favour of the open operation,[21-24] though with the admission that in developing countries (where mitral stenosis remains common and facilities are limited) the cheapness and simplicity of the closed procedure still justify its use for a mobile valve in young patients.[25 26] Yet experience in Teheran on the outcome of closed or open operation in 39 children points to improved results with the open approach.[27] There is in addition increasing advocacy of earlier operation in symptomless patients with valve areas that would not be regarded as critical to arrest the process of progressive fibrosis and delay or possibly avoid the need for later valve replacement.[21 23 24]

In Britain closed valvotomy has remained an acceptable form of palliative treatment for stenosis with mobile cusps as indicated by a loud mitral closure sound and a good opening snap. A recent report from two London hospitals now points to the demise of this method, describing the results of open valvotomy performed in the years 1968 to 1980 in 157 patients with mitral disease and dominant stenosis.[28] In that series there was only one death within one month of operation. The authors do not state in how many patients they have employed closed valvotomy but comment that when using a Tubbs dilator they have found that only one commissure is adequately split. In common with numerous earlier reports they emphasise that open operation permits the safe removal of left atrial thrombus, decalcification of the valve, separation of fused chordae tendineae, and functional assessment of mitral incompetence by injecting saline into the left ventricular cavity. Some 124 patients were followed up for one to 14 years (mean 7·5) with seven deaths and eight valve replacements. Actuarial curves predict a 10 year survival of 90%, and the estimated restenosis rate was 16% at 10 years.[29]

In the next decade in Britain medical students and doctors will see ever fewer new patients with mitral stenosis. If current trends continue, those diagnosed will be operated on early in the presymptomatic phase of the disease, in a way parallel to the prophylactic closure of an atrial septal defect or patent ductus: they will have open operations which in most cases will preserve the valve. Closed valvotomy will vanish from the operating list, though it will linger nostalgically in the minds of the senior thoracic surgeons who made such fine use of it in its heyday—and it will continue in use in Third World countries provided that surgeons are available who are trained in the technique.[30]

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# Pain and diabetic neuropathy

Neuropathies in diabetes occur either as symmetrical sensory polyneuropathy affecting chiefly the feet and legs or as mononeuropathies of individual nerves or nerve roots. Pain may develop in both forms of neuropathy, either affecting the feet symmetrically or, in the mononeuropathies, causing pain in the thighs from femoral neuropathy (amyotrophy) or root pains, chiefly of the trunk.[1 2] (Painful paraesthesiae in the hands are more commonly due to the carpal tunnel syndrome, to which diabetics are prone.) The good prognosis for the pain and debility of femoral neuropathy has been well documented,[3] but the painful neuropathy affecting the feet is not so well understood.

We do not know the exact prevalence of painful sensations in the feet in patients with diabetic neuropathy, but commonly they complain of paraesthesiae and perhaps a burning sensation. The symptoms need not cause too much distress but they may take the form of unremitting pain which persists day and night and causes insomnia and depression. Patients complain that their feet feel swollen, noting particularly an intense discomfort on contact with clothing and bedclothes. The distress is made worse by severe searing pains, which

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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# 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