occur at any time. Some describe life as "hardly worth living." Loss of weight is a striking feature, often preceding symptoms and amounting on average to one fifth of body weight,[4] a feature which has led to the appropriate description of this syndrome as "neuropathic cachexia."[5]

This form of painful neuropathy appears to be a distinct entity unrelated to other complications of diabetes. It may occur at any age, in insulin or non-insulin dependent diabetics of any duration, often without trace of other diabetic complications, and it is much more common in men than in women.[4] Sometimes neurological abnormalities may be strikingly absent—which may be confusing for the clinician—and the area affected by the pains may extend proximally far beyond any demonstrable signs. The results of electrophysiological investigations correlate poorly with symptoms.[6] Even tests of autonomic function give normal results in some cases, despite most men reporting impotence. The relative lack of objective evidence for neuropathy sometimes leads to a spurious label of "malingering."

Nerve fibres of all types may be damaged by diabetes mellitus, but the pathological features of painful neuropathy have not been much studied. One recent report has described active degeneration of myelinated fibres of all sizes, together with that of unmyelinated fibres[4]; and other workers have found a predominant degeneration of small fibres[7] or an association with active fibre breakdown.[8] Sympathetic (small fibre) denervation is also implied by the finding that blood flow in the foot is substantially increased in patients with diabetic neuropathy[9] (including painful neuropathy). The anatomical cause for the pain is uncertain (though it has been extensively discussed elsewhere); it has been attributed to the presence of small regenerating unmyelinated sprouts.[10]

The treatment of painful neuropathy is exceptionally difficult. Its management requires skilful use of non-addictive analgesics, antidepressants, and hypnotics. A combination of nortriptyline and fluphenazine may be effective, and (as in other forms of neuralgia) the use of carbamazepine and phenytoin might be helpful. Possibly aldose reductase inhibitors, which prevent accumulation of sorbitol, may be of value.[11] Cutaneous nerve stimulation is not usually of particular value but offers patients an active manoeuvre in times of distress. Some sufferers find that cooling the feet is helpful, which leads to speculation whether other modes of reducing the excessive blood flow might help. Good control of diabetes, usually with insulin, is recommended, and continuous subcutaneous insulin infusion may hasten improvement,[12] though this may occur spontaneously regardless of diabetic control.

Patients with disabling pain should be reassured that their most severe symptoms will resolve in less than a year and subsequently altogether.[4] Symptoms do not recur, and even after six years there is usually no sign of the evolution of conventional neuropathy. Unfortunately a group of patients remain whose symptoms are less dramatic but none the less disagreeable, and they report the persistence of their pains over many years.[13]

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[1] Garland H. Diabetic amyotrophy. *Br Med J* 1955;ii:1287-90.
[2] Longstreth GF, Newcomer AD. Abdominal pain caused by diabetic radiculopathy. *Ann Intern Med* 1977;**86**:166-8.
[3] Casey EB, Harrison MJG. Diabetic amyotrophy: a follow-up study. *Br Med J* 1972;i:656-9.
[4] Archer AG, Watkins PJ, Thomas PK, Sharma AK, Payan J. The natural history of acute painful neuropathy in diabetes mellitus. *J Neurol Neurosurg Psychiatry* 1983;**46**:491-9.
[5] Ellenburg M. Diabetic neuropathic cachexia. *Diabetes* 1974;**23**:418-23.
[6] Greene DA, Brown MJ, Braunstein SN, Schwartz SS, Asbury AK, Winegrad AI. Comparison of clinical course and sequential electrophysiological tests in diabetics with symptomatic polyneuropathy and its implications for clinical trials. *Diabetes* 1981;**30**:139-47.
[7] Brown MJ, Martin JR, Asbury AK. Painful diabetic neuropathy: a morphometric study. *Arch Neurol* 1976;**33**:164-71.
[8] Dyck PJ, Lambert EH, O'Brien PC. Pain in peripheral neuropathy related to rate and kind of fiber degeneration. *Neurology (Minneapolis)* 1976; **26**:466-71.
[9] Edmonds ME, Roberts VC, Watkins PJ. Blood flow in the diabetic neuropathic foot. *Diabetologia* 1982;**22**:9-15.
[10] Thomas PK. Pain in peripheral neuropathy: clinical and morphological aspects. In: Culp WJ, Ochoa J, eds. *Abnormal nerves and muscles as impulse generators*. Oxford: Oxford University Press, 1982:553-67.
[11] Jaspan J, Herold K, Maselli R, Bartkus C. Treatment of severely painful diabetic neuropathy with an aldose reductase inhibitor: relief of pain and improved somatic and autonomic nerve function. *Lancet* 1983;ii:758-62.
[12] Boulton AJ, Drury J, Clarke B, Ward JD. Continuous subcutaneous infusion in the management of painful diabetic neuropathy. *Diabetes Care* 1982;**5**:386-90.
[13] Boulton AJM, Scarpello JHB, Armstrong WD, Ward JD. The natural history of painful diabetic neuropathy: a 4 year study. *Postgrad Med J* 1983;**59**:556-9.

Post traumatic pain syndromes

Post traumatic pain syndromes are characterised by chronic pain which is not necessarily related to the site or extent of the injury. Neurological and general examination may confound the clinician by the lack of abnormal findings in the face of extreme suffering. We can understand the pathogenesis of acute traumatic pain, which generally follows the distribution of the injured nerve. This sort of pain is conducted through a defined pathway. In contrast, chronic post traumatic pain may be poorly localised and its pathway of conduction is not understood. The mechanism of this form of pain may be in territory between neurology and psychiatry. The philosophical approach by doctors to this problem is thus of great interest.

A caricature would portray the neurosurgeon as a determinist, arguing that pain is the result of a biological system. This implies that there are pathways amenable to either destruction or electrical stimulation which may modify the brain's and hence the mind's creation of pain. In contrast, the psychiatrist is entitled to an existentialist viewpoint—for example, pain may be the choice of the conscious mind in place of depression. In practice both views are useful in the diagnosis and management of post traumatic pain.

Miller coined the term accident neurosis to encompass postconcussive syndrome as well as other post traumatic symptoms.[1] He maintained that most of the symptoms after a minor injury such as headache, dizziness, irritability, decreased libido, and pain were not organically determined and would subside if the compensation claim was settled. Symonds, however, argued that many apparently neurotic complaints after head injury may have an organic basis, and supported this conclusion with the evidence of the frequent finding of cellular or diffuse damage to the white matter.[2] Medicolegal practice presents special problems; the adept clinician can usually detect those patients who are malingering, but this still leaves a core of patients who have organically determined pain either somatic or psychogenic, and many with both components. Clearly post traumatic pain may be

the sole or most prominent feature of an affective depressive disorder. Blumer has called this the pain prone disorder.[3] Patients are usually rigid, hard working introverts who deny psychiatric problems. Associated features such as disturbance of sleep and lack of energy, or any sensation of pleasure, should be sought, for these support the diagnosis. Treatment with tricyclic antidepressants may be successful.

Causalgia, though tainted with a psychoneurosis in the past, is clearly organically mediated, though secondary depression is a common accompaniment. Weir Mitchell described the condition in soldiers in the American civil war.[4] Causalgia is characterised by a burning pain, which is not confined to a specific dermatome, and with autonomic dysfunction as evidenced by oedema, abnormal sweating, and trophic changes. It may be the result of partial nerve injury or apparently minor injuries without lasting sequelae. The pain may not date from the neurological insult, and a delay of weeks or months is characteristic. Movement provokes the pain so that the affected limb is kept immobile and joints become stiff. The shoulder-hand syndrome is just one variety of this. Patients cannot bear to be touched, and clothing and bedclothes may trigger off excruciating pain. Causalgia is probably an example of pain arising from abnormal discharges from afferent nerve fibres, arising from a gradual alteration in the relation between different afferent pathways, and perhaps resulting in a central state of oversensitivity to denervation.

Sudeck described a further example of reflex sympathetic dystrophy.[5] These cases occurred after minor trauma to the wrist or ankle joint. Early changes include spotty decalcification of the joint with hyperaemia and oedema; later atrophic changes are seen, and the limb becomes cyanotic, cold, and clammy. Pain may be severe and provoked by slight movement. Marsden *et al* (p 173) have described sustained muscle spasms and rhythmical myoclonic jerks associated with Sudeck's atrophy. The pathophysiology is again thought to be related to abnormal discharges from afferent fibres—this leads to interesting speculations about the genesis of other movement disorders. The history of movement disorders is littered with psychiatric explanations, but most neurologists now accept that disorders such as spasmodic torticollis, writer's cramp, and so on, have an organic basis. Similarly, we may have been slow in recognising that patients with post traumatic pain may have movement disorders. Painful legs and moving toes (which could be more simply called Spillane's syndrome, as he first described it) can come on after apparently minor nerve injury or arise spontaneously.[6] Pain tends to be peripheral and is associated with bizarre writhing movements of the toes, although proprioception is normal. These cases appear to be separate from those described by Marsden and his colleagues, as the movements are sinuous and athetoid rather than myoclonic and the pain is less severe. Possibly, however, Spillane's and Marsden's syndromes form part of a spectrum with the same underlying pathogenesis, the initial event being damage to a peripheral nerve and subsequent secondary spinal cord and central elaboration.

What about the treatment of these disorders? All authors agree that early intervention is important. The psychiatric findings should be assessed early; are they primary or secondary, and does the pain suggest causalgia? Physiotherapy started early may prevent the development of joint stiffness and dystrophy and hence speed return to work. An early return to work is an important prognostic factor in determining ultimate functional capacity. Causalgia is notoriously difficult to treat once it is established, and the sympathetic doctor must not become frustrated by the apparent futility of his treatment. Drugs such as carbamazepine are often tried, occasionally successfully, but analgesics are rarely effective for long term use. Not infrequently patients are referred for a more invasive form of treatment. Some patients will improve with most techniques, but no single procedure has a good enough success rate to recommend it to every patient. Local anaesthetic block will often take away pain for the duration of the block but surgical section at the same site may not work. There has been some success using guanethidine to block sympathetic outflow and this may be more related to a central action due to noradrenergic hypersensitivity. Deafferentation pain may respond to stimulation, be it at the level of the peripheral nerve, spinal cord, ventrocaudal thalamic, or internal capsule. About two fifths of patients will derive an excellent result from this procedure.[7]

Creating radiofrequency lesions central to the dorsal root is an alternative procedure which may be successful. Such procedures obviously require careful selection as to whether they are appropriate by a specialist in pain relief. Further definition of the role of endorphins and that of the substantia gelatinosa may lead to more selective means of controlling pain. Lest we forget, tricyclic antidepressants should always be tried in chronic pain, not only for their antidepressant properties but also for their possible analgesic effects.

Post traumatic pain is a difficult subject but it may be illuminated considerably by correct attention to the neurological findings and the psychiatric history. It should then become clear which patient may benefit from a predominantly determinist or psychiatric approach—many patients will require both.

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[1] Miller H. Accident neurosis. *Br Med J* 1961;i:919-25.
[2] Symonds C Sir. Concussion and its sequelae. *Lancet* 1962;i:1-5.
[3] Blumer D. The pain-prone disorder. In: Benson DF, Blumer D, eds. *Psychiatric aspects of neurologic disease*. Vol II. New York: Grune and Stratton Inc, 1982:179-94. (Seminars in Psychiatry Monograph.)
[4] Weir Mitchell S. *Injuries of nerves and their consequences*. New York: Dover Publications, 1977.
[5] Sudeck P. Uber die akute Entzündliche Knockenatrophie. *Arch Klin Chir* 1900;**62**:147-56.
[6] Spillane JD, Nathan PW, Kelly RE, Marsden CD. Painful legs and moving toes. *Brain* 1971;**94**:541-56.
[7] Tasker RR, Organ LW, Hawrylyshyn P. In: Bonica U, ed. *Pain*. New York: Raven Press, 1980:305-29.

Are public health education campaigns worth while?

Communities are sometimes subjected to intensive health education activity through mass communication for limited periods. Campaigns such as these usually aim at improving or maintaining the health of the population by persuading people to adopt or avoid certain types of behaviour. The topic concerned may be general, such as dental disease, or apply to a particular subgroup, such as cervical cancer. It may be one of immediate public concern, such as the need to contain an outbreak of rabies, or less threatening, such as the risk of developing cardiovascular disease. The message may be communicated through the press, the broadcasting media,