Overuse Syndrome and the Overuse Concept

Hunter J.H. Fry

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Discussion Papers on the Pathology of Work-Related Neck and Upper Limb Disorders and the Implications for Diagnosis and Treatment

Gabriele Bammer
editor and coordinator

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INTRODUCTION

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This is the third in a series of working papers designed to promote discussion about the pathological basis of work-related neck and upper limb disorders. The ultimate aim of the papers is to allow some resolution about pathology to be achieved, so that diagnoses can be more accurate and treatments more useful.

The first two papers in this series "The neurogenic hypothesis of RSI" by John Quintner and Bob Elvey (1) and "The relevance of concepts of hyperalgesia to "RSI"" by Milton Cohen, Jesus Arroyo and David Champion (2) concentrated on the nervous system. This paper "Overuse syndrome and the overuse concept" by Hunter Fry examines pathology in muscles. There are 12 commentaries which add supporting information and/or probe potential weaknesses and these are then responded to by Hunter Fry.

Both the commentaries and Hunter Fry's response highlight some of the conflicts which still influence discussion of these disorders. The damaging effects of the unresolved debates, particularly about whether or not these disorders are 'real' are well recognised but just beginning to be documented. Reid and co-workers (3) point out that for individuals with these disorders not being believed is a contributor to symptoms becoming chronic. They suggest that affected people may visit doctor after doctor and describe this as "a pilgrimage in search of moral affirmation" (p 602). In many cases the pilgrimage is not initiated by the affected person, but is requested by the insurance company or the lawyers handling the compensation case. The conflicting diagnoses and advice and the all-too-common assaults on personal integrity may significantly exacerbate existing problems. The process by which this may occur has been outlined by Lloyd and Stagoll (4).

Conflicting advice and disbelief may also impair the rehabilitation process. The Australian Capital Territory RSI Support Group has undertaken one of the first studies in this area (5). Thirty people, chosen to represent a range of rehabilitation experiences, were interviewed and 28 were reinterviewed about 9 months later.

The rehabilitation process was clearly shown to involve the mobilisation of a complex array of resources: government and/or private agencies, doctors, unions, case managers, rehabilitation providers, supervisors and co-workers. They often interrelated in adversarial ways. Despite, or perhaps because of, the range of players, the people with the disorders found it difficult to obtain the information they needed to successfully manage their condition. This ranged from information about eligibility for benefits such as home help, rights to change rehabilitation providers and eligibility for vocational training.
The study showed that people who were able to take personal control of the rehabilitation process and who were able to find meaningful work were most likely to be successfully reintegrated into the workplace. Supervisors played an important role in enabling people with restrictions and limited capacities to be accommodated rather than further marginalised. None of the participants were 'cured' and all had to restrict activities in their private lives.

Improved understanding of these conditions will enable those affected to have more confidence in the professional advice they are given and in consequently taking control of their lives and being successfully reintegrated into the workforce.

I am grateful to Mr Fry and to the commentators for generously devoting time to this project. Further contributions to the debate, either commentaries on this paper or expositions of other hypotheses are invited.

REFERENCES


OVERUSE SYNDROME AND THE OVERUSE CONCEPT

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All body tissues and indeed all materials can under certain circumstances be taken beyond their limits with some sort of injurious change resulting. I have found the most useful definition of overuse syndrome as it affects the upper limbs (of, for instance, musicians) to be “a condition of pain and loss of function in muscles and ligaments, which have been subjected to excessive or unaccustomed use.”

One of the great problems about this common condition is that, like many spinal problems, there may be no definite objective physical signs which incontrovertibly demonstrate the cause for the symptoms. In overuse syndrome the physical findings are semi-objective and must rest partly on the credibility of the patient. In highly motivated groups such as musicians, the condition can be studied with the least amount of distortion by pressure from psycho-social factors produced by the compensation and welfare systems. Some musicians have free access to these (orchestral players), others do not (conservatorium students, school-aged musicians, freelancers). This is the group I know best.

Overuse syndrome as it is now known fits well into a family of disorders involving muscular and ligamentous overuse. While a great deal is not known about this condition, there are nevertheless historical, clinical, histological, biological and therapeutic aspects which support this hypothesis.

I have personally examined over two thousand musicians with overuse syndrome and at least three thousand others with the same disorder from other hand-use-intensive work such as keyboard operators.

HISTORICAL

In the second half of the last century there was a considerable amount of writing on use-related pain from hand-use-intensive activities and muscular failure associated with it. This extensive literature as it relates to musicians has been explored and reviewed in recent times.

Telegraphers (morse code operators) and writers were the other two groups which were quite extensively studied. The descriptive literature was so good that despite the heterogeneity amongst the cases reported, there was a clear view that a large group of these people suffered from muscular overuse, which responded only to rest. Painless loss of muscle function was also reported in terms of weakness and loss of coordination (now called focal dystonia).

Poore, Gowers and Waller all carried out basic investigations on affected muscles. In some of the cases there was active debate as to whether the lesion was actually in the muscles or the central nervous system. Waller believed the neuromuscular junction to be the weakest link in the neuromuscular claim and likely to fail first in prolonged physical activity.

Terms used for this disorder were ‘cramp’ which referred most often to cases where there was painless loss of muscle function but also apparently to the painful condition as well. The term occupational neurosis (a neurological disorder) was also frequently used. This was prior to the days of Sigmund Freud who coined the term ‘psychoneurosis’, which itself ultimately became shortened to neurosis! The term overuse was used a great deal and the
historical review quotes much of the writing accepting the premise of a muscular overuse condition. This is impressive simply because the medical and social context was so different, yet some of these descriptions could have been written in present time.

The modern equivalent of this is the painful musculo-ligamentous overuse without any loss of coordination at all at one end of the spectrum and the focal dystonia (or painless incoordination syndrome) at the other. There appear to be many points along a spectrum where a proportion of the people with a painful condition now have dystonic features, i.e. musicians report impairment of technique.10

The parallels between past times and today hold good.

CLINICAL EVIDENCE

In most serious cases of overuse of the upper extremity, the position of pain is matched by tender muscle groups and ligaments in the appropriate area. In the less severe cases or the very chronic cases, the match may not be so good. Nevertheless, tenderness, albeit a semi-objective physical sign, is usually present in highly motivated groups such as musicians.7 There are, however, a number of other clinical states of overuse of this type.

The classical ballet student would be lucky to survive much beyond 25 years of age as a performing dancer. While the acute injuries suffered by these artists are obviously well known, it is generally acknowledged that it is the chronic muscular overuse which is the main problem. To quote Sammarco, in an editorial: "Most dance injuries are of the chronic and overuse category with only a small percentage resulting from direct or indirect acute trauma."26

The treadle sewing machine was described as causing lower limb overuse last century.6 I have seen two patients who used these machines for extended hours and who developed painful quadriceps muscles with some tenderness persisting long after the work was given up. Another example of muscular overuse in the lower limbs is seen in marathon runners, who appear to suffer quite extensive damage on their long runs. Creatinine kinase enzymes originating from muscles are regularly found in the urine, and muscle biopsy studies clearly show muscle fibre necrosis. Fortunately the muscles have a remarkable capacity to regenerate.

During the 1939-45 war, military recruits were hurriedly put into service. Some were less than fit and often put into an extensive exercise program. A number of them developed not only painful muscles but gross myoglobinuria demonstrating clearly the muscle injury due to this unaccustomed exercise.12

Sports medicine has developed a great deal in the last twenty years and musculo-ligamentous overuse, common amongst sportspeople was described in some detail in a whole issue of the Sports Clinics of North America16. A recent editorial in the Lancet summarised the clinical as well as the histological features of strenuous and exhaustive exercise.5

Dalakis at the National Institute of Health has become very interested in the overuse which appears to develop in the surviving muscular fibres after childhood poliomyelitis. These fibres appear to be rather prone to develop overuse later on. This group of people has also been studied clinically by Jacqueline Perry at Ranchos Los Amigos Hospital in Downey, California. She has collected an impressive array of clinical evidence. The overuse can be disabling for these people who may have to return to knee braces and the like.

'Iron pumpers' are another group who frequently develop some degree of muscular overuse. This group is well known to sports medicine practitioners but I am not aware of any formal documentation of the condition in this group. There also appear to be no muscle biopsy studies.

All the groups above appear to recover with the appropriate degree of rest with the exception of dancers. If the dancers rest they lose their technique so it is unfortunately a 'catch 22' situation for them.
HISTOLOGICAL EVIDENCE

There is now quite a mass of histological studies indicating changes in the muscles with overuse. These investigations began with Nelson Howard in 1939. Recently the first dorsal interosseous muscle has been studied in keyboard operators and this showed the following significant changes when compared with controls: loss of type 2 fibres, type 1 fibre grouping, significant fibre hypertrophy, mitochondrial lesions and a number of ultra microscopic findings including fingerprint inclusions, paracrystalline inclusion bodies, z band streaming and others.

A Lancet editorial detailed a number of histological studies as did the Clinics in Sports Medicine of North America and Dalakis. There is really no biopsy material available for ligamentous strain. Clinically this is usually attended by tenderness of the involved structure but its biopsy would be likely to make the patient clinically worse so it is not justifiable. Nevertheless, chronic ligamentous strain is frequently seen in orthopaedic practice.

THE CONCEPT OF SELF INJURY

Can muscle damage itself by its own contractions? Sometimes there appears to be strong resistance to this proposition.

Acute muscle tears appear frequently. In addition, prolonged sub-maximal contractions such as seen in the marathon runner can certainly be attended by damage caused by the muscle action itself. In acute and chronic injurious change to muscle therefore, the main factor operating is the contraction of the muscle itself rather than some external agent.

DISCUSSION

The historical, clinical and histological evidence allows overuse syndrome such as seen in musicians and others with hand-use-intense activity to fit into the family of disorders referred to above. There are a number of other 'theories' which must be addressed if the musculo-ligamentous overuse hypothesis is to hold good.

Psychogenic Theory

Though this is sometimes propounded by rheumatologists and some others, I am aware of no evidence for it and no cures have resulted from psychotherapy based on this proposition.

Malingering

This is a conscious process of exaggerating, distorting and fabricating for gain. In compensation medicine there will always be a small proportion of people who will mangle but this does not mean that others do not suffer from a real disorder. Taking this argument one step further it would be very difficult to explain a whole family of muscular overuse disorders by malingering.

Hysteria

This is an unconscious process whereby the patient's anxiety is converted into a physical symptom with no underlying physical basis. There are occasional devotees of this theory. Not only is this hypothesis unattractive but no evidence at all of which I am aware has been put forward to support it. Removal of the factor causing the conversion and appropriate psychotherapy should cure the symptoms in true hysterical conversion. This certainly does not occur with overuse syndrome.

Spinal Nerve Roots

This is an area which has attracted some attention recently on the basis of some 'signs' which cannot be duplicated by others. The bony spine is accessible, frequently operated on and its disease processes are often well documented by direct inspection and even cured by operation. Until or unless practitioners are able to demonstrate these physical signs in a reproducible way, the hypothesis must lack conviction. The anatomical parts are too well known.
Treatment based on this proposition should produce cure of symptoms and to date I am not aware of any reported success.

Central Nervous System

I have referred to the two main ‘pure’ presentations of overuse which form the two ends of the spectrum between painless focal dystonia and painful musculo-ligamentous overuse. The pure form of focal dystonia was previously thought by some to resemble hysterical paralysis. Using single fibre electromyography it has been shown incontrovertably to have an organic basis. Abnormal impulses reaching the muscles through the nerves were demonstrated by Cohen and Hallett. For this to happen central nervous dysfunction must be occurring, although the reason for it is not clear. Neither is it clear why approximately 25% of musicians who suffer from painful ligamentous overuse also show some dystonic features. Certain aspects of the histological study of the first dorsal interosseous muscle suggest some central nervous system involvement, as do some clinical aspects (e.g. both upper limbs developing symptoms at the same time).

One might speculate on a number of mechanisms, such as a virus related to poliomyelitis affecting the central nervous system; or there may be a role played by herbicides, pesticides, heavy metals or other toxic substances which lower the threshold of overuse or affect the central nervous system directly. Much research needs to be done in this area.

RESPONSE TO TREATMENT

The only therapy which appears to have any success in cure of symptoms is that of reducing the level of physical activity of the affected parts to a degree that all pain causing activities are avoided. Muscle and joint movements must be continued to preserve normal function. This was noted last century and quotations from the writings of those doctors made it very clear that rest was the only treatment which helped. To quote Lockwood, “Rest is the cornerstone of treatment”. Smythies describing cure of the condition in his own hand by rest, describes the failure and indeed ill-effects of other treatment. My own figures in musician patients indicate the success of this treatment and the hypothesis on which it is based.

CONCLUSIONS

While there is much which is still not understood about this overuse disorder, the hypothesis of actual musculo-ligamentous overuse is well supported by historical, clinical and biological evidence and the response to treatment based on that hypothesis.

It is likely that within the next five years or so, scientifically based studies will be carried out in the United States which will throw some further light on the features of this disabling disorder. In that country, the Department of Labour and Industry and the National Institute for Occupational Safety and Health have decided that, notwithstanding the information gap, they are prepared to spend a great deal of money on preventative programs and have rejected Dr Nortin Hadler's contention that the disorder is a non-problem basically caused by recognising something that is not there.

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23. Poore GV. An analysis of 93 cases of writers cramp and impaired writing power; making, with 75 cases previously reported a total of 168 cases. *Medico-Chirurgical Trans.* 1887; 52: 301-333.


The exact nature of the hypothesis is unclear. Hunter Fry appears to suggest that the condition is one affecting muscle and ligamentous tissue, as a result of extreme or unaccustomed use.

Under "Clinical Evidence" we feel a more precise definition of stages would be valuable. The terms used "serious cases", "less severe cases" and "very chronic cases" lack definition. The information provided in this section is too broad and anecdotal and specific clinical evidence alluded to is not detailed.

The inclusion of two ideas in the paragraph commencing "The treadle sewing machine..." is confusing and the logic is further complicated by the statement that "muscles have a remarkable capacity to regenerate". An acute muscle strain in a marathon runner is likely to regenerate quickly, but this is not so in the case of the sewing machine operator who may have been performing this activity over a long period of time.

Again in the paragraph beginning "Sports medicine..." Hunter Fry does not expand sufficiently on the clinical differences between an acute strain and a long term overuse injury.

It would be valuable if the studies supporting rest as a curative technique were detailed and appropriate supporting evidence provided. The basis for excluding dancers from the category of groups that will respond to rest is unclear, as many other occupational and recreational groups would also run the risk of loss of technique with rest programmes.

In the section on "Histological Evidence" Hunter Fry briefly mentions his work with Xenia Dennett (Dennett & Fry, 1988), which is interesting, but he fails to refer to the more recent and extensive biopsy studies by Awad (Awad, 1990) on the presence of fibrocytic nodules in affected muscles.

The section headed "The Concept of Self Injury" needs further expansion and clarification to warrant inclusion in this paper.

In the section "Discussion", the exact composition of the "family of disorders referred to above" is not stated and is unclear. The discussion of the various theories is cursory and needs to be detailed and expanded further. The theory of malingering is an area where Hunter Fry's wide experience with musicians could be introduced and argued to good effect.

Under the subheading "Spinal Nerve Roots" Fry inaccurately dismisses Quintner & Elvey's signs as not being able to be duplicated by others. Both authors have written extensively on this technique since 1983. The examination technique has been taught at both undergraduate and postgraduate levels and is well documented. Additionally there appears to be some confusion in this paragraph between the concept of spinal nerve roots and the boney spine.

The subsection "Central Nervous System" would benefit from further clarification and expansion. Many mechanisms of injury are mentioned without sufficient discussion.
The section "Response to Treatment" presents the ideal platform for Mr Fry to detail his extensive experience in the management of one particular group of RSI sufferers (ie. musicians). Information on rest routines, average duration of symptoms and rehabilitation would be most interesting. For example, how does Mr Fry maintain the critical balance between curative rest and skill maintenance in his patients? A greater proportion of this paper could have been devoted to treatment methods and thus have provided a valuable forum for future discussion and research.

REFERENCES


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Dr Fry has narrowed the focus on overuse syndrome by restricting his review mainly to muscle disorder with some comments on ligamentous dysfunction. The essence of his theory is that repetitive use of muscles has been shown to be associated with structural and functional abnormalities, also strained ligaments are common clinical problems, therefore the broader spectrum of clinical overuse disorder is attributable to such primary musculo-ligamentous pathogenesis. The main factor operating is considered to be contraction of the muscle itself, rather than some external agent.

Much of the background discussion, including the historical perspective and the diversity of overuse sufferers such as musicians and dancers, is of considerable interest. Dr Fry’s contributions in these areas have enriched the debate.

It is a reasonable proposition, supported by physiological data, that sustained high level repetitive isokinetic or isometric muscle contractions ie muscle "overuse" (although this has not been adequately defined) may cause morphological and functional abnormalities of muscle. There is no problem with Dr Fry’s paper to this point. However it does not necessarily follow that such abnormalities explain the full clinical picture and the time course of disorder (refractory cervicobrachialgia in the occupational context).

The primary musculo-ligamentous hypothesis does not adequately and convincingly explain the majority of the observations concerning the diffuse neck and arm disorder which I and my colleagues have observed in many hundreds of patients from diverse occupations. I refer to the wide spread of pain; the hyperalgesia which is elicited from muscles, tendons, ligaments, vertebrae, joint capsules, subcutaneous tissue and skin; the hyperpathia; the allodyniae and the increased mechanosensitivity of peripheral nerves; cutaneous sensory impairment; the sympathetic nerve dysfunction; the range of dystonic phenomena; and the remarkable chronicity of many of these cases.

Marathon runners do not develop diffuse and persistent hyperalgesia in the lower limbs in the manner that is observed from repetitive use of the upper limbs. In the context of the neck and arm syndromes (diffuse cervico-
brachalgia), it is possible that small diameter nociceptive afferents from muscle contribute to the hyperalgesic state. However this is a relatively weak area of knowledge in pain physiology, and there is little evidence in support of the concept.

Recent studies from St. Vincent's Hospital have shown that semiselective block of polymodal nociceptor fibres in the brachial plexus by infusion of low concentrations of bupivicaine can arrest the cervicobrachialgia. If the block is continued for several days, the hyperalgesic state reduces, and sustained relief (up to 3-6 months to date) can be observed. This experience is difficult to reconcile with the musculo-ligamentous hypothesis of Fry, unless sensitised and activated small diameter nociceptive afferents from musculo-ligamentous tissue are contributing very substantially to the hyperalgesic state. Whatever the peripheral nociceptor sensitization and activation that occurs in these conditions, our observations and evidence point strongly to activated nociception with secondary motor and sympathetic reflex responses in the central nervous system.1-3

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The main problem I find with Dr Fry's paper is that he fails to articulate precisely what is his hypothesis. He talks about an "overuse syndrome" and the "overuse concept": the first acknowledges a phenomenon but the second involves a teleological argument. The fundamental question, namely what is the pathophysiology of chronic overuse has not been addressed.

HISTORICAL

This section recognises that the present problems have been well identified in the past literature. A spectrum of disorders at the phenomenological level has been reported, ranging from the neuralgic to the paralytic forms of occupational "neurosis". Dr Fry is correct in identifying the latter as focal dystonia. The basic studies quoted failed to identify a pathophysiology for either.

CLINICAL

Dr Fry correctly identifies the phenomenon of hyperalgiesia (which he calls "tenderness", as do most people) although he acknowledges that hyperalgiesia is not always concordant with complaints. This of course impinges on the
very difficult area of clinical psychophysics where the read-out ("pain") is influenced by more than just nociception. This section then describes certain examples of acute muscle "overuse", all situations in which damage to muscle is able to be demonstrated. That these groups of acute problems tend to recover with the appropriate degree of rest or "non-use" does not necessarily imply that overuse is a mechanism. That is, one must be aware of the teleological fallacy in that argument.

HISTOLOGICAL EVIDENCE

This short section refers to five studies which report histological changes in muscles; there are no data on the histology of "ligamentous strain". I have not reviewed the muscle studies critically myself.

DISCUSSION

The unstated hypothesis which I infer is that the origin of the pain is muscle and ligament and that the cause is "overuse". Not only does the teleological problem again emerge but also it is now accompanied by the problem of tautology, namely that any pain occurring in a context of "overuse" is to be not only explicable by a homogeneous mechanism but also attributable to "overuse". It is somewhat alarming to read that "psychogenic theory" is "sometimes propounded by rheumatologists" when some of us have taken pains to distance ourselves from that. There is no evidence for primary psychogenesis of this problem. The same comments apply to malingering and hysteria. Dr Fry dismisses the Quintner/Elvey hypothesis all too simply: the "signs" have been recognised by others. That treatment based on concepts of peripheral nociception has not produced "cure" must prompt re-evaluation of those concepts, in particular to consider the possibility of a peripheral or central neuropathic pathogenesis. It is difficult to demonstrate and measure psychophysical phenomena but that should not detract from hypotheses based thereon.

CONCLUSION

Dr Fry's hypothesis suffers from the problems of teleology and tautology. It may be well-based on the historical, clinical and histological evidence and the response to treatment based on that hypothesis but, as Medawar has pointed out, the evidence on which a hypothesis is formulated cannot be taken as evidence for its veracity.
are greatly increased. Vague terminology and entrenched views combine to confuse the dispassionate clinical observer. The two schools of "real illness" and "malingers" shape up to each other in the courtroom, provide a field day for the lawyers, but do little to help the patients. 

How can we get closer to resolving these issues? After all, many of the "sufferers" are poorly paid manual workers undertaking soul destroying repetitive tasks. They have much to gain from compensation and a respite from occupational boredom. They also are likely to slip inexorably into chronicity.

The search for truth in the patient's symptoms is thus frequently dogged by the knowledge that they would be advantaged by stopping work and indeed their intellectual powers may preclude a good history in the first place. The position of professional and student musicians is in marked contrast. Here are a group of intelligent, highly motivated individuals who have everything to lose if they develop a disabling pain and yet their 'work' involves repetitive movements and abnormal postures.

In my view, there is no doubt that musicians develop symptom-sign complexes of overuse syndrome indistinguishable from those of less gifted and less motivated workers undertaking work practices which involve comparable hand-arm movements. Ballet dancers develop equally disabling pain and tenderness but in the lower limbs. Again, here is another group for whom changing employment would be a disaster.

If we "believe" the disease to be genuine in dedicated artists, should we not approach the lowly manual worker with more open mindedness? After all, the overuse is genuine and obvious in both groups. The belief in the effects is being driven by our preconceptions of patients and of their motivations. Dr Fry has done us all a service by his work into the occupational hazards of the performing arts. It does not provide the incontrovertible proof we all seek but should make those who espouse the malingering theory to sleep less easily in their beds.

There are a number of different views of the causes underlying "RSI" and it is interesting to note that authors can come to radically different conclusions about basically similar groups of patients.

Reviewers come to their task biassed by their own training, experience, and individual values and judgements. As I will be criticizing the views offered, it should help to put this criticism into perspective if I present, briefly, my own view of RSI at the outset: RSI is a label used to refer to many different conditions. In the Australian epidemic most complainants had a combination of some initial minor muscular problem with a large psychological problem they were talked into by the prevailing public climate. They were persuaded to believe they had structural injuries in their arms or neck by people with strong ideas on the pathology—whether clinical or political—or people searching for a sense of mission in their lives, especially journalists.

The writer assumes that the subjects he deals with are rational, like the mythical "rational man" of economic theorists. In the case of musicians, Fry ignores the frustrated ambition of those who want to be much better than their physique or talent allows. In the case of factory and office workers writers ignore the mind-set of "them and us". Reality is different. A large part of the population believes in Flying Saucers, iridology, infallibility of computers, Stalinism, Total Allergy Syndrome, and fairies at the bottom of the garden.
Hunter Fry trained as a plastic surgeon. Earlier in his career he was on the staff of one of the leading teaching hospitals in Melbourne. Now he writes and speaks prolifically on the overuse problems of musicians.

“All body tissues and indeed all materials can under certain circumstances be taken beyond their limits with some sort of injurious change resulting”, he writes. The same taking beyond limits presumably applies to interpretations of observations. The question in the case of Hunter Fry's assertions is whether the conclusions he has come to about the nature of overuse injury are sustainable scientifically.

His high point was his paper in the Lancet (Dennett & Fry, 1988). His co-author was a left-handed histopathologist who herself suffered from writers' cramp. In this study muscle samples from the thumb cleft were taken from 8 controls and 21 patients affected on one side, and 8 affected on both sides. Various subtle changes in “injured” muscle were described. Their significance was presented as a statistical inference, and hotly debated by opponents of the injury theory in later issues of the journal.

Fry says "There is really no biopsy material available for ligamentous strain”. Such material is indeed scarce, though some is available from specimens excised during surgery for “tennis elbow”. However the sufferers from RSI are not the marathon runners or the tennis champions, but nearly all people on wages paid by others. Fry discounts a psychogenic cause, because “no cures have resulted from psychotherapy”. Would that psychogenic conditions were so predictably curable! He brushes aside hysteria and malingering, and his dismissive approach to theories other than his own reaches a climax when he says, referring to spinal nerve roots, “Treatment based on this proposition should produce cure of symptoms and to date I am not aware of any reported success”. He should read the papers by Quintner and Elvey (one of which he actually quotes) to find large numbers of successes reported.

There are many problem areas with strong analogies to the disagreements over the “causes of RSI”. One is the causes of the common problem of back pain, one similarly bedevilled with questions of psychology, money for compensation, lawyers, and politics. A leading Australian researcher and anatomist (Bogduk 1992) says of various theories “... none of these concepts has been studied in this way with positive results. It seems easier to win arguments by saying the same thing often enough and loudly enough than to conduct appropriate studies, the guidelines for which are well established.”

The tragedy in what has passed for scientific debate over RSI is not only its poor quality and dogmatism, but damage to the credibility of scientific method. Time will solve this, not as long as the century or two after Galileo's killing, but still long in an age when such great technical advances are being made in so many other areas.

REFERENCES


A) EVIDENCE SUPPORTING THE HYPOTHESIS

The concept of overuse syndrome as the end point for uses of the hand and arm requiring repetitive movement is one that is congruent with my experience and those of other physicians.

B) EVIDENCE AGAINST THE HYPOTHESIS

I disagree that the problems of what Mr Fry calls “Overuse Syndrome” can be studied in its pure form in musicians. There are very many pressures on musicians, both from their peers and from their teachers, which put enormous pressures on them and distort the presentation of their condition.

Overuse syndrome does not fit into a family of disorders involving muscular and ligamentous overuse only, although in many patients early on in the development of the condition adverse mechanical tension signs are present and therefore there has to be evidence of involvement of the peripheral nervous system as well.

I disagree that chronic overuse conditions involving muscles are the cause of retirement of most ballet dancers. In my experience acute injuries and persistent pain therefrom are the commonest cause of medical retirement.

In his discussion on psychogenic theory, malingering and hysteria Mr Fry does not make the obvious but important point that people who are in pain and concerned about their future often become very disturbed.

In regard to response to treatment I strongly disagree that rest alone is the important modality of treatment. I would suggest that if rest alone does not produce complete relief of symptoms within a very few days then expert physiotherapy including adverse mechanical tension techniques (Butler, 1991) should be employed.

REFERENCE


INTRODUCTION

Fry opens by stating a truth which is self-evident - living tissues cannot adapt to physical forces which exceed their biological tolerance. The definition of overuse syndrome which follows is faulty: as well as being circular, it begs the question of underlying pathology. All upper limb and/or cervical pain and loss of function reported by those who perform repetitive manual work (e.g. musicians) originates, ipso facto, in musculo-ligamentous tissues which have been severely overused, i.e. “taken beyond their limits”. By presuming musculoskeletal injury, Fry can then state that overuse syndrome is a member of a family of musculoskeletal disorders caused by overuse.
The elements of the "overuse concept" as proposed by Fry are:

(i) that forces generated during manual work of a repetitive nature can overload and therefore injure upper limb and/or cervical musculo-ligamentous tissues.

(ii) that these patients usually do not exhibit objective physical signs of injury.

(iii) that the credibility of musician patients strengthens the hypothesis that musculo-ligamentous injury (without objective physical signs) is the cause of their upper limb pain and loss of function.

[NOTE: before considering the evidence offered in support of the overuse injury concept, the following summary has been prepared for the benefit of readers unfamiliar with the work of Fry16-20]

CLINICAL FEATURES OF OVERUSE SYNDROME AND ITS DIFFERENTIAL DIAGNOSIS

Symptoms

1. Prodromal symptoms may include upper limb weakness, stiffness, heaviness, pins and needles.17

2. Sensory: pain is the dominant symptom.16-18

   (i) Anatomical location: hand & wrist, forearm, elbow, shoulder, scapular region, or neck.

   (ii) Pattern: localised or spreading from muscle group to muscle group both proximally and distally. May spread to opposite side ("without much in the way of extra muscle work to account for it"). Can arise simultaneously in both arms.17

   (iii) Time course: pain worsens and becomes persistent unless the causal activity is modified or ceased.19,20


4. Fluctuating mental depression (sic).17

Examination findings

1. Alldynia (pain due to a stimulus that is not normally painful when applied elsewhere to the body): tenderness of muscles or joint ligaments found at the anatomical site(s) of presumed overuse injury.16-19

2. Swelling overlying region of tenderness.17

3. Patchy cutaneous sensory change - altered perceptions of hot and cold.17

4. Hyperpathia response (pain persisting many hours after physical examination).17

Grades of severity

Grade 1. Pain in one site on causal activity.

Grade 2. Pain in multiple sites on causal activity.

Grade 3. Pain with some other uses of the hand, tender structures demonstrable, may show pain at rest or loss of muscle function.

Grade 4. Pain with all uses of the hand, post activity pain with minor uses, pain at rest and at night, marked physical signs of tenderness, loss of motor function (loss of response or control), weakness.

Grade 5. Loss of capacity for use because of pain which is continuous, loss of muscle function, particularly weakness, gross physical signs.

Comment:

1. This classification infers that patients with Grades 3 to 5 severity of overuse syndrome, will have widespread pain.

2. From the outset, it can be seen that the features of overuse syndrome have the general characteristics of referred pain syndromes. 1,14

3. What is meant by "gross physical signs" is not explained.17,18

4. Hand cramps are not included in the symptomatology of overuse syndrome (see comments on differential diagnosis).
Differential diagnosis [Note: any of these conditions can exist "alongside" overuse syndrome]

1. Cervical radiculopathy
2. Thoracic outlet syndrome
3. Reflex sympathetic dystrophy
4. Ulnar nerve entrapment or neuritis at the elbow
5. Carpal tunnel syndrome
6. Other nerve entrapment syndromes
7. Tenosynovitis of the De Quervain's type
8. Flexor tenosynovitis
9. Osteoarthritis at the base of the thumb
10. Rheumatoid disease

Comment: In one study, Fry diagnosed overuse syndrome in 379 of 900 (42%) musicians (age range, 8 to 70 years) who were referred directly to him. In another study, he found this condition in 658 of 2000 (33%) musicians (age range, 11 to 78 years) referred to him for "occupational maladies." Neither study contained an estimate of the prevalence of these other conditions which Fry claimed may coexist with, but are distinguishable from, overuse syndrome. This omission is surprising, as these conditions are said by Fry to be either "aggravated or unmasked by music making." Also noteworthy, is that the number of musicians with focal dystonias does not appear in these studies. Elsewhere in this discussion paper, Fry estimates that approximately 25% of musicians who suffer from painful ligamentous overuse also show some dystonic features. Why did he not include them in his seminal studies?

EVIDENCE ADDUCED IN SUPPORT OF THE HYPOTHESIS

Historical

Fry has extensively reviewed the 19th century literature on work-related upper limb symptomatology. There was heterogeneity between the cases reported. However, for Fry to claim that the debate revolved solely around "whether the lesion was actually in the muscles or the central nervous system" is untrue. There was a body of opinion which considered that peripheral neural tissues were also involved. Indeed, Poore favoured a peripheral neuro-muscular origin for the symptoms of the conditions which he designated the "true" and "neuralgic" forms of writers' cramp. In these cases, he frequently observed both nerve trunk tenderness and altered (depressed or exalted) electrical excitability of muscles. Both phenomena were thought by him to be the consequence of muscular exertion. Poore reasoned that:

... alterations (especially depressions) of irritability occur in muscles which have been overworked, and notably in those which have been subjected to prolonged strain. This condition occurring in overworked muscles I have called "fatigue."... but whether it be primarily dependent upon change in the muscular fibre, the nerve end, the nerve trunk, the cord or the brain, there is not sufficient evidence to show (page 129).

Assuming (incorrectly) that Poore favoured "a primary condition of muscle failure", Fry claimed that Poore's next logical step would have been to biopsy the first dorsal interosseous muscle (because of its important role in pen prehension). To support this claim, Fry quoted selectively from Poore's important paper on the upper limb difficulties experienced by 21 professional pianists. Fry singled out Poore's reference to various vague conditions known as muscular rheumatism and myalgia in order to support his theory of overuse syndrome. He neglected to mention that, when discussing the "machinery involved in the delicate manipulations" performed by a pianist, Poore had emphasised the importance of a healthy state of the entire nervous system as well as of the muscles involved in piano playing. He recognised that difficulties in piano playing could arise from a variety of causes, "a fact which I believe to be true in the great majority of professional ailments." Finally, Fry did not mention the tenderness and intolerance to stretch of the different nerves of the forearm which Poore observed in most of his musician patients.
As correctly pointed out by Fry, Gowers did not favour a peripheral (neuromuscular) origin for the symptoms of the Occupation Neuroses. Contrary to Fry's assertion (p. 621), Gowers reported that in cases of the spasmodic form of writers' cramp, the electrical irritability of the nerves and muscles "may be perfectly normal, or present a slight change, increase or diminution, chiefly in cases that have lasted for some time."

The animal experimental research studies performed by Waller formed part of his enquiry into mechanisms of muscle fatigue and recovery. If Fry claims that failure at the neuromuscular junction is the primary physiological disturbance in overuse syndrome, he has yet to argue this case.

The features of overuse syndrome in musicians, as described by Fry, do not include focal dystonia. Nevertheless, he states elsewhere that a number of patients present to him with a primary incoordination as well as with pain. The focal dystonia is thought by him to be of central origin, the pain to be of peripheral (musculo-ligamentous) origin.

The spasmodic form of writers' cramp is regarded by neurologists as a focal motor disorder, with a close relation to dystonia. Pain and paraesthesiae which develop in some patients may be due to median nerve injury in the carpal tunnel as a result of dystonic spasms of the wrist. The historical view that focal dystonia and painful musculo-ligamentous overuse without any loss of coordination are at opposite ends of a clinical spectrum derives from Gowers, who was an avowed centralist. Fry's concept of overuse syndrome clearly aligns him with the 19th century peripheralists.

The clinical picture of overuse syndrome described by Fry is reminiscent of a localised form of the condition originally known as fibrositis, but now as fibromyalgia syndrome. The concept of fibrositis was initially put forward by Gowers.

We are thus compelled to regard lumbago in particular, and muscular rheumatism in general, as a form of inflammation of the fibrous tissue of the muscles ... We may conveniently follow the analogy of "cellulitis," and term it "fibrositis".

Gowers also described a condition which he called muscular fibrositis of the arm or brachial myalgia. It was, he stated, "met with in every degree of severity, and when intense it is a very terrible malady, distressing and prolonged ... Its great feature is pain in the muscles, not spontaneous, but induced by their contraction, or by sudden tension, however slight." This condition could affect any part of the arm although proximal muscles were said to be more commonly affected. Bilateral involvement could occur. The process of inflammation "may spread and reach the nerve sheaths of the arm, and in them set up a secondary brachial neuritis with all its consequences, tenderness of the nerves, pain along their course and in their distribution, oedema of the hand, and even muscular wasting and impaired sensation." In the 1930s and 1940s, chronic myalgia (fibrositis) was described in workers in many occupations, e.g. labourers, coal miners, gun layers and bulldozer operators. Brachial fibrositis was a condition to which telephone operators, laundry workers and typists were particularly liable.

The work of Kellgren on the characteristics of pain arising from deep structures led to the eventual abandonment of the fibrositis concept by English physicians. He showed that local pain evoked from saline injections into structures deep to the skin may be accompanied by referred pain. Referred pain is defined as pain falsely localised, and thus interpreted by the sufferer as arising from one deep tissue, when, in fact, it had originated in another. In his experiments, diffuse pain arising from muscle followed a spinal segmental pattern (sclerotomal, as distinct from dermatomal distribution) and was accompanied by referred tenderness of the deep structures. Feinstein et al., using similar methods to provoke pain from deep somatic tissues, found that areas of deep tenderness did not closely correspond with aching pain. They also noted that referred pain may not necessarily involve all muscles in the related spinal segment.
Kellgren and Hotchkiss found that pain which is worsened by cold and eased by heat is a feature of many painful conditions of the extremities (e.g. post-traumatic syndromes, painful nerve injuries, glomus tumours, many forms of arthritis and rheumatism). Such pain is accompanied by hyperalgesia of the affected deep tissues, a lowered threshold to mechanical stimuli, and "their spontaneous pain is also deep, and when severe it spreads proximally up the affected limb following the segmental pattern of deep pain."

It is important to view the concept of overuse syndrome against this historical background, noting in particular the work of Kellgren, out of which has developed our current understanding of the behaviour of deep pain arising from musculoskeletal structures.

Clinical evidence

1. Tenderness

Fry has stated that in the overuse syndrome, "Pain is commonly felt in the muscle groups of the hand, wrist, forearm (including common origins), also upper arm, shoulder and neck according to what (sic) structures have been overused ... In the less severe examples, pain occurs only with music-making and the overused structures are not really tender to palpation (my italics added). In the more severe examples, the overused structures are tender to palpation..."

In his opening remarks in this section of the discussion paper, he moves away from his previous opinion: "In the less severe or the very (sic) chronic cases, the match may not be so good. Nevertheless, tenderness, albeit a semi-objective physical sign, is usually present (my italics) in highly motivated groups such as musicians." The alleged nexus between pain, tenderness and musculo-ligamentous overuse is not properly supported by the reference quoted. This paper contains the following imprecise statements: "Physical signs are those of tenderness in the overused muscles and joint ligaments. Where there is pain in the hand and wrist the intrinsic muscles of the hand are usually tender to pressure, and the ligaments of the carpometacarpal joint of the thumb and wrist capsule, particularly on the radial side, will become painful when stretched. More proximal symptoms can be matched to tenderness in other muscle groups."

2. Other examples of overuse

(i) Ballet dancers: Fry's quotation from Sammarco refers to dance injuries (my italics) in the category of chronic overuse. Fry has not addressed the question as to whether or not dancers develop the lower limb equivalent of the overuse syndrome affecting the upper limb.

Poore considered that dancer's cramp was a symptom of fatigue induced by the "violent and prolonged exertion" necessary to maintain balance on the points of the toes. Oppenheim described dancers' cramp as a painful tonic contraction of the thigh muscles occurring in ballet dancers "in the beginning of dancing when gliding forward upon the toes." Another German neurologist, Cassirer, noted occupation spasms of the lower extremity to be quite rare, but to have been reported in professional dancers. Dana commented that under the name of ballet-dancers' cramp certain painful and paralytic troubles occurring in ballet dancers had been described. In his opinion, these were not co-ordinate problems but were either neuralgic or local (musculo-skeletal) strains.

(ii) Treadlers: Those whose occupation involves excessive work with their legs were known to be at risk of developing sciatica. Cassirer noted that rigidity, stiffness, fatigue and pain may develop in workmen who are compelled to use pedals. He recommended that these cases be carefully examined for evidence of occupation neuritis.

(iii) Marathon runners: The study on marathon runners performed by Hikida et al. was quoted by Fry to support his theory that direct muscle self-injury is the underlying cause of the upper limb overuse syndrome. This group showed that muscle fibre necrosis and inflammatory changes were present in biopsies of the gastrocnemius muscles of human marathon runners taken both before and up to 7 days after a marathon. They hypothesised that the inflammatory reaction may be a major factor in localized leg soreness for days following the marathon. They gave no
indication that marathon runners develop diffuse and chronic lower limb pain.

(iv) Military trainees: Fry17 quotes Greenberg and Arneson25 who studied 586 candidates for officer training. They found 23 cases of proved myoglobinuria associated with intense physical exertion (involving both upper and lower limb musculature). The salient clinical features included muscle weakness, pain and swelling. EMG changes (small amplitude motor units and fibrillation potentials) were found in the 4 patients so examined. Muscle biopsy, undertaken in three subjects, revealed changes interpreted as a myopathic lesion manifested by widespread regeneration and inflammation. Recovery (restoration of exercise tolerance) had taken place by the third month in all but one recruit. Graduated physical training with frequent rest periods was not associated with myoglobinuria. Although Fry16-21 has not documented rhabdomyolysis or myoglobinuria in sufferers of overuse syndrome, he infers that these phenomena support his hypothesis of muscle injury!

(v) The post-polio myelitis syndrome: It is difficult to understand why Fry put forward this poorly understood syndrome as clinical evidence supporting the overuse concept in those not afflicted with poliomyelitis. The various pathologic mechanisms that may underlie the late complications of polio have recently been summarised by Halstead.28 Musculoskeletal overuse in these patients is discussed in relation to the cardinal symptom of progressive late-onset weakness. Chronic mechanical strain on joints, ligaments and soft tissues that have been inadequately supported for 30 or more years undoubtedly do contribute to weakness. Theories to explain new motor unit dysfunction include persistence of the polio virus, immunological mechanisms, premature aging, spinal cord changes compromising motor neurones or motor neuron overwork.

3. Aching muscles after exercise

In a recent study, upper limb pain, soreness and joint stiffness were noted by five healthy untrained subjects who had performed exhaustive biceps exercise by doing isolated eccentric contractions (forced lengthening or stretching of muscle).53 If concentric contractions were performed, no symptoms developed. Serial magnetic resonance imaging showed changes consistent with muscle injury only in muscle performing eccentric contractions. These MR changes persisted in some, but at a subclinical level, for up to 75 days after the disappearance of symptoms.

The Lancet editorial14 quoted by Fry fails to support his hypothesis. It highlights the known dissociation between muscle pain and damage, the reversibility of tissue damage after exercise, and the fact that both the pain and the damage are reduced, and eventually eliminated, by repeated activity.33,43

4. Evidence from other studies of musculoskeletal problems in instrumental musicians

Over the past decade, interest in the medical problems of musicians has been increasing.30 Hoppmann and Patrone32 reviewed the pertinent literature. Medical problems in musicians were classifiable as musculotendinous overuse (52%), nerve entrapment/thoracic outlet syndrome (18%), or motor dysfunction (10%). These authors also noted that difficulties with performance could be caused by joint hypermobility and degenerative joint disease. Owing to uncertainty over the basic pathophysiological lesion(s), these authors regard musculotendinous overuse syndromes as a diagnoses of exclusion: "We consider musculotendinous overuse to be any musculoskeletal problem, whether inflammatory or not, that appears to result from excessive use, presents with pain as the major symptom, shows minimal motor dysfunction, and is not better defined as nerve entrapment or thoracic outlet syndrome."32 Their uncertainty as to the pathophysiology of development of overuse syndromes is shared by Lockwood.40

5. Static muscle loading

The possible consequences of static loading of musculoskeletal tissues are not discussed by Fry in the discussion paper. He has, elsewhere, advised "regular respite from the constrained position of music making so that muscular and mental tension can be dissipated" and
recommended reduction of static loading caused by musical instruments.\textsuperscript{17}

The conventional wisdom has been that neck and shoulder pain are more likely to develop in those workers who develop a high static load in the musculature of their neck and shoulder girdle.\textsuperscript{26,27}

A recent controlled experimental study from Finland has challenged this hypothesis.\textsuperscript{55} These researchers failed to show an association between increased shoulder muscle activity (as assessed by surface electromyography over the upper trapezius and rhomboids/erector spinae muscles) and neck-shoulder pain. In addition, the matched asymptomatic controls reported taking fewer rest periods than those with frequent neck-shoulder pain. However, their opening statement - "muscular pain in the neck-shoulder region is common in many sedentary occupations" - provides yet another example of the flawed thinking that underlies the concept of muscular overuse injury.

**Histological evidence**

1. The study performed by Dennett and Fry\textsuperscript{12}

This study attracted a number of criticisms:\textsuperscript{6,31,52} (a) there was no evidence adduced that overuse syndrome is a specific syndrome; (b) the study was poorly controlled for age; (c) the clinical grading system had not been validated and it ignored the duration of symptoms; (d) the origin of the patient sample was not described; (e) the choice of muscle taken for biopsy appeared an arbitrary one; (f) mitochondrial and ultrastructural changes of the degree reported were non-specific and were present in both affected and control muscle; (g) the number of fibres containing paracrystalline or fingerprint inclusions was not stated, and these changes occurred in only 3 of 31 muscles sampled; (h) a possible contribution of therapy to muscle changes was not considered.

2. Other studies

(i) Post-polio syndrome: It is inappropriate for Fry to cite the biopsy study by Dalakas.\textsuperscript{10} This study concerned 27 patients with postpoliomyelitis progressive muscular atrophy (muscle weakness and atrophy which some patients develop years after recovery from acute paralytic poliomyelitis). The newly weakened muscles showed signs of reinnervation and recent denervation. Muscles that had been left weak since the original illness showed a mixture of myopathy with new and old denervation. This study was thought to shed light upon the degree of compensation of the surviving motor neurones and their apparent failure for further reinnervation via axonal sprouting. The relevance of this study to overuse syndrome remains unclear.

(ii) The Lancet editorial summarised the morphological changes in muscles sampled after eccentric contractions; extensive disruption of muscle structure, with lesions localised particularly in the region of the Z-disc affecting up to half of all muscle fibres examined, and infiltration of mononuclear cells.\textsuperscript{4} Changes indicative of damage and inflammation were not prominent in the biopsy study of Dennett and Fry.\textsuperscript{12}

(iii) Forearm muscle changes associated with tenosynovitis: Fry\textsuperscript{17} drew attention to abnormalities in forearm muscle reported from biopsy specimens taken by Thompson \textit{et al.}\textsuperscript{56} from patients with acute symptoms associated with hand-use-intensive occupations. In fact, only 5 of 544 cases were biopsied, and there were no reports of abnormality of forearm muscles.

(iv) Work-related changes in the trapezius muscle: Although not mentioned by Fry, reduced blood flow in the trapezius muscle and its fascia (recorded using a laser Doppler flowmeter) has been postulated as the cause of the pain as well as the mitochondrial damage and biochemical changes found in patients with "work-related chronic myalgia."\textsuperscript{38,39}

**The concept of self injury**

This is the least satisfactory section of Fry's discussion paper. Having posed the question, "Can muscle damage itself by its own contraction", he mentions, but does not discuss, the strong resistance to this proposition. If voluntary muscle can be damaged when performing repetitive work, then
further questions arise. First, is this muscle damage sufficient to cause local symptoms (pain, weakness)? Second, why does the pain persist and become widespread in some sufferers?

1. Damage to muscle

Jones and Round\textsuperscript{33} (pages 158-174) have outlined the ways in which muscles may be damaged: as a result of direct physical trauma, as a consequence of metabolic depletion with excessive use or due to some pathological process. According to these researchers (p. 163), “it seems unlikely that metabolite changes which occur in normal muscle as a result of exercise are ever sufficient to result in the type of damage that can be produced in isolated preparations by prolonged stimulation under anaerobic conditions or treatment with metabolic inhibitors.\textsuperscript{33}

For the individual muscles involved, delayed-onset pain and muscle damage after unfamiliar severe exercise are reduced, and eventually eliminated, by repeated activity.\textsuperscript{43} Tenderness can also be abolished by training (pages 158-174).\textsuperscript{33} However, the current explanation for delayed onset muscle tenderness after unaccustomed exercise postulates inflammatory changes occurring in the connective tissues in and around muscles (page 185).\textsuperscript{33} Polymodal receptors in these tissues are likely to be sensitized by this inflammatory process and to then react excessively to pressure or tension.

It is of some interest that one explanatory model for the deep tender points observed in patients with myofascial pain syndromes and fibromyalgia syndrome postulates an inflammatory process in the deep fascia (overlying muscles) damaged by intense muscular contractions.\textsuperscript{34}

2. Persistence and spread of pain

Peripheral sensitization of polymodal receptors in damaged musculoskeletal tissues may give rise to hyperexcitable foci in the spinal cord, thus explaining both the persistence and spread of pain, as well as associated allodynia and hyperpathia phenomena.\textsuperscript{9,42,57} The difficulty in accepting this hypothesis is the rarity of neuropathic pain states in such conditions as arthritic inflammation\textsuperscript{60} and muscle tissue injury,\textsuperscript{43} where a similar process of polymodal receptor sensitization is likely to be present.

Another model, which may explain deep cramp-like muscle pain which can persist and become widespread, has been proposed by Ochoa \textit{et al.} \textsuperscript{44} In this model, sympathetic-dependent muscle pain develops when the normal sympathetic outflow plays upon a damaged afferent system in the muscle fascicles.

DISCUSSION

In this section, Fry shows a poor understanding of the other explanatory models (theories) proposed for the overuse syndrome (also known as \textit{Occupational Overuse Syndrome} and \textit{Repetitive Strain Injury}). Accordingly, any further commentary is inappropriate.

RESPONSE TO TREATMENT

The treatment of overuse syndrome by a radical rest programme is the corollary of Fry's explanatory model.\textsuperscript{20} To support his advocacy of rest, he uses the self-reported case of Smythies\textsuperscript{54} who “whilst doing some art work, had occasion to keep the left wrist in maximum strained flexion for several periods of some 10-20 minutes each.” From the detailed description of symptoms he gave, Smythies “brush with tenosynovitis” was more likely to have been a “brush” with neuropathic pain consequent upon an insult to the median nerve, either in the carpal tunnel or between the heads of pronator teres. Whilst Smythies\textsuperscript{54} did highlight the lack of efficacy of conventional methods of physical treatment in his case, he mused as to why people with acute tenosynovitis are not sent more often by orthopaedic departments for acupuncture, the form of treatment which he had found to be of great benefit.

The quotation from Lockwood\textsuperscript{40} is incomplete. It should read “Rest is the cornerstone of treatment during the acute stage.” Lockwood\textsuperscript{40} goes on to advocate splinting, supplemented by ice and non-steroidal anti-inflammatory agents. When the acute symptoms of the musicians settle, he advocates rehabilitative exercises and
attention to deficiencies in playing technique, as well as alterations to the instrument to prevent recurrences.

Fry's treatment methods in musician patients are based upon his own (untested) system of grading for severity of symptoms. His results are difficult, if not impossible, to interpret as they are completely uncontrolled. For example, he treated 44 pianists and was able to "cure" 6 of the 7 with Grade 1 and Grade 2 overuse. The remaining 37 (presumably with Grades 3-5 overuse) were advised to follow his programme of "radical rest". His programme was rejected outright by 6, and subsequently by 8 patients. Fry claimed that one patient showed no improvement over 12 months, 16 were free of pain and are playing their instruments and 6 are "progressing satisfactorily".

CONCLUSIONS

Contrary to Fry's opinion, his hypothesis of "actual musculo-ligamentous overuse" lacks support from the historical, clinical and biological evidence presented in this discussion paper. Even his data on response to treatment do not support this hypothesis.

There are diverse medical conditions which may respond to rest. This response is surely not to be taken as evidence that they were originally caused by some form of bodily overuse.

I share Fry's optimism that scientifically based studies will throw some further light upon the features of this disabling pain syndrome.

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This is an original and unique concept which has been proposed for almost a decade now by Mr. Fry. The essence of the concept is that certain people when carrying out a particular action for a considerable length of time, develop changes in tissues such as muscles and ligaments which produce pain and inability to use the part. The vast majority of the complaints or conditions relate to the upper limb, and most of Fry's original work derives from musicians, although he has also studied other groups. The particular damage which occurs to these tissues is not specified, although Dennett and Fry published work on electron microscopy of what were considered to be affected intrinsic muscles in the hand in a number of patients. No other clinical or histological studies have ever been carried out to support Fry's theory and, as far as this reviewer is aware, Fry's work stands alone without any supportive confirmatory studies. In this paper Fry describes a number of historical papers which he considers support his arguments, but I do not consider that the original writer's cramp paper by Gowers, or the polio papers from Rancho Los Amigos can be remotely considered as relating to Fry's overuse concept.

It is commonsense knowledge that when people use muscles, joints, etc., more than normal they become tired and ache. They generally recover rapidly within a day or so, and indeed if one carries on such work, in other words gets into training, then the muscles, ligaments, etc., become more used to such work and a person can achieve a far higher level of power, repetition etc., in their particular work. Fry would have us believe that in certain patients this "training" mechanism breaks down, but he does not specify how, nor does he specify which particular tissues are affected, or what the specific pathological process is.

The arguments against the overuse concept are considerable. Conventional medicine, and here I include myself, is not prepared to accept that a physical injury or disease process occurred in an upper limb, or indeed elsewhere, unless there are convincing and reproducible physical signs. The accepted signs of an injury and associated healing are those of swelling, tenderness, loss of function, and associated histological findings of inflammation. Fry's theory is based on the findings of pain, loss of function, and tenderness in various parts of the upper limb, and it must be stressed that none of these findings are objective. Fry contends that his original study was carried out on musicians and he argues that financial reward is not involved.
with musicians, and they are not to be considered as having an opportunity for secondary gain in complaining of these symptoms. In this he is being remarkably naive, and most hand surgeons are well aware of the many pressures, often psychological, on musicians to achieve a particular level of performance, and the fact that they may find difficulty in playing their instrument can have many causes without possible physical problems in the upper limb itself. Fry's theories have produced considerable reaction in the medical press, particularly in Australia, and while it would not be fair to say he has no supporters, the majority of informed comment has been critical of his theories.

The claimed histological support for his work, his own paper with Dennett, a histo-pathologist, was not convincing and was firmly condemned when it was published in the Lancet, and it has not been repeated or confirmed by anyone else.

**GENERAL CONCLUSION**

This is a unique and isolated theory propounded by Hunter Fry, which has not received any support or backing from any other scientific study. Until such independent support is forthcoming, the overuse concept should remain an eccentric and unproven hypothesis.

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This paper supports the hypothesis that the overuse syndrome is located in the peripheral tissues such as muscles, tendons, and ligaments. The cases referred to are musicians, ballet dancers, and treadle sewing machine operators. The relative load on muscles and tendons is probably high in these occupations and the concept of overuse correct. This is true mechanically as well as metabolically for the musculo-tendinous tissues involved, as can be documented by many physiological studies. This causes a catabolic effect in the tissues which exceeds the anabolic processes and consequently a net breakdown of tissues. This is similar to the over-training concept in sports. In line with this "Rest is the cornerstone of treatment" (Lockwood 1989) allowing sufficient time for recovery. Interestingly, sewing machine operators working with modern technology machines also seem to develop overuse syndromes. These are located in the neck and shoulder areas and this is in spite of the relative load being rather low. Recent studies by our research group on motor control have shown that during low force precision work a stereotype recruitment pattern of motor units may occur. This means that a few muscle fibres perform all the work. These may then work at relatively very high workloads, even when the load on the muscle as a whole is low. The result may then be an overuse syndrome even in these occupations. In such cases variation of the work tasks causing a variation in muscle fibre recruitment, may be a treatment superior to rest.
This paper describes the overuse syndrome as it is seen by a practicing clinician with wide experience, especially in examining musicians and keyboard workers. It has been noted by many clinicians that the problems of musicians and other workers in repetitive jobs do not fit well into the diagnostic entities of musculoskeletal disorders described in medical textbooks. Mr. Fry sees the overuse syndrome as a continuum with painful musculoskeletal overuse at one end and focal dystonia at the other. Focal dystonia is a poorly understood condition that is difficult to cure. The inclusion of focal dystonia under overuse syndrome should be discussed in more depth, and I am not sure whether such an inclusion is justified.

Although much clinical experience has been gained in the examination and treatment of workers in repetitive jobs, including musicians and keyboard workers, more epidemiological studies are needed to obtain information about the causes of and the factors contributing to overuse symptoms. Some musical instruments seem to be more problematic than others, especially for players with certain individual characteristics. If we knew more about possible mismatches between the player and the instrument, we could do primary prevention by, for example, advising a young student in the selection of the instrument.

I do agree that the primary modality of treatment in overuse syndrome is rest, especially in acute pain or in an exacerbation of more chronic pain. The dose or regimen of this treatment should, however, be discussed. If too long a period of rest is given and if activity is not resumed by gradually increasing the hours of work, we will see a relapse of pain as a direct result of the inactivity and being unaccustomed to work. In my opinion, proper work-rest schedules should be emphasized to a greater extent in the treatment of those with musculoskeletal problems, even performing artists. In addition, I advise my patients to try to replace part of their daily practice with so-called ideational practice, i.e. carefully and slowly thinking through the piece of music with all its artistic and technical details without playing it. Maybe some musicians are too enthusiastic or ambitious, and simply play too many hours!

It is a pity that Mr. Fry did not mention the importance of ergonomics in the treatment of musicians. The playing of many instruments involves deviated wrist postures and prolonged elevation of the arm. The presence and extent of these well-known risk factors has been diminished in some cases by redesign of the instrument. I have seen mainly redesigned wind instruments, and it is certainly more difficult to redesign string instruments without losing authenticity. But we can emphasize improving the positioning of the instrument, and the posture and the technique of the stringed instrument player.
The hypothesis as indicated by the definition given; "overuse syndrome as it affects the upper limbs (of, for instance, musicians)... a condition of pain and loss of function in muscles and ligaments, which have been subjected to excessive or unaccustomed use" can only be tested by comparison with cases with the same symptom complex not considered the result of overuse. This implies that cases would have to be selected from a population based sample and then compared with unaffected matched controls (case control study). Despite the proliferation of emotive literature on this subject this basic first step does not seem to have been taken.

The causal role of overuse has been disputed mainly by tertiary referral specialists who see a highly selected subset of cases in an office chair, without the inconvenience of investigating the workplace. Industrial physicians and ergonomists who see unselected populations from the onset of symptoms, before secondary and tertiary complications arise are not in doubt that almost all cases are work related. Fry also, having undertaken epidemiological work himself on musicians, is able to see the whole perspective of the syndrome.

His exclusion of the possibility of tendon lesions and tenosynovitis in the pathogenesis of symptoms is based perhaps on failure to see tenosynovitis at operation for carpal tunnel syndrome on such cases. Against this, considerable evidence that tendons can be involved is presented by Chaffin and Andersson (1990). As with muscle their estimates of forces generated in the carpal tunnel under various loads indicate that capillary pressure will frequently be exceeded in sustained postures with the likelihood of ischaemic injury. These tensions could be great enough to cause direct strain of tendons.

Fry does include ligamentous lesions though the evidence for these, as he states, is minimal. His view that there is much histological evidence of changes in muscle due to overuse is not shared by Cohen et al (1992). More carefully controlled studies are needed perhaps together with non-invasive magnetic resonance biochemistry in the resting and active state.

Movement abnormalities (focal dystonia) occur with and without other features of the syndrome and so are not readily explained by the ischaemia hypothesis. Most now agree (Cohen et al, 1992) with his contention that the self sustaining pain cycle in this condition is due to a disorder of the nervous system and that it is not purely psychological.

Till there are generally agreed diagnostic criteria papers will be difficult to compare with respect to causation, treatment response and prevention.

His claim that "all appear to recover with the appropriate degree of rest" is controversial. The writer considers that valuable time and morale is lost if a reeducation program is not started from the time of first consultation or stopping work. Definitive controlled epidemiological and treatment studies are long overdue.

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This paper proposes a specific deep tissue source for a range of clinically encountered overuse syndromes. Clinical, histological, biochemical, neurological and therapeutic evidence is offered to support a myopathic (-ligamentous) basis for these presentations. Together the evidence tends to endorse the possibility that under certain circumstances, muscle can damage itself.

The myopathic basis for overuse syndromes proposed here has some backing from the sort of evidence not presently forthcoming, but ultimately necessary, with certain other proposals. The author does not specifically nominate the RSI phenomenon. However, reference is made to the paper by Quintner and Elvey (1991) that does. With respect to the two proposals, myopathic and neurogenic, on clinical grounds alone the authors appear to be dealing with somewhat different presentations. Even relatively prolonged periods of rest do not cure all cases of RSI.

Not surprisingly, certain features of deep tissue (muscle) syndromes described here are suggestive of a central nervous system contribution. The referral of tenderness and pain with mechanical stimulation, their persistence long after the precipitating activity (eg sewing machine operation) had ceased and their occasional appearance contralaterally, are examples. Spinal cord neurone hyper-responsiveness can account for abnormal motoneurone discharges following discreet peripheral tissue pathology (Woolf 1983). Painful or painless muscle weakness and fatigue without obvious wasting may be a consequence of complex central nervous system processing, including reduced gamma motoneurone drive (Mense, 1991). Multiple, receptive fields, lowered thresholds for a mechanical stimulation, the appearance of novel distant fields and widespread referral are characteristic of myopathic and joint syndromes (Guildbaud, 1991; Mense, 1991). Mense (1991) has described likely spinal cord neurone mechanisms in a plausible model for referral and other clinical features observed following muscle pathology.

Optimal management of overuse syndromes obviously requires clear histological, anatomical and physiological evidence for their basis. Hypothetical causes which currently lack this background will either eventually be supported by the necessary evidence or excluded. Apparently some overuse presentations are relatively straightforward and easy to diagnose and treat. Others appear to be complex, and more difficult to categorise and manage.

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My paper was a summary rather than a rewrite of all of my published papers. A detailed response to these commentaries would be far too long to be readable. Quintner’s commentary alone is many times longer than the paper on which he comments. The further information requested by Beswick and Cursley appears in a number of my previous publications and this is also true of my interest in ergonomics1 about which I have published from 1984 onwards. I will try to concentrate, therefore, on what appear to be the main areas of difficulty.

PSYCHOGENESIS

I agree with Cohen that no evidence has been put forward to suggest psychogenesis as the principal or majority cause of use-related pain. In industrial and compensation medicine, one normally expects to see a clinical presentation tailored for gain in a small proportion of patients, but this is not restricted to use-related upper limb pain, for it occurs right across the board in industrial complaints. The National Occupational Health and Safety Commission, in all its submissions, however, could not find one piece of evidence to support the psychogenic theory2.

The medical profession in Australia is already feeling the long term effects of the damage caused by doctors writing inaccurate and intemperate letters and articles in the popular press and the Medical Journal of Australia. These writings almost invariably supported the psychogenic theory and were often demeaning to patients. The sociological result was predictable, as it became obvious to other professional groups (including lawyers), that as a profession doctors were not debating the issue rationally and were seen as having little sympathy for such patients. Some medical reports, where the heat almost rose from the paper, echoed the intensity of the debate.

Those already antagonistic to the medical profession were able to act in such a way as to make life quite difficult for independent examining doctors who tried to remain truly independent. Those supporting the psychogenic position are largely to thank for this. It was this inability to seek research and clinically based information on use-related pain that led to 'The Australian Phenomenon'.

While the “all in the mind” theories are most closely associated with some Australian rheumatologists, there are also some proponents in the UK and USA. Semple, for instance, appears to allow of no possibility whatsoever that the syndrome has other than a psychogenic base (see also ref. 3). He has not so far as I am aware offered any evidence to support the theory in which he so fervently believes. In the United States, Hadler, a rheumatologist, appears to believe that this is a non-problem and that the mistake being made by the medical profession is to recognise it as a problem. He says that the National Institute of Occupational Safety and Health (NIOSH) in America, employers, affected workers, insurance companies, doctors in practice, are all wrong and therefore responsible for the problem4.

In Australia, Brooks, a rheumatologist, in sworn evidence in the case of Cooper vs The Commonwealth, attributed the symptoms of overuse syndrome to the patient’s belief system. He stated twice during his evidence that there was no difference between pain and tenderness and that he saw no possibility that overuse syndrome in musicians could have an organic basis5. Although he has been challenged to produce actual research evidence to back his claims6, he has not done so7 at least in refereed journals or at public meetings.

Awerbuch, an Adelaide based rheumatologist, is an especially impassioned believer in the theory of psychogenesis, and will be remembered for the term 'Kangaroo Paw', his synonym for use-related pain. He continues to this day to write strong letters about his theory and criticising all those who disagree with him without offering any evidence for his own theory, and without reporting results of treatment or even claiming expertise in clinical management of this group.
Milazzo, another Adelaide rheumatologist, has written at least one medical report accepting the psychogenic basis of use-related pain but did not give reasons for this acceptance. He mentions the possibility of this being incorrect but concludes by saying "the likelihood of this, however, seems exceedingly remote".9

Littlejohn (also a rheumatologist) and Miller wrote in the Lancet "the syndrome is thus, psychosomatic"10. This conclusion was based on theories to do with pain pathways but no clinical evidence was presented to support the theories.

Luciere, a psychiatrist, has written about her view that the use-related pain of overuse syndrome is due to conversion hysteria.11 She is quoted freely and favourably by Brooks and Awerbuch, yet she has not published any series of which I am aware giving details about patients and she has acknowledged in a newspaper interview that she cannot claim success for treatment based on her theories.12

This is 1992, ten full years after the beginning of the controversy in Australia. I believe it is fair and reasonable at this stage to ask the psychogenecists for the last time to 'put up or shut up'. Semple, Luciere, and the Australian rheumatologists should produce prevalence and incidence figures, treatment modes, return to work and symptomatic cures rather than just destructive criticisms of others who try to do these things. Hadler, though acknowledged in his field as being expert in the assessment of patients with rheumatoid arthritis, must establish credibility in the same way.

Harrington makes an important point on the matter of believing the patient. Again, talking about the group I know best (the instrumental musicians), it goes against basic commonsense to approach such a patient on the premise that he/she is a liar and must have some hidden motive for the complaints other than those which are transparently obvious.

TERMINOLOGY

'RSI' is a junk term - at least in Australia. We were asked not to use it again after 1986 by the National Occupational Health and Safety Commission (NOHSC) by way of a statement in December of that year from the Honourable Ralph Willis, the Minister of Labour at the time. The NOHSC originally used 'RSI' to replace tenosynovitis, however, 'RSI' then became a collective term for many unrelated causes of upper limb pain and then became a pejorative term for industrial upper limb pain of doubtful origin. Such junk terms are better buried and not replaced by other junk terms, whose substitution can only compound the confusion. There must be many doctors scratching their heads over terms such as 'cervico-brachial disorder', 'regional pain syndrome', 'repetition strain injury', 'tenosynovitis', 'cumulative trauma disorders' and now 'refractory cervicobrachial pain' (RCBP).

Historically, use-related pain (and its consequences) was prevalent 100 years ago amongst musicians and other groups in the USA, UK and Europe13,14. It was then described in more logical terms, quite different from the terminological morass in Australia today. In the USA now, only one collective term is commonly used: 'Cumulative Trauma Disorder'. This term includes any condition which can be aggravated or brought about by hand use intense activity on the basis that some element of micro-trauma is involved. The other term now becoming used commonly in the United States is overuse syndrome (OS) - a specific term for what used to be called tenosynovitis. OS can be defined as "pain and loss of function in muscle groups and ligaments subjected to excessive or unaccustomed use" (Fry)15 or "Overuse injury occurs when a tissue is stressed beyond its anatomic or physiologic limit" (Lederman & Calabrese)16 or "injuries caused by excessive use of body parts so that cumulative effects of repeated small trauma occur that exceed physiologic limits" (1990 edition of Dawson, Hallett & Millender on Entrapment Neuropathies)17. I agree with this terminology.

There seems to be no special need therefore to invent yet more junk terms; however, for those who are attracted to such terms I would put forward Myodysaesthetic Algofibropathic Dystrophia as being better than those so far offered in Australia. These are new words, from new combinations, so that they can be made to mean whatever one wishes them to mean. In addition the acronym is superior.
Newspeak

The other fruitful cause of confusion and scrambled communication is the development of an alternative set of words to replace those in common usage. Some are arcane, others fabricated. The words inspire eccentric syntax and suggest spurious expertise.

Not only does this make for greater difficulty in communication (i.e. to other professional groups), but it also makes it more difficult to communicate with other doctors. Tenderness and analgesia for instance are found in the two volume Oxford English Dictionary (OED), but hyperalgesia, allodynia and hyperpathic are not. Neither can these terms be found in two commonly used medical dictionaries.

Newly introduced words have a more flexible meaning since they have not been legitimated by common usage. Thus they can be made to mean whatever one wishes them to mean. In the Medical Journal of Australia (August 1991) doctors were even urged to adopt this alternative terminology (hereinafter, nosology, and taxonomy, instead of terminology and nomenclature) to use in medical reports for lawyers! 18

A good example of the misfortune that may befall even established words is seen in the history of the word neurosis. In the 19th century this meant a disorder of the nervous system and included such entities as motor neurone disease, lead poisoning, tertiary syphilis and the like. With the advent of Sigmund Freud there came the term psychoneurosis which meant a psychological or a psychiatric neurosis (disorder). All would have been well if psychoneurosis had not then been abbreviated back again to neurosis! In this way the whole meaning of the original word was changed, and best exemplified by one of the most famous quotations from Freud himself: 19

"With a normal sex life, a neurosis ... is not possible."

THE CENTRAL NERVOUS SYSTEM (CNS)

There is ample evidence that the CNS is a participant in some way in the overuse disorder and I agree broadly with what Pearson and Champion have said. Quite apart from anything else, some of these patients develop a type of reflex sympathetic dystrophy which cannot be explained in any other way. In the case of musicians particularly, a proportion of them develop movement disorders which can be demonstrated clinically and quite precisely by laboratory testing. This could not happen without some participation of the nervous system.

In the muscle biopsy study published by Dennett and Fry (see below) there were features also suggesting CNS participation20. In this respect, it is a pity that Quintner chose to 'throw out the baby with the bath water', so keen was he to discredit the study.

THE OVERUSE CONCEPT

In my brief paper I believe I gave adequate reason for stating that injurious change can be brought about in muscle by prolonged submaximal contraction. This occurs in a number of patient and sporting groups and anybody seriously contending now that injurious change cannot be brought about by the muscle's own contractions is not facing the facts.

MUSCLE HISTOLOGY

Some of the commentators disputed the importance of the changes reported in the muscle biopsy study by Dennett and Fry20 and suggest that they are subtle or minor.

The changes found were neither subtle nor minor. For subjects who had been off work for many months, the muscle fibre hypertrophy (as measured against controls) was considerable as well as significant. There was an increase in Type 1 fibres with type grouping significant against controls. Mitochondrial changes were present which tended to mirror the severity of the clinical state. The number of ultrastructural changes was also significant against controls and a number of other abnormalities were noted as well. In general, the severity of the changes paralleled the grade of clinical severity.

In all affected patients, the muscle which was biopsied (the first dorsal interosseous) was
tender to palpation and therefore deemed to be involved in the process. What would be the point of biopsying a muscle which was not involved as implied by Quintner? Normally, pressure over a normal, unaffected first dorsal interosseous muscle does not produce pain. In all the affected individuals in the study, modest pressure did produce pain in this muscle and they had previously complained of pain in the hand.

Quintner has stated that it is inappropriate for me to cite the biopsy study of Dalakas. Dalakas himself would not agree with this. There are some fibres in the post-polio patients that were apparently never affected in the initial attack. These are the ones that suffer histological changes similar to those of our patients. In my conversations with Dalakas himself, he was clearly of the view that there was an important link between the two studies.

The muscle biopsy study itself was published in the *Lancet* on April 23, 1988 and inspired four letters from people apparently deeply and irrevocably committed to a belief system of psychogenesis. Their attack on the study was indiscriminate and inexpert. I should point out that the paper was a non-interpretative one and that because the numbers were not large, the differences between the affected and control values had to be large in order to achieve the significance they did. The four letters were in marked contrast to acknowledged experts, for instance; the editorial in the *British Medical Journal*, the review paper on musicians in the *New England Journal of Medicine*, and also the second edition of *Entrapment Neuropathies* of Dawson, Hallett and Millender, where people in the field discussed the study.

There were some very odd suggestions in these *Lancet* letters, particularly from Brooks, who suggested *inter alia* that changes found (which included gross hypertrophy) might be due to splinting for long periods leading to disuse! Hocking suggested steroids as well as splinting as a possible cause (steroids cause atrophy too).

There are now many published muscle biopsy studies showing injurious change to muscle with sub-maximal muscular activity. Apart from those mentioned in my paper here, there are quite a number listed in the quoted *Lancet* Editorial and a number also listed in the field of sports medicine. Only those ignorant of this literature could, in 1992, seriously contend that overuse/self injury from certain muscular activity cannot occur. However, the study of Dennett and Fry is really the only study which relates directly to overuse syndrome in the hand. The experts in the field saw no particular reason to write to the *Lancet*. For the record, however, our reply to the four psychogenecists in the *Lancet* is reproduced below (with permission).

*We reported morphometric changes in the first dorsal interosseous muscles from women with clinical grade 3, 4, 5 overuse syndrome.

In the most severely affected women (grades 4, 5) significant changes were found - an increased percentage of type I fibres with hypertrophy, a decreased percentage of type II fibres with hypertrophy, and increases in mitochondrial rearrangements, including moth-eaten and scalloped changes. In the grade 3 group similar trends were found although the pooled values were not significant.

We agree that controls should ideally be one for one, matched for age, sex, race, occupation, and pattern of musculoskeletal activity. The mean age of our controls (29.5) was less than the mean age of the most severely affected patients (45.5). The mean age difference was due to the ages of those who volunteered together with the natural reluctance of ourselves and ethics committees to enlarge such a group.

Both Professor Brooks and Dr. Hocking imply that many of the changes could well be secondary to splinting and/or steroids. In two patients there had been perfunctory splinting: No patient had received local or systemic steroids. In disuse atrophy and steroid myopathy selective type 2 fibre atrophy occurs, while in our study the changes were very different, type 2 fibre hypertrophy.
We do not believe the changes reported in the most affected group of patients can be attributed to ageing. From a large experience of muscle biopsies from nearly 4000 patients of all ages, one of us (X.D.) is familiar with the commonly encountered changes with age in otherwise active people. These changes are consistent with minor loss of anterior horn cells causing small group atrophy with some reinnervation and, less commonly, mitochondrial diminution with subsarcolemmal loss. The changes we emphasised were very different.

Brooks may have misunderstood the results of the bilateral biopsies, done to compare the clinically affected with the apparently clinically non-affected side in what is usually a bilateral disorder. We think that the results of the bilateral biopsies indicate subclinical disease on the (as yet) unaffected side*. Any changes in the muscles from affected individuals must be compared with control material from unaffected individuals.

It is wrong to imply that overuse syndrome is singularly prevalent in Australia. The apparent differences in various countries is not in the prevalence of this disorder but in the nomenclature.*

*Treatment

In my view, the results of treatment are crucial. Any treatment in overuse syndrome which at the end produces a patient who is painfree and, in the case of a musician is able to resume playing the instrument with sustainable practice habits, becomes very valuable data. I have published my results in musicians providing considerable detail and showing approximately 80% cure of symptoms. There is an important difference between cure of symptoms and cure of the disease. Although the 'cured' musician may perform and practice painfree with technique restored, there is always the possibility that the basic disorder is dormant and might be reactivated if any liberties are taken with the strict control of physical activity in music making which follows rehabilitation. It is therefore not justifiable in my view to talk about cure of the disease.

I was surprised to see Patkin claim that Quintner and Elvey had produced cures. I have reread their paper in this present series and I cannot find any claims of patient cures as Patkin states.

Lucire believes that overuse syndrome and chronic fatigue syndrome (CFS) are examples of mass hysteria (despite the clear evidence that CFS is caused by damage to the immune system). Lucire was apparently prepared to admit that she had no successes from psychotherapy based on her theory.

Traditionally, conversion hysteria is treated by removing the patient from the 'intolerable' stress which causes the 'intolerable' anxiety which is converted into a physical symptom. The soldier in the trench about to go 'over the top' into a hail of bullets complains of hysterical paralysis or blindness, which can be successfully treated back at a base hospital, but will return if he returns to that same situation. Patients with overuse syndrome who cease work (supposed to be the stressor), but continue to use their hands in other respects, do not get better. It is impossible to reconcile this with a diagnosis of mass conversion hysteria.

The onus of proof is on the proponents of these psychogenic theories. We have waited 10 years - they have been found wanting.

The 'Rest' Treatment

This means rest from pain causing or pain aggravating activities. This was what Poore, Gowers, De Waterville, Waller and many others in the 19th century advocated as being the only treatment that worked. It is certainly the only treatment which has worked for me, but its nature and purpose is somewhat misunderstood.

The rest treatment does not mean splintage or restriction of motion, both of which are undesirable. It is important that the muscles and joints move as normally and frequently as possible to minimise loss of muscle bulk and...
prevention of loss of movement in joints. 
Alternative ways of carrying out some day to day actions must be found and often assistance is required. The person living alone may have difficulties with this program. Patients who are resourceful and well organised have a greater chance than those who are not so endowed. In the case of instrumental musicians with more severe degrees of overuse, four out of five will achieve a painfree state after some months and very gradually can start strictly controlled hand exercises and then start playing their instruments again.

I have not been able to duplicate these results with other groups, no matter how highly selected are the individuals. In the non musician group the best I have been able to achieve is one in three who go back to some sort of work painfree (unpublished). This may have to do with the single mindedness and determination of musicians to return to their instruments or possibly it may also be related to the fact that most professional performing musicians begin their music making in earnest in the first decade of life so the musculo-ligamentous structures are trained well before growth had been completed. For the clarinettist whose overuse is attributable to loading 830 grams continuously on the end of the right thumb, return to clarinet playing is accomplished by the use of a small supporting post which takes the weight completely off the thumb. Without this or a similar device, the problem will of course recur. This is an example of essential ergonomic help and also applies to other instruments. Once cured of symptoms, however, the practice habits must be modified so that overlong segments of practice are avoided and adequate breaks are taken between segments.

Sjøgaard’s work is likely to provide more understanding of muscular deficiency problems. I was greatly interested in this as I was not aware of this research on fundamental muscle function. We take for granted that the muscle fibre recruitment mechanism will not fail but supposing it does? The neuromuscular programs must be incredibly complex and there is no justification for assuming that such staggeringly complex neuro-muscular circuitry cannot break down under load.

Overuse syndrome, real or alleged, is now approximately 50% of all reported industrial disease in the United States and forceful measures are being taken to reduce what has come to be known as "ergonomic hazards". OSHA has considerable powers to enforce settlements and agreements in this respect and may exact huge fines in the case of non-compliance. Agreements and settlements may include medical audits which have to be accurate to within 5%. OSHA does not seem one bit interested in debating whether or not cumulative traumas exist only in the minds of the sufferers. Ergonomic reforms are being forced at a rapid pace.

In general, research of any kind appears to be vigorously opposed by the psychogenesists. A good example is Hadler’s attack on the impressive study of Silverstein and Fine. This study showed basically more danger being present with the short cycle rather than long cycle repetitive work. Hadler’s editorial attacked everyone indiscriminately, the investigators, NIOSH (research), the employers, the employees, the insurance carriers, the medical profession, and the study itself, on the basis that he was right and all others were wrong. Fortunately, doctors will ultimately decide these questions on merit, rather than on the say-so of the loudest voice.

RESEARCH

There is interesting research in the pipeline, notably at the National Institute of Health in Bethesda, Maryland, USA. This includes the very difficult technique of recording muscle afferent impulses and the investigation of the movement disorders in musicians. Other studies are starting to appear in the United States such as those of Silverstein & Fine and Harber et al.
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