The Journal of Laryngology and Otology
November 1996, Vol. **110,** pp. 1081–1083

Hemifacial spasm associated with external carotid artery compression of the facial nerve

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Abstract
We report a unique case of hemifacial spasm due to compression of the facial nerve by the main trunk of the external carotid artery within the parotid space. Decompression of the facial nerve as well as partial section of the proximal trunk of the nerve, caused the hemifacial spasm to disappear.

Our case gives support to the theory that hemifacial spasm can be caused by pressure on the facial nerve along all its course and not only in its intra-cranial portion.

Key words: Facial nerve; Hemifacial spasm

Introduction

Hemifacial spasm (HFS) is characterised by paroxysmal bursts of involuntary tonic or clonic activity occurring in muscles innervated by the facial nerve on one side of the face (Ehni and Woltman, 1945).

Jannetta *et al.* (1977) claim that the cause of spasm is compression of the facial nerve by vascular structures at its exit from the brainstem. Therefore, they performed microvascular decompression of the facial nerve in the cerebello-pontine angle (Jannetta *et al.*, 1977; Barker *et al.*, 1995).

In some other cases, the aetiology of this phenomenon remains obscure. The treatment in these cases is only symptomatic. Destructive surgical (Scoville, 1955; Fisch, 1986) or pharmacological procedures (Laskawi *et al.*, 1994) have been used in order to cause weakening of the facial muscles by injuring the peripheral branches of the facial nerve. These methods met with limited success and resulted in partial facial palsy.

Identification of the specific cause can make the treatment more successful. Therefore, we report, for the first time, a case of HFS in the presence of compression of the facial nerve within the parotid space by the external carotid artery.

Case report

A 55-year-old male patient had left hemifacial spasm for four years. At first, there were clonic contractions of the orbicularis oculi, but over the years, the spasm became severe and more frequent. The contractions involved the upper lip levator muscle and all the other muscles innervated by the left facial nerve.

On examination, there was paroxysmal involuntary spasm of the left side of the face involving primarily the orbicularis oris and orbicularis oculi. The contractions were painless and worsened by emotional upset. No facial weakness was seen. Corneal reflexes, facial sensation, taste, hearing and ocular movement were normal, as well as the remainder of the examination.

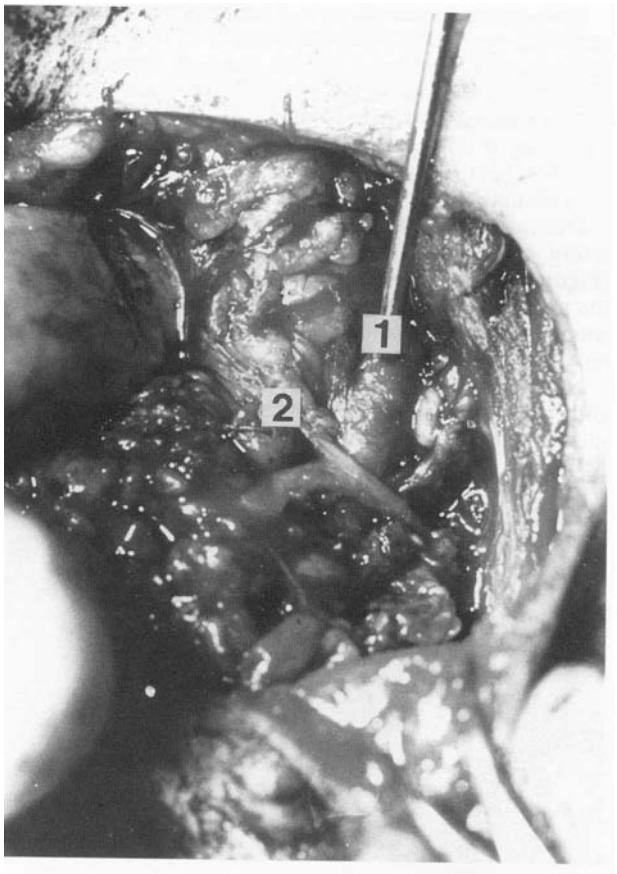

Fig. 1
The external carotid artery (1) compressed by the main trunk of the facial nerve (2).

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Accepted for publication: 13 August 1996.

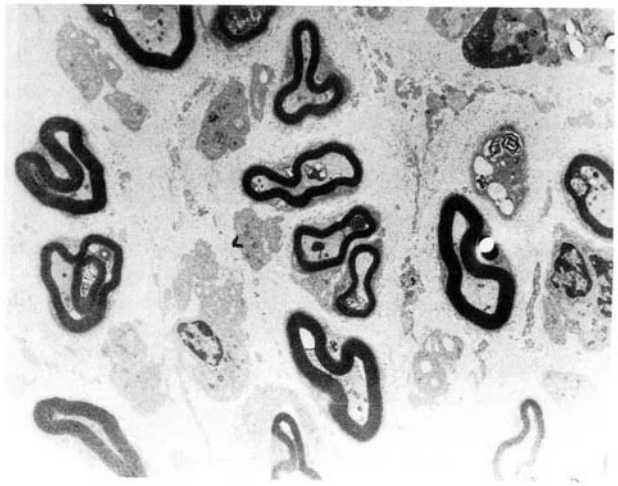

FIG. 2
Cross section, myelinated fibres showing variability in thickness and a demyelinated fibre. (×5,600).

Electromyography of the left face recorded sudden spontaneous synkinetic bursts of activity synchronous in the orbicularis oris and the upper lip levator muscles. The potential activity was normal.

Operative finding and clinical course

With the diagnosis of HFS, a left superficial parotidectomy was performed. The main trunk of the facial nerve was exposed and freed to its primary branching. By surgical microscope, the nerve trunk was sectioned approximately two thirds through its total diameter beginning at its superficial surface. The sectioned portion was then ligated tightly, turned back on itself and stripped for a distance of 0.5 cm as described by Scoville (1955).

During the operation, we found that the external carotid artery compressed the main trunk of the facial nerve (Figure 1). The external carotid artery was separated from the nerve and fixed in position using an absorbable gelatin sponge (Gelfoam) so that it no longer compressed the nerve.

Histological examination of the nerve showed fibres with areas of hypertrophy and thickening of the myelin sheath and areas of demyelinated fibres (Figure 2 and 3).

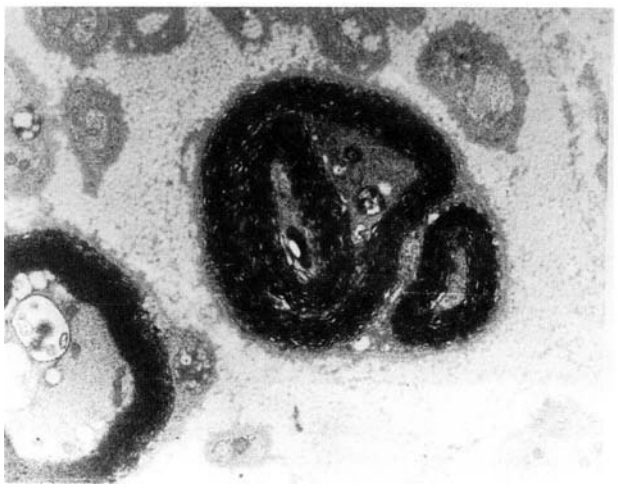

FIG. 3
Marked hypertrophy of myelin sheath (×16,800).

Post-operatively, the HFS immediately diminished and disappeared within one month. The weakness of the left facial muscles which was noticed after the operation, gradually improved, becoming almost undetectable six months later. Electromyography was without any spontaneous activity.

Five years later, the patient has no complaints and the HFS has not returned since the operation.

Discussion

Most theories on the aetiology of HFS include the assumption that the facial nerve is compressed somewhere along its course. One hypothesis claims that the compression injures the myelin, thereby allowing bare axons to contact each other closely and that this close contact between the bare axons promotes direct electrical communication between the individual nerve fibres (ephaptic transmission). Another hypothesis claims that the symptoms of HFS are a result of abnormal function of the facial motor nucleus as a result of irritation of the nerve (Moller, 1951).

In the great majority of cases, the HFS is caused by a vascular compression of the facial nerve at the root exit zone at the brain stem (46 cases of 47 patients of Jannetta *et al.*, 1977). Tumours of the cerebello-pontine angle can also cause HFS as reported by Levin and Lee (1987) and Nagata *et al.* (1992).

Within the temporal bone course of the facial nerve, the nerve can be compressed by haemangiomas causing HFS (Eby *et al.*, 1992).

HFS due to parotid lesions which compress the nerve distal to the styloid within the parotid space is extremely rare. To our knowledge, only two cases of parotid tumour causing HFS have been reported (Nussbaum, 1977; Destee *et al.*, 1985).

The finding of the external carotid artery compressing the facial nerve, as was noted in our case, has not previously been reported. We assumed that this was the reason for HFS in our patient. Our histological findings were similar to those reported by Kumagami (1974), which appears to lend some support to our assumption. Decompression of the facial nerve as well as partial section of the proximal trunk of the nerve, caused the HFS to disappear.

Our case gives support to the theory that HFS can be caused by pressure on the facial nerve along all its course and not only in its intra-cranial portion. Therefore, we suggest a complete evaluation of the entire course of the facial nerve should be made in cases of HFS before deciding on the operation.

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https://doi.org/10.1017/S0022215100135819 Published online by Cambridge University Press

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