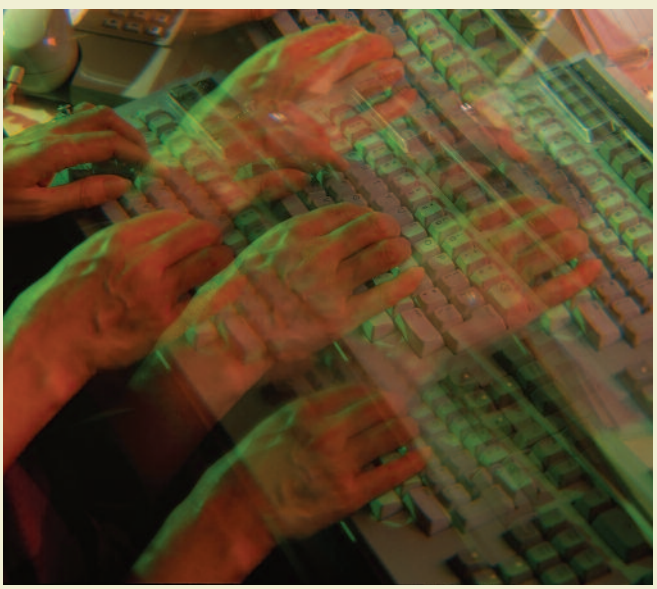
Whatever happened to RSI?

MILTON L. COHEN MD, FRACP, FFPMANZCA

Persistent pain in the absence of discernible disease or damage presents a challenge to Western medicine. Repetitive strain injury (RSI) is a reminder that medicine itself is a social construct.

'It is neither reasonable nor possible to divorce the practice of medicine from the societal culture in which it is practised.' Thus proclaimed Professors Gorman and Scott in their Forum article in the November issue of this journal.[1] As an example of this truism, they referred to the 'medicalisation of normality' and specifically cited the 'epidemic of upper limb pain in telephonists [in New Zealand, who were]…diagnosed as having an occupational overuse syndrome, formerly called repetitive strain injury (RSI)'.[1] How timely then that this article is asked to address, 'Whatever happened to RSI?'.

It is well over a decade since the sound and fury of the RSI debate resonated in the columns of medical journals and lay editorials. It may be that the howls of 'fabrication' and 'iatrogenesis' were more acceptable to society than the pleas of sociobiologists and neurobiologists who struggled – then and now – to understand the nature of pain without obvious disease, which lies at the heart of RSI.



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Associate Professor Cohen is Senior Staff Specialist, Rheumatology and Pain Medicine, St Vincent's Hospital Sydney, and a Conjoint Associate Professor of Medicine, University of New South Wales, Sydney, NSW.

What was RSI?

The acronym RSI (for repetitive strain injury) suffered from two defects. The first, the one that should have been fatal, was a failure of denotation. It was never clear to which clinical problem, let alone which physical findings, the name should be applied. In fact, the acronym ultimately was applied, in the plural, to any pain in the neck and/or pectoral girdle and/or upper limb that apparently occurred in a work-related context, even where diagnoses such as arthropathy or nerve compression could be made. This fundamental error was compounded by the labelling of some RSI cases as arthritis, tenosynovitis or cervical spondylitis (sic), even in the face of treatment failure.

The second defect of RSI, shared by its main synonyms 'occupational overuse disorder' and 'cumulative trauma disorder', was the incorporation of a hypothesis of causation in its name. Thus society and medicine were confronted with a label that was so loosely applied as to encompass anything, yet simultaneously proclaimed its aetiological connection.

It remains a cause for sober contemplation that a profession that prides itself on its scientific foundation could have so transgressed principles as basic as not confusing hypothesis with fact, defining the population of interest as a prelude to systematic study, and seeking to understand pathophysiological mechanisms before inferring disease.

What was the issue?

The transgressions described above obscured the fundamental clinical problem of RSI, namely persistent pain in the absence of a discernible disease or damage process (injury), a situation that is still a challenge to the reductionist tradition of Western medicine.[2] As labelling and treatment of patients with chronic cervicobrachial pain faltered, resort was taken in the untestable concept of psychogenesis. This caused more errors, including the failure to distinguish the consequences of persistent pain (such as frustration, depression and loss) from underlying mental illness and the imposition by default of psychiatric labels despite the requirement that positive diagnostic criteria for such illnesses be applied.

This led to RSIs being thought of as examples of somatisation, a psychological construct without a neurobiological substrate. However, it was not clear who was at fault: the doctors trying to medicalise complaints that defied understanding or the powerless workers projecting their angst onto their bodies and becoming patients.

The twofold nature of the medical debate surrounding the clinical scenario of RSI had a parallel in the adversarial nature of industrial relations at the time: the psychological default of the one lining up with the establishment default of the other. That

RSI emerged during a period of rapid change in economic policy and technological development – and the resultant reorganisation of work and social adjustment – might have offered insight into factors that could amplify distress but not into the nature of the distress itself.

What was the clinical problem?

In retrospect, it is remarkable that the fundamental clinical feature of RSIs, namely pain, was overlooked. By 1990, when the 'epidemic' of RSI had peaked, a quarter of a century had passed since the publication by Melzack and Wall of the gate control theory of pain.[3] This proposition, which identified firstly the brain as the organ responsible for pain and secondly the possibility of modulating nociceptive information by both upward and downward processes in the central nervous system, stimulated a vast amount of neurobiological and psychobiological research that continues to transform understanding.

'Surely pain – let alone tenderness – is not solely a product of society. There must be a neurological basis.'

The RSI phenomenon provided a critical test of the medical community's ability to absorb changing concepts of pain and to integrate them into practice. Yet, more than 25 years later, even in the era of unprecedented access to information, that test was not passed. It seems that the concepts of pain as a biopsychosocial phenomenon, of the plasticity of the nervous system to afferent input, and of the cognitive effects of experience and environment tended to be drowned in the clamour of labelling and attribution.

The clinical problem, however, was not only pain but also the intriguing phenomenon of tenderness. Technically, tenderness embraces both hyperalgesia (increased pain in response to a noxious stimulus) and allodynia (pain in response to a non-noxious stimulus); in clinical medicine, allodynia may be the more appropriate term. The group of patients with diffuse cervicobrachial pain in whom no inflammatory, degenerative, rheumatological or neurological disease state could be identified was characterised by allodynia to mechanical stimuli, such as pressure, vibration and movement, often associated with hypoaesthetic responses to cutaneous stimuli. This apparent paradox of a painful yet hypoaesthetic limb was not new to the literature (anaesthesia dolorosa), and neither were motor features such as rapid fatigue and dystonia (which are common features of writers' cramp). The occurrence of these phenomena in tissue that is otherwise normal requires explanation.

By 1950, there was already literature from the experimental physiologists concerning this tenderness, labelled by them as 'secondary hyperalgesia'. By 1990, the concept of central sensitisation of nociception being responsible for secondary hyperalgesia was widely known and has continued to be developed.[4] Essentially this proposition is that nociceptive or sustained subnociceptive inputs may sensitise wide dynamic range neurons in the dorsal horns of the spinal cord, leading to spontaneous discharge manifesting as pain, discharge in response to non-noxious stimuli manifesting as allodynia, and enlargement of receptive fields manifesting as spreading of pain.

Of course, central sensitisation can be inferred only indirectly in humans. Experimental work – psychophysical studies,[5] cerebral event-related responses[6] and studies of activation of nervi nervorum – in patients with persistent cervicobrachial pain has provided evidence for altered central nociceptive processes. Other work has suggested that the associated vasomotor changes in cervicobrachial pain are due to the sensitivity of neural tissue to circulating catecholamines rather than to increased sympathetic outflow and that the localised condition of lateral epicondylalgia (erroneously called epicondylitis) is also probably centrally mediated.[7]

The concept of central sensitisation provides the best explanation yet of the phenomenon of pain associated with tenderness. Important issues are raised by this concept, including that mechanical modalities of treatment may make matters worse, that chasing nociception – especially with needles and scalpels – is unlikely to be of benefit, and that pharmacotherapeutic modification of symptoms and, if possible, mechanisms may be a necessary adjunct to behavioural change.

Nociceptive versus neuropathic pain

What has emerged is the probability that many, if not most, chronic musculoskeletal pain problems characterised by allodynia/hyperalgesia of normal tissue are neuropathic in pathogenesis – that is, due to dysfunction of nociceptive pathways rather than to activation of peripheral nociceptors. Central sensitisation may be influenced upward (by sustained nociception) or downward (by thoughts, beliefs and emotions). Indeed, once central sensitisation is established, it may be impossible to distinguish between the contribution from enhanced nociception and that from increased attention (hypervigilance). Neurobiologically however, increased attention is more likely to be driven by enhanced nociception, whereas how can one test the proposition that ideas cause allodynia?

So, whatever happened to RSI?

If the criteria for index cases of a putative condition cannot be defined, how can prevalence, incidence and outcome be monitored? The fall back position is to rely on anecdotes or case

studies. From this stance, the clinical phenomenon of RSI has not disappeared. New cases continue to emerge and old cases to persist of cervicobrachial pain with mechanical allodynia but no obvious peripheral nociceptive process. These cases may represent the endemic occurrence of the problem, given that the epidemic was too heterogeneous for any label to stick.

But the epidemic has left a legacy. In the workplace, ergonomic changes have been made and incorporated into standard work practices. Pre-emptive strategies have been introduced; anecdotally, current incident cases do seem to be associated with violation of those strategies. Recognition of the importance of workplace attitude and culture was highlighted. Yet the pressures of economic and industrial reform have not relented, and anxiety in the workplace remains a powerful amplifying factor for other sources of distress.

RSI is a reminder that medicine itself is a social construct and that people with illnesses that are difficult to define are a challenge to orthodoxy. But surely pain – let alone tenderness – is not solely a product of society. There must be a neurobiological basis for this complaint. The default positions of the profession and society preferred to ignore that possibility, as well as the voices of sufferers and their advocates. If the RSI phenomenon has helped the superseding of the biomedical model of illness by a biocultural view grounded in neuroscience, then the pain of so many has not been in vain. MT

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Further reading

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