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Hemifacial spasm; a long term follow-up of patients treated by posterior fossa surgery and facial nerve wrapping

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SUMMARY Sixteen patients with hemifacial spasm were treated by posterior fossa surgery and wrapping of sponge around their facial nerve. A good or excellent result has been obtained in fourteen of the sixteen cases, and in seven cases followed for four years or longer. Two patients had a mild recurrence of their hemifacial spasm after a cessation of their spasm for eighteen months and two years following surgery. Contrary to the experience of other authors a definite vascular abnormality was found in only four cases. In the other twelve cases circumferential fibrosis about the nerve is again proposed as a mechanism for the effectiveness of the procedure.

Hemifacial spasm is a benign, but distressing movement disorder of the face, which occurs particularly in middle aged women and tends to be gradually progressive. Although it may be rarely associated with a definite cause, such as compression by a local tumour or basilar artery aneurysm, in the vast majority of cases the aetiology is still disputed. The treatment of this condition has involved either operations on the peripheral part of the facial nerve, such as percutaneous fractional thermolysis,[1] alcohol injections, partial facial nerve section, or facio hypoglossal or facio accessary anastomoses,[2] or treatment involving posterior fossa exploration.[3-10] The peripheral facial nerve operations produce a temporary relief of spasm while facial weakness is present, but as the weakness resolves the spasm often recurs. Janetta[7] has reported a good or excellent result in 76 out of 85 patients treated by posterior fossa exploration and separation of a vessel from the facial nerve. We now report the results of sixteen patients, including a long term follow up of eight cases of hemifacial spasm, treated by posterior fossa exploration and wrapping of the facial nerve.

Patients and methods

Sixteen patients with hemifacial spasm have been treated at the Radcliffe Infirmary since 1976, by posterior fossa exploration and wrapping of the facial nerve. Eight of these patients have been reported previously, and this study includes the long term follow up of these patients.[9] One patient (no 9) from that series, is not included in this report as he had a meningioma as the cause of his hemifacial spasm. He died three years after his operation from unrelated causes and had no recurrence of his spasm. The patients range in age from 33 to 76 and most are female. All patients presented with hemifacial spasm, which had begun in the orbicularis oculi muscle, and had progressed to adjacent muscles until the entire musculature innervated by the facial nerve was involved.

Operative technique

The operative technique has been described in a previous report.[9] In brief, it consisted of a unilateral posterior fossa craniectomy and thorough exploration of the seventh cranial nerve, using microneurosurgical techniques. When there was no evidence of any pathology the seventh nerve was dissected free and a small triangular piece of non-absorbable (Ivalon) sponge was introduced between the eighth and seventh nerves, and then wrapped around the seventh nerve.

Possible causes were found in four cases. The facial nerve had been split by an artery into two unequal parts in two cases, once by a small branch of the anterior inferior cerebellar artery (AICA), and in another case by the AICA itself. Both were treated by division of the smaller bundle of nerve roots and separation of the artery away from the remaining nerve by the sponge which was wrapped around the remaining larger part of the seventh nerve. A prominent vertebral artery impinged on the nerve in one patient and a piece of sponge was interposed between the nerve and the vessel. The posterior inferior cerebellar artery (PICA) was lying between the

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facial nerve and eighth nerve in one patient. However, this was not grooving the facial nerve and a piece of Ivalon sponge was wrapped around the facial nerve in such a way as to push the arterial loop away from the nerve.

Results

The table gives details of the patients and results of treatment. There was no mortality and no serious post-operative sequelae. One patient had a significant permanent facial weakness. This patient had severe pre-operative facial weakness, which was temporarily worse after surgery. A mild facial weakness was present in three other patients pre-operatively. In one patient it disappeared post-operatively, and in the other two it was unchanged from its pre-operative state. In one patient there was a mild permanent unilateral nerve deafness. Four other patients had a mild temporary conductive deafness post-operatively, probably because of small amounts of blood entering the middle ear via mastoid air cells. Cerebro-spinal fluid otorrhoea occurred in one patient for two days post-operatively, then ceased spontaneously. The follow up period extended from two months to five and a half years. Eight patients have been followed for longer than four years, including five for over five years. One patient was lost to follow up after four and a half years. In each case there has been marked diminution of the hemifacial spasm. Ten patients have no spasm, five of them having a follow up period of over four years. In assessing those patients who still have spasm, the patients and their closest relative were asked to give a grading of the severity of the spasm

Table *Results*

Patient no.	*Unit no.*	*Age (yr)*	*Sex*	*Date of Operation*	*Operative findings*	*Complications*	*Results*
1	39078	57	F	11 Feb 76	Prominent vertebral artery compressing facial nerve	Temporary vertigo and minimal facial weakness	No spasm
2	39143	76	F	29 Mar 76	No apparent abnormality	Increased pre-existing facial weakness; mild nerve deafness	No spasm
3	37476	55	F	12 Apr 76	No apparent abnormality	None	Immediate relief Mild blepharospasm returned after eighteen months. 70% improvement
4	37943	38	M	14 Sept 76	Artery found splitting nerve	None	Spasm ceased completely over several weeks
5	39976	41	F	30 Nov 76	No apparent abnormality	Temporary mild conductive deafness	Very slight blepharospasm returned after two years No increase since then 85% improvement
6	38339	40	F	12 May 76	No apparent abnormality	Temporary mild conductive deafness	Spasm ceased completely over several weeks
7	40815	42	F	1 Aug 77	No apparent abnormality	Temporary nausea and temporary mild conductive deafness	Occasional spasm 80% improvement No change over last three years
8	36685	59	F	8 Aug 77	No apparent abnormality	None	No spasm
9	42489	55	F	1 May 79	No apparent abnormality	Temporary mild conductive deafness	No spasm
10	43932	50	M	29 May 80	PICA between facial nerve and vestibular cochlear nerve, but not grooving facial nerve	Nil	No spasm. Gradually diminished over six months
11	43930	54	M	29 May 80	No apparent abnormality	Nil	Very occasional blepharospasm 95% improvement
12	44404	53	F	28 Oct 80	No apparent abnormality	Nil	No spasm
13	44881	38	F	22 Jan 81	No apparent abnormality	Mild pre-operative facial weakness gradually completely resolved	Mild occasional blepharospasm. 90% improvement
14	45032	44	F	16 Jan 81	No apparent abnormality	Slight CSF otorrhea for two days. Mild pre-existing facial weakness persists.	Spasm has gradually improved, and is continuing to do so 75% improvement
15	45457	33	F	23 June 81	No apparent abnormality	Nil	No spasm
16	45462	64	F	2 July 81	AICA split facial nerve	Nil	No spasm

in a percentage form compared with their pre-operative state. All of those patients and their relatives felt that the facial spasm had improved by at least 70%, being slight blepharospasm only in four cases.

The results were graded into three arbitary groups. The result was considered to be excellent if the patient had no spasm at the time of review, ten being in this category. The result was assessed as good if the patient had only a very mild persistent spasm, which was regarded by both the patient and their nearest relative as being at least an 80% improvement compared with their state before operation, four were in this group. The result was considered fair in two patients in whom it was thought that the spasm was at least 70% improved after operation. One of these patients had an interval free period of eighteen months, and the other patient has had a gradual resolution of her spasm since her operation and feels that she probably is still continuing to improve. The spasm returned following a period of two years absence in a further patient. This spasm, involving her eye only, was regarded as being 85% better than her pre-operative state and has not progressed over three years. That is, there was a recurrence of spasm in two patients following a period of absence after operation.

All four patients with a definite pathological finding at operation now have no spasm. In two of these patients the spasm has gradually resolved over several weeks. However, this comparison with those patients who had no evidence of pathology is not significant. Four patients did not experience an immediate cessation of their facial spasm, but rather had a gradual improvement over weeks to months until the spasm disappeared completely in three patients and has not recurred.

Discussion

The aetiology and treatment of hemifacial spasm is still controversial. While some theories postulate a central or brain stem mechanism for the origin of hemifacial spasm,[11-13] others have suggested that the causative lesion is within the facial nerve, either within the posterior fossa,[3 5 6 14 15] or more distally.[16 17] Vascular compression of the facial nerve within the posterior fossa was seen in 14 of 19 patients described by Gardiner and Sava,[3] and in 84 of the 85 patients described by Janetta.[7] He has reported a good or excellent result in 76 of 85 patients operated upon for hemifacial spasm in which he has separated a vessel from the nerve. His patients were first operated upon in 1966, and a comprehensive review of the long term results would be most useful and is awaited. This vascular compression has been recorded by other authors.[4 8 10] but the problem remains that there may be varying interpretations of what exactly constitutes a pathologically placed vessel.[18]

We are not, however, inclined to believe that the effect of the operation is due to separation of a compressing vessel from the nerve, as in only four of our 16 patients was there a definite vascular compression and there was no significant difference in the outcome between that group and those with no pathology demonstrable. We believe that the relief of spasm is brought about rather by the mild degree of trauma associated with wrapping the facial nerve with non-absorbable sponge and that the delayed slow reduction in spasm in four patients could be attributable to fibrosis occurring around the nerve. Whilst we do not dispute that definite compression of the nerve might produce, or assist, in the development of hemifacial spasm, we believe that this is an unusual situation, rather than being the cause in nearly all cases, as suggested by Janetta.[7]

We are not able to explain the sudden cessation of hemifacial spasm in those 11 patients who had no definite vascular abnormality and whose spasm ceased immediately after operation. However, it is most probably due to manipulation of the facial nerve producing an immediate, but slight degree of trauma sufficient to abort the spasm. After some weeks the circumferential fibrosis then takes over. In support of this contention is the immediate good result seen in trigeminal neuralgia after the "compression decompression" operation of Taarnhøj.[19]

The good or excellent result obtained in 14 of our 16 cases and which has been maintained in seven of the eight patients followed for four years or more shows that this technique is effective in the long term, and the relative paucity of complications indicates that it is a safe operation.

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