Operative findings on microsurgical exploration of the cerebello-pontine angle in trigeminal neuralgia

P RICHARDS,* H SHAWDON, R ILLINGWORTH*

From the Regional Department of Neurosurgery* and Department of Radiology, Central Middlesex Hospital, London, UK.

SUMMARY The anatomical findings in 52 patients undergoing posterior fossa exploration for idiopathic trigeminal neuralgia are described. Anatomical abnormalities in the cerebello-pontine angle were found in 46. The commonest was an arterial loop indenting or distorting the nerve at the root entry zone. In view of this the operative procedure has been changed from root section to microvascular decompression.

Although trigeminal neuralgia can occur in patients with multiple sclerosis and with tumours of the cerebello-pontine angle, in the majority the condition has been considered to be idiopathic. In 1934 Dandy first drew attention to the finding of anatomical and pathological structures impinging on the trigeminal nerve in the cerebello-pontine angle in 60% of 215 patients with apparently idiopathic trigeminal neuralgia, but it was 25 years before Gardner confirmed the findings and performed the first operation to dissect the compressing artery off the trigeminal nerve. Jannetta has since developed this operation to relieve trigeminal neuralgia, but only he and Apfelbaum have reported large series of such microvascular decompressions. Several smaller series have described the operative findings in the cerebello-pontine angle but the concept of an anatomical or pathological structure compressing the nerve and causing trigeminal neuralgia remains controversial. This paper describes the operative findings in the cerebello-pontine angle in 52 patients with clinically idiopathic trigeminal neuralgia and discusses these findings in relation to the treatment used.

Patients and methods

The patients were treated between June, 1972 and November, 1982. The sex, side and divisions involved, together with the age at onset and length of history before operation, are given in table 1. Pre-operative vertebral angiography was performed in 26 earlier patients and computed tomography (CT) in 24 later patients. Two patients had both investigations. All patients had taken carbamazepine but 41 had ceased to respond to maximum doses and 11 had developed side effects preventing continued treatment. The surgical procedure consisted of exploration of the cerebello-pontine angle through a small retro-mastoid craniectomy with the patient in the lateral decubitus position with hyperventilation anaesthesia. The operating microscope at magnifications of ×10 and ×16 was used for all cases and photographs were taken of the operative findings in 22.

Operative findings

These are detailed in table 2. Arterial loops were found in contact with the trigeminal nerve, usually indenting and displacing the nerve, in 37 (71%). In two other patients arterial loops were found close to the nerve but not in contact. The commonest vessel involved was the superior cerebellar artery (SCA) in 25 patients, usually making a downward loop medial to the nerve near the back of the petrous bone and then turning back upwards, often as a double loop, and producing compression at the point where the nerve enters the side of the pons. The compression at this point was often from the medial side, the distal part of the vessel then turning away upwards and posteriorly. Because the SCA usually lay a little deeper in the operative field than the nerve, it was not always immediately recognised when the nerve was exposed. An upward looping anterior inferior cerebellar artery was identified as the compressing vessel in two cases, while in seven the vessel could not be identified. Ectatic arteries were responsible for compression in three instances, two cases being caused by the basilar artery and one by the contralateral vertebral artery. Four patients were found to have veins indenting or displacing the nerve.
and there were three clinically unsuspected tumours (one each of meningioma, trigeminal neurinoma and epidermoid tumour). Thickened arachnoid was found in two cases and there were four negative explorations. These four were all in the first 25 patients and it may be that inexperience led to vascular compression or some other abnormality being missed.

**Radiological investigations**

The radiological investigations were reviewed by a neuroradiologist (HS) without access to clinical information. Twenty-one angiograms were available for review. The two ecstatic basilar arteries and a single ectatic vertebral artery were clearly shown. Two angiograms were performed in patients with tumours, but only the epidermoid tumour was demonstrated. Of the remaining 16 angiograms, nine showed the SCA on the side of the tumour looping lower than on the opposite side. In 24 patients who had CT scans only the patient with the epidermoid tumour had an abnormal scan.

We now feel that the vertebral angiography is not indicated in the routine pre-operative assessment for microvascular decompression and perform CT scanning with contrast in all patients to exclude clinically unsuspected cerebello-pontine angle tumours.

**Discussion**

Since Dandy’s original description of anatomical and pathological abnormalities occurring in the cerebello-pontine angle of patients with apparently idiopathic trigeminal neuralgia, there has been considerable controversy as to the significance of the findings. Working without the operating microscope Dandy found arterial loops in 30-7%, veins in contact with the nerve in 14% and tumours in 5-6%. Other vessels, both arteries and veins, were often found in close proximity to the trigeminal nerve, or sometimes in contact. However, like Jannetta we have not considered this relevant unless contact was at the point of entry of the nerve into the pons, the root entry zone.

These findings are similar to those of Jannetta, who in a series of 411 operated patients with trigeminal neuralgia found arterial contact with the nerve in 82-2%, venous contact alone in 13-9%, and unsuspected tumours in 3-6%. Apfelbaum in a series of 200 had similar findings of 82-2%, 12-5% and 3-0% for arteries, veins and tumours. Provost and Hardy described one case of vascular compression of the trigeminal root while Constans et al described a giant aneurysm of the basilar artery causing trigeminal neuralgia. Wegrzy n has described vascular compression in 68-7% of 80 cases of trigeminal neuralgia. The SCA was the responsible vessel in 24 of the 55 cases with vascular compression. There were seven cases with tumours in the cerebello-pontine angle and five had thickened arachnoid.

It has been argued that these findings are purely coincidental and unrelated to trigeminal neuralgia. Adams, Kaye and Teddy performed posterior fossa exploration in 57 patients with trigeminal neuralgia. They saw a vascular structure close to the trigeminal nerve in a large percentage of cases, but considered it relevant in only 11%. They performed microvascular decompression in those six patients and posterior root section in the others. Pertu is et al found arterial compression in only one case out of 14.

Three anatomical studies have been published examining the relationship of nerves and vessels in the cerebello-pontine angle of unselected cadavers without history of trigeminal neuralgia. Sunderland...
dissected 210 brains in subjects of whom no clinical
details were known. He found the superior cerebellar
artery looping downwards and indenting the nerve in
two specimens and perforating the nerve in one. In
three specimens the posterior inferior cerebellar
artery looped upwards and grooved the undersurface
of the nerve and in two specimens ectatic basilar
arteries reached and compressed the inner margin of
the sensory root. Hardy and Rhoton examined 50
nerves in 25 elderly cadavers. In 26 nerves there was
contact between the nerve and superior cerebellar
artery and in four there was contact with the anterior
inferior cerebellar artery. In only six cases was the
point of contact at the root entry zone of the nerve
and it was uncommon for the arterial contact to
produce distortion of the nerve. Mehta, Fatani and
Rao studied 60 nerves in 30 elderly cadavers and
found five superior cerebellar artery loops and three
anterior inferior cerebellar artery loops in contact
with the nerve. Distortion of the nerve due to
vascular compression was not seen.

These papers suggest that although arterial contact
with the trigeminal nerve in the cerebello-pontine
angle may be common in unselected cadavers, it is
rare to find vascular contact with the nerve at the root
entry zone causing indentation or distortion of the
nerve. It is this part of the nerve which has been stated
to be the important area for causation of trigeminal neuralgia and we believe that vascular
compression, indenting or distorting a nerve at this
point, is relevant to the trigeminal neuralgia in
the patients we have described. It may be argued that our
series is a sub-group within the broad spectrum of the
condition. However, comparing our patients with a
large series of 637 patients with trigeminal neuralgia
of Ruge, Brochner and Davis, they are similar in
being mainly in the middle and later years of life,
having a preponderance of female sufferers, more
right sided involvement and a distribution more
commonly in the lower part of the face. All had taken
carbamazepine but it became ineffective in 41, the
remaining 11 stopping the drug because of unac-
tetable side effects. This also suggests that vascular
loops are not found only in a sub-group of patients
who are refractory to carbamazepine.

In view of our belief that these anatomical
abnormalities are aetiologically relevant we have
changed our operative procedure from fractional
section in the earlier patients (22 cases) to decom-
pression in the later group (25 cases). Two patients
had decompression combined with small fractional
sections. The patients with tumours had them
removed, although in the case of the trigeminal
neurinoma subtotal removal was combined with
fractional section. Microvascular decompression was
achieved by dissecting the vessel free and interposing
non-absorbent material between the vessel and the
nerve. The material we have used is a small piece of
cotton gauze, measuring 1 cm x 0.5 cm, sometimes
folded double.

The second patient treated by decompression
failed to respond and was re-explored one week later.
The decompression was found to be inadequate but
in view of the age of the patient and that she had
undergone a second operation a fractional section
was performed. One other patient suffered some
pain in the face eight months after decompression.
This pain was not typical trigeminal neuralgia and
responded to dental treatment. The rest of the
patients who underwent microvascular decompression
remain pain-free between five years and six
months since operation (mean 30 months). There
was no mortality from decompression and no sensory
loss was produced. Deafness occurred post-oper-
atively on the side of the operation in one patient, and
shows no sign of recovering six months after the
procedure.

These findings appear to confirm the concept
proposed initially by Dandy, first utilised therapeu-
tically by Gardner and later extensively by
Jannetta of an extrinsic anatomical cause in the
cerebello-pontine angle of most patients with
trigeminal neuralgia. There is probably also an
additional intrinsic cause in the posterior root and
progressive loss of myelin with age leading to
artificial synapses has been suggested. Age may also
be a factor in producing vascular contact as the
cerebral arteries lengthen and become more tortu-
ous. This vascular contact at the root entry zone
where the central myelin sheath is replaced by the
Schwann cell layer may increase demyelination as
well as act as an irritant to the nerve. King and
Barnett using acute lesions of the trigeminal nerve
in anaesthetised cats have shown that an afferent
impulse in the damaged trigeminal nerve leads to a
volaey of delayed efferent impulses in the same nerve.
Normallly a tactile stimulus on the face sends an
impulse to the brainstem, leading to a return volley of
impulses for each stimulus. If this volley of impulses
reaches an artificial synapse at the trigeminal root
further impulses may be sent back to the brainstem,
resulting in pain until the neuron is exhausted.

Microvascular decompression appears to be an
effective and safe treatment for trigeminal neuralgia
and is the procedure of choice for fit patients who
wish to avoid the risk of unpleasant sensory symp-
toms which can follow any sensory loss. Relevant
structural anomalies such as we have described are
likely to be found in most patients. For patients not
concerned about sensory loss, and for those who are
old, unfit or fearful of operation, percutaneous
thermocoagulation will continue to be a very safe
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alternative.

We are grateful to Mrs V Roberts and Miss Anne Mahony for much secretarial assistance.

References