

# Transient Muteness Followed by Dysarthria in Patients with Pontomesencephalic Stroke

## Report of Two Cases

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### Key Words

Transient muteness · Dysarthria · Pontomesencephalic stroke · Speech

### Abstract

Dysarthria is the principal motor abnormality following vascular damage to pontine paramedian structures, owing to the involvement of corticobulbar fibres. Here we describe 2 cases of adults affected by dysarthria following transient muteness as the result of a stroke in pontomesencephalic structures. Their clinical outcome was very similar to that of young patients who have undergone surgery of the 4th ventricle. Recently the importance of pons involvement has also been underlined in these cases. This case report suggests the existence of a functional network for speech, in which the pontomesencephalon is an important station for the triggering and the efficacy of verbal production.

### Introduction

Dysarthria is considered the main motor abnormality in pontine paramedian infarctions by Kataoka et al. [1], with a frequency of 100% in basal lesions and 71.4% in tegmental lesions, as a consequence of the involvement of corticobulbar fibres.

Pontine paramedian strokes may also be responsible for pure dysarthria syndromes, as described by Kim [2] in 1994, suggesting that the involvement of corticobulbar fibres in vascular damage of this area is much more constant than that of corticospinal motor fibres.

Here we report 2 case studies of patients who presented transient muteness following pontomesencephalic stroke that only later evolved into dysarthria. A similar clinical entity, named transient mutism followed by dysarthria (TMFD), has already been described after surgery of the posterior cranial fossa.

We suggest that a vascular injury of the pontomesencephalon may evoke a clinical entity similar to TMFD syndrome, by altering a crucial step of triggering verbal production.

### Case Reports

#### Case 1

F.A.M., a 56-year-old right-handed woman, with a history of hypertension, showed sudden loss of consciousness which lasted some hours with right-sided hemiplegia, with a deficit of the cranial nerves VII and XII of the central kind and dysphagia. A cranial CT scan carried out a few hours after loss of consciousness showed a haemorrhagic lesion in the median and left paramedian pontomesencephalic region. An MRI scan of the cranium showed a haemorrhage in the left paramedian portion of the pontomesencephalic joint foot. A few hours after the stroke, with recovery of consciousness, the patient was unable to speak, without any vocalization, whilst comprehension of spoken language was preserved. One month later, dys-

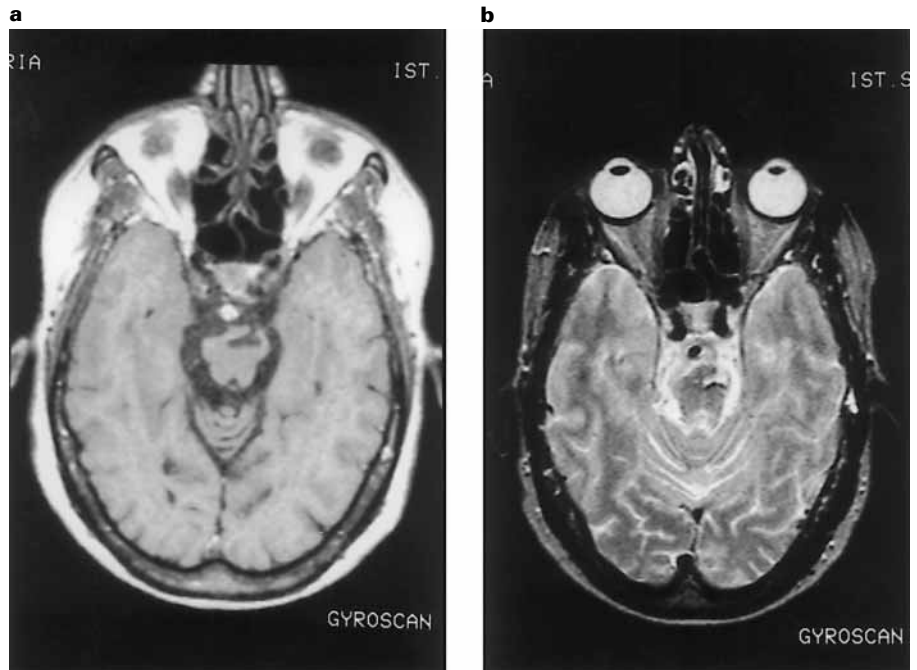
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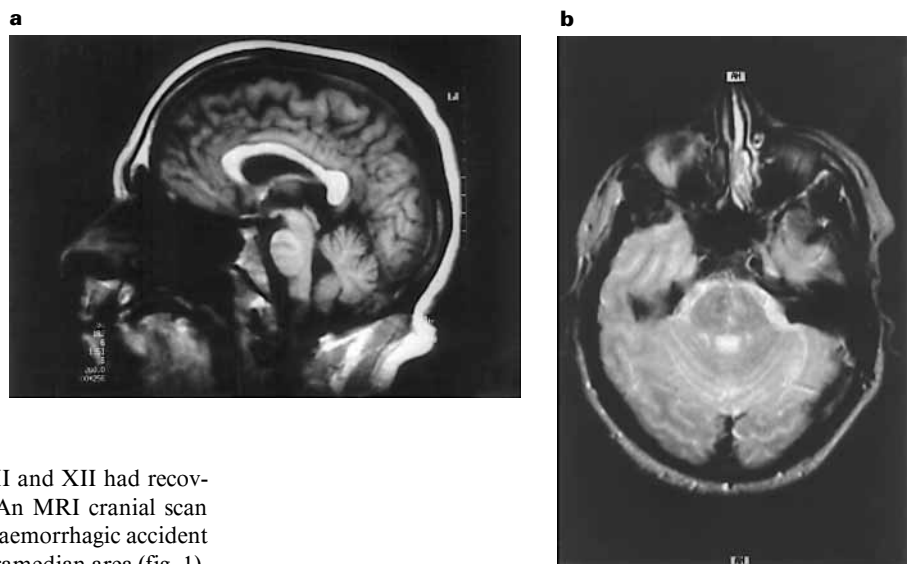
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**Fig. 1.** MRI T<sub>1</sub>- (a) and T<sub>2</sub>-weighted (b) axial images. Pontomesencephalic median and left paramedian area of hypo-intensity in a and hyperintensity in b, due to haemorrhagic accident.



**Fig. 2.** MRI sagittal T<sub>1</sub>-weighted image (a) and axial T<sub>2</sub>-weighted image (b). Pontine left paramedian area of hypo-intensity in a and hyperintensity in b due to ischaemic infarction.

phagia and the deficit of the cranial nerves VII and XII had recovered completely, while hemiplegia persisted. An MRI cranial scan performed at this time showed the result of a haemorrhagic accident in the pontomesencephalic median and left paramedian area (fig. 1). In this period the patient began to pronounce short words and she underwent examination for dysarthria, performed by an expert speech pathologist. According to criteria proposed by Darley et al. [3], her oral production was characterized by imprecise consonants, monopitch, low pitch, restrained voice and by short phrases and slow rate. After 2 more weeks, her voice was no longer hypophonic, although the pronunciation was still slurred and laboured. A few days after, she was able to pronounce complete sentences but with an evident dysarthria. After about 1 year, her dysarthric defect persists.

#### Case 2

R.C., a right-handed male of 44 years, smoker (more than 20 cigarettes a day), with a history of unstable arterial hypertension, presented with intense headache and vomiting, gradual worsening tetraparesis, dysphagia more marked for liquids, left cerebellar hemisindrome, paresis of the right facial and XIIth nerve of the central kind,

muteness and stupor. A cranial MRI scan, performed 1 month later, showed the presence of a small pontomesencephalic median and left paramedian area of ischaemia (fig. 2).

After 15 days, left-sided motility and strength recovered. After 1 month, tongue motility improved, dysphagia disappeared and there was partial recovery of the cerebellar hemisindrome and right-sided motility. At this time, the patient underwent examination of dysarthria performed by the same speech pathologist as in the preceding case. The patient's oral production was characterized by imprecise consonants, monopitch, low pitch, restrained voice, hypernasality and by short phrases and slow rate. During the following days oral expression slowly but constantly improved, and he was able to pronounce long phrases, without hypophonia, but in a dysarthric way. At the 1-year follow-up, the dysarthria is still evident.

## Discussion

The first description of the classical TMFD syndrome was made by Rekate et al. [4] in 1985. This syndrome, as described after cerebellar surgery, is characterized by: (1) onset of the disorder usually after an interval varying from a few hours to 2–3 days; (2) temporariness varying from 3 to a maximum of 16 weeks; (3) gradual recovery of oral expression: first phonemes, then single words and finally complete phrases, with scanning speech (cerebellar dysarthria). All the cases reported in the literature regard children, with the exception of one young adult [5].

In our 2 cases muteness developed immediately after a vascular pontomesencephalic lesion. Unlike patients after posterior cranial fossa surgery [6–8], in our cases there was no interval between parenchymal injury and the onset of language impairment. Van Dongen et al. [9] were the first authors to underline the importance of pontine tegmentum involvement in TMFD genesis, suggesting that, together with hydrocephalus and localization of tumour in the 4th ventricle, it was an indispensable condition for the genesis of so-called cerebellar postsurgical muteness.

We suggest that muteness could be due to a *diaschisis* that temporarily alters the supranuclear, nuclear and cerebellar connections of the cranial nerves VII, IX, X and XII, devoted to phonation and to verbal articulation control. This interpretation assigns a crucial role to the pontomesencephalic lesion site in determining the initial muteness. It implies the existence of a network devoted to triggering articulation so that damage to one of these structures produces a muteness, but only transiently because the other components would be capable of activating speech. Among the putative structures devoted to triggering articulation (damage to which produces TMFD)

there could be pontomesencephalic structures. This interpretation is an interesting hypothesis, but here it cannot be supported.

Another interpretative problem is represented by the explanation of cerebellar-like dysarthria in our cases, in which the vascular damage involves only the brainstem.

Dysarthria has been described as a consequence of vascular injuries in different cerebellar territories, but some authors underlined the contemporary involvement of the brainstem in patients who developed dysarthria [7, 8, 10, 11]. Moreover, it has been remarked that pure cerebellar strokes in the territory of the posterior inferior cerebellar artery do not cause dysarthria, while when the brainstem is involved this speech alteration develops [12]. The genesis of dysarthric disorders has also been attributed to mesencephalic structures [7], and the important role played by the mesencephalic reticular substance and by the grey peri-aqueductal substance in language function is indeed well known. The pontomesencephalic site, therefore, seems to be crucial for its anatomical and/or functional links with the cerebellum. Injury to this structure allows the recovery of speech but with a functional defect linked to a lack of cerebellar control.

Dysarthria from vascular lesions at different levels of the brainstem, cerebellar muteness and transient muteness observed in our 2 patients could all be the expression of the involvement, at different levels, of the same anatomofunctional pathway. The appearance of one symptom or the other could depend on the lesion extension and on the level at which the pathway is interrupted.

In conclusion, TMFD caused by pontomesencephalic lesions can be explained by considering this region as a crucial station in a complex network that allows the triggering of verbal production and the efficacy of such a production.

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