fever, headache, nausea and vomiting. Two days later, he appeared acutely ill and physical examination revealed mild nuchal rigidity. A lumbar puncture showed a turbid CSF, which contained 6,400 white blood cells/mm$^3$ with a polymorphonuclear predominance, glucose 41 mg/dl (blood glucose 219 mg/dl) and protein 4.1 g/l. Gram stain showed no microorganisms. Cultures for aerobic and anaerobic bacteria were sterile. The patient was given a ten days course of cefotaxime plus metronidazole with rapid improvement. The patient was asymptomatic when discharged. No other complications have appeared after 2 months of follow-up.

Meningitis is seldom reported as a complication of radiofrequency trigeminal rhizotomy in the current literature. In a review of more than 14,000 cases from 33 reports, Sweet recorded meningitis in seven patients, brief aseptic meningeal reactions in nine and temporal-lobe abscesses in another two. Usually characteristics of these meningitis are not mentioned. Silverberg and Britt described a streptococcal aetiology for a case of meningitis after radiofrequency trigeminal rhizotomy. In our experience, purulent meningitis complicated a 0.4% of all such rhizotomies performed (2 out of 504). Streptococcus mutans was recovered in one of the cases. In the other patient, prior antibiotic therapy (penicillin) may account for the sterile CSF cultures. In both cases, meningitis and radiofrequency trigeminal rhizotomy are related, since the infection was detected shortly after the procedure was performed. Invasion of the CSF space by the oral flora accidentally carried on the electrode, seems the most likely mechanism for production of meningitis. This is supported by the fact that a usual oral bacteria (S mutans) was the aetiologic agent in one of the cases. However, there was no evidence of mucosal puncture during the procedures.

In summary, these two cases illustrate purulent meningitis as a complication of radiofrequency trigeminal rhizotomy. Clinical suspicion of this possibility may lead to a prompt diagnosis and adequate treatment with a good prognosis. Streptococcal aetiology seems most likely.

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Accepted 30 January 1987

Oblique pectoral crease and "scapular hump" in shoulder contour are signs of trapezius muscle weakness

Sir: Patients with shoulder girdle weakness from neuromuscular disease may develop an oblique skin crease overlying the pectoral muscles and a hump in the contour of the shoulder between the neck and the acromion. The mechanisms underlying these signs have not been clearly delineated. We have seen oblique pectoral folds and humps in the shoulder contours of seven patients with trapezius muscle paralysis.

A 47 year old man was moving a refrigerator when it fell against the right side of his neck, pinning him for 2 hours, leaving him with pain and weakness in the right shoulder. On examination, he had a pigmented scar over the right spinal accessory nerve in the posterior triangle of the neck, with weakness and wasting of his right trapezius muscle. EMG showed abnormal insertion potentials and markedly reduced voluntary activity in the trapezius alone. All other muscles of the right arm and shoulder girdle were normal clinically and electromyographically. On the affected side, a pectoral crease was obliquely oriented. A hump produced by the superior medial corner of the scapula was visible from the front in the contour of the upper trapezius (fig).

Six other patients were seen with an oblique pectoral crease and a scapular hump resulting from trapezius weakness. Four patients had these signs unilaterally resulting from spinal accessory nerve injury during surgical exploration of the posterior triangle. A fifth patient had trauma to the posterior triangle followed by reparative surgery. He had total paralysis of trapezius, levator scapula and rhomboid, with mild weakness of biceps and deltoid muscles. The last patient had syringomyelia with no trapezius

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Fig  Right trapezius paralysis from accessory nerve injury in posterior triangle of neck. Single solid arrow = oblique pectoral crease. Double solid arrow = normal pectoral crease. Open arrow = scapular hump in shoulder contour.
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. 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: (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 Woodall 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 sternocavicular joint. Brooke and Ringel 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 midportion 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 serratius anterior weakness abduct the arm, causing the scapula to rise up over the shoulder. Walton 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. DeJong and Mumenthaler 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 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-tabes

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 urinary incontinence with bone 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.
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*J Neurol Neurosurg Psychiatry* 1987 50: 1082-1083
doi: 10.1136/jnnp.50.8.1082

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