Clinical Focus: Pain Management

WHAT IS CHRONIC PAIN AND HOW SHOULD IT BE MANAGED?

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There is a scene in the film ‘Titanic’ in which the salvage experts are showing an old lady a computer simulation of the last moments of the fated voyage. She, a survivor of the voyage, smiles wisely, compliments them on the accuracy of their deductions, and then says words to this effect: “That’s a neat forensic view of what happened. Now I’ll tell you what really went on”. To a pain specialist there are parallels. Orthodox medical examination and investigation will give the clinician a ‘forensic’ view of the problem. But the forensic view may miss the point.

The wording of an X-ray report will illustrate the point. “Severe degenerative narrowing is present at L4/5” leads the clinician and patient to believe that the abnormality causes the pain, even at age 82. By contrast, if one reads “there are age-related changes at all levels including L4/5” then the possibility of this spine being normal is included. Disc narrowing might be the cause of pain but it is not certain.

Words such as ‘arthritis’, ‘degeneration’ and ‘wear and tear’ are used regularly to describe changes in X-rays of the cervical and lumbar spine. To the professional they may imply normality (the problem is not one of metastatic cancer but of degeneration), but to the patient the description may be devastating. It is surprising how often the patient who believes he has an ‘arthritic spine’ will admit to having an elderly disabled relative awaiting a hip replacement.

The orthodox or ‘forensic’ view of the chronic pain sufferer makes two omissions. Firstly, it ignores completely the possibility that the pain is not a symptom of an abnormality but the abnormality itself. Secondly, it assumes a peripheral source for the pain and ignores the changes in the condition overwhelmed rheumatology clinics with depressed, insomniac, polysymptomatic women with incurable muscular symptoms.

Adaptation of the central nervous system explains the variable response to noxious stimuli. As an easily understood example, Beecher’s description of wounded World War II soldiers suffering no pain when guaranteed safe passage from the battle zone is a classic one, but the danger of applying this example to the management of chronic pain is that the problem and its solution are seen to be psychological. Patients, in particular those who have defied the medical profession with puzzling pain symptoms, do not take kindly to a suggestion that there are psychological factors maintaining the chronic pain state. Such a suggestion may be taken to imply malingering, and some clinicians actually believe this. A credible alternative is needed, offering a scientific explanation and respectability without blaming the sufferer for the psychological consequences. Such an explanation is at the heart of the pain clinic philosophy.

THE GATE CONTROL THEORY

Melzack and Wall’s theory of a relay system in the outer layers of the dorsal horn of the spinal cord is dated, but accurately describes how painful and non-painful sensations compete at spinal cord level. According to the theory, fast conducting fibres conveying touch sensation are capable of inhibiting the discharge of spinal interneurons responding to painful sensation. Such a model describes in simple terms the observation that ‘rubbing makes the pain feel better’, and provides a rationale for the use of techniques such as transcutaneous electrical nerve stimulation (TENS).

CENTRAL SENSITIZATION

Central sensitization is the process whereby the central nervous system becomes progressively more alerted to pain stimuli. Continuous painful stimulation causes predictable changes in the behaviour of cells in the spinal cord. An early change is the production of a protein called c-fos, one of the gene regulatory proteins and a sign that synthetic capacity has been triggered. Neurotransmitters are produced, not merely to replenish those already used, but in greater quantities and of new types. Cell membrane proteins, the targets of neurotransmitter action, are produced and existing ones are modified by changes in the cell membrane. These changes have far-reaching consequences. The ‘sensitized’ dorsal horn is exquisitely sensitive to painful and non-painful stimulation, and the sensitization may last a long time after cessation of the original stimulus. The patient with postherpetic neuralgia will testify to this. The ‘gate’ of the gate control theory is effectively jammed open. The changes which occur in cells at molecular level may make this process irreversible.

THE NEUROMATRIX THEORY OF PAIN

The idea of pain having its origin within a specific but diffusely organized set of neurons within the brain is the antithesis of the peripheral or ‘forensic’ view. But it remains an attractive one that explains many phenomena. The patient with a complete spinal cord injury may continue to experience specific pain problems even when there is no neurological continuity with the part of the body in which pain is felt. The pain may be a complicated experience. Thus a painful muscle like cramp may be felt which, if it cannot be relieved, is followed by a sense of fatigue.
THE BIOPSYCHOSOCIAL MODEL

The biopsychosocial model sees pain as a disease in its own right, irrespective of causation. The disease 'chronic pain syndrome' has its own symptoms, signs and complications. The symptoms are similar to depression, the signs are known as 'pain behaviours' and include inappropriate reaction to examination and adoption of the sick role. The complications are progressive disability, loss of cardiovascular and musculoskeletal fitness and dependence upon others for the activities of daily living. Treatment is aimed at relieving not the subjective symptoms of pain, but the depression and disability that is a consequence of it.

There is no consensus amongst people who treat chronic pain sufferers concerning the respective relevance of each model. Some continue to treat the damaged tissue, using investigations of increasing sophistication, inventing plausible-sounding diagnoses and treating 'lesions' that others consider variants of normal. Theirs is the 'forensic' view of chronic pain: there is no smoke without fire, pain means damaged tissue and repair will eliminate not only the pain but also the changes that have occurred in the central nervous system. But repair will often require a lengthy search for the broken part or the smouldering ember. Others take a radically different view: chronic pain is a disease. It is generated within the central nervous system as a consequence of the activation of specific neural networks. This activation may have been triggered by a peripheral event but something else has prompted the chronic pain state. The peripheral event - the broken part or smouldering ember - has become an irrelevance, and treatment of it may not stop the pain.

There is an interesting case report in the pain literature about a patient with 'whiplash' injury to the cervical spine following a road accident. Chronic whiplash, you will know, is a controversial condition. There are who claim that it is a disease that has been invented by the lawyers and can be treated by financial compensation. In contrast, my 'forensic' colleagues claim that the syndrome is caused by 'arthritic' cervical facet joints, and that the pain can be permanently abolished by destroying the nerve supply to these joints. The case report states that a man with a chronic whiplash type of pain became progressively depressed with his pain and committed suicide. At his post mortem he was found to have a single arthritic facet joint. My 'forensic' colleagues claim to have the answer: if this joint had been treated by a nerve destruction procedure the patient might not have died. One has to marvel at the boldness of the claim: it begs the question of why this patient, of all the sufferers of chronic whiplash pain, should have developed suicidal tendencies, and what other traumatic life events and premorbid personality, together with brain serotonin deficiency or other biochemical processes, contributed to the ultimate outcome.

One could be pessimistic and say that these opposing views of the pathology of the persistent pain state are irreconcilable, like the opposing views of wave and particle physics in the early years of the century. A forthcoming lecture by one of the northwest's most outspoken 'forensic' clinicians has been provocatively entitled "The biopsychosocial model of pain: a refuge for the diagnostically destitute". On an international level, rivalry between the opposing views dominates the medico-political scene with arguments about the appropriateness of technology and the rôle of benefits agencies in the care and rehabilitation of pain sufferers. Disagreement between exponents of the 'biopsychosocial' approach and other opinions climaxed following the publication of a monograph called 'Back Pain in the Workplace'. This describes back pain persisting beyond six weeks as 'a problem of activity intolerance and not a medical problem' and was criticised by both the 'forensic' and the 'neuromatrix' camps. One authority, Patrick Wall (of the gate control theory and a champion of the neuromatrix persuasion) commented "One seeks in vain for a word of sympathy or hope for those who cross the six-week threshold".

One has to ask the question "Is there a unifying solution to the problem?" Possibly there is. In the same way that Rutherford's and Einstein's views of the universe could be explained as 'waves of particles' there may be a middle road that allows some patients to be treated predominantly within the medical model and others predominantly within a biopsychosocial model. Certainly there is a need for a sharing of common ground and resources between orthopaedic, rheumatology, radiology, physiotherapy, pain relief and psychology personnel, and the interface between them and primary care has to be such that a patient is not given conflicting messages. The reconfiguration of the health services around Morecambe Bay may give us a unique opportunity to plan this. In Lancaster we have developed a team approach to managing pain according to the biopsychosocial model (see accompanying article) and are seeking to extend the application of this approach. The small, mobile team could, in theory, offer its services in a variety of locations for a few weeks at a time. In Barrow there is the facility for a more sophisticated assessment method and individualized management of the more disabled patients according to the same principles. The development of other services across the bay will determine how well we can integrate the pain service. If, for example, there is irresistible demand and pressure for locally available, open access magnetic resonance imaging, then the rôle of peripheral factors in the pain experience may receive more attention, as there will be an expectation that magnetic resonance imaging is part of the routine investigation of back pain. On the other hand, a service which is more restricted may encourage more widespread acceptance of the psychosocial model, with a willingness on the part of the sufferer to forgo detailed 'forensic' examination if an alternative way of relieving the disability can be offered.

REFERENCES

1 Gunn CC Fibromyalgia: what have we created? (letter) Pain 1995;60(3):349-350

