more commonly encountered viral encephalides.^{10,11} Simple blood tests may reveal eosinophilia which only develops when the larva is dead, CSF examination may also demonstrate eosinophils or lymphocytes. Serological assay can be performed on serum or CSF: indirect haemagglutination is regarded as diagnostic although false positives are encountered and complement fixation in the CSF has almost the same diagnostic rate.^{12,13} Radioimmunoscreening has been developed recently.

Plain radiographs of the soft tissues and of the skull may show multiple small round calcified lesions and where the disease is endemic this is diagnostic. Cerebral lesions are not invariably calcified. Contrast ventriculography, cisternography and myelography may show solitary or multiple filling defects which may be mobile.^{14,15} CT will reveal the location of parenchymatous lesions with characteristic calcification or ring enhancement and variable surrounding oedema. Scanning after cisternal contrast will demonstrate basal or ventricular cysts^{6,14} but magnetic resonance imaging is better able to show ventricular cysts and the scolex within a cyst.

When the diagnosis has been made, treatment is symptomatic and aimed at the management of seizures and at the relief of local or generalised intracranial pressure. Undiagnosed mass lesions, especially if solitary, are best treated by resection. If excision is planned a complete resection is essential as the wall of the larva may reform a cyst.¹⁶ Definitive medical treatment with Praziquantel is now proven to be effective and is also useful in disseminated central nervous system cysticercosis and the use of steroids is also advocated.^{17,18} Hydrocephalus when present can safely be treated by CSF diversion.¹⁹

Cysticercosis is said to be the commonest parasitic infestation of the central nervous system.⁸ Worldwide reports of cerebral cysticercosis have increased recently, both from endemic areas and from population centres previously believed free from the disease. Only five cases of cerebral cysticercosis (and five of non-cerebral disease) have been reported to the Public Health Laboratory Service in this country since 1975, making cerebral cysticercosis rarer here than the travelling habits and multinational origins of the population would lead us to expect. There are, therefore, grounds to believe either that these figures do not reflect the true situation and that cysticercosis is more widespread or that we can expect an increase in this common international disorder. Should comparatively simple serological

investigations fail to provide sufficient information then more sophisticated techniques such as stereotactic excision may be required.

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References

- Bickerstaff ER, Cloake PCP, Hughes B, Smith WT. The racemose form of cerebral cysticercosis. *Brain* 1952;**75**:1-18.
- Bickerstaff ER. Cerebral cysticercosis, common but unfamiliar manifestations. *Br Med J* 1955;**1**:1055-8.
- Bickerstaff ER, Small JM, Woolf AL. Cysticercosis of the posterior fossa. *Brain* 1956;**79**:622-34.
- Stepien L. Cerebral cysticercosis in Poland. Clinical symptoms and operative results in 132 cases. *J Neurosurg* 1962;**19**:505-13.
- Olive JI, Angulo-Rivero P. Cysticercosis of the nervous system. I: Introduction and general aspects. *J Neurosurg* 1962;**19**:632-4.
- McCormick GF, Chi-Shing Zee, Heiden J. Cysticercosis cerebri, review of 127 cases. *Arch Neurol* 1982;**39**:534-9.
- Grisolia JS, Wiederholt WC. CNS cysticercosis. *Arch Neurol* 1982;**39**:540-4.
- Wierholt WC, Grisolia JS. Cysticercosis, an old scourge revisited. *Arch Neurol* 1982;**39**:533.
- Brown WJ, Voge M. Cysticercosis. In: Brown WJ, Voge M, eds. *Neuropathology of Parasitic Infections*. Oxford: Oxford University Press, 1982:108-36.
- Srinivas HV, Rao TV, Deshpande DH. Cerebral cysticercosis: clinical and pathological observations with emphasis on the encephalitic type. *Clin Neurol Neurosurg* 1980;**82**:187-97.
- Madrado I, Olhagary B, Becerra M, Sandoval MA, Soto R. Acute cysticercosis encephalitis: description of a histologically confirmed case. *Neurosurgery* 1983;**13**:593-5.
- Latovitzki N, Abrams G, Clark C, Mayeux R, Ascherl G, Sciarra D. Cerebral cysticercosis. *Neurology* 1978;**28**:838-42.
- Nieto D. Cysticercosis of the nervous system; diagnosis by means of the spinal fluid complement fixation test. In: Palacios E, Rodriguez-Carbajal J, Teveras JM, eds. *Cysticercosis of the Central Nervous System*. Springfield: Charles C Thomas, 1983:55-62.
- Rodriguez-Carbajal J, Palacios E, Chi-Shing Zee. Neuro-radiology of cysticercosis of the central nervous system. In: Palacios E, Rodriguez-Carbajal J, Teveras JM, eds. *Cysticercosis of the Central Nervous System*. Springfield: Charles C Thomas, 1983:101-13.
- Kim KS, Weinberg PE. Spinal cysticercosis. *Surg Neurol* 1985;**24**:80-2.
- Stern WE. Neurosurgical considerations of cysticercosis of the central nervous system. *J Neurosurg* 1981;**55**:382-9.
- Sotelo J, Escobedo F, Rodriguez-Carbajal F, Torres B, Rubio-Donnadieu F. Therapy of parenchymal brain cysticercosis with Praziquantel. *N Engl J Med* 1984;**310**:1001-7.
- Sotelo J, Tomes B, Rubio-Donnadieu F, Escobedo F, Rodriguez-Carbajal J. Praziquantel in the treatment of neurocysticercosis: long term follow-up. *Neurology* 1985;**35**:752-5.
- Lobeto RD, Lamas E, Portillo JM, et al. Hydrocephalus in cerebral cysticercosis. *J Neurosurg* 1981;**55**:786-93.

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Purulent meningitis after percutaneous radiofrequency trigeminal rhizotomy

Sir: Since it was first described by Sweet in 1965,¹ percutaneous radiofrequency trigeminal rhizotomy (RFTR) has been considered the procedure of choice for surgical treatment of most patients with trigeminal pain. The simplicity of the technique, the effectiveness, the low morbidity and almost non-existent mortality justify its wide use and acceptance.^{2,3} Although some complications occur, meningitis is seldom included among them. We describe two patients in whom purulent meningitis appeared after this treatment of idiopathic trigeminal neuralgia.

A 60 year old man suffered left-sided trigeminal pain involving the third division for 6 years. He had received carbamazepine which had to be stopped because of gastritis intolerance and development of glaucoma. Then, radiofrequency trigeminal rhizotomy was performed. Two days later the patient became febrile with nausea, vomiting, headache and mild alteration of consciousness. Meningeal signs were present at physical examination. Cerebrospinal fluid (CSF) contained 1,180 white blood cells/mm³ with 75% granulocytes, glucose 2 mg/dl and protein 2.2 g/l. No microorganisms were seen on Gram stain. Culture of CSF produced the growth of Gram positive cocci which were later identified as *Streptococcus mutans*. Blood cultures were sterile. Empiric therapy was changed to penicillin G, which was given for 14 days. The patient was discharged with no further complications. He remains asymptomatic after 6 months of follow-up.

A 66 year old man had received carbamazepine for 14 months for right trigeminal pain in the distribution of the third division. As the pain was not controlled with medical treatment, radiofrequency trigeminal rhizotomy was performed. Ten hours after the procedure the patient developed

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/mm³ 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 abscess in another two.⁴ Usually characteristics of these meningitis are not mentioned.^{5,6} Silverberg and Britt⁷ described a streptococcal aetiology for a case of meningitis after radiofrequency trigeminal rhizotomy. In our experience, purulent meningitis has 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.¹ This is supported by the fact that a usual oral bacteria (*S. mutans*) was the aetiological 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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References

- 1 Sweet WH, Wepsic JG. Controlled thermocoagulation of trigeminal ganglion and rootlets for differential destruction of pain fibers. Part 1: Trigeminal neuralgia. *J Neurosurg* 1974;39:143-56.
- 2 Schwarcz JR. Percutaneous thermocontrolled differential retrogasserian rhizotomy for idiopathic trigeminal neuralgia. *Acta Neurochir* 1982;64:51-8.
- 3 Siegfried J. 500 percutaneous thermocoagulations of the gasserian ganglion for trigeminal pain. *Surg Neurol* 1977;8:126-31.
- 4 Sweet WH. The treatment of trigeminal neuralgia (Tic douloureux). *N Engl J Med* 1986;315:174-7.
- 5 Tew JM, Jr. Treatment of trigeminal neuralgia by percutaneous rhizotomy. In: Youmans JR, ed. *Neurological Surgery*, ed 2. Philadelphia: Saunders Co, 1982;Vol 6:3564-90.
- 6 Van Loveren H, Tew JM, Keller JK, et al. A 10-year experience in the treatment of trigeminal neuralgia. *J Neurosurg* 1982;57:757-64.
- 7 Silverberg GD, Britt RH. Percutaneous radiofrequency rhizotomy in the treatment of trigeminal neuralgia. *West J Med* 1978;129:97-100.

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

Sir: Patients with shoulder girdle weakness

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.

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.¹⁻³ 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