Letters


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High-resolution CT scan in pontine ataxic hemiparesis

Sir: In 1978, Fisher demonstrated pathologically that lacunar infarcts of the basis pontis can produce ataxic hemiparesis.1 Two cases with this syndrome caused by pontine infarcts, confirmed by CT scan, have been reported.2,3 We report a third case documented by high-resolution CT brain scan.

A hypertensive man, aged 40 yr, was admitted to hospital with sudden onset of weakness of the left limbs and unsteadiness of gait. Examination showed a very slight weakness of the left side of the face and grade 4/5 power in the left upper and lower extremities. The tendon jerks were brisker on the left, and a left Babinski’s sign was present. Dysmetria, intention tremor and adiadochokinesis on the left limbs were prominent. There was horizontal nystagmus on both sides on lateral gaze. No other neurological sign was seen. Somatosensory cortical evoked potentials to digital nerve stimulation were normal. The CSF, CT brain scan, echocardiography and vertebral angiography were normal. Examination a month later showed slight spasticity of the left limbs. Power and coordination were normal. A high-resolution CT brain scan demonstrated two small areas of the low density in the right basis pontis, at about the junction of the upper one third and lower two thirds of the pons (figure).

In this patient, the presence of nystagmus and the normal somatosensory cortical responses suggested a lesion in the pons rather than in the corona radiata or internal capsula.2 The first CT brain scan excluded pontine haemorrhage and a supratentorial lesion. The high resolution CT brain scan demonstrated clearly the existence of two small adjacent infarcts in the basis pontis one month later. Our case closely resembles case 1 of Fisher.1 The vessel most likely occluded is a lateral penetrating artery arising from short circumferential branches of the basilar artery.1,2 High-resolution CT brain scan allowed accurate clinico-anatomical correlation in this patient and confirmed Fisher’s original finding, which indicated that a lesion of the basis pontis at the junction of the upper one third and lower two thirds of pons.2 Why the ataxia is not bilateral is still unknown.1,2

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References


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Pseudoankylosis of the mandible following a fronto-temporal craniotomy

Sir: Head injuries with evidence of intracranial haemorrhage, intracranial aneurysms and tumours are the more common indications for a neurosurgical procedure. If the procedure involves sectioning of the temporalis muscle, fibrosis of the muscle give rise to complications during and after healing of the surgical wound. Since the temporalis muscle is one of the principal elevators of the mandible, a restriction of its action can result in pseudoankylosis of the mandible and atrophy of the muscle may also occur. Inspite of the large number of neurosurgical procedures undertaken, the incidence of pseudoankylosis is very low.1–4 A further case, in this instance following fronto-temporal craniotomy, is reported. It is of interest to note that while the previous cases in the literature were treated surgically, this patient was managed conservatively with satisfactory results.

In September, 1983, a 48-year-old white female patient was referred by a neurosurgeon for a maxillofacial surgical opinion regarding limitation in mouth opening which was affecting her mastication. The patient had undergone a right fronto-temporal craniotomy to clip an aneurysm arising from the internal carotid artery on that side in August, 1982. Immediately following this, the patient experienced limitation in opening her mouth which persisted. The past medical history indicated that she had been in good health until she developed the symptoms which led to neurosurgical intervention.

Clinical examination revealed an obvious depression in the region of the craniotomy site. On palpation, there was neither tenderness nor a sensation of stretching of fibres of the temporalis muscle when the patient attempted to open her mouth. She had a near full complement of permanent teeth and an interincisal opening of 10 mm. The mandible deviated slightly to the right side on opening. Palpation of the temporomandibular joint during movement of the mandible suggested that this was not a case of true ankylosis. Radiographic examination showed no bony changes in the joint or the surrounding structures and a diagnosis of pseudoankylosis of the mandible due to temporalis muscle fibrosis was made.

Although coronoidectomy to detach the temporalis muscle from the mandible was considered, it was decided initially to treat the patient by conservative measures. A trismus screw made out of hard acrylic was provided and instructions given to use it gradually to produce progressive opening of the mouth by distraction in the incisor region. Over a six month period, the mouth

Fig High resolution CT brain scan without contrast. Axial view.
opening increased by 28 mm with satisfactory lateral excursions of the mandible. The patient was pleased with her progress and no operative treatment was thought to be necessary. She had been advised to continue with the same self physiotherapeutic measures and will remain under review.

Coronoidection in order to release the temporalis muscle attachment from the mandible has been the preferred method of treatment for this condition, as temporalis myotomy is likely to produce only a limited period of improvement before fibrosis recommences at the site of myotomy. Coronoidection has been carried out in the cases previously reported.1-4 In the case of Sanders et al2 a bilateral procedure was carried out in order to release fibrosis which occurred following a transcortical incision and bifrontal craniotomy for excision of an olfactory meningioma.

In the case reported here, the patient was seen in the Maxillofacial Surgery Department about a year after the neurosurgical procedure with significant limitation to mandibular opening. However, institution of active jaw exercise utilising a trismus screw was seen to have produced significant improvement in condition. It is suggested that this condition be initially treated by active jaw opening exercises, if possible in the early neurosurgical postoperative period. By doing so, surgical correction of the disability may be avoided.

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References


Materials arising

Measurement of thermal thresholds

Sir: I read the papers 'An improved automated method for the measurement of thermal thresholds 1. normal subjects. . . .2. patients with peripheral neuropathy'1,2 with great interest, since quite independently, we also have been developing a microcomputer-based thermal testing system. By placing the power supply, signal converters, thermometer and other meters in a single peripheral device and using a portable computer, the system we have developed is transportable, but operates on very similar principles to that described by Jamal et al.1,2

The results obtained by these authors are impressive. There are, however two points arising from the discussion, which should be considered further.

Spatial summation is an important determinant of thermal sensitivity, particularly for warming, and values obtained for threshold are therefore very dependent on contact area.6 Our thermode has a contact area of 7.5 cm² and is sufficiently small to be used on the face and the dorsum of the hand. With this thermode we are obtaining higher values for threshold measurements than those given by Jamal et al, who used a 12.5 cm² thermode.3 Dyck et al use a 3.5 cm² thermode and give results from the dorsum of the foot for 303 healthy subjects.4 The details of thermode size and the number of patients studied are relevant to the comparison made by Jamal et al between their results and those of Dyck et al for inter-subject variation. One would expect lower values from a 12.5 cm² contact area thermode and the absolute range of thresholds cannot be entirely unrelated to the number of subjects in the selected age bands, studied by each group. By using a relatively large thermode, Jamal et al may have lost the discrimination necessary to demonstrate the variations in regional sensitivity, which other workers have found.3-6

To present their data on intra-individual variation, Jamal et al take the mean value of thresholds from all subjects together, measured on one or more occasion and give the maximum differences between these means, with the difference expressed as a percentage of the first observation.1 Fagius and Wahren6 presented their data for intra-individual variability as the range of differences found with paired observations in individuals, expressing the difference as a percentage of the first measurement. It was therefore incorrect of Jamal et al to use the figure of 150% for intra-individual variation from the work of Fagius and Wahren5 in direct comparison with theirs of 5%, since the two studies calculated variability in a different way.

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References


Jamal et al reply

We have read Dr Fowler's comments with interest. Dr Fowler suggests that the differences in results obtained by ourselves compared with Dyck et al are a consequence of thermode size. We agree that spatial summation determined in part by thermode
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