Mucosal trigeminal stimulation to investigate trigeminofacial excitability in hemifacial spasm and healthy controls.

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INTRODUCTION

Hemifacial spasm (HFS) is a movement disorder characterized by intermittent twitching of facial innervated muscles affecting 10/100.000 people.

Two main pathophysiologic mechanisms have been postulated to explain the origin of the spasm: the peripheral ephaptic transmission and the VII c.n. nucleus hyperexcitability. Neurophysiologic testings gave contrasting results because of the co-stimulation of VII and V cranial nerves during standard trigemino-facial reflex recordings, not allowing a distinction between the peripheral ephaptic transmission and the nuclear hyperexcitability.

To this intent, to avoid facial nerve co-activation, we looked for a trigemino-facial reflex elicited by endo-oral mucosal stimulation in controls and in HFS patients.

MATERIALS and METHODS

Mucosal Blink Reflex (mBR) was bilaterally recorded from orbicularis oculi (OOc) and depressor anguli oris (DAO) muscles, after electrical stimulation of the medial inferior gingival surface of both sides. We recordeded the mBR from eleven HFS patients treated with BoNT (age: 60.9±10.3) at TO (three months from the last injection) and T1 (three weeks after BoNT injection), and from five healthy controls (age: 53.8±9.9).

Presence, latency and frequency of early (mR1) and late (mR2) components of mBR were measured and compared in HFS and controls, as well as before and after BoNT. Nonparametric tests were used for statistical analysis. A p value <0.05 was considered significant.

RESULTS

In controls, mucosal stimulation evoked an mR2 in 100% of subjects from OOc and in 40% from DAO; no mR1 was recorded.

In HFS patients, mR1 was recorded in 45% and in 18% from affected and unaffected OOc, respectively, and in 45% and 9% from affected and unaffected DAO, respectively. The mR2 was recorded in 64% of affected and 73% of unaffected OOc, and in 73% of affected and 27% of unaffected DAO.

In OOc muscles, we found that mR1 frequency was significantly increased (p=0.049) and mR2 frequency was significantly reduced (p=0.042) in HFS subjects compared to controls. In DAO muscles, we found no difference between patients and controls, but mR2 was more frequent in the affected DAO compared to unaffected one (p=0.018). We also found a significant increase of mR2 frequency on the unaffected DAO (p=0.034) after BoNT treatment compared to baseline testing.



Mucosal Blink Reflex (mBR) recorded from representative HFS patient and controls (control3, control4, and HFS1). Five superimposed traces, recorded from left (L) and right (R) Ooc and DAO muscles, are shown. Note the presence of an early response (mR1) only from the Ooc muscle of HFS patient.

CONCLUSIONS

Trigeminal mucosal stimulation, in healthy subjects, evoked a late mR2 in OOc and DAO while no early mR1 response was observed. On the contrary, in HFS patients trigeminal mucosal stimulation evoked a clear mR1, while mR2 frequency was lower than in controls on both affected and unaffected sides. Furthermore in HFS patients, mR2 frequency of the unaffected side was modulated by BoNT treatment.

We can speculate that, in HFS subjects, oligosynaptic mR1 circuits are enhanced while polysynaptic mR2 circuits, modulated by suprasegmental systems, are inhibited. This finding supports the hypothesis of a brainstem hyperexcitability in HFS, partially inhibited by suprasegmental control and secondarily modulated by the spasm reduction after BoNT treatment. Facial nerve stimulation is avoided in this experimental setup, so the ephaptic origin or a peripheral modulation of the responses can be excluded.

These data demonstrate for the first time that mucosal trigeminal stimulation evokes reflex responses in OOc of healthy subjects and is a useful tool to explore trigemino-facial excitability in facial movement disorders.

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