



Writer's cramp: is focal dystonia the best explanation?

Michael H Pritchard

Consultant Rheumatologist, Cardiff, Wales, UK (retired, University Hospital of Wales, Cardiff)

Correspondence to: Michael H Pritchard. Email: mhpritchard41@gmail.com

Summary

Often considered no more than an historical curiosity, writer's cramp remains an important disability in the workplace and the mechanism, which has puzzled the best medical minds for generations, remains contentious. A remarkable range of hypotheses has been put forward to try and explain a disability which periodically reached epidemic and economically worrying levels, but in the end medical opinion has accepted the explanation put forward by neurologists Sheehy and Marsden in 1983 that this was caused by a form of focal dystonia. However, the majority of the historical descriptions of writer's cramp do not fit the classical parameters of focal dystonia and are more accurately described as a progressive forearm muscle fatigue. Today's keyboard operators continue to complain of symptoms identical to their clerical forebears demonstrating that this is a problem which has evolved but not disappeared; this has the paradoxical advantage that modern research techniques enable this complaint to be revisited. The result shows that two varieties of writer's cramp have always existed and while focal dystonia remains a valid explanation for a minority of cases, the much more common fatigue-based complaint is better explained by chronic compartment syndrome of the forearm.

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Introduction

Have there always been two forms of writer's Cramp? There are some flaws in Sheehy and Marsden's paper,¹ but focal dystonia is a perfectly valid mechanism. Clear accounts over many decades have described the sudden spasm of the hand or fingers that followed certain actions connected with writing, especially where the writer was in a stressful situation. The reaction resembles a conditioned reflex in a Pavlovian sense. However, all musculoskeletal specialists recognize chronic forearm pain in the workplace but do not call it writer's cramp. It is called Repetitive Strain Injury,² Overuse Syndrome,³ Diffuse Forearm Pain⁴ and many other generally indecisive terms, yet a careful history shows that the chronic, diffuse, activity-related discomfort and weakness in the forearm experienced by today's keyboard operators is identical to the descriptions given by 19th century clerks, whose doctors would have called it writer's cramp.

Methods

To study this condition comprehensively, this paper has combined historical review, to take advantage of the excellent descriptions of writer's cramp in the medical literature, with contemporary research from a number of disparate sources including ourselves. If a fatigue-based writing-related complaint remains alive and well, although concealed under alternative names, an opportunity exists to investigate it using technology not available a century ago, and if today's signs and symptoms are identical to the descriptions of the Victorian clerks' complaint, it follows that it is not only possible to treat the condition rationally for the first time but also to extrapolate any modern explanation to the clerks, scribes and telegraphists of the past. Logically, people using the same muscles under similar circumstances risk suffering from the same complaints even though they are living a century later. Technology changes but forearms do not and it is not to what you do, but the way that you do it which counts.

Many aspects of writer's cramp can be explained using modern techniques, but these have never been either pulled together into a coherent whole or put into their historical context but when collated and combined with the data from the Victorian authors, a convincing basis for a muscle fatigue form of writer's cramp emerges.

Historical review

In 1700, Bernardino Ramazzini wrote⁵:

The Diseases of Persons incident to this work arise from three Causes; firstly, constant sitting, secondly the perpetual motion of the hand in the same manner, and thirdly the attention and application of the mind.

Constant writing considerably fatigues the hand and whole arm on account of the almost continual and almost tense Tension of the muscles and tendons.

Victorian surgeon Samuel Solly had described the problem in bank clerks (scriveners),⁶ and there is no better description:

When Scriveners' palsy first commences the victim of it only feels its direful influence after a hard day's work. He regards it only as a sign of fatigue and as he starts fresh the next morning attaches no importance to it as the first attack of a serious enemy; but in a short time he is obliged to rest earlier in the day and hails his early dinner with joy, as giving him some respite from the fangs of his tormentor'

He reported neurological disturbances accompanying the muscle symptoms:

There is generally a feeling of weariness and slight pins and needles but not usually numbness. Virchow says: Ordinarily no disturbances of the sensibilities show themselves, except an undefined feeling of strain and fatigue; sometimes a pressure in that part of the muscles; a painful drawing of the nerves in the direction of the trunk; a cold feeling in the whole arm.

This is writer's cramp as everyone understood it, but this is not the description of focal dystonia.

The essential point is that the majority of the clinical descriptions of writer's cramp at this time describe a progressive increase in aching fatigue in the forearm muscles on prolonged usage accompanied by vague paraesthesiae and burning pain, not the sudden cessation of movement or spasm seen in focal dystonia, and today's keyboard operators describe the same symptoms. The situation is not that writer's cramp is never due to focal dystonia but that two forms have always existed, albeit with different visibilities in different eras. There is nothing new about this; G-B Duchenne in 1855 clearly recognized two versions of the complaint among his patients, to which he gave separate names: *spasme fonctionnel* (occupational spasm) equivalent to focal dystonia and *paralysie musculaire fonctionnelle* (occupational muscle paralysis) which describes the other.⁷ Unfortunately, no-one else for the next century was so perceptive.

This dilemma is illustrated by the work of two London physicians in the 1870s, William Gowers and Vivian Poore. Both wrote extensively about writer's cramp, Gowers as a neurologist⁸ insisting on a dystonia-based central nervous system disorder even though he was also an enthusiast for improving writing techniques, while Poore, who was a general physician, considered that peripheral muscle fatigue was the cause.⁹ Both were aware of each other's work but each called his own observations writer's cramp. Why they were unable to agree that there might be two conditions is unclear; perhaps to have one inexplicable medical disorder was bad enough, to have two was unacceptable.

Victorian writers,^{6,8,10} while describing patients with both forms, considered they were variants of the same complaint, one the precursor of the other. The prevailing theory was that the cause was a failure of a 'writing centre' in the brain or upper spinal cord even though no such centre had ever been identified and it was not clear what had gone wrong with it. This 'one form' approach was made even more untenable by the fact that work-related arm pain was occurring in most occupations, not just writing, but Gowers confidently predicted that one (neurological) aetiology would, *mutatis mutandis*, fit all cases; in other words, not only explaining the two clinical

forms of writer's cramp itself but even the arm pains of manual workers such as blacksmiths and milkmaids who did no writing at all.⁸

Writer's cramp has tended to occur in epidemics. In the 1830s, the rise in prevalence among scribes and clerks was attributed to the change from quill pens to steel nibs. Whether true or not, crucially writers did not have to stop periodically to sharpen a steel nib as was required with a quill, an action which gave the writer a brief but therapeutic rest break from what would otherwise be continuous writing. There is no reason why such a change should affect focal dystonia since the only change was the actual technique of writing, but cutting out even short breaks would certainly affect a condition based on muscular fatigue.

The diagnostic confusion deteriorated further in the 1890s with the introduction of potential psychological causes for Medically Unexplained Symptoms such as writer's cramp, as now the patient's personality and mental state were investigated rather than the details of the physical complaints. Gowers had described writer's cramp as an 'Occupational Neurosis',⁸ a technically correct description at the time for an apparently neurological condition of unknown origin, but Sigmund Freud was also using the term 'neurosis' to describe his own psychological concepts. As a result of this nominal confusion, writer's cramp, with no physical explanation, could from then on be conveniently reclassified as a psychological disorder.

At the same time, the increasingly widespread use of the typewriter was making clerks and scribes, the main victims of the disorder, redundant anyway so it began to disappear from the office scene and medical interest declined. Typists never got writer's (or typist's) cramp, a fact which was recognized by contemporary clinicians but which they could not explain. However, telegraphy was the new growth industry and telegraphists suffered in large numbers. No technical breaks were required; the work was continuous, static and stressful thus fulfilling Ramazzini's work description precisely. Typing on the other hand required frequent breaks from keystrokes at the end of each line and page so the work was never continuous.

The General Post Office, who ran the telegraph service, commissioned a large scale

survey in 1912.¹¹ The authors again identified more than one clinical presentation of telegraphist's cramp (which had been specifically shown¹² to be clinically identical to writer's cramp in scribes) and described both a gradual onset of worsening muscle fatigue in some workers and a sudden spasmodic form in others. Their explanation was that the work itself was irrelevant but could be explained on a eugenic basis; the problem would go away if trainees were screened properly to eliminate those of a 'neurotic' tendency. A second GPO report in the 1920s¹³ tried to establish the same outcome, but even though they found that telegraphists were no more neurotic than anyone else, again the work was not blamed.

The third epidemic of writer's cramp was not recognized as such. This was in Australia and again coincided with the introduction of new technology, this time the electronic keyboard, which by allowing continuous working eliminated the short, frequent but beneficial breaks required by the typewriter. Occupational physician David Ferguson was at a diagnostic watershed. He wrote two papers in 1971, one on Morse code telegraphists blaming their 'neurotic personalities'¹⁴ but simultaneously describing a group of factory workers who, while suffering from the same upper limb complaints, were not neurotic but responding adversely to their stressful working conditions.¹⁵

The resulting epidemic of arm pain at work spawned the term 'Repetitive Strain Injury' but clinical definitions were irrelevant as any arm pain would do for compensation. In psychologist Yolande Lucire's opinion,¹⁶ it was all mass hysteria egged on by venal doctors, greedy union leaders, dishonest lawyers and compliant politicians. Ferguson, however, did analyse his cases later and found that once the obvious arm complaints (i.e. tennis elbow, tendonitis, etc.) had been eliminated there remained a group which puzzled him. He wrote

the majority of cases of repetition injury were not localised syndromes, (i.e. such as tennis elbow which was easily recognised) but of a more diffuse order, apparently of muscles. This disorder whose symptoms are those of aching, weakness and tenderness of muscles...but had been confused with and

may coexist with occupational cramp which should be considered a variant of repetition injury.¹⁷

Although reference was made to Ramazzini, focal dystonia was never mentioned but there seems little doubt that many of those caught up in the Australian epidemic were describing symptoms identical to the muscular form of writer's cramp.

Contemporary investigations

On the assumption that historical descriptions suggest two types of writer's cramp exist, one due to a form of focal dystonia (although without identifying a mechanism), what is the background of the other? We have investigated this disorder ourselves in two papers,^{18,19} which have identified two pathological abnormalities in patients complaining of progressive aching and disabling forearm pain on prolonged and continuous usage. In the first paper, using Doppler ultrasound on the radial artery at the wrist at rest and after a specific 2-min repetitive gripping exercise (20 patients, 19 controls aged 18–55, mostly keyboard workers), we found that the pain coincided with failure to mount a normal vasodilatory response to exertion in the affected forearm.¹⁸ This abnormality did not occur in either the asymptomatic arm or in controls where normal vasodilatation followed the exercise. Since the working muscle was unable to call on an adequate blood supply for sustained activity the inevitable result was a degree of relative ischaemia, a convincing mechanism for pain and disability.

This relative ischaemia has been shown by other authors using other techniques, for example by ¹³³Xenon clearance²⁰ and a form of intermittent claudication was also predicted by several Victorian authors²¹: failure of the normal vasodilatory response to exertion, even if the mechanism is unclear, is a simple and consistent explanation for this observation.

In the second paper¹⁹ using different patients but with the same complaint and work history (30 patients, 24 women and 6 men, aged 30–59), we found by direct measurement of intra-compartment pressure (Kodiag, Braun) that there was a rapid increase in pressure in the forearm extensor compartment on exertion

accompanied by pain and paraesthesiae, the typical symptoms of chronic compartment syndrome. The symptoms were identical to both those of the first group, in whom ischaemia had been identified, and the classic descriptions of writer's cramp.

The muscles involved are those of the extensor forearm compartment, not the flexor, demonstrating the vulnerability of fixed wrist and hand position for long periods, whether keying, writing or typing. Poore predicted this²² when he drew attention to the importance of the strain on muscles that maintain a fixed position, as opposed to those involved in repetitive movement. It is therefore a 'static strain injury', a term also used by Hazleman,²³ not a repetitive one. Modern technology supports this prediction: T2-weighted MRI images indicate that the main oedema accumulation on usage (the presumed cause of the increase in pressure) occurs in the extensor carpi radialis muscles,²⁴ the chief maintainers of forearm and wrist posture whatever the fingers are doing.

Chronic (exertional) compartment syndrome, CeCS (exertional since the symptoms are specifically usage related²⁵), is a poorly understood condition best recognized in the lower legs of runners where it is known as shin splints.²⁶ In this condition, intra-compartmental pressure rises rapidly and painfully on exertion, usually from a raised baseline and settles slowly over about 15–30 min after resting. The pressure is high enough to inhibit function inside the compartment, but not to cause permanent damage to the muscle. American vascular surgeon William Turnipseed²⁷ described such complaints as 'atypical claudication' which he resolved in almost every case by compartment decompression. The symptoms on prolonged exertion in the arms are identical to those in the legs, but while upper limb CeCS is occasionally described in case reports in the medical literature, it has usually been associated with strenuous sports, including motocross and bodybuilding, not repetitive work.²⁸

It cannot be coincidence that these patients, when working, should be complaining of both CeCS and muscle ischaemia simultaneously so there must be a connection.

Vasodilation in response to exercise is activated via the sympathetic nervous system and therefore the loss of vasodilation on exertion suggests that

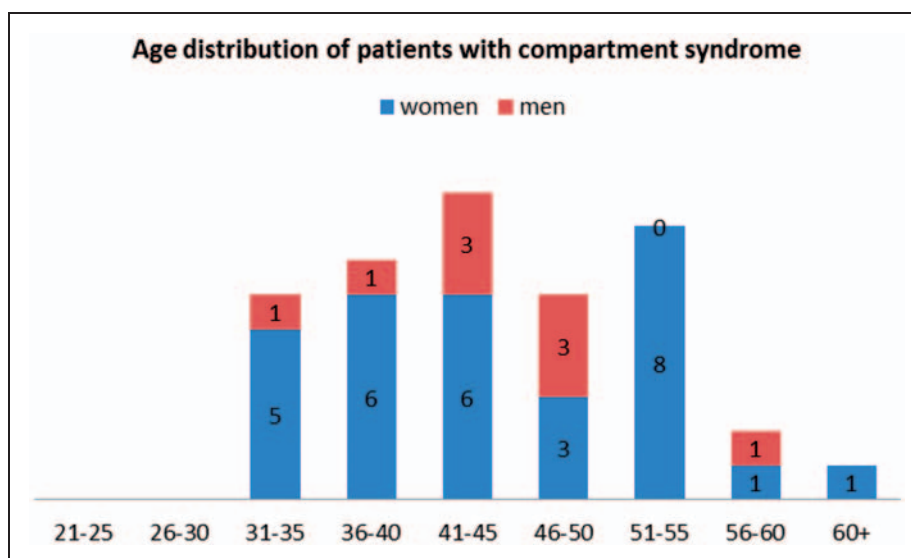


Figure 1. Demographic data relating to patients undergoing decompression for compartment syndrome.

this is an autonomic (autonomic nervous system, ANS) response failure. Chronic increase in pressure inside a muscle compartment will eventually affect all adjacent nerve trunks, including those of the ANS, and we have found in occasional cases that CeCS can occur as a consequence of nerve injury at the radial tunnel and that this recovers after decompression. It is of interest that Edmundsson *et al.*²⁹ found that patients with chronic diabetes and autonomic neuropathy in the lower leg complaining of intermittent claudication were often found to have CeCS rather than arteriopathy and their symptoms resolved on compartment decompression, again demonstrating a connection between autonomic dysfunction and compartment syndrome.

If this is true then compartment decompression, by releasing the pressure build-up and the resulting compression neuropathy, should cure writer's cramp. As a continuation of our second paper, we have decompressed 46 arms in 39 patients (30 women and 9 men, aged 30–64 all of whom were either unable to work or having increasing difficulties) and reviewed the results two to four years later. The result was described as good or excellent by 75%, enabling most to be able to return to work, and the rest complained of some on-going neuropathic pain, possibly due to

the prolonged compression of the radial nerve prior to decompression.

Demographically, writer's cramp in its modern form is uncommon but appears to occur sporadically and unpredictably across the whole working population doing rapid and unremitting repetitive work (Figure 1). It is not possible either to predict the risk or to estimate the prevalence since there are too many variables in job description, working pressure, ergonomics and so on as well as the low level of diagnosis. Also, younger sufferers are more likely to change their job than complain. Remarkably, the only study with a cohesive and defined population was the GPO 1912 survey of telegraphist's cramp,¹¹ in one office finding 13 cases out of 148 operatives, i.e. around 8%, half being work-threatening.

Whether there is a preceding abnormality of the autonomic responses in the muscular form of writer's cramp remains unproven, but some susceptibility is likely. Possibly individual response to stress is as important as overuse since although it commonly follows changes in working practice specifically designed to increase output by reducing natural breaks and is commonly reported after a period of intense work, stress, either domestic or work-related, has been observed for decades in many sufferers. Once

started, however, the chronicity can be explained by coupling compartment pressure with failure of vascular response via a self-perpetuating compression neuropathy.

Results and management

CeCS and a failure of vasodilatation on exertion, possibly attributable to ANS dysfunction, are not only demonstrable abnormalities in the muscular form of writer's cramp, but can be shown to be directly interconnected via a compression neuropathy and can account for all the signs and symptoms in this condition. There is no doubt that early recognition of the symptoms is essential for simple treatment as once it has become self-perpetuating compartment decompression is the only option although long-term neuropathic injury may by then be present. This is a simple day case procedure.

Conclusion

Writer's cramp is alive and well but renamed so often that the original term has largely disappeared and the perception of the complaint with it. There is room for two writer's cramps, both work-related but proceeding down separate pathological paths for reasons which, while not clear, suggest different individual susceptibilities and responses when subjected to specific physical and psychological stresses. Focal dystonia is a perfectly valid mechanism but only explains a minority of cases. The majority are much more in keeping with a diagnosis of chronic compartment syndrome, an easily treated condition once recognized in today's workplace and, given the clinical similarities, applicable to the historical situation as well.

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