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SHORT REPORT

# Volitional type of facial palsy associated with pontine ischaemia

Rudolf Töpper, Christoph Kosinski, Michael Mull

**Abstract**

**A dissociation between voluntary and emotional facial innervation is described in a patient with a pure motor stroke due to a unilateral ischaemic pontine infarction. Voluntary facial innervation of the contralateral orbicularis oris muscle was affected whereas emotionally induced innervation of the same muscle was spared. This report provides evidence that fibres conveying voluntary and emotional commands are still separated in the pons. Whereas corticobulbar tracts carry the information for voluntary facial innervation, efferents from the amygdala and the lateral hypothalamus are candidates for the somatomotor aspects of emotions.**

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**Department of Neurology**
R Töpper
C Kosinski

**Department of Neuroradiology, Technical University of Aachen, Aachen, Germany**
M Mull

Correspondence to:
Dr Rudolf Töpper, Department of Neurology, Pauwelsstrasse 30, 52057 Aachen, Germany.

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In patients with the volitional type of central facial palsy facial involvement is most pronounced during voluntary contraction whereas emotionally triggered contractions are preserved or at times even exaggerated on the paretic side.[1] In the emotional type of facial palsy the opposite phenomenon can be seen: whereas voluntary triggered contractions are normal, there is facial impairment during emotionally triggered movements. Automatic voluntary dissociation is not restricted to muscles supplied by the facial nerve: in the bilateral anterior opercular syndrome automatic voluntary dissociation is also seen in masticatory muscles supplied by the trigeminal nerve, in the tongue, and in muscles involved in swallowing.[2] Whereas the volitional type of facial palsy is thought to be caused by a lesion in the motor cortex or in the corticobulbar pathways, lesions in the basal ganglia, the hypothalamus, or the thalamus can cause an emotional facial palsy.[3] The precise neuroanatomical basis of automatic voluntary dissociation, however, has remained obscure. It is not clear at what level above the facial nucleus fibres conveying volitional and emotional information converge. We describe a patient who had an ischaemic

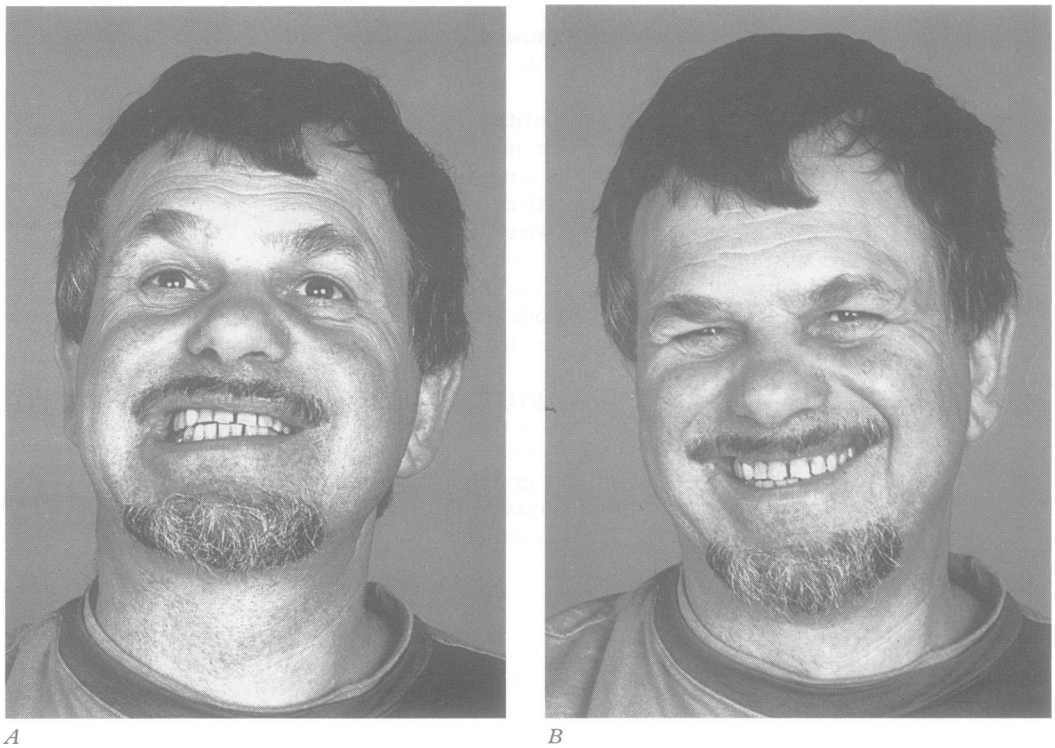

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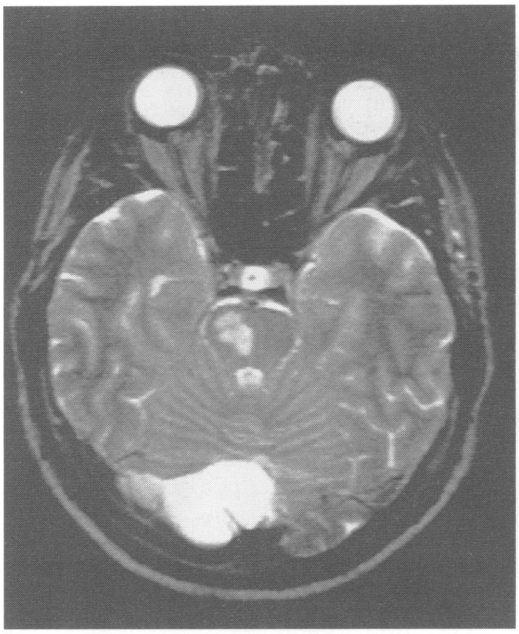
*Figure 1 Volitional facial paresis when patient was asked to bare his teeth (A). Symmetric movement with emotional responses (laughing) (B).*



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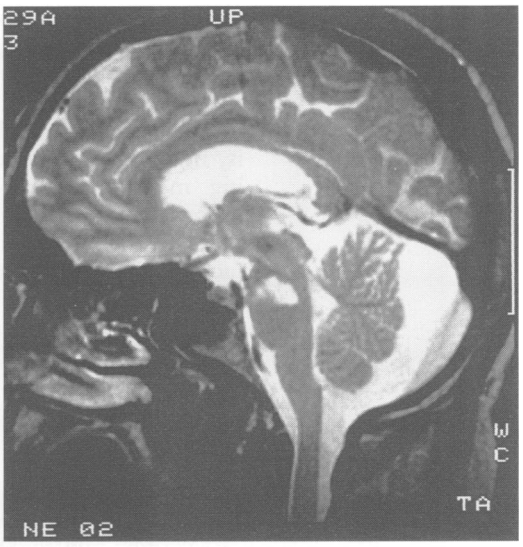


*B*

*Figure 2 T2 weighted MRI (SE 3·0 s/90 ms) in axial (A) and sagittal (B) planes. Hyperintense lesion of the right upper pons is visible consistent with anteromedial/anterolateral pontine infarction. Note the typical wedge shaped lesion in the sagittal plane (B) that spares the pontine tegmentum.*

stroke in the upper pons; he presented with a volitional type of facial paresis indicating that the pathways subserving volitional and emotional input on to the facial nucleus are still anatomically separated in the upper pons.

## Case report

A 57 year old man was admitted to the neurology department because of a central paresis of his left arm and leg with sudden onset. His medical history was unremarkable. There was no trauma preceding the episode. On neurological examination the patient was alert and oriented. He had a facial paresis with relative sparing of the upper portion of the face. When asked to bare his teeth there was drooping of the angle of the left side of his mouth (fig 1A). On involuntary contraction, however, there was symmetric innervation of the muscles of the mouth (fig 1B). The remainder of the cranial nerve examination was unremarkable; specifically there was no disturbance of oculomotor function. He had a 3/5 paresis of the left arm and leg with reflexes being more pronounced on the left. Babinski's sign was positive on the left, negative on the right side. Cerebellar and sensory function were normal.

A brain CT obtained eight hours after the onset of symptoms showed no brainstem lesion. Fourteen days after the event MRI showed a large hyperintense lesion of the right upper pons on T2 weighted images (fig 2A and B). This lesion respected the midline, had contact with the ventral circumference, and spared the lateral pontine border and the pontine tegmentum.

Extracranial and transcranial Doppler sonography showed no evidence of stenosis or dissection of extracranial or accessible cranial arteries. Transthoracic and transoesophageal echocardiography was also unremarkable.

A diagnosis of an anteromedial/anterolateral ischaemic pontine infarction with volitional facial paresis was made.

## Discussion

A dissociation of volitional and emotional facial nerves was found in this patient with a unilateral pure motor stroke due to ischaemia in the pons. This finding may provide new information on the neuroanatomical organisation of facial innervation. Voluntary motor impulses from the motor cortex descend through the internal capsule and either make direct connections with the facial nucleus or terminate in the pontine reticular formation.[4 5] The pathway by which emotional commands reach the facial nucleus is not clear in humans. Information from the amygdala and the lateral hypothalamus, which are thought to be involved in the generation of emotions, reach the brainstem via the medial forebrain bundle and the dorsal longitudinal fasciculus.[6] Unlike corticobulbar tracts the amygdala and the lateral hypothalamus do not have direct projections to the motor nuclei of the facial nerve but send axons to the lateral tegmentum where interneurons for the facial nerve are located.[7] These polysynaptic pathways are considered to form an anatomical basis for somatomotor components of affective behaviour[4 8] and are therefore likely candidates for conveying emotional responses to the facial nucleus. That a lesion of these tracts to the lateral tegmentum may cause an emotional facial paresis has already been postulated by Wilson.[9] The dissociation in our patient can, therefore, be explained by a pyramidal tract lesion in the upper pons that disrupts the corticobulbar fibres to the facial nucleus whereas efferents from the amygdala and the lateral hypothalamus to the lateral tegmental area are spared. Because emotional facial responses such as laughter and crying involve stereotyped bilateral innervation of facial muscles[10] it can be argued that the efferents

from the amygdala and the hypothalamus are organised bilaterally. In our patient one could, therefore, postulate that emotional information reached the facial nucleus via the undamaged ipsilateral fibres. This hypothesis is, however, contradicted by the overwhelming majority of patients with hemispheric lesions of descending motor tracts in whom there is both a volitional and an emotional facial palsy. The voluntary and emotional type of innervation of the facial nucleus therefore seems to follow a common pattern with bilateral projections to the muscles of the upper two thirds of the face and unilateral projection to the lower third.

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