MICROVASCULAR DECOMPRESSION FOR TREATMENT OF HEMIFACIAL SPASM
Operative techniques and results in 40 patients

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SUMMARY :

The clinical and operative findings are reviewed in 40 patients with hemifacial spasm. Vascular cross-compression of the facial nerve adjacent to the brain stem was noted in 39 patients. In 38 the abnormality was usually by an arterial loop. In one patient, osseus contact was discovered and drilled away. Mortality was zero and, morbidity was minimal. Complete cure of spasm was achieved in 36 cases (90%) and a further 2 patients improved.

KEY WORDS :

Facial nerve; Hemifacial spasma; Microvascular decompression; Root/exit zone

INTRODUCTION

Classical hemifacial spasm (HFS), is a disorder characterized by hyperactive dysfunction of the facial nerve leading to progressive involuntary twitching of the muscles of face. Over a period of months or years, mild progressive muscular weakness may be noted and synkinesis often occurs. Pain is not a prominent symptom, although patients with the tonus phenomenon may develop an aching discomfort. The spasm is most often unilateral, occurs more commonly in women, and affects the left side more often than the right. The problem is often confused with a nervous habit spasm or "tic", and patients are frequently sent for psychiatric help. This benign disorder may become a serious social and psychological disability.

Treatment of this condition has involved either operations on the peripheral part of the facial nerve, such as percutaneous fractional thermolysis, injection of alcohol or botulinum toxin, partial nerve section or faciohypoglossal or facioaccessory anastomoses or treatment involving posterior fossa exploration (4,7,11,12,16,22,34).

Optimal neurosurgical management of HFS must relieve symptoms and preserve neurological function. There is now extensive evidence that the spasms are usually caused by cross compression of the facial nerve at its root/exit zone (REZ) (6,10,11,19,20,24,28). Microvascular decompression (MVD) of the REZ of the affected nerve more closely approaches this ideal than any other treatment in the long history of treatment of this condition.

The purpose of this report is to analyze the operative findings and results of MVD within the posterior fossa in our series of 40 patients with HFS and to compare our observations with those of other published series.

PATIENTS AND METHODS

From 1983 to January, 1990, 40 patients with HFS underwent MVD by one surgeon (HBC) in the Department of Neurosurgery, Frenchay Hospital, Bristol, UK. Ages at operation ranged from 30 to 77 years, with a mean of 53 years, and duration of symptoms averaged 7 years, with range of 1 to 17 years. Fourteen patients had previously undergone other types of operation without relief and mild preoperative facial weakness was noted in 15 patients. Twentyeight patients had spasms on the left side, 11 spasms on the right, and 1 had bilateral spasms (Table 1). None of the patients had a history of Bell's palsy or other predisposing factors.

The diagnosis in each case was made on a clinical basis. In some cases computed tomographic (CT) scanning, audiometry and brain stem evoked potential studies were performed before operation.

The techniques of MVD are well known and are described here only briefly. These patients were operated on in the lateral park bench position using the keyhole microsurgical approach (12). A small (2-2.5 cm) retromasdoid craniectomy was performed through a linear, vertical incision. After opening the dura the subarachnoid cisterns were opened and the cerebellar flocculus was gently elevated to expose the glossopharyngeal and vagus nerves. In the last 16 patients the brain stem auditory evoked potentials (BAEP) were monitored throughout. A tapered

microsurgical retractor was placed on the choroid plexus of the lateral recess of the 4th ventricle in order to expose the 7th nerve REZ at the ponto-medullary junction. Arterial contact was asserted and decompression of the nerve was accomplished by transpositioning and fixing the offending vessels with various techniques (Table 2). Large, doligoectatic arterial loops needed repositioning with silastic slings (29).

TABLE 1 : Characteristics of Patients With HFS

SEX	
Male (no.cases)	15
Female "	25
Ratio	M:F-1
AGE AT OPERATION (yr)	
Mean	53
Range	30-77
DURATION OF SYMPTOMS (yr)	
Median	2.5
Range	1-17
PREVIOUS TREATMENT (no. cases)	14
ASSOCIATED FACIAL WEAKNESS	15
LATERALITY (no. cases)	
Right	11
Left	28
Bilateral	1

TABLE 2

Operative Techinique	HFS
Ivalon Sponge	20
Teflon wool Sling	11
Silastic Sling	5
Tissel Fibrin Glue	1
Combined Method (Sling and Tissel)	3

OPERATIVE FINDINGS AND RESULTS

Exploration of the REZ revealed vascular cross-compression of the 7th nerve in 38 of 40 cases, 1 case of osseus contact and 1 case where no significant pathological condition was found. The impinging vessels were the anterior inferior cerebellar artery (AICA) in 15 cases, posterior inferior cerebellar artery (PICA) 13 cases and the vertebral artery in the other cases (Table 3). MVD proved to be of benefit in 38 of the 40 cases; 36 patients (90%) were completely relieved of spasm. Two patients failed to benefit from the operation (Table 4). The duration of postoperative follow-up ranged from 1 month to 7 years with mean of 2.5 years.

There were 4 patients with loss of hearing which was compelete in only 2, and 3 patients had 7th nerve paresis, which was permanent in one. Thus, of 40 operations 31 were free of complications, 3 resulted in longterm deficits, and 6 led to transient dysfunction. There was no mortality (Table 5).

TABLE 3

Operative Findings	HFS
AICA	16
PICA	13
VA	5
VA-AICA	1
VA-PICA	3
Osseous Contact	1
No Compression	1

TABLE 4
Symptomatic Outcome at last Follow-up

HFS (Mean Follow-up 2.5 yr)	
Good	36
Improved	2
Failure	2
TOTAL	40

TABLE 5

Complication		HFS
Deafness	Compelet	2
	Partial	3
Tinnitus		1
Facial Weakness	Temporary	2
	Permanent	1
Mild Bulbar Paresis		
	Temporary	2
TOTAL : 40 cases		11

DISCUSSION

Hemifacial spasm is a distressing, relatively uncommon, condition characterized by the insidious development of paroxysmal, involuntary, unilateral hyperkinetic facial movements. It almost always starts as a mild intermittent twitch of the orbicularis oculi. The abnormal movements gradually progress down the afflicted side, sometimes sparing the musculi frontalis to include perioral and other facial muscles, although the platysma is frequently involved. Spontaneous remission is rare, and with the passage of time the spasms tend to become increasingly frequent and vigorous.

The first observation as to the aetiology of HFS was made in 1875 by Schultz. He recorded the case of a man with typical HFS of approximately a year's

duration, who died as a result of pulmonary tuberculosis. Autopsy examination of the brain disclosed an aneurysm of the left vertebral artery which lay against the left 7th and 8th nerves (31).

Almost a century after the first clear description of HFS, the pathogenesis of this motor disorder remains in dispute. Much of the current controversy centres around a hypothesis originally put forth by Campbell and Keedy that vascular compression of the facial nerve in the posterior fossa may be responsible (8). Gardner and Sava made the observation that arterial contact with the facial nerve was present in 7 of 19 patients with HFS upon whom they operated to perform neurolysis of the facial nerve (13,14). Maroon et al. reviewed the vascular causes of HFS and described a patient with a 6-year history of HFS in whom a saccular aneurysm of the PICA was found compressing the facial nerve (24).

In a series of papers published from 1976 onwards, Jannetta has elaborated the concept of microvascular compression (19,21). Other investigators, however, have questioned the significance of his findings on the basis of Sunderland's study of 210 unselected autopsy specimens where arterial compression of the 7th nerve at its origin was found in 27 cases (33).

In Janetta's view the cross-compression usually arises from the development with age of elongated, redundant arterial loops, a phenomenon observed at autopsy by Sacks and Linderburg (20,30). He stated HFS is due to pulsatile compression by arteries at the REZ of the 7th nerve (19,26). This zone was defined as a junctional area between thinner central and thicker peripheral myelin. Cross-compression at this site is responsible for demyelination and axo-axonal conduction (ephaptic transmission) leading to classical HFS. If such compressive vessels are surgically displaced from the REZ then the symptoms disappear. This procedure is called MVD.

In recent years MVD surgery has been popularized in neurosurgery. Although there have been many papers reporting favourable results (3,5,11,12,19,22,28, 36) some authors published opposing results claiming a relatively high recurrence rate and incomplete cure (15,22). Some investigators even doubted the neurovascular compression mechanism, attributing the surgical effect to trauma to the nerve root (2), but we believe the neurovascular compression theory is correct in the majority of cases and MVD of the 7th nerve to be a highly effective treatment for HFS.

The diagnosis of hemifacial spasm can be established with a careful history and clinical examination. A variety of similar conditions (post paralytic hemifacial spasm, Habit spasm, Facial myokmia, Essential blepharospasm, Tardive dyskinesia) can be separated from HFS on the basis of clinical presentation. It is very uncommon for diagnostic studies such as routine CT scanning, skull films, angiography, electromyography, to shed any light on the pathogenesis, if the only finding is HFS, although, CT scan with thin posterior fossa cuts has revealed significant abnormalities in 83% of cases (9). Since hearing loss is a potential complication of MVD operations involving the facial nerve (27), we routinely tested the 8th nerve function in the last 20 patients in our series preoperatively and during operation with BAEP and electrocochleography.

Numerous medical therapies for HFS have been proposed, including sedative-hypnotics, vasodilators, central anticholinergics, anticonvulsant medications (17,32), but in the vast majority of cases HFS has proven refractory to medical management.

Surgical procedures have affected the facial nerve at various points from its posterior origin to the periphery. Older methods disrupt the main nerve trunk or its branches and include the injection of alcohols, avulsion, acupuncture, neurotomy, needle insertion, decompression within the temporal bone (25,32,34). Dissatisfaction with these lesioning procedures has prompted the development of newer techniques including percutaneous thermolysis, radiofrequency coagulation and MVD (7,18,35,36). There is no question that these operations offer a higher success rate and less facial paresis than any others.

Contemporary experience with posterior fossa exploration for HFS was reviewed by Loeser and Chen (23). From the data on 450 procedures in 433 cure patients they calculated that 84% of first operations resulted in cure and a additional 5% of patients were improved but not cured. Very recently Fukushima presented his experience with 2381 patients (personal communication with Mr. Coakham) (12). There were 54 cases with incomplete results and 86 late recurrences in his series. Of these 140 patients, 69 were cured by reoperation, making final cure rate of 97%. Loeser and Chen also tabulated the operative findings in 450 explorations for HFS. There were only 16 in which no source of neural compression was found. All but 4 of these cases were contributed by one group, that of Kaye and Adams, who discovered pathological neurovascular relationships in only 25% of their series of 16 patients (1). In Fukushima's series of 2381 HFS, 17 cases had some organic lesions compressing the facial nerve. In the other 2364 he found

that they all had typical arterial compression but only one had vein compression. There was no case with negative findings in this recent study. In the published series, the most common complication was 8th nerve dysfunction, which was transient in 14% and long term in 10%. We feel this complication can be prevented by BAEP monitoring during operation. Operative mortality has been virtually nonexistent, with only a single case reported in the literature (36).

It can be seen that our results are essentially identical to these overall figures from the literature (Table 6).

TABLE 6

Author	Year	No.of pat	% of cure
Neagoy	1974	14	64
Kelly	1977	5	100
Fabinyi	1978	9	78
Janetta	1980	229	93
Wilson	1980	22	82
Kaye & Adams	1981	16	63
Loesser & Chen	1983	20	60
Piatt & Wilkins	1984	48	62
Apfelbaum	1984	53	88
Aoki & Nagas	1986	30	100
Panagapoulas	1987	29	89
Fukushima	1989	2381	97
Hepgül & Rowlinson & Coakham	1990	40	90

In conclusion, HFS is effectively managed surgically in the vast majority of cases and MVD is one of the most safe and rewarding operative procedures in neurosurgery. Complications are few and not often significant; mortality is virtually zero.

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