Journal of Neurology, Neurosurgery, and Psychiatry 1996;**60**:72–77

Hemifacial spasm: a prospective long term follow up of 83 cases treated by microvascular decompression at two neurosurgical centres in the United Kingdom

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Abstract

***Objective*—To evaluate the use of microvascular decompression (MVD) for the treatment of hemifacial spasm (HFS).**

***Methods*—Eighty three patients with HFS who underwent MVD via a suboccipital craniectomy are presented.**

***Results*—Seventy two out of seventy eight patients available for follow up remained free of any spasms at a mean follow up period of eight years. Two patients continued to have minor intermittent muscle twitches and three had recurrence of HFS. One patient's operation was not completed. Twenty had a transient complication and eight were left with permanent postoperative deficits, the commonest being unilateral sensorineural deafness. Seventy one patients declared themselves satisfied with the procedure. A causative vessel was found on the root exit zone of the seventh cranial nerve in 81 patients.**

***Conclusion*—The procedure seems to provide lasting relief for most patients. The correct operative technique is essential if complications are to be avoided.**

(*J Neurol Neurosurg Psychiatry* 1996;**60**:72–77)

Keywords: hemifacial spasm; microvascular decompression

Hemifacial spasm (HFS) is a condition consisting of unilateral paroxysmal involuntary tonic clonic spasms occurring in the muscles innervated by the facial nerve. The involuntary contractions usually begin in the orbicularis oculi muscle and gradually spread to the other muscles of facial expression.

It is a rare condition. The average annual incidence has been reported as 0·74 per 100 000 for men and 0·81 per 100 000 for women, and the average prevalence as 7·4 per 100 000 in men and 14·5 per 100 000 in women for the white population of the United States.[1] Thus it is not often encountered in neurosurgical practice and results have mainly been reported by tertiary referral centres in the United States.[2-4]

Causative lesions that have been reported include aneurysms of the posterior circulation,[5] epidermoid tumours,[6] and arteriovenous malformations.[7] However, most cases are due to arteries, usually the posterior inferior cerebellar artery, compressing the facial nerve at the root exit zone.[8-10]

Despite the use of microvascular decompression (MVD) to treat HFS for many years there are still only very limited results of long term follow up. We present 83 cases of HFS treated by MVD at two neurosurgical centres by two neurosurgeons who undertake this procedure only as part of a wider neurosurgical practice, and we discuss the relative merits of MVD compared with other treatments.

Patients and methods

Eighty three patients were treated at two neurosurgical centres in the United Kingdom between 1978 and 1992. Forty seven were women and 36 were men, with ages ranging from 25 to 78 years (mean 53·3 years) at the time of operation. The spasms were on the right side in 42 and the left side in 41 patients. The preoperative duration of symptoms was from eight months to 25 years with a mean duration of 6·15 years. As well as the muscular spasms, other neurological findings consistent with the diagnosis were present in 50 patients. Table 1 summarises these.

After investigations including brain CT (vertebral angiography in the earlier cases) to exclude an underlying causative mass lesion, MVD was performed. The posterior fossa approach as described by Jannetta[11] and modified by Sugita[12] was used. The patients were placed in the lateral decubitus position and a small anterior inferior craniectomy, about 3 cm in diameter, extending up to the posterior margin of the inferior part of the sigmoid sinus was performed. On opening the dura, the ninth and 10th cranial nerves were exposed as they passed from the jugular foramen towards the side of the medulla. These nerves were followed with careful microsurgical dissection back to the side of the medulla by freeing the

Table 1 Preoperative neurological signs in 83 patients with HFS

Neurological signs	*No of patients*
Seventh nerve paresis alone	32
Seventh nerve synkinesis alone	7
Seventh nerve paresis with synkinesis	9
Hemifacial spasm with "trigeminal neuralgia"	2
Total	50

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Received 18 April 1995
and in revised form
30 August 1995
Accepted 6 September 1995

nerves first from the flocculus of the cerebellum, and then, more medially, from the choroid plexus of the lateral recess of the fourth ventricle. At this point the eighth nerve is found passing transversely across the line of dissection. The seventh nerve is found there as a white elongated triangle, lying slightly caudal and medial to the eighth nerve. The compressing artery was usually discovered before this point was reached, looping across the side of the brainstem. Little dissection was required to lift the artery away from the seventh nerve, which is usually deeply grooved at the point where it leaves the side of the brainstem. The causative artery was mobilised and held away from the nerve by a piece of Ivalon sponge, lintine patty, or shredded teflon.

Intraoperative monitoring of auditory and facial nerve functions has not been used.

All operative procedures were undertaken by RDI or JJ. Assessment of the results was conducted by outpatient follow up and recently by a standard questionnaire sent to each patient. Information regarding the exact vascular relation to the facial nerve was obtained from the operative notes. If the patient did not respond, an attempt was made to contact them by telephone or by an outpatient interview. Information on three deceased patients was obtained from family or general medical practitioners. In 78 of 83 patients follow up was achieved but the other five could not be contacted. Three are resident overseas and have not responded to letters, and two have not been contacted in this country.

Results

OPERATIVE FINDINGS

A vascular structure was found to be compressing the facial nerve, which was grooved transversely at the root exit zone, in 81 (97·5%) out of 83 patients in our series. The compressing vessel in most cases was the posterior inferior cerebellar artery but mostly it was not possible to identify the artery with confidence (table 2). In 12 patients two separate arterial loops were causing compression and in one patient three loops. If two or more loops were found then all were mobilised and held away from the facial nerve. One case had to be abandoned because of an unstable blood pressure due to haemorrhage from an accessory occipital sinus before the facial nerve was explored.

Table 2 Source of seventh nerve compression by a vascular structure in 82 patients with HFS*

Compressing vessel	*Arteries*
Posterior inferior cerebellar artery	49
Anterior inferior cerebellar artery	14
Ectatic vertebral artery	8
Ectatic basilar artery	2
Unknown vessel	16
Vein alone	5
No vascular compression	1
Total	95

*Operation abandoned in one patient.

Table 3 Timing for 10 patients who had a delay to total cessation of HFS after MVD

Delay to total cessation of spasm	*Length of time in years of follow up*
4 days	14·3
4 days	7·1
7 days	5·5
7 days	8·0
7 days	7·0
2 weeks	7·7
3 weeks	8·8
3 weeks	13·0
2 months	16·8
7 months	7·3

RELIEF OF SYMPTOMS

In the 78 patients in whom long term follow up was achieved, 72 (92%) were completely relieved of their HFS by MVD. Spasms ceased immediately in 62 patients and after a delay of up to seven months in 10 patients (table 3). In other patients spasms often stopped immediately after the operation but then returned for one or two days before finally ceasing. Two patients had major improvement but continued to have minimal twitching around the eye on the side of the previous spasms.

In response to the questionnaire, 71 of 78 (91%) of the patients declared themselves satisfied with the results of the operation.

Four patients had an intermittent drumming sound in the ear on the affected side before the operation. This was relieved by the operation in all cases.

Three cases of HFS treated by MVD recurred and one patient's spasm remained unchanged in the immediate postoperative period. The recurrences occurred at six, nine, and 24 months. The first patient chose to have no further treatment and the condition spontaneously resolved. The second patient delayed further intervention until four years from the original procedure and was re-explored uneventfully. No causative vessel was found. The spasm improved but recurred two years later. No further surgery was undertaken. The last patient had a recurrence of HFS at two years. A further exploration was undertaken and the facial nerve was found to have been inadequately decompressed from the ectatic basilar artery at the original procedure. Further packing was introduced to decompress the nerve and the patient was relieved of spasm for two years, since when he has been lost to follow up and thus cannot be included in the final results analysis.

In the patient whose HFS remained unchanged in the immediate postoperative period, a re-exploration two weeks after the first procedure found a small loop of the posterior inferior cerebellar artery at the root exit zone that had been missed at the first operation. The root exit zone was decompressed with complete relief of the spasms. The patient's spasm returned after two years but he declined further surgery.

In the patient whose operation was abandoned before the facial nerve was exposed, the spasms continued and showed progressive worsening.

There have been no recurrences during a mean follow up period of 9·6 (range 5·5–16·8)

Table 4 Postoperative complications in 82 patients after MVD for HFS*

Postoperative complications	*No (%)*
Permanent:	
Seventh nerve palsy	1 (1·2)
Complete unilateral sensorineural deafness	4 (4·7)
Partial unilateral sensorineural deafness	3 (3·5)
Total	8 (9·4)
Temporary:	
Seventh nerve paresis	9 (10·6)
Deafness	4 (4·7)
Subjective facial numbness	1 (1·2)
Vertigo or dizziness	4 (4·7)
CSF leak	1 (1·2)
Meningitis	1 (1·2)
Total	20 (23·5)

*Eighty five microvascular decompressions were performed on 82 patients.

years, in the 10 patients who had a delayed response to the operation. This is by contrast with the experience of Barker *et al* who found that patients with evidence of postoperative spasm, even for short periods, had a higher incidence of recurrence.[4]

Five patients were lost to long term follow up. One patient has not been seen since discharge when he was free of spasms and one patient, who was free of HFS after MVD, was only followed up for one month. Two further patients, one seen two years and the other 18 months after their operation were free of spasms. The fifth case lost to follow up has been described above.

ASSOCIATION WITH TRIGEMINAL NEURALGIA

Two patients in our series had both HFS and trigeminal neuralgia. This association has been previously described.[13] Both patients underwent MVD for each disease with resolution of symptoms in the immediate postoperative period. Only one patient was available to follow up and was still asymptomatic.

FOLLOW UP

The mean time of follow up in our series is eight (range 2–17·25) years with 43 patients (54%) followed up for a minimum of eight years.

COMPLICATIONS

There was no operative mortality in this series.

Complications occurred in 28 (32·9%) patients. There were 20 (23·5%) with a transient complication and subsequent complete recovery. Eight (9·4%) had a permanent postoperative deficit. Table 4 illustrates the postoperative complications.

The commonest permanent neurological deficit after the procedure was unilateral sensorineural deafness, which occurred in four patients. Three other patients experienced a mild permanent reduction in hearing on the operated side. One patient had a permanent postoperative facial palsy. One patient had subjective but not objective facial numbness in the distribution of the first and second divisions of the trigeminal nerve on the operated side.

Transient facial weakness occurred in nine patients. This was often delayed for two to three weeks after the operation and all fully recovered after a mean duration of two months (range 10 days to three months). A mild temporary hearing reduction was noted by four patients and four complained of temporary postoperative dizziness and vertigo. There was a single case of meningitis and one CSF leak that spontaneously resolved. One patient returned six weeks after the MVD with symptoms from a subtentorial arachnoid cyst on the contralateral side. The cyst was not present when the preoperative scan was reviewed. The symptoms were relieved by a cystoperitoneal shunt.

In one case the procedure was abandoned, due to an unstable blood pressure from profuse bleeding at the cervico-occipital junction from an accessory occipital venous sinus. Exploration of the seventh nerve was not carried out.

Discussion

HFS is a condition consisting of unilateral paroxysmal involuntary contractions of the muscles innervated by the facial nerve. The condition can be very disabling with personal embarrassment resulting in self imposed social isolation, and problems with personal interaction interfering with employment. In some cases the eye on the affected side may remain closed sufficiently long to interfere with activities such as driving, which require binocular vision.

Medical treatment of HFS, using carbamazepine, clonazepam, or orphenedrine is largely disappointing.[14-16] Treatment with botulinum A toxin for facial dyskinesiae including HFS is well known.[17-19] This technique requires multiple injections into the muscles and is effective for two to three months, when it must be repeated. Thus injections are for an indefinite time. The quoted success rate for significant relief of symptoms is 70–75%.[18 19] The procedure can give rise to complications including ptosis, exposure keratitis, diplopia, epiphora, drooling, and strabismus.[20] Excess dosage at the time of injection can result in temporary facial paralysis. The reported complication rate with this technique is 2% to 14% per treatment.[20 21] The method has the major advantage of saving patients the risk of major intracranial surgery and is therefore often preferred to MVD by both patients and physicians. The need to repeat the injections at frequent intervals is however, a considerable (and expensive) disadvantage. Also, some patients undergoing MVD after a course of injections have an impaired cosmetic effect due to a loss of fine muscle movement which is often permanent. Apfelbaum[22] makes a strong point regarding the difference in quality of life if a patient is not constantly reminded of his symptoms. Although raised as an issue in the management of trigeminal neuralgia, this is equally applicable to the management of HFS.

The only curative treatment is an operation. Previous surgical treatment was aimed at destructive procedures. Total or partial destruction of the peripheral trunks or branches of the facial nerve by surgical expo-

Table 5 Long term results in published series of MVD for HFS

Author (ref)	*No in series*	*Operations*	*Follow up (mean)*	*Complete relief*	*Partial relief*	*No benefit*	*Recurrence*	*Vascular cross compression*
Auger *et al*[3]	54	NA	range 3 months–10y (3·9y)	44 (81%)	5 (9%)	5 (9%)	6 (11%)	53 (98%)
Barker *et al*[4]	612	612	range 1–20y (8y)	86%	5%	9%	NA	612 (100%)
Iwakuma *et al*[26]	74	NA	1 month–3y	72 (97%)	1 (1·3%)	1 (1·3%)	1 (1·3%)	73 (98·6%)
Loeser and Chen[10] (personal series)	20	21	range 4 months–7y (2·5y)	17 (85%)		3 (15%)	5 (25%)	75%
Loeser and Chen[10] (literature review)	433	450	NA	88%	5%	2%	19 (4%)	NA
Panagopoulos *et al*[36]	29	NA	6 months–8·5y	26 (90%)	NA	3 (10%)	NA	29 (100%)
Piatt and Wilkins[37]	48	NA	(3·5y)	30 (62·5%)	12 (25%)	NA	6 (12·5%)	46 (96%)
Wilkins (review)[38]	41	NA	range 5–12y (8·1y)	30 (73%)	6 (15%)	NA	5 (12%)	NA
Illingworth *et al* (this paper)	83	86	range 2–17·25y (8y)	72 (92·2%)*	2 (2·6%)*	1 (1·3%)*	3 (4%)*	81 (97·5%)

*Percentage calculations based on 78 patients for long term follow up.
NA = not available.

sure or percutaneous glycerol injection substitutes facial weakness for abnormal facial movement. Relief from the spasms was often immediate but returned after three to six months due to nerve regeneration.[23] Division of the facial nerve beyond the stylomastoid foramen with a faciohypoglossal anastomosis has been carried out. There is a facial palsy for two months then recovery to reasonable symmetry at rest. Active movement using the tongue can be learnt but mass contraction movements can remain troublesome. In a series of 13 patients treated by this method 12 were satisfied, although the duration of follow up was not specified.[24]

Wakasugi[25] treated 239 patients by placing a needle percutaneously into the facial nerve trunk at the stylomastoid foramen and performing a radiofrequency lesion. The facial palsy disappeared at one to two months in most but only 47% of patients were relieved of their spasms at eight months. Iwakuma *et al*[26] noted that 30 patients who had previously received this treatment, refused further injections because of the severe pain, excessive paralysis, and early return of the spasms. Fisch and Esslen favour a selective neurectomy of the facial nerve branches involved in the spasms.[27] Using this method Iwakuma *et al*[26] found that HFS recurred at one year in 60% of patients and only 15% had relief of spasm for two to three years. Ludman and Choa[28] described their experience with a group of 62 patients, all with HFS, in whom the facial nerve was exposed within the middle ear, via a transtympanic route. A hook was then pushed through the thickness of the facial nerve and moved longitudinally. Between three and six punctures of the facial nerve were made in this way. Analysis of the results showed a mean remission of 9·3 months after the first operation but at subsequent procedures the benefit was less and less apparent, with mean remission falling to 5·6 months at the second, 2·9 months at the third, and only one month at the fourth procedure.

The pathophysiology of HFS and whether the compressing vessel is causative is debated. Jannetta maintains that the vascular cross compression is the cause of the spasm and Moller and Jannetta[29 30] and Nielsen and Jannetta[31] have performed preoperative, intraoperative, and postoperative recordings in patient cohorts to try to support this hypothesis.[29-31] Several other authors agree with this explanation.[32 33] However, the concept is challenged by Adams and Kaye[34] and Adams,[35] who regard the relief of the spasm as secondary to a mild degree of operative trauma and later fibrosis around the nerve.

Despite this and because of the unsatisfactory results from the operations described above, MVD as described and popularised by Jannetta has become the preferred operative treatment for HFS.

Table 5 summarises the operative findings and postoperative results of MVD for HFS in other series.

Barker *et al*[4] reported long term results for MVD with 86% complete and 5% partial response rates (> 75% relief of symptoms), with a mean follow up of eight (range 1–20) years. Iwakuma *et al* reported a series of 74 patients with a follow up ranging from one month to three years.[26] Seventy two (97%) had complete relief, one patient partial relief, in one patient there was no benefit, and there was one recurrence.

Panagopoulos *et al*[36] performed the operation on 29 patients with HFS. The follow up in this series ranged from six months to 8·5 years, no mean value being mentioned. Twenty six patients (90%) had complete relief of their spasms and three failed to benefit.

Piatt and Wilkins[37] described a series of 48 patients in whom 62·5% had an excellent and 25% a good response. There were six failures or recurrences. Forty one patients followed up for a mean of 8·1 years showed 30 (73%) patients with a continuing excellent response and six (16%) with a good response. Only five patients had significant residual or recurrent HFS.[38]

Auger *et al*[3] reported a series of 54 cases, followed up for a mean of 3·9 years, with 44 patients having a complete and five a partial response. Five patients had no benefit and in six patients the spasms recurred.

By comparison, we report the data on 83 cases, of whom 78 were available for long-term follow up with a mean of 8·0 years. Complete relief of symptoms was obtained in 72 (92·5%) and two had partial relief. In one case there was no benefit and three patients had a recurrence (table 5).

Table 6 Complications encountered in published series for patients undergoing MVD for HFS

Author	*No in series*	*No of completed operations*	*No of patients with no complications*	*Total permanent complications*	*Total temporary complications*	*Seventh nerve permanent*	*Seventh nerve temporary*	*Eighth nerve permanent*	*Eighth nerve temporary*
Auger *et al*[3]	54	NA	35 (65%)	15 (28%)*	18 (33%)*	2 (4%)	9 (17%)	8 (15%)	NA
Barker *et al*[4]	648	648	NA	NA	NA	22 (3·4%)	25 (3·9%)	21 (3·3%)	0
Iwakuma *et al*[26]	74	NA	NA	NA	NA	NA	NA	12 (16%)	
Loeser and Chen[16] (personal series)	20	21	12 (57%)	5 (24%)	4 (19%)	1 (4·8%)	1 (4·8%)	4 (19·2%)	
Loeser and Chen[16] (literature review)	433	450	341 (76%)	71 (16%)	38 (8%)	26 (6%)		v58 (13%)	
Panagopoulos *et al*[36]	29	NA	21 (72·5%)	8 (26%)*	5 (17%)*	1 (3·5%)	3 (29%)	4 (14%)	1 (3·5%)
Piatt and Wilkins[37]	48	NA	28 (60·5%)	6 (12·5%)	14 (30%)	0	2 (4%)	2 (4%)	1 (2%)
Illingworth *et al* (this paper)	83	85†	57 (67·1%)	8 (9·4%)	20 (23·5%)	1 (1·2%)	9 (10·6%)	7 (8·3%)‡	4 (4·7%)§

*A patient may have had more than one post-operative complication.
†One procedure abandoned.
‡3% in the last 63 patients.
§1·6% in the last 63 patients.
NA = not available.

MVD for trigeminal neuralgia was followed by a long term recurrence rate of 5% per year in the series of Burchiel *et al*[39] and this finding is supported by Breeze and Ignelzi,[10] who noted a 13% recurrence in a series of 52 patients followed up for an average of 23 (range 1–53) months. In our series of patients with HFS treated by MVD a late recurrence rate has not been found.

Table 6 describes the complications encountered in series published to date. The most frequent complication was damage to the seventh and eighth cranial nerves within the cerebellopontine angle.

Hanakita and Kondo[11] reported the serious complications encountered in a series of 278 patients who underwent MVD, of which 239 operations were for HFS. They encountered nine complications including an acute intracerebellar haematoma, delayed cerebellar swelling, status epilepticus due to supratentorial air, and immediate brainstem infarction, none of which were found in our series. Using intraoperative monitoring of auditory brain stem evoked potentials, the rate of postoperative hearing disturbance was 4% in this series. Other complications were not discussed.

In our series there was a temporary complication rate of 23·5%. A permanent neurological deficit occurred in eight (9·4%) patients, the commonest being unilateral sensorineural deafness.

Three quarters of the complications occurred in the first 20 patients. In the other 63 patients the total, comprising both permanent and temporary complications, fell to 11%, by contrast with 32·9% for the entire series. As experience increased it was realised that the only significant vascular compression occurred at the root exit zone. This meant that there was no need to explore the full subarachnoid course of the seventh nerve, and much less dissection was required. The operation therefore can be very precisely targeted to expose the root exit zone of the seventh nerve as described earlier. The importance of this approach is that it brings the surgeon directly down on to the root exit zone and because the seventh and eighth nerves are approached transversely to the line of the nerves. This approach, using a narrow retractor blade to lift the cerebellum and choroid plexus off the ninth and tenth nerves, therefore avoids any axial traction on the seventh and eighth nerves. It is this axial traction which can cause deafness, and the approach described should avoid this distressing complication. This finding is supported by the fact that in our series the permanent deafness rate fell to 3% and the partial deafness rate to 1·6% in the final 63 patients.

Further refinements suggested by Jannetta are the intraoperative monitoring of audiographic responses to protect against inadvertent eighth nerve damage,[12] and by monitoring the facial EMG, and continuing the operation until lateral spread responses are eliminated to improve the cure rate.[43] Neither of us had brain stem auditory evoked potentials or facial EMG available during the series. We think that evoked potentials are only of limited value to protect hearing because of the time necessary to summate multiple responses, and that the method is no substitute for the surgical technique we have described. The surgical procedure requires complete decompression of the seventh nerve root entry zone by relieving vascular compression. The use of intraoperative facial nerve EMG does change this requirement. However desirable these monitoring techniques may be we do not think that they are mandatory. The high level of relief of HFS and the low incidence of deafness in the last 63 patients once the technique was refined are compatible with other series (tables 5 and 6) and, we think, support our opinion.

The tensor tympani and stapedius muscles are innervated by the facial nerve. When an acoustic stimulus is applied to the contralateral ear, the stapedius muscle contracts and compliance decreases. This is the acoustic-stapedial reflex. Diamant *et al*[44] reported two patients in whom stapedius muscle contractions occurred synchronously with the facial twitches. Kim and Fukushima reported the results of impedance audiometery in a group of 15 patients who had simultaneous "tympanic noise"—which may be the same as the drumming described by four of our patients—and HFS.[45] The abnormalities found—namely, tonic contraction of the stapedius muscle during tonic facial spasm and an absence of the stapedial reflex—were abolished by surgery. In a similar manner to the series of Kim and Fukushima,[45] all our patients were free of this distressing symptom after surgery and have remained so at the time of follow up.

MVD of the facial nerve performed by experienced surgeons using the correct approach to the root exit zone is a relatively safe procedure and gives the patient the only chance of permanent relief. It should be offered to all patients with HFS, who are deemed fit for surgery. The alternative therapeutic option of botulinum toxin should be available for patients who are unfit or unwilling to undergo major surgery.

These two methods are very different approaches to the treatment of HFS. Both have advantages and disadvantages. Ideally all patients with this condition should be provided with sufficiently detailed information to allow them to make a well informed and considered decision.

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