function bilaterally, but all other shoulder girdle muscles were spared.

The trapezius is the major muscle acting upon the scapula and works in concert with other shoulder girdle muscles to position and stabilise the scapula during movements of the upper limb.⁴ The action of the trapezius is a composite of actions by upper, middle and lower segments of the muscle. The upper trapezius elevates the scapula and shoulder. The middle portion retracts the scapula toward the midline. The lower fibres move the scapula down and medially. Contraction of the entire trapezius draws the point of the shoulder upward and medially while the lower angle of the scapula moves laterally. This action tilts the glenoid fossa upward and allows full abduction of the arm by the deltoid. Weakness of the trapezius has been recognised as causing a number of abnormalities of motion and posture:^{5,6} (1) Elevation and retraction of the shoulder are weak, (2) Head tilt to the affected side is impaired, (3) Fingertips on the involved side hang lower and project further forward when the arms are extended anteriorly, (4) Upper portion of the scapula falls laterally, (5) The scapula wings when the arm is abducted or extended anteriorly, (6) Shoulder contour is lowered.

A hump in the shoulder contour is not listed as a sign of trapezius weakness in earlier reports, (Haymaker and Woodall⁷ illustrate a hump in their fig 137, but do not mention the hump in the text), nor do we find reference to an oblique pectoral crease as a sign of trapezius weakness.

In normal individuals there is often a vertical crease where the upper arm meets the pectoral tissues in the anterior axillary fold. With shoulder weakness, a crease is angled medially and obliquely towards the sternoclavicular joint. Brooke¹ and Ringel² believe this oblique crease develops when shoulder weakness permits the shoulder to fall anteriorly; pectoral muscle atrophy may accentuate the fold. We agree, but emphasise that none of our patients had generalised shoulder girdle weakness or wasting, and none had significant pectoral weakness or atrophy. Obesity probably accentuated the oblique pectoral crease in our case with syringomyelia and in the patient illustrated in Ringel's fig 8.

Brooke describes a prominence in the mid-portion of the upper trapezius in patients with muscle disease. This hump is easily viewed from the front and accentuated when the arm is abducted. Ringel mentions a prominence in the mid portion of the trapezius when patients with serratus anterior weakness abduct the arm, causing the scapula to rise up over the shoulder.

Walton³ presents the photograph of a patient with facioscapulohumeral dystrophy who is abducting her arms. He identifies the humps in the shoulder contours as elevated scapulae, but does not remark on the basis of this sign. Brooke speculates that the hump in shoulder contour may result from upward displacement of the scapula or activity of the trapezius to stabilise the shoulder. However, in our patients the hump was evident despite total paralysis of the trapezius. DeJong⁵ and Mumenthaler⁶ comment on the position of the levator scapula directly beneath the skin when the trapezius has atrophied, but they do not state that it is overactive or visible in the shoulder contour. The levator scapula does not appear responsible for the hump since our case 5 had a typical hump despite complete paralysis of the levator scapula and rhomboid. We believe the hump is produced by the superior angle of the scapula which is displaced upward and laterally because of middle and lower trapezius weakness. The superior scapular angle becomes visible above the trapezius contour which is lower than normal due to atrophy and weakness of upper trapezius fibres. We propose the name "scapular hump" to designate this feature and indicate its cause.

Trapezius weakness displaces the shoulder down and forward to produce an oblique pectoral crease. Weakness and atrophy of the upper trapezius flattens the shoulder contour; middle and lower trapezius weakness allows the superior angle of the scapula to rise and produce the "scapular hump" between the neck and point of the shoulder. We believe trapezius weakness is the key element in the origin of an oblique pectoral crease and scapular hump in patients with diffuse shoulder girdle weakness. These signs can be seen with trapezius weakness alone; they need not imply more widespread neuromuscular disease or pectoral involvement.

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Pain produced by spinal cord stimulation in a patient with allodynia and pseudo-tapes

Sir: Spinal cord stimulation can be a useful procedure in relieving previously intractable pain associated with deafferentation syndromes, as for example, in phantom limb pain. A case is presented in which spinal cord stimulation actually exacerbated such a pain.

A 47 year old man had vague lumbosacral and right thigh pain for several years before 1968 when he presented with a sudden acute pain down both legs. This was managed conservatively and eased. Subsequently, however the pain returned and he began to notice that he was dragging the right leg. By February 1983 when he re-presented he had developed saddle anaesthesia, weakness of the legs and was found to have absent ankle reflexes. A myelogram showed a complete extradural block at L4-5 and he underwent a laminectomy for a central disc protrusion.

After the operation power improved but his saddle anaesthesia increased. He also developed urinary incontinence and began to notice severe burning pain of the thighs when touched, with reduction of sensation over the feet. A myelogram in August 1983 showed residual narrowing at L4-5 and arachnoiditis. A full L5 laminectomy was performed with bone removal at L3-4 and an attempt made to remove some of the extensive fibrous tissue from around the lumbar roots. Following the second operation his mobility improved to allow him to walk 20 m or so, but he was doubly incontinent and had reduced sensation in the feet and saddle anaesthesia. The severe pain remained. Unfortunately he also developed severe lightning pain in the legs which would come on intermittently for 5 minutes or so every 2 weeks for 3 days.

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 throughout 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 laminectomy, 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.^{1,2} The severe burning dysaesthetic pain in the thighs following light touch and associated with absent pin prick sensation in that area has been termed allodynia.³ 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.⁴

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,⁵ 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 of 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,⁶ the spinothalamic tract does appear the most likely to have been involved.

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Matters arising

Apraxia in subcortical lesions

Sir: Of great interest was the paper by De Renzi *et al*¹ confirming our previous report² 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*¹ 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 Sala³ 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 sub-

cortical structures. All the seven patients we described, had ideomotor apraxia; five also had constructional apraxia and one had bucco-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*¹), the reports of apraxia in patients with deep cerebral lesions open new evidence of the functions of the basal ganglia. This is not surprising if one considers the close anatomical linkages between the cerebral cortex and the basal ganglia.

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De Renzi *et al* reply

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