He was considered for spinal cord stimulation in early 1986. At that time he was able to stand and walk a few metres. Formal testing of power showed that it was grade 4 bilaterally at the hips. Reflexes were symmetrically and abnormally brisk at the knees and absent at the ankles. The right plantar reflex was absent, the left flexor. There was complete analgesia of the sacral and scrotal areas and absent pin prick sensation below L4 bilaterally. However, in this distribution he also had severe pain on light touch.

An epidural electrode was inserted through the T10-11 interspace and positioned at T7-8. It was extradural and dorsal to the cord through its epidural passage and in the midline. On stimulating with a 0.2 ms pulse at about 5 mA and 33 Hz he experienced severe pain initially at around the level of his laminecetomy, instead of the usual pleasant tingling other patients have reported. Turning the current up reproduced the severe pain in his thighs down to the knees but not beyond. He also experienced a tingling pain in his penis and scrotum, an area which had previously been anaesthetic for 4 years. In other subjects spinal cord stimulation has only been described as painful if dorsal roots are stimulated directly. Then pain is referred to a local level, whereas in this case pain was felt at levels some segments caudal to the electrode. Bladder evacuation was also more complete (urine volume passed 500 ml rather than the usual 50 ml). All these phenomena were present on spinal cord stimulation, but ceased on stopping the stimulation, and were reproducible. After a period of about 20 hours spinal cord stimulation was discontinued and he returned to his previous state.

Pseudo-tabetic pain after laminectomy is a severe if rare complication of surgery and like tabes dorsalis has been ascribed to root or dorsal root ganglion damage. The severe burning dysesthetic pain in the thighs following light touch and associated with absent pin prick sensation in that area has been termed allodynia. Whether this pain is of dorsal root origin or a consequence of central reorganisation in the spinal cord is not known, although the latter view has received support from Loh and Nathan.

How spinal cord stimulation relieves deafferentation pain is not known. Effects at a mid brain level and antidromic effects at a segmental level involving dorsal column collaterals have been proposed. The current employed in the present case was such that only some dorsal cord fibres are likely to have been stimulated, and under normal conditions such stimulation leads to a pleasant tingling sensation. In those cases in which the ventral spinal cord tracts have been stimulated by a ventrally located electrode, pain has resulted. Thus it is suggested that in this case allodynic type pain was produced by stimulation of the dorsal tracts on the spinal cord. Although there can be no proof on this matter it is thought likely that this was due to antidromic conduction along dorsal column fibres to segmental neurons via axon collaterals. These segmental cells, as a result of the neurological damage, had altered receptive field and response properties and the resultant transmitted impulses were interpreted as pain. While the tracts necessary for transmission of these impulses rostrally might not have been limited to the ventral cord, the spinothalamic tract does appear the most likely to have been involved.

References

Matters arising

Apraxia in subcortical lesions

Sir: Of great interest was the paper by De Renzi et al confirming our previous report in this Journal of apraxia in seven patients with deep cerebral lesions, that is the basal ganglia and the thalamus. Unfortunately De Renzi et al missed this reference and claimed that evidence of the occurrence of apraxia in subcortical lesions was scarce and that no emphasis had been given to this point in the literature. Recently also Basso and Della Sala have described a single case of ideomotor apraxia in a patient with a haemorrhage confined to the head of the left caudate nucleus.

It is therefore well documented that apraxia, generally considered a cortical sign, may also occur when the lesion involves subcortical structures. All the seven patients we described, had ideomotor apraxia; five also had constructional apraxia and one had buccal-facial apraxia. None of the patients had utilisation apraxia. The lesions (demonstrated by computed tomography) were located in the lenticular body in five patients, in the thalamus in the other two. In three patients the lesion was in the right hemisphere.

Not all patients with lesions of the basal ganglia and/or the thalamus have apraxia. However, the limited number of cases so far reported does allow a definite conclusion concerning anatomo-clinical correlations. As we discussed (and it has been recalled by De Renzi et al), the reports of apraxia in patients with deep cerebral lesions open new evidence of the function of the basal ganglia. This is not surprising if one considers the close anatomical linkages between the cerebral cortex and the basal ganglia.

De Renzi et al reply

We apologise to Dr Agostoni et al for having omitted the quotation of their paper in 1986.

References