

Brain Control of Facial Muscles

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The cerebral cortex plays a major role in mediating higher-order, or "executive" behaviors which include the organization and production of complex motor activity. The specific region of the cerebral cortex controlling motor activity is commonly referred to as "motor cortex". A number of recent studies conducted on focal dystonia patients have shown that abnormal metabolic patterns, in the form of decreased and increased activity occur in different parts of the motor cortex (Tempel and Perlmutter 1993; Ceballos-Bauman et al., 1997; Playford et al., 1998; Feiwell et al., 1999). Given this, it becomes likely that the descending projections, or pathways which link the motor cortex to key subcortical motor centers such as the brainstem motor nuclei (e.g., facial nucleus) and basal ganglia are too, adversely affected in focal dystonia patients and perhaps involved in the expression of the symptoms. Particularly, if circuitry exists which links discrete parts of the motor cortex controlling the head and neck to individual muscle groups. The fact that these diseases often target a unique group of muscles implies to us that a highly specific neural system is altered. This view being in contrast to a potentially common and widespread cellular alteration that may underlie the more generalized forms of dystonia.

To advance our understanding of these issues as they relate to blepharospasm, we investigated the descending projection from motor cortex to musculotopically defined subsectors of the facial nucleus (Morecraft et al., submitted). Specifically, we studied the descending projection from the face representation of the primary (M1), supplementary (M2), rostral cingulate (M3), caudal cingulate (M4) and ventral lateral pre- (LPMCv) motor cortices in the rhesus monkey. The descending projection was defined by injecting anterograde tracers into the face representation of each motor cortex. In the same animals, the musculotopic organization of the facial nucleus was defined by injecting fluorescent retrograde tracers into individual muscles of the upper and lower face.

The facial nucleus received input from all cortical face representations. M1 and LPMCv gave rise to the heaviest projection with progressively diminished intensity occurring in the M2, M3 and M4 projections respectively. Injections in all cortical face representations labeled terminals in all nuclear subdivisions of the facial nucleus (dorsal, intermediate, medial and lateral). However, significant differences occurred in the proportion of labeled boutons found within each functionally characterized subdivision. M1, LPMCv and M4 projected primarily to the contralateral lateral subnucleus which innervated the perioral (mouth) musculature. M2 projected bilaterally to the medial subnucleus which supplied the orbitoauriculars of the ear. M3 projected

bilaterally to the dorsal and intermediate subnuclei which innervated the frontalis and orbicularis oculi muscles respectively.

Our results indicate that the various cortical face representations may mediate different elements of facial expression. Corticofacial afferents from M1, M4 and LPMCv innervate primarily, the contralateral lower facial muscles. Bilateral innervation of the upper face is supplied by M2 and M3. The widespread origin of these projections indicate selective vulnerability of corticofacial control following localized brain damage. If in fact the cerebral cortex plays a major role in the manifestation of blepharospasm, our observations indicate that the rostral cingulate motor cortex (M3) may contribute to the symptoms by conveying potentially altered cortical influence to the upper facial muscles.

In a parallel set of experiments examining neural inputs to the motor cortex, we have found that the face representation of M3 receives heavy and concentrated input from both the limbic lobe (including area prostriata of the calcarine sulcus) and the amygdala (Morecraft and Van Hoesen 1998; Morecraft et al., 1998; Morecraft et al., in press). The limbic lobe and the amygdala are well known for their central roles in mediating emotional behavior. On the other hand area prostriata, which directly abuts the peripheral representation of the primary visual field (V1) in the calcarine sulcus of the occipital lobe, is responsive to bright flashes of light (photic stimulation) and itself receives direct inputs from the peripheral visual representation of V1 and visual association area V2. Of possible significance to our findings is the fact in many patients, primary initiators of sustained muscle contractions in the upper facial musculature include bright and intense flashes of light (visual stimuli) and normal but significant stresses (emotional stimuli).

We plan to continue investigating the local circuit interactions of the M3 projection to the facial nucleus. Our goal is to determine if direct synaptic contacts occur between terminal axons of the M3 projection and membrane profiles of facial motor neurons innervating the orbicularis oculi and frontalis/corrugator muscles. We will also initiate a study examining the M3 projection to the basal ganglia and thalamus since basal ganglia-thalamic circuitry has long been implicated as a potential candidate in the development of focal dystonia. We suspect that if alterations occur in the rostral cingulate motor area (M3), both the descending projection to the facial nucleus and the descending projection to the basal ganglia may collectively contribute to sustained muscle contractions. Particularly if the basal ganglia circuitry which initiates within M3, is directed back to its origin.

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