The Journal of Laryngology and Otology
July 1997, Vol. **111**, pp. 688–690

Letters to the Editor

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

Dear Sir,

I read with interest the recent paper entitled 'Hemifacial spasm associated with external carotid artery compression of the facial nerve' by Rakover *et al.* published in the *JLO* in November 1996. I agree with the authors' opinion that hemifacial spasm (HFS) can be caused by compression of the facial nerve along all its course and not only in its intracranial portion. There are, however, some points which I cannot agree with in their paper.

The authors performed a superficial parotidectomy, with the diagnosis of HFS, to section two thirds of the total diameter of the facial nerve. The surgical technique reported by Scoville (1955) which they used was unique, but it is destructive and is already an obsolete one for treatment of HFS. On the other hand, the concept of HFS due to neurovascular compression of the facial nerve at its root exit zone (REZ) is well accepted world wide, and many neurosurgeons are performing neurovascular decompression at the REZ. This surgical technique is not destructive and the operative results were remarkable Huang *et al.*, 1992; Barker *et al.*, 1995; Kondo, 1997). The authors, therefore, should consider neurovascular decompression instead of partial section of the facial nerve as the first choice of treatment.

In order to confirm that the authors' patient had compression of the facial nerve at its extracranial instead of intracranial portion, the authors should first have performed magnetic resonance imaging (MRI) (Felber *et al.*, 1992; Furuya *et al.*, 1992; Nagaseki *et al.*, 1992) to rule out neurovascular compression at the REZ of the facial nerve, before they went to partial nerve section, as they aptly stated in their paper that a complete evaluation of the entire course of the facial nerve should be made.

The authors also stated that they found compression of the main trunk of the facial nerve by the external carotid artery during the operation, and the artery was separated from the nerve. They assumed that compression of the facial nerve by the external carotid artery was the reason for HFS since HFS disappeared within one month after the operation. They carried out two procedures for this patient: partial nerve section and neurovascular decompression. Partial nerve section alone can heal HFS whatever its cause, though some complications and recurrence may be expected. Therefore, one cannot conclude that compression of the facial nerve by the external carotid artery is the cause of HFS, if both partial nerve section and neurovascular decompression are performed at the same time. Besides, compression of the cranial nerve by blood vessels does not always provoke hyperactive disorders of the nerve. We neurosurgeons sometimes have a chance to observe blood vessels compressing the fifth, seventh or eighth cranial nerve although the patients had no functional disorders. It is, therefore, inadequate to conclude that HFS is due to compression of the facial nerve by the external carotid artery simply because the authors found compression of the facial nerve by the artery. They still cannot exclude the possibility of intracranial neurovascular compression of the facial nerve.

I will fully agree with the authors' conclusion if they confirmed that there was no vascular compression of the facial nerve intracranially, and performed only neurovascular decompression extracranially without partial nerve section and HFS disappeared as a result.

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Authors' reply

Dear Sir,

We thank Dr Ryu for his comments regarding our paper. We agree that magnetic resonance imaging

(MRI) should be performed in the evaluation of a patient with hemifacial spasm (HFS). Unfortunately, at that time, it was not as available as it is today.

Selective neurectomy of peripheral facial nerve branches is still one of the best procedures that can be performed in HFS with an 80 per cent success rate (Bauer and Coker, 1996). We believe that the patient must be given the choice to decide the type of operation to be performed, either the intra-cranial approach, or the selective peripheral neurectomy.

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Abductor vocal fold palsy in the Shy-Drager syndrome presenting with snoring and sleep apnoea

Dear Sir,

I read with interest the excellent case report entitled 'Abductor vocal fold palsy in the Shy-Drager syndrome presenting with snoring and sleep apnoea' by McBrien *et al.* (1996). The authors rightly point out the risks associated with anaesthesia in these patients, but do not mention the reports of sudden death during sleep in Shy-Drager patients with vocal fold dysfunction and obstructive sleep apnoea. Sleep apnoea from laryngeal obstruction in Shy-Drager syndrome carries a far worse prognosis than common idiopathic sleep apnoea, and deserves further discussion.

Reports of sleep-related stridor in Shy-Drager syndrome date back to 1967 (Bannister *et al.* 1967). Lehrman *et al.* (1978) noted the association between Shy-Drager syndrome, vocal fold paresis, and obstructive sleep apnoea; although tracheostomy was planned in their patient, sudden death during sleep occurred before definitive treatment of upper airway obstruction could take place. Since 1978, several additional case reports and series have supported the concept that obstructive sleep apnoea related to vocal fold abductor paralysis can be lethal in Shy-Drager syndrome (Briskin *et al.,* 1978; Williams *et al.,* 1979; Kavey *et al.,* 1989; Munschauer *et al.,* 1990). The compelling nature of these reports has supported recommendations that symptoms or signs of obstructive sleep apnoea in these patients should be immediately evaluated by overnight polysomnography, followed by emergent tracheostomy if obstruction is demonstrated (Kavey *et al.,* 1989; Munschauer *et al.,* 1990). In addition, diurnal vocal fold dysfunction appears to be highly prevalent in these patients, and may be present even in early stages of the disease and/or without symptoms of stridor or snoring (Williams *et al.,* 1979). It may well be prudent to suggest that all Shy-Drager patients undergo routine laryngological examination (Williams *et al.,* 1979), followed by overnight polysomnography if vocal fold paresis is noted. Maximal inspiratory/expiratory flow-volume loops should also be useful in screening these patients for upper airway obstruction.

It is not certain why patients with Shy-Drager syndrome and obstructive sleep apnoea, in contrast to those with idiopathic obstructive sleep apnoea, are so prone to sudden death during sleep. Autonomic cardiovascular instability from their disease may be responsible, or it may be a consequence of the laryngeal nature of the obstruction. While tracheostomy has been most frequently performed for definitive treatment of the upper airway obstruction, laryngofissure and fold lateralization has also been reported (Kenyon *et al.,* 1984). Unfortunately, sudden death during sleep still occurs in some Shy-Drager patients even after relief of upper airway obstruction, presumably from the central apnoeas also noted to occur in this disease, or perhaps from cardiovascular instability (Briskin *et al.,* 1978; Bannister *et al.,* 1981).

Incidentally, Isozaki and colleagues (1994) have recently provided some intriguing information concerning the mechanism of laryngeal obstruction in these patients. Using a novel catheter electrode array, they demonstrated both abductor paresis (posterior cricoarytenoid muscle) as well as persistence of adductor tone (thyroarytenoid muscle) during inspiration in some Shy-Drager patients with vocal fold paralysis, raising the possibility that laryngeal obstruction in this disorder may in part be dyskinetic as well as paralytic.

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