC. *Modern Clinical Psychiatry*, 9ed. Philadelphia: WB Saunders; 1977.
23. Cameron N. *Personality Development and Psychopathology*. Boston: Houghton Mifflin; 1963.
24. Crisp AH, Moldofsky H. A psychosomatic study of writer’s cramp. *Br J Psychiatry* 1965 Sep;111(478):841–58.
25. Bindman E, Tibbetts RW. Writer’s cramp--a rational approach to treatment? *Br J Psychiatry* 1977 Aug;131:143–8.
26. Sylvester JD, Liversedge LA. Conditioning and the occupational cramps. In: Eysenk HJ ed. *Behaviour Therapy and the Neuroses*. Oxford: Pergamon Press; 1960. p. 334–48.
27. Walton JN. *Brain’s Diseases of the Nervous System*, 8ed. Oxford: Oxford University Press; 1977.
28. Awerbuch M. *RIS. Med J Aust* 1986 Oct 6;145(7):362–4.
29. Beard GM. Conclusions from the study of one hundred and twentyfive cases of writer’s cramp and allied affections. *The Medical Record* 1879;244–47.
30. Paul WE. The etiology of the occupation neuroses and neuritides. *J Nerv Ment Dis* 1911;38(8):449–66.
31. Lance JW, McLeod JG. *A Physiological Approach to Clinical Neurology*, 3ed. London: Butterworths; 1981.
32. Sheehy MP, Marsden CD. Writers’ cramp-a focal dystonia. *Brain* 1982 Sep;105 (Pt 3):461–80.
33. Merskey H. Regional pain is rarely hysterical. *Arch Neurol* 1988 Aug;45(8):915–8.
34. Macfarlane GJ, Hunt IM, Silman AJ. Role of mechanical and psychosocial factors in the onset of forearm pain: prospective population based study. *BMJ* 2000 Sep 16;321(7262):676–9.
35. Beard G. Neurasthenia, or nervous exhaustion. *Boston Med Surg J* 1869;80:217–21.
36. Norstrom G. A study of the affection “writer’s cramp”. *New York Medical Journal and Philadelphia Medical Journal* 1904;79:491–7.
37. Maeda K. Occupational cervicobrachial disorder and its causative factors. *J Hum Ergol (Tokyo)* 1977 Dec;6(2):193–202.
38. Quintner J. The RSI syndrome in historical perspective. *Int Disabil Stud* 1991 Jul-Sep;13(3):99–104.
39. Ireland DC. Repetitive strain injury. *Aust Fam Physician* 1986 Apr;15(4):415–6, 418.
40. Littlejohn GO. Repetitive strain syndrome: an Australian experience. *J Rheumatol* 1986 Dec;13(6):1004–6.
41. Hadler NM. Cumulative trauma disorders. An iatrogenic concept. *J Occup Med* 1990 Jan;32(1):38–41.
42. Ferguson D. The “new” industrial epidemic. *Med J Aust* 1984 Mar 17;140(6):318–9.
43. Hadler NM. *Occupational musculoskeletal disorders*, 1ed. New York: Raven Press; 1993.
44. Ferguson D. An Australian study of telegraphists’ cramp. *Br J Ind Med* 1971 Jul;28(3):280–5.
45. Ferguson D. Repetition injuries in process workers. *Med J Aust* 1971 Aug 21;2(8):408–12.
46. Stone WE. Repetitive strain injuries. *Med J Aust* 1983 Dec 10-24;2(12):616–8.
47. RSI, “kangaroo paw”, or what? *Med J Aust* 1985 Mar 18;142(6):376–7.
48. Fry HJ. Overuse syndrome of the upper limb in musicians. *Med J Aust* 1986 Feb 17;144(4):182–3, 185.
49. Wigley RD. Repetitive strain syndrome--fact not fiction. *N Z Med J* 1990 Feb 28;103(884):75–6.
50. Dennett X, Fry HJ. Overuse syndrome: a muscle biopsy study. *Lancet* 1988 Apr 23;1(8591):905–8.
51. Champion GD, Cohen ML, Quintner JL. Fibromyalgia in the workplace. *Ann Rheum Dis* 1993 Nov;52(11):836–7.
52. Miller MH, Topliss DJ. Chronic upper limb pain syndrome (repetitive strain injury) in the

- Australian workforce: a systematic cross sectional rheumatological study of 229 patients. *J Rheumatol* 1988 Nov;15(11):1705–12.
53. Cooke ED, Steinberg MD, Pearson RM, Fleming CE, Toms SL, Elusade JA. Reflex sympathetic dystrophy and repetitive strain injury: temperature and microcirculatory changes following mild cold stress. *J R Soc Med* 1993 Dec;86(12):690–3.
 54. Nakashima K, Rothwell JC, Day BL, Thompson PD, Shannon K, Marsden CD. Reciprocal inhibition between forearm muscles in patients with writer's cramp and other occupational cramps, symptomatic hemidystonia and hemiparesis due to stroke. *Brain* 1989 Jun;112 (Pt 3):681–97.
 55. Pritchard MH, Pugh N, Wright I, Brownlee M. A vascular basis for repetitive strain injury. *Rheumatology (Oxford)* 1999 Jul;38(7):636–9.
 56. Pritchard MH, Williams RL, Heath JP. Chronic compartment syndrome, an important cause of work-related upper limb disorder. *Rheumatology Oxford* 2005 Nov;44(11):1442–6.
 57. Pritchard MH. Writer's cramp: is focal dystonia the best explanation? *JRSM Short Rep* 2013 Jun 5;4(7):1–7.
 58. Greening J, Lynn B. Minor peripheral nerve injuries: an underestimated source of pain. *Man Ther* 1998;3(4):187–94.
 59. Spaans F. Occupational nerve lesions. In: Vinken PJ, Bruyn GW eds. *Diseases of Nerves. Handbook of Clinical Neurology*. Amsterdam: North-Holland Publishing Company; 1970. p. 326–43.
 60. Butler DS. *The sensitive nervous system*. Adelaide: Noigroup Publications; 2000.
 61. Arumugam V, Selvam S, MacDermid JC. Radial nerve mobilization reduces lateral elbow pain and provides short-term relief in computer users. *Open Orthop J* 2014 Oct 17;8:368–71.
 62. Jepsen JR. Clinical neurological examination vs electrophysiological studies: Reflections from experiences in occupational medicine. *World J Methodol* 2015 Jun 26;5(2):26–30.
 63. Jepsen JR. Upper limb neuropathy in computer operators? A clinical case study of 21 patients. *BMC Musculoskelet Disord* 2004 Aug 13;5:26.
 64. Cheng CF, Cheng JK, Chen CY, et al. Mirror-image pain is mediated by nerve growth factor produced from tumor necrosis factor alpha-activated satellite glia after peripheral nerve injury. *Pain* 2014 May;155(5):906–20.

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