

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

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## ABSTRACT

**The historical descriptions of chronic upper limb “overuse” pain syndromes reflect contemporary work-related conditions. Therefore, the former theories about the character and pathogenesis of these conditions are still of interest. The first case studies and case series related these conditions to abnormalities in the nervous system or muscles. The general opinion gradually changed and a dysfunction of the central nervous system, a “neurosis” was assumed, because no underlying lesion of the nervous system could be identified and many patients appeared to be mentally ill. This narrative review discusses the symptoms and findings in the physical examination reported in the first historical descriptions and interpretations at that time. There is evidence for an involvement of the peripheral nerves in the pathogenesis of these syndromes although this was not demonstrated by systematical neurological examinations at the time.**

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

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## INTRODUCTION

This article aims to review some of the historical descriptions and interpretations of work-related upper limb “overuse” conditions with a special emphasis on “writer’s cramp”, which was their common designation. The clinical manifestations and the interpretations at that time are presented and discussed with emphasis on symptoms and physical findings that may indicate an involvement of the peripheral nervous system.

The initial perception that peripheral pathology caused by adverse work was responsible for the workers’ symptoms was gradually changed to emphasizing a somatic dysfunction of the central nervous system, which according to some clinicians was influenced by a nervous state in the patients [1]. Later interpretations went even further and argued that these conditions were purely psychiatric.

The diagnosis of work-related upper limb disorders remains a challenge today, as a major proportion of work-related upper limb disorders are regarded as diagnostically unclassifiable [2]. Therefore, a look back on the historical descriptions is interesting and the previous descriptions and interpretations may potentially contribute to the

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current understanding of work-related “overuse” upper limb disorders.

## METHODS

This is a narrative rather than a systematically composed review. Literature dealing with the history of work-related upper limb disorders was searched for in PubMed, and further references came up from the reference lists of the retrieved articles.

## RESULTS

### Historical descriptions of work-related “overuse” conditions

Most historical literature from the nineteenth century termed chronic work-related upper limb “overuse” conditions as spasms, paralyses, or dyskinesias, but cramp was the main designation. These ailments were mainly described among writers – “writer’s cramp” – but “telegraphist’s cramp” and “cotton-twister’s cramp” are other frequently reported comparable conditions that occurred endemically in exposed occupations. Analogous conditions were described from numerous additional occupations, industries, arts and sports where the arm and hand is used intensively (Table 1). Examples are shoemaker cramps characterized by severe bouts of pain at night that mostly occurred in apprentices, and milkmaid’s and tailor’s cramp, which could be transient and disappear on stopping work. Comparable syndromes were described in the lower limbs, the head and neck, as well as in the tongue [3].

### Ramazzini – the first comprehensive description

The prototype of the classic occupational cramp – “writer’s cramp” – was first described by Ramazzini (Figure 1) three hundred years ago [4] (Table 2). Ramazzini described three major triggering factors: Constant sitting, perpetual motion of the hand in the same manner, and the attention and application of the mind [4]. He described the clinical features and speculated about the character of “writer’s cramp” (Table 2). He was concerned about the well-being of his patients and suggested several different treatment options.

“Writer’s cramp” was again described by Bell in 1830 [5] (Table 3) who underlined the selective handicap of patients and emphasized their happiness.

### Solly – central nervous system affliction

An epidemic of “writer’s cramp” in office workers was attributed to the introduction of the steel pen to replace the goose feather quill [6]. In 1864, Solly described



Figure 1: Bernardino Ramazzini—the father of occupational medicine.

Table 1: Risk occupations for so-called occupational cramps (after Kalmus 1934 [3]).

Writers, telegraphists, cotton twisters, tailors, drapers, seamstresses, sailmakers, knitters, hairdressers, ironers, bowlers, metal workers, hammermen, turners, engravers, goldbeaters, cabinet makers, sawyers, locksmiths, tinsmiths, nailmakers, masons, painters, enamel workers, compositors, watchmakers, shoemakers, saddlers, sailors, fencers, diamond cutters, money counters, letter sorters, cigarette rollers, cigar makers, pianists, organists, violinists, violoncellists, harpists, flautists, drummers, orchestra conductors, typists, comptometer workers, waiters, florists, artificial flower makers, folders of newspapers, and milkers of cows.

writers who worked during extended time periods. Following initial cramp in the hand they developed severe and persistent burning arm pain. The pain was accompanied by fatigue and weakness, a feeling of cold, and peripheral tingling, stinging and/or numbness. The severity of the symptoms could result in discontinuation of their profession [7]. Solly explained the condition by overloading of a nervous center in the spinal cord or cerebellum [7, 8].

Table 2: Ramazzini's description of "writer's cramp" [4].

"That there were a far greater number of scribes and notaries among the ancients than there are in our day because of the invention of the art of printing, is certain; nevertheless, everyone knows that in every city there are many people who support themselves and their families solely by writing. Incessant driving of the pen over the paper causes intense fatigue in the hand and the whole arm because of the continuous and almost tonic strain on the muscles and tendons, which in course of time result in failure of power in the right hand. An acquaintance of mine, a notary by profession, still living, used to spend his whole life continuously engaged in writing, and he made a good deal of money by it; first he began to complain of intense fatigue in the whole arm, but no remedy could remove this, and finally the whole right arm became completely paralyzed. In order to offset this infirmity he began to train himself to write with his left hand, but it was not very long before it too was attacked by the same malady. But what tortures these workers most acutely is the intense and incessant application of the mind, for in such work as this the whole brain, its nerves and fibers, must be kept constantly on the stretch; hence ensues loss of tonus. From this results headaches, heavy colds, sore throats and fluxes to the eyes keeping them fixed on the paper; those who suffer most from these ailments are book-keepers and accountants, as they are called, for example those who hire themselves to work for merchants. Also we must reckon in the same category the private secretaries of princes. For weakness of the arm and right hand it will be beneficial to apply rubbing but it must be rather gentle and done with oil of sweet almonds to which you can add a small portion of brandy to strengthen the part affected. In winter they must take care not to let the hands grow numb from excessive cold, so they must be protected by good thick gloves."

Table 3: Bell's description of "writer's cramp" [5].

"The paralyzed scrivener, though he cannot write, can amuse himself in his garden, can shoot, and cut his meat at the dinner table, indeed he can do almost anything he likes, except earn his daily bread as a scribbler... Upon your early correct diagnosis may depend the health and happiness of your subject. If you mistake its real nature and regard it as a sign of incipient softening of the brain, a mistake I have known to occur, you may destroy the happiness of your patient ..."

## Peripheral upper limb affliction

### Poore

In 1872, Poore described "writer's cramp" as "a remarkable example of a rare and difficult condition" [9]. Three years later, he presented 42 patients, whose main symptom was loss of power and compromised writing ability. He interpreted these constraints as fatigue in the muscles or their antagonists [10]. Poore

identified various subgroups of the impairment in an analysis of 75 cases out of which most had "neuralgic" or "real" "writer's cramp" [11]. Their symptoms were the same as those previously described by Solly [7]. Characteristic features were neuralgic pain or fatigue after use of the arm, numbness and a cramped sensation in the hand, and difficulty in finding a comfortable arm-position at night. There was soreness and excessive irritability (tested by Faradism) of the radial nerve and the extensor muscles, weakened supination, pain over the distal radius by passive flexion of the thumb and hyperesthesia on the dorsal hand. More than half of his patients had soreness of the brachial plexus [11]. Less frequent were "adynamic" cases with paresis related to peripheral nerve lesions, "spasmodic" cases where clear spasms caused the writing difficulties, "degenerative" cases where upper limb tremor impaired writing, and an "anomalous" group with other manifestations [11]. In a larger series of patients published 10 years later, Poore excluded paralytic and degenerative cases. His main physical findings were still tenderness of nerves, especially the median nerve, changed irritability in certain muscles with Faraday's examination, and tremor [12]. He interpreted the findings as manifestations of muscle fatigue due to a peripheral neuromuscular disorder. The subsequent examination of 21 pianists who could not play due to upper limb pain consistently revealed nerve trunk tenderness and extremely painful extension of the sore nerves. He assigned simple stretching for each of the three main forearm nerves, the median, radial and ulnar nerve [13].

### Robinson

The discovery in 1831 of electromagnetic induction permitted the development of the telegraph, which was in global use in 1860. Pressing the Morse-key lever in a series of rapid wrist flexions/extensions with pronated palm turns the electric connection on/off and transmits the code's dots and dashes. The cumulative effect of work led to telegraphist's cramp, which was described in 1880 [14]. The loss of control over the movements required for use of the Morse key was interpreted as muscular weakness. Suggested risk factors included the hours and style of work, the workers' seniority, the type of Morse key, as well as personal characteristics of the telegraphists such as neurasthenia or nervous instability. The incapacity could be limited to a single letter, selective for specific muscular activities or associated with general muscular weakness and spasm [15]. Upper limb pain and dropping things were common. Some patients had no pain, except when a particular letter or group of letters were to be sent. The telegraphist who could not send a particular letter felt panic. The receiving telegraphist could not read the sent message, and this contributed to the patient's painful mental state and constraints in coping with the work. Many telegraphists had to be released from their work although remission of mild cases could

occur after long-term relief. The improved condition was regarded as due to improved general health, to the absence of fear of cramp, the elimination of anxiety and better temperamental stability [14]. Robinson described different types of telegraphist's cramp – identical with the previous descriptions of “writer's cramp” – such as a gradually increasing muscle fatigue in some workers and a sudden spasmodic form in others [16]. Telegraphist's cramp disappeared when teleprinters replaced the Morse key, and with the increasing use of the telephone [14].

## Bridge

In 1920, Bridge described occupational cramp in the English cotton industry, in which twistors join the warp treads by twisting the two warp ends between the flexed left thumb and index finger. In this way, a worker could join 2000 ends per hour. The twistors' symptoms were pain, cramps and loss of power in the affected muscles. Half of the patients had thenar atrophy [17].

## Beard

In an analysis of 125 patients the neurologist Beard (Figure 2) emphasized that cramp was but one of a large number of symptoms and that cramp was mostly absent. He found no two cases alike. Symptoms included muscular fatigue, aching and burning pain, tremor, numbness and tingling, abnormal sensitiveness to touch and cold and actual paralysis. He also mentioned nervousness and noted that this was rarer in the southern states of USA. Beard regarded “writer's cramp” and comparable “overuse” upper limb conditions as primarily peripheral neuromuscular disorders. He noted however, that they may secondarily and rarely become central and general. Symptoms could spread to include larger parts of the limb and even the opposite arm. He found workers of strong constitution more in risk than nervous and delicate persons and that the condition was easier relieved and cured in the latter than when it occurred in strong persons. It was less likely to occur in those who did original work, as authors, journalists, or composers, than in workers with routine tasks such as clerks and book-keepers. He emphasized that “writer's cramp” can be curable in early stages but absolutely hopeless when symptoms have persisted for years [18].

## Duchenne and Reynolds

Duchenne distinguished between occupational spasm and occupational paralysis. He described neuralgic pain in a pianist, in which muscular dysfunction was not observed [19]. Reynolds perceived paralysis and cramp as reflecting the same condition but emphasized the presence of involuntary spastic symptoms as when an attempt is made to carry out a special and complicated movement. He found similar conditions among artists, musicians, seamstresses, blacksmiths and milking maids



Figure 2: George M. Beard

[20]. Flint characterized “writer's cramp” as a “local spasmodic affection” [21].

The prevailing theory in the nineteenth century was that the occupational “overuse” upper limb disorders were caused by “physical fatigue” in muscle or nerve tissue although structural changes in the central or peripheral nervous system or muscle were not identified.

Clinical descriptions of work-related upper limb disorders in the 1860s and '70s were followed by joint efforts towards an etiologic classification. In the late nineteenth century the spasmodic, paralytic and neuralgic syndromes became increasingly perceived as various clinical manifestations of a dysfunction of the central nervous system. Theories of a peripheral neuromuscular dysfunction lost support.

## Gowers – development of the central hypothesis

William Gowers (Figure 3), a prominent neurologist towards the end of the 19<sup>th</sup> century, termed “writer's cramp” and comparable work-related disorders as occupational neuroses. By this concept he understood conditions, which according to the existing knowledge lack underlying lesions of the nervous system. At the time neurosis was thus a technically correct description for an apparently neurological condition of unknown etiology (although Sigmund Freud used the same term to describe psychological concepts).

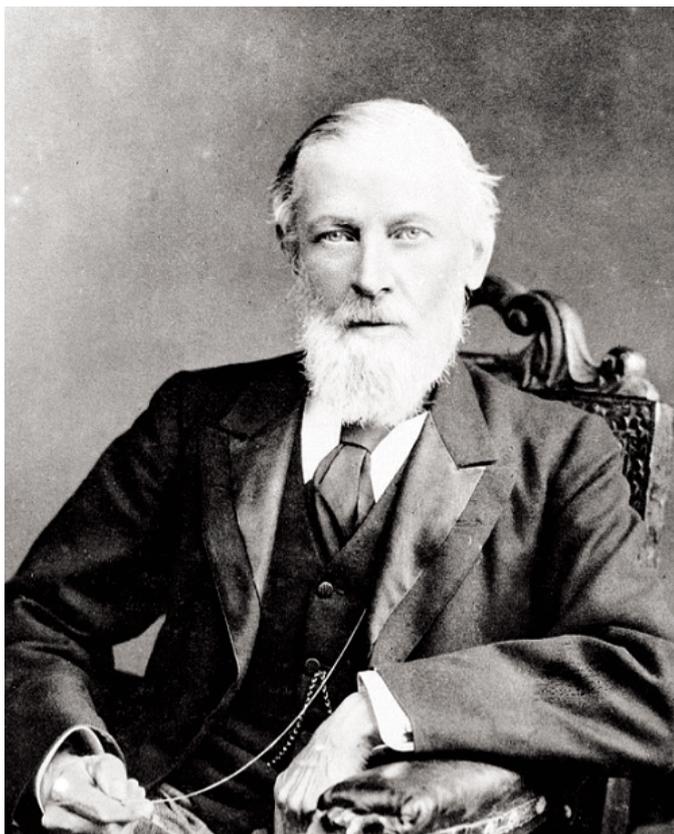


Figure 3: William Gowers.

Gowers stated that symptoms were 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 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 that showed signs and symptoms of pathological anxiety. He also noted their frequent concerns about work and family issues and about heavy burdens of responsibility [1].

Gowers made a distinction between a motor (spasmodic) and sensory (neuralgic) form of "writer's cramp" both of which could be present in isolation or in combination. Motor disturbances were most important but he regarded paresis as rare although he did describe local weakness to an extent reminiscent of spasms. Unable to identify any underlying lesion in the nervous system he interpreted both forms by a derangement in the cerebral centers that controlled the specific motor function. He tried to incorporate Poore's observations [12] by explaining secondary pathological changes in the sensory nerves as a result of pain that was central in its emergence

and in the motor nerves consequent to centrally induced spasm, respectively. He supported this theory by the fact that the opposite hand was often involved, and because the pain could subsequently spread to other areas [1].

Although Gowers emphasized that the loss of sensibility and significant muscle atrophy could occur [1] he never classified "writer's cramp" as a kind of brachial neuritis or neuralgia. He insisted on a central hypothesis for "occupational neuroses" [22] but acknowledged that a local disorder or lesion of the limb could trigger the condition. He described the development as progressive (Table 4) and regarded the volume of work as less important for the development than the way of writing, including how the pen was held and moved. Two out of various modes of writing were regarded as particularly risky: One with the hand supported on the little finger and the pen being moved with the fingers and thumb. The other was with the hand supported at the wrist and with lateral movement by wrist abduction. Forearm support was beneficial while he regarded free arm movements from the shoulder with fingers hardly moving as protective against the disease. He perceived bad workplace ergonomics as a particular risk factor. Cassirer and Oppenheim also understood "occupational neuroses" as conditions, which according to the existing knowledge lack any pathological anatomical background such as underlying lesions of the nervous system [23, 24]. They emphasized the relation of symptoms to neurasthenia, persistent emotions, functional conditions, or exhaustion neuroses based in the centers in the central nervous system that control the associative muscle activities required for writing [24].

### **Critical opponents to the concepts of "occupational neuroses"**

Other researchers were critical of the importance of worries and nervousness for the development of "writer's cramp". Reynolds found psychological factors of minor etiological importance [20]. Paul emphasized the variations in somatic symptomatology and in the same way as Beard [18] suggested that frequent symptoms should receive more attention. Total 177 out of his 200 patients with work-related upper limb symptoms had pain in muscles and joints or the adjoining regions. Numbness and weakness were present in 38 and 26 patients, respectively. No patients had cramp. Paul argued that the symptoms perceived as occupational neuroses were caused by local injuries from repeated impacts and tensions affecting muscles and nerves near tendons, fascia and joints [25].

Norstrom described chronic muscular tension in the neck as a frequent cause of headaches. He found similar indurated areas in the upper limb muscles in 34 out of 47 patients with spasmodic "writer's cramp". The symptoms were interpreted as secondary to chronic muscle infiltrations ("myositis"), or –in the absence of these infiltrations– to disturbed coordination.

Table 4: Gowers' description of "writer's cramp" [1].

"After writing for some time the patient experiences something unusual about his writing; the pen does not move quite as he intended to do; a stroke now and again is irregular, extends too high or too low; a slight involuntary movement causes an unintended mark. He finds that he is grasping the pen too tightly, and cannot help doing so; that the fingers do not keep in their accustomed place; and the first finger has a tendency to slip off the pen, so that this gets between the first and second finger. He endeavours to mend matters by taking a firmer hold, but this seems to increase the difficulty, and he finds that he writes slowly, as if a weight was attached to the hand. The hand feels strangely tired, and an aching pain in the finger or thumb or the first metacarpal bone, or in the wrist/forearm, makes it still more difficult for him to go on writing. These symptoms may continue, with only slight impairment of the power of writing, for weeks or months, but they occur after writing for a shorter time: they increase in degree, and now and then there is distinct spasm, which cannot be controlled. The first finger or the thumb tends to be flexed at the middle joint, so that its tip moves up the pen, or, less commonly, the fingers become extended across the pen. The characters of the writing become still more irregular, the down-strokes become too thick, the point of the pen may be driven through the paper, and in its irregular of form and force the writing resembles that done in a jolting carriage."

"Rarely the chief spasm is in the fourth finger, or in the third and fourth fingers, and pain may be felt in the long flexors of these fingers, and the ulnar flexors of the wrist. Sometimes the whole hand seems to get stiff, and its movements slow; in such cases there is a tendency for the letters that are formed to become smaller and smaller as the writing proceeds, until they are illegible. As the spasm increases in degree, it extends in range, and involves more of the muscles of the forearm. There is a tendency for the wrist to become flexed or extended, or supinated, and in the effort to prevent the disturbing movements, the opponens contract strongly, until at last all the muscles of the forearm may be in such energetic spasm as to render movement of the pen impossible. Various devices are at first employed to counteract the spasm. The mode of holding the pen is changed; it is held between the first two fingers or fixed in a piece of cork, which is grasped by the hand, and the movements in writing are effected by the upper arm or the patient fixes the right hand by the help of the other, for instance, some fingers of the left hand between the two last fingers of the right. For a time these devices give a little help somewhat, but the spasm gradually increases in degree, and overcome the fixing help, or it spreads to the muscles of the upper arm".

"The best and only free method is to write from the upper arm and shoulder, with not fixation in the arm; the forearm, wrist and little finger rest on the table, so as to take some of the weight of the limb from the shoulder muscles, but both wrist and forearm move along the table as the writing progresses from left to right. In this way the pen can be held lightly; very little of the movement is affected by the small muscles of the hand; the fingers scarcely alter their position, except when a stroke is carried far above or below the line; and even for this, a movement of the fingers is not always necessary. No one style can be considered free unless it is easy to write a whole line across the page of foolscap without once breaking contact between the pen and the paper".

The recommended treatment was massage [26] –a therapeutic modality that is still widely applied in spite of little evidence of effect [27, 28]. He did, however, concede that a neurotic reaction manner with established disease could worsen the prognosis [26].

Although Paul emphasized a peripheral neurogenic explanation for the pain in "writer's cramp", he also thought that excessive use of force could cause myolytic changes leading to upper limb pain [25].

Monell described the progression of disease. Starting from a mainly reversible condition with acute muscle fatigue, a persistent state of heaviness, fatigue, paresis, and pain could develop. The symptoms could be relieved by rest, but would return when resuming work. He regarded "writer's cramp" as a state of chronic fatigue due to muscle injury by toxic metabolites [29]. Without regeneration in rest periods, the continued overload would compromise the nutrition of the tissues and lead to less effective rest [30].

The symptoms reported by Poore [11–13] and Beard [18] were recognized by Dana [31] who reported 100 workers with upper limb symptoms relating to their occupation, e.g. clerks, stenographers, typists, telegraphers, pressers, ironers, tailors, and musicians. Most of Dana's patients had occupational neuralgias and neuritis. Only 23 patients had "real" occupational cramp, which accompanied brachialgia or neuritis in six patients. Dana explained "writer's cramp" as a peripheral disorder with neuritis that caused major arm pain accompanied by nerve trunk tenderness and peripheral tingling and numbness [32]. Windscheid suggested that cervical and brachial plexopathy in e.g., telegraphists, blacksmiths, locksmiths, file cutters, turners, and tailors could be prevented by sparing the arm from overstrain [33].

The identification by neurologists in the late nineteenth century of discrete upper limb peripheral nerve dysfunction led to the suggestion that the symptoms could be due to minor peripheral nerve lesions that could develop secondary to occupational exposures. Spaans [34] proposed that the many conditions referred to as "occupational neuroses" could well be due to lesions of the peripheral nerves. Oppenheim conceded that a combination of occupational neuroses with neuritis might occur [24].

At the beginning of the twentieth century, there was an increasing awareness in the scientific community that "writer's cramp" and other "occupational neuroses" could represent several diseases. Following Paul's article on the subject [25], it became accepted that the symptoms in many patients had a peripheral neurological background. Ramsay Hunt noted descriptions from different professions of more than fifty different types of occupational neuritis. He recommended these conditions to be kept separate from "occupational neuroses of central etiology", which were rather characterized by "real" cramp or spasm [35]. The neuralgic kind of occupational "overuse" syndrome was recognized by many in the beginning of the twentieth century [1, 11, 14, 21, 23, 24,

32, 36], but the condition required definitive sensory or motor outcomes to be termed occupational neuritis [23]. Three categories of patients with occupational neuritis were identified in 1912:

1. Patients with repeated direct trauma to the nerves during their work
2. Patients exposed to toxic substances, e.g., lead
3. Patients in which with “fatigue” of certain muscle groups has induced nerve lesions (this group probably including entrapment neuropathies) [37].

## DISCUSSION

Looking at the historical descriptions of symptoms and signs there was a variable picture which may well encompass various diseases. However, it is clear that many of the described symptoms and findings may well reflect peripheral nerve afflictions of the upper limb. Biological plausibility supports the described interpretations about pathogenesis (Table 5).

### Symptoms

Muscle cramp can be related to muscle overuse and strain such as holding a position for a prolonged period of time, as well as to inadequate blood supply and nerve compression. Despite the designation “writer’s cramp”, Ramazzini [4], Bell [5], Solly [7, 8], Poore [11–13] and

Beard [18] emphasized the motor and sensory symptoms such as fatigue, pareses, paresthesia and pain rather than cramp. Poore did describe a cramped sensation in the hand and difficulty in finding a comfortable arm-position at night [11]. None of Paul’s patients had cramp [25]. On the other hand, cramp was regarded as typical among cotton-twisters [17] and Norstrom regarded cramp as secondary to chronic myositis, and—in the absence of muscular infiltrations—as secondary to disturbed coordination [26].

A spasm is a sudden, involuntary contraction of a single muscle or a group of muscles. Most commonly spasm refers to involuntary muscle activity including muscle contractions due to abnormal nerve stimulation, or abnormal activity of the muscle itself. Poore found the writing difficulties in “spasmodic” “writer’s cramp” to be rarely caused by clear spasms [11]. Most of Beards patients had spasm, which, however, was neither the most important nor a necessary symptom [18]. Reynolds noted involuntary spastic symptoms [20] and flint a “local spasmodic affection” [21]. Spasm was also described in telegraphists [15]. Gowers described stiffness and slow movements and found that grasping the pen too tightly caused spasm in the third and fourth fingers, and the occurrence of pain, tiredness and heaviness in the long flexor of these fingers, and of the ulnar flexors of the wrist. With increased spasm, additional forearm muscles were involved and the wrist tended to become flexed, extended, or supinated [1]. Other neurologists perceived spasmodic “writer’s cramp” as a focal dystonia [38, 39], which according to the current general opinion is the

Table 5: Symptoms and findings in the ancient descriptions of work-related “overuse” upper limb disorder suggesting peripheral neuropathy

Authors [References]	
<b>Symptoms</b>	
Muscle cramp	Ramazzini [4], Bell [5], Solly [7, 8], Poore [11–13], Bridge [17], Norstrom [26]
Spasm	Poore [11], Beard [18], Reynolds [20], Flint [21], Hunter [47], Gowers [1]
Pain	Solly [7], Hunter [47], Poore [11–13], Bridge [17], Paul [25], Beard [18], Monell [29], Dana [31], Gowers [14].
Weakness	Ramazzini [4], Solly [7], Poore [10, 11], Hunter [47], Bridge [17], Paul [25], Gowers [14], Monell [29, 30],
Sensory symptoms	Solly [7], Poore [11], Gowers [14], Dana [32], Paul [25], Hunter [47]
Dropping things	Hunter [47]
Contralateral spread	Beard [18], Gowers [1]
<b>Findings</b>	
Mechanical allodynia of nerve trunks	Poore [11–13], Dana [32], Gowers [1]
Weakness – paresis	Beard [18], Gowers [1], Poore [11], Monell [29, 30],
Sensory dysfunction	Ramazzini [4], Solly [7], Poore [11].

result of a basal ganglia and/or sensorimotor cortex malfunction in the brain.

Pain is the main symptom in upper limb “overuse” conditions. The severe and persistent burning and neuralgic pains described by Solly [7, 11, 19] represent characteristic features of neuropathy. Wrist or more generalized pain was common in telegraphists [15] as well as in cotton-twisters [17]. The vast majority of Paul’s patients had pain especially in muscles and joints or adjoining regions [25]. The neuralgic pain described by Poore [11–13] and Beard [18] was also evident in Dana’s patients, most of which had neuralgias and only few had cramp [31].

Weakness may have various meanings to patients and can be understood as fatigue manifested as tiredness, lack of energy, weariness or exhaustion from labor, exertion and stress. In contrast to fatigue, true motor weakness refers to loss of muscle strength involving the motor pathways [40]. Ramazzini described intense fatigue and reduced upper limb power, which could develop into complete paralysis [4]. Fatigue and weakness was also reported by Solly [7] and Poore [10], who specifically noted impaired supination [11]. Definite paralysis was also described by Beard [18]. Muscular weakness was common in telegraphists [15] and cotton-twisters [17] but only identified in 1/8 of Paul’s patients [25]. Gowers emphasized spastic, tremulous or neuralgic motor disturbances with associated tiredness. He conceded that fine movements could be affected in progressed disease with reduced muscular strength but found atrophy to be rare [14]. Monell regarded the progression of “writer’s cramp” as an irreversible condition with fatigue, heaviness, and paresis of the limb [29, 30].

Solly mentioned sensory symptoms such as a feeling of cold, peripheral tingling, stinging, and numbness [7]. Most of Gowers’ patients had pronounced sensory symptoms such as progressing and spreading finger-paresthesia when writing [14]. Paresthesia was present in approximately 1/5 of Paul’s patients [25]. According to Dana, paresthesia developed typically secondary to neuritis [32]. Dropping things was reported as common among telegraphists [15].

Gowers observed that half of the patients, who started to write with the contralateral hand, developed symmetric symptoms that tended to progress even more rapidly. According to current knowledge, his interpretation of this as psychological rather than somatic [14] was a misconception [41].

## Signs

The historical descriptions of occupational “overuse” conditions report many findings that indicate the character of these upper limb disorders. While muscular weakness was a frequently reported symptom in the historical literature, pareses were rarely demonstrated at the physical examination. Today it is acknowledged that a careful neurological examination of the distribution

of weakness will provide diagnostic assistance and help in distinguishing between diseases of muscles, neuromuscular junction, peripheral nerve, or central nervous system. In addition, the quantification of the severity of weakness allows for tracking over time [40].

When testing a muscle, which is weak due to impaired innervation, there is a tendency for the emergence of tremor. Initially, Poore interpreted tremor as related to a degenerative neurological disease [11] but later he perceived tremor as a major physical findings in the “degenerative” type of “writer’s cramp” [12].

Atrophy reflecting muscular weakness was rarely described in the historical descriptions [1, 14], but thenar atrophy was present in half of the patients with cotton-twisters cramp [17]. This is one of the few indications of a specific condition that could explain symptoms in a defined group of workers –in this case suggesting that cotton-twisters were prone to median nerve affliction. In general, atrophy is a late sign occurring after long standing nerve affliction.

Poore described brachial plexus and radial nerve soreness. He also demonstrated mechanical nerve-allodynia by provoking pain over the distal radius with passive flexion of the thumb [11]. Tenderness of nerves, especially the median nerve was later described as key findings at the physical examination that could reflect a peripheral neuromuscular disorder [12]. Nerve trunk soreness was also found in pianists who could no longer play due to upper limb pain, and extension of the sore nerves was extremely painful. Poore’s assignment of stretching the median, radial and ulnar nerves [13] is currently forming the basic in the concepts of adverse nervous tension [42]. Dana interpreted tenderness along the course of the nerves as reflecting a peripheral nerve affliction [32]. Even Gowers noted pain extending along the nerves’ course along with fatigue and local tenderness [14]. Nerve trunk pain is now an accepted component of peripheral neuropathy [43]. The peripheral nerves can and should be examined by assessing the presence of abnormal soreness—mechanical allodynia— whereas Tinel phenomena rather reflect nerve regeneration [44] –not only at the carpal tunnel but at any location along any nerve.

Faradic current, which is an interrupted low-frequency direct electrical current, can induce alternate contraction and relaxation of voluntary muscles by stimulation through their nerves. Gowers found that electrical reactions were not or only slightly disturbed [14] while other clinicians [12] emphasized positive electrical findings.

Sensory abnormalities at the physical examination such as paresthesia, dysesthesia and sensory deficits indicate a disorder of the peripheral nerves or spinal cord. The distribution of sensory abnormalities may provide clues to localize the disorder [40]. The classical descriptions of occupational “overuse” syndromes, however, rarely specify the locations of sensory disturbances although Solly reported hyperesthesia on

the dorsal hand in patients with “writer’s cramp” [11]. Cold allodynia as described already by Ramazzini [4] and Beard [18] is a typical feature of nerve afflictions.

Since the descriptions by Norstrom [26] of indurations of the upper limb muscles interpreted as chronic myositis, [26] many researchers and clinicians regard myogenic infiltrations as frequent sources of pain, in particular in the neck and shoulder region. The so-called myofascial pain syndrome is a condition in which chronic myofascial pain develops secondary to occupational repetitive motions. Pressure on sensitive points in muscles (trigger points) causes referred pain in seemingly unrelated parts of the body. There is controversy as to whether myofascial pain syndromes represent a specific pathology or are merely a term to describe a clinical condition that provides patients with the reassurance that their symptoms are real [45].

## The confusion continues

The historical descriptions of work-related upper limb “overuse” conditions differ significantly with regard to symptoms and signs. Cramp, spasm, pain, weakness and sensory symptoms such as paresthesia were commonly reported. The physical examination, in contrast, revealed fewer consistent findings. There were, however, regular reports of muscular weakness, tremor, or atrophy, as well as sensory symptoms such as paresthesia, dysaesthesia, hyperesthesia or hypoesthesia and cold allodynia. Nerve trunk allodynia consequent to nerve palpation or adverse positioning of the limb was the most consistent finding in the historical descriptions. Taken together, the historical descriptions of patients resemble the features that characterize many of the patients with work-related upper limb complaints that we see today [46, 47].

## CONCLUSION

Looking at the symptoms and signs in the ancient descriptions of work-related “overuse” disorders of the upper limb it is remarkable how they remind of the features that characterize the patients with upper limb disorders that we see in the context of clinical occupational medicine today. Taking into account that the major proportion of work-related upper limb disorders cannot currently be diagnostically classified according to current consensus criteria, we might learn from the ancient descriptions. Looking at symptoms and findings, upper limb peripheral neuropathies are likely to be responsible for a major proportion of occupational upper limb “overuse” syndromes in the historical literature as well as for many of today’s work-related upper limb disorders. We should therefore pay attention to the historical descriptions of symptoms and findings that suggest an involvement of the peripheral nerves. The physical neurological examination was less developed at the time, and consequently there were confusions and disagreements regarding the diagnostic

interpretation. The current physical neurological examination, however, is a strong diagnostic tool, which can contribute significantly to the diagnostic assessment. I, therefore, emphasize the importance of consequently and systematically studying the physical neurological qualities of the upper limb in our patients.

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## 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

The Author declare no conflict of interest.

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