Central facial weakness due to medial medullary infarction: the course of facial corticobulbar fibres

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Abstract
Two patients are reported with contralateral hemiparesis including a face of supranuclear type, caused by an infarct of the unilateral ventromedial part of the upper medulla. Data from these patients support the hypothesis that part of the corticobulbar fibres supplying the lower facial muscles descend ipsilaterally in the ventromedial part of the upper medulla and then, after decussation, ascend rostrally to the contralateral facial nucleus.

Keywords: facial nerve; corticobulbar tract; brain stem infarction; magnetic resonance imaging

The anatomical distribution and the function of human peripheral facial nerves are well known, but little is known of the course of the corticobulbar fibres that connect the motor cortex with the facial nucleus.1–3 The facial corticobulbar fibres provide strongly unilateral innervation to the contralateral lower facial muscle and bilateral innervation to the upper facial muscles.1–3 Classic symptomatic localisation postulates that lesions rostral to the upper medulla (fig 1A) and then, after decussation, ascend rostrally to the contralateral facial nucleus.1–3

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gram, ECG, and Holter monitoring findings were all normal. Brain MRI performed 10 days after onset showed an infarct of the left ventromedial part of the upper medulla (fig 1B) and old multiple lacunar infarctions of the deep white matter of the cerebral hemispheres. Two weeks after onset MR angiography disclosed atheromatous changes involving the left vertebrobasilar arteries, although no apparent stenosis or occlusion was found. The right hemiparesis remained two months after the onset, yet the right facial weakness resolved within three weeks.

Discussion

Although contralateral facial weakness is usually caused by lesions rostral to the facial nucleus, it is important to realise that lesions caudal to the facial nucleus can also cause contralateral facial weakness.

There have also been several reports of mild facial weakness caused by medial medullary infarction, but there were few details on the responsible lesion. Currier hypothesised that the corticobulbar fibres to the lower facial nucleus leave the pyramid at the pontomedullary junction and descend caudally to at least...
Figure 2 Schematic diagram of the pathways of the facial corticobulbar fibres. (A) (our patients) and (B) (lateral medullary infarction) show affected sites of the corticobulbar fibres. CST represents corticospinal tract; CF represents facial corticobulbar fibres.

mid-medullary levels, and then most fibres cross to the opposite facial nucleus. It has been postulated that interruption of these fibres by infarction at a predecussation level results in the contralateral central facial weakness. There have been some reports of facial weakness resulting from a similar mechanism by medial medullary infarction, although it has been established pathologically that the facial nucleus received such corticobulbar fibres.

Recently, Cavazos et al. reported a patient with an infarction of the medullary pyramid with pure motor hemiparesis including the lower face. Based on the MRI findings, they hypothesised that some of the facial corticobulbar fibres descend ipsilaterally, making a loop as low as the upper medulla before decussating and ascending to the contralateral facial nucleus. We suggest that the findings in our patients serve to support Cavazos's description on the course of the facial corticobulbar fibres.

The absence of facial weakness in several reported cases may be consistent with a more caudal medullary lesion. On the other hand, lateral medullary infarction relatively often results in facial weakness on the side of the lesion. It has also been postulated that facial weakness on the side of the lesion results from corticobulbar fibres being interrupted while they ascend contralaterally after decussating.

We have proposed a scheme for the facial corticobulbar fibres (fig 2). In our patients, left ventromedial lesion of the upper medulla may give rise to right central facial weakness because the corticobulbar fibres are affected at predecussation levels (fig 2A). On the other hand, the lateral medullary infarction is likely to result in ipsilateral central facial weakness because the corticobulbar fibres are interrupted at postdecussation levels (fig 2B). Therefore, it is suggested that part of the pathways of the facial corticobulbar fibres descend ipsilaterally adjacent to the ventromedial part of the corticospinal tract to the upper medulla before decussating, and ascend contralaterally in the dorsolateral part of the medulla to the facial nucleus.

MRI allows a clear delineation of lesions of the brain stem and spinal cord. It is hoped that further neuroanatomical study will clarify in detail the courses of central pathways including the corticobulbar and corticospinal tracts.

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