Supranuclear Pathways for Facial Movements


Abstract

The authors report on the case of a young man with a mild head injury and an isolated palsy of voluntary facial movements, attributed to a midbrain traumatic hematoma. This exception to the generally accepted conjunction between brainstem contusion and poor prognosis pertains to a special entity of midbrain laceration due to hyperextension of the head, with minimal influence on the level of consciousness. The clinical presentation of this lesion with facial palsy sparing emotion-related movement has rarely been described and offers a clue for exploring the neuroanatomy of facial movement.

According to the authors, this is a case of supranuclear facial palsy at the level of the cerebral peduncle. This explanation is plausible, because “in supranuclear lesions there may be a dissociation of emotional and voluntary facial movements; often, some degree of paralysis of the arm and leg or aphasia (in dominant hemisphere lesions) is conjoined,” as has happened in the described patient.

First of all, there is the problem that the contusion seems to be outside the cerebral peduncle (Figs. 1A and 3A of the article), while, as correctly described by the authors, “the corticobulbar fibers for face movement lie medially to the ones for the extremities in the cerebral pedicle.” The corticospinal tract of this patient was probably injured at the level of the internal capsule and not at the level of the midbrain peduncle. However, the main problem is that this is obviously a case of upper facial paresis, which should not occur in a supranuclear facial injury, given that the corticopontine innervation of the upper facial muscles is bilateral. This bilateral innervation occurs at the level of the pons, near the facial nucleus. An alternative hypothesis is that this case could be a kind of facial apraxia.

Facial (including orofacial and buccofacial) apraxia occurs in areas connected to the premotor cortex. We describe such a case from our department. A 25-year-old woman suffered a head injury after a car accident. She was initially lethargic, but her level of consciousness quickly improved to normal. She had right facial paresis, as is typical in cases of facial nerve palsy. Facial movements were easier during mimicry. She could sometimes perform spontaneous movements of her face, which she could not do on demand. She had difficulty in her verbal expression; verbal pronunciation was better during repetition. She also has difficulty swallowing, with diminution of her gag reflex, which was not accompanied by hypesthesia of the oropharynx. All these clinical signs fluctuated at consecutive clinical examinations (Fig. 1).

A neuroradiological investigation revealed contusions in the areas of Broca and left lower parts of the precentral gyrus, in central motor areas for the face and oropharynx, as well as adjacent perisylvian areas. There were no brainstem abnormalities (Figs. 2–4).

This was a case of orofacial dyspraxia, which is characterized by difficulty in facial movements, easier movements after mimicry, and spontaneously and fluctuation in their clinical presentation. It is also characterized by dyspraxia in talking, especially in difficult syllables, easier mimicry, and fluctuation in expression of language. Finally, it is characterized by diminished gag reflex without hypesthesia of the oropharynx.

Clinical and imaging studies show a strong correlation between orofacial dyspraxia and lesions in the frontal operculum. This condition may also occur with subcortical lesions involving periventricular and/or peristriatal white matter as well as the basal ganglia (circuit of cortex/basal ganglia/thalamus/cortex).
Finally, it is well known that oropharyngeal dyspraxia can accompany the mutism associated with removal of vermian tumors (circuit of cortex/cerebellum/thalamus/cortex).1 Orofacial dyspraxia can happen in patients with lesions of areas connected to the premotor area. We must remark that subcortical and basal ganglia lesions tend to produce mainly emotional disturbance of facial movements.

The case described by the authors is quite different from the one that we have described.

In our opinion, the patient the authors have described has suffered a mixed lesion. If we compare Fig. 3A of the original article with a standard anatomical atlas,2 we can see that the upper part of the lesion is partly within the internal capsule, but mainly within the ventral anterior nucleus of the thalamus, which is well known as part of the prefrontal loop.3 It is probably an interesting case combining the clinical picture of supranuclear facial nerve palsy with that of orofacial dyspraxia. Such a combination could explain the entire spectrum of the clinical picture of the patient.

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References


RESPONSE: In regard to the point of view expressed by Drs. Sakellaridis and Kelesis, there are a number of facts we wish to point out.

At first, our patient’s photographs have probably been misinterpreted, as it is clear that he suffers from central facial palsy. In addition, the presence of a definite supranuclear lesion combined with the absence of petrous bone fractures renders the hypothesis of peripheral facial lesion unacceptable.

Drs. Sakellaridis and Kelesis propose an alternative concept to explain the findings in our patient, the one of facial