Letters to the Editor

Abnormalities of ocular motility in myotonic dystrophy

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Abbreviations: OKN = optokinetic nystagmus; SP = smooth pursuit; VOR-S = vestibulo-ocular reflex suppression

Anastasopoulos et al. (1996) reported a parallel degradation of smooth pursuit (SP) and vestibulo-ocular reflex suppression (VOR-S) indicating a central nervous system dysfunction in patients with myotonic dystrophy, who all had visual acuity better than 0.6. However, in our study on 13 myotonic dystrophy patients (Verhagen et al., 1992), we found SP impairment only in the two cases with the lowest visual acuity (0.3 and 0.5), but optokinetic nystagmus (OKN) responses were normal in all cases. We were therefore inclined to attribute the poor SP response in these latter cases to their poor visual acuity. Because OKN responses are less dependent on foveal vision than SP, we wrote that in view of the fact that most myotonic dystrophy patients have reduced vision due to cataract and retinal abnormalities (Miller, 1985), we would not be inclined to accept a deficit in visual following responses, unless it showed up in both the SP and the OKN responses (Verhagen et al., 1992). Unfortunately OKN was not examined by Anastasopoulos et al. (1996). In fact, OKN responses have never been included in reports on myotonic dystrophy, as far as we know, except in ours (Verhagen et al., 1992) and in a case report by Emre and Henn (1985). In our opinion, the VOR-S findings add nothing substantial to the SP findings in the study by Anastasopoulos et al. (1996), as the visual target (a laser spot) was identical in both cases, as was, apparently, the visual background (Ganzfeld-like structure of the cylindrical screen or darkness?) which, if not identical, must have been at least so designed that background structures could not compete with SP of the moving target by eliciting OKN responses in the opposite direction. In other words, the method was such that the emphasis was very much, in a fairly similar way, on foveal vision in both cases. Therefore an additional finding of OKN impairment might have made it more easy to accept central dysfunction as the underlying cause. Studying VOR-S using the whole (structured) visual surround instead of a laser spot might be a suitable alternative.

Anastasopoulos et al. (1996) suggested that the accentuation in SP/VOR-S deficits at higher frequencies might be related to a similar frequency-dependent performance of parieto-occipital visual association areas. Frequency-dependent impairment of SP/VOR-S on the basis of suboptimal visual acuity (foveal function) might be a more trivial suggestion. We could not find any report on the dependence of SP gain on visual acuity in a 1966–96 Medline search, other than one example of impairment of accommodation degrading SP (Penetar et al., 1988), but it is common knowledge that SP deteriorates in persons with refraction errors when they are not using their glasses and, presumably, so does VOR-S. It could well be that such deterioration is more marked at the higher frequencies of stimulation.

References


Reply

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We thank Verhagen and Huynge for their comments. These authors suggest that the deficit for SP and VOR-S, which we observed in our myotonic dystrophy patients (Anastasopoulos et al., 1996), could possibly be due to low visual acuity. However, visual acuity in all of our patients was good (>0.6) and clearly better than in the two patients in their study (Verhagen et al., 1993), who were the only ones showing a clear deficit in SP and visual acuity (0.3 and 0.5).

It should be mentioned, however, that the two patients in their study had, in addition to the SP and visual acuity deficit, convergence paralysis and divergent strabismus. While slowing of SP with low visual acuity appears not to be documented in the vast SP literature (at least we did not find an indication for it, either), it is known that abnormalities of SP are found in a considerable proportion of patients with strabismus. Therefore, we may have to look for reasons other than visual acuity, which could explain the discrepancy between the SP deficit in our patients and the negative findings in the vast majority of their patients.

Important differences concern the methods used to elicit SP and to analyse the responses. The authors applied only one stimulus, consisting of a spot moving in a circle of 10° radius at a horizontal/vertical peak velocity of 20°/s. Thus, the horizontal and vertical components of their stimulus had a frequency of ~0.32 Hz. We mentioned that the SP deficit in our patients was most pronounced at higher frequencies (i.e. at 0.4 and 0.8 Hz). Furthermore, the authors used conventional EOG, the spatial resolution of which is clearly less than that of the infrared device which we used. This point might be of relevance for detecting small catch-up saccades which compensate for SP slowing. Unfortunately, we do not learn from their article whether they separated saccades at all from the responses. Saccade separation in their study might have been more difficult with the strong low pass filtering (LP 30 Hz) and an apparently rather low (but not precisely specified) sampling rate. Such methodological differences might be the reason why our data are more comparable with the work of Bollen et al. (1992) who did observe an SP slowing, using methods for data acquisition and analysis similar to ours.

The authors appear to suggest that a deficit in visual-following responses in patients can only be proved if it shows up in both SP and OKN responses. We have not tested OKN in our patients, so that we cannot make a statement about their OKN performance. However, we would disagree with the authors’ suggestion. It is well known that both OKN and SP are often impaired in parallel in patients with parietal lesions, for instance, but there are cases in which OKN is relatively spared compared with SP (Baloh et al., 1980; Chambers and Gresty, 1982; Heide et al., 1990). Furthermore, we find it difficult to accept the authors’ finding of a normal OKN in their two patients with defective SP when considering the broad variation of their normative data (“the lower 5% confidence limit of response velocity was 20°/s, with the stimulus velocity being 40 and 60°/s”).

Coming back to the question as to what extent SP may be affected by low visual acuity, it is our experience from clinical nystagmography that a moderate or medium impairment has little effect on SP. This experience is based, for example, on our observations in patients suffering from low vision of one eye due to optic neuritis; when comparing SP in the normal versus the impaired eye, we noticed hardly any difference. This observation is in line with the generally accepted notion that the internal (open loop) gain of the SP system is very high (Gresty and Hamalgyi, 1979; Leigh et al., 1982) and that central visual motion processing depends mainly on the so-called magnocellular visual route, which favors high temporal and low spatial properties of visual stimuli (see Merigan and Maunsell, 1993).

Finally, we address the authors’ question about the background conditions in our SP and VOR-S tests. They were identical (darkness), as was the visual stimulus (light spot). We disagree with the authors’ claim that our VOR-S test added nothing substantial to the SP findings; we hold that the parallel degradation of SP and VOR-S performance, which we observed in our myotonic dystrophy patients, clearly indicates a central rather than a muscular origin of the SP deficit.

References


The course of cortico-hypoglossal projections in the human brainstem: functional testing using transcranial magnetic stimulation

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Abbreviations: CMAP = compound muscle action potential; PES = peripheral electrical stimulation; TMS = transcranial magnetic stimulation

Urban et al. (1996) recently assessed the course of the cortico-hypoglossal projections in the human brainstem using transcranial magnetic stimulation (TMS) of the motor cortex. To this interesting study we want to add some important findings we have made with a similar technique, especially concerning the uncrossed central motor pathways.

A special bipolar surface electrode was used to record the compound muscle action potentials (CMAPs) from the lingual muscles after TMS of the motor cortex and peripheral electrical stimulation (PES) of the hypoglossal nerve medial to the angle of the jaw. With this technique, which was described previously in detail (Muellbacher et al., 1991, 1994; Urban et al., 1994), the assumed bihemispheric motor representation of the lingual muscles was confirmed. In their study, Urban et al. (1996) used a similar recording technique, but PES was only performed in subjects with a medullary lesion (Urban et al., 1996). However, for a reliable estimation of the pattern of crossed and uncrossed central innervation, not only TMS, but also PES with simultaneous recording from either side of the tongue should be performed in every subject.

TMS of one hemisphere produces CMAPs in the contralateral and in the ipsilateral lingual muscles. The ipsilateral CMAPs usually represent responses propagated via the uncrossed corticobulbar pathways, but they may partially be derived from a peripheral ‘contamination’. Interestingly, some individuals do show CMAPs in the contralateral half of the tongue after PES (Fig. 1), despite a careful positioning of the mouthpiece in the midline (Muellbacher et al., 1994; montage B). In our last series of 40 control subjects (80 sides), 23 presented contralateral CMAPs with a sharp initial negative deflection from the baseline with amplitudes up to 64% of the ipsilateral responses. The exact origin of these contralateral responses after PES is unclear, but a similar peripheral ‘cross-over’ of the CMAPs from one side to the other also seems possible after cortical stimulation. The ipsilateral responses after TMS may therefore be derived from the contralateral lingual side, thus not exactly representing CMAPs propagated via the uncrossed cortico-hypoglossal projections.

In their study, Urban et al. (1996) analysed the ipsi- and contralateral responses after cortical stimulation in patients with different brainstem lesions. As a peripheral cross-over...
has not been excluded, the exact origin of the ipsilateral CMAPs after cortical stimulation remains unclear in their series. We therefore propose, that simultaneous recording after PES should be performed in every subject; whenever bilateral responses do occur, the relative amplitudes after PES and after TMS should be given to provide an appropriate estimation of the pattern of crossed and uncrossed cortico-hypoglossal projections.

References

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We read with great interest the article of Urban et al. (1996) entitled ‘The course of cortico-hypoglossal projections in the human brainstem. Functional testing using transcranial magnetic stimulation’. Studying motor function in the tongue muscles and other muscles of the oral pharyngeal cavity is very important due to the severe impairments of dysphagia and dysarthria that patients with neurological disease sometimes experience. We would like to challenge, however, the statement ‘in healthy subjects stimulation in either hemisphere always evokes bilateral responses.’

We are conducting a study in stroke patients with speech and/or swallowing impairment and in normal controls. We perform cortical and subcortical TMS with a Cadwell MES 10 and peripheral stimulation of the hypoglossal nerve using a conventional electrical stimulator. A figure-of-eight coil is used to perform cortical TMS. This type of coil has the ability to produce focal stimulation, permitting the mapping of excitable cortical areas, and a selective stimulation of one hemisphere without affecting the opposite one. All recordings are made on a Nicolet Viking IV instrument.

The responses are recorded with custom EMG surface recording electrodes mounted onto intraoral mandibular and palatal splints similar to those used in orthodontistry. Simultaneous recordings are made at eight locations within the oral cavity. Three recording electrodes are mounted, under the tongue muscle, equally spaced on either side of the mandibular splint. Right and left recording electrodes are also mounted posteriorly onto the palatal splint against the soft palate. Single reference electrodes are placed on each side on the anterior part of the palatal splint (in contact with the hard palate) and the ground electrode is located against the centre of the hard palate. This placement strategy allows an optimal distance between active and reference electrodes, and a neutral or less active location for the reference electrodes (Maloney et al., 1996).

In contrast to the study of Urban et al. (1996), we have been often able to obtain a unilateral response after focal cortical stimulation in normal subjects. This is illustrated in Fig. 1, which demonstrates unilateral motor evoked potentials in the tongue after TMS of the contralateral hemisphere. Our preliminary data suggest a predominantly crossed corticospinal control of the tongue, in contrast to the observations of Urban et al. (1996) and others.

References
Fig. 1 The top panels show unilateral responses from the left anterior, middle and posterior tongue and soft palate (traces 1–4) following magnetic stimulation of the right cerebral cortex in three subjects. The bottom panels show unilateral responses from the right anterior, middle and posterior tongue and soft palate (traces 5–8) following magnetic stimulation of the left cerebral cortex in the same three subjects.

Reply

Technical considerations of electromyographic tongue muscle recordings using transcranial magnetic stimulation

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We are grateful to Muellbacher et al. and Maloney et al. for their remarks concerning possible pitfalls in electromyographic tongue muscle recording techniques. They actually speculate that ipsilateral CMAPs following TMS represent volume conducted activity ('cross-over') from the contralateral half of the tongue instead of responses propagated via the uncrossed corticobulbar pathways.

As an assumption for selective recordings from both halves of the tongue it has been shown that each half of the tongue is innervated separately by the ipsilateral nerve without crossing the midline (Brodal, 1981). A dense sheet of fibrous tissue runs longitudinally down the midline of the tongue dividing it into two symmetric halves (Malek, 1939).

In a previous study (Urban et al., 1994) we carefully
Evaluated our recording technique to assure selective recording from either half of the tongue. To avoid the registration of volume conducted activity pairs of electrodes were arranged in the mouthpiece to ensure placement at the lateral edges of the tongue. In three patients with unilateral hypoglossal nerve section due to carcinoma operations, bilateral TMS and peripheral electrical stimulation of the hypoglossal nerve at the angle of the jaw (PES) demonstrated CMAPs only on the healthy half of the tongue but no relevant volume conducted activity was recorded on the paretic side (fig. 3 in Urban et al., 1994). These results have recently been confirmed in two further patients.

In the study under discussion (Urban et al., 1996a) we analysed ipsi- and contralateral responses from the tongue after TMS in 11 patients with ischaemic brainstem lesions. In five of these patients TMS of one hemisphere demonstrated an isolated absence of the response on only one half of the tongue: in three patients (3, 4 and 9) CMAPs were absent only on the contralateral half of the tongue, but present on the ipsilateral half, and in the other two patients (1 and 11) CMAPs were absent only on the ipsilateral half, but present on the contralateral half of the tongue (see table 1 and figs 1 and 3 in Urban et al., 1996). This further excludes registration of volume conducted potentials over the passive half of the tongue. In case of volume conduction, unilateral responses should not occur. We concluded that we can be sure of selective assessment of the ipsi- and contralateral corticobulbar projections in our series.

Drs. Muellbacher and Mamoli recommend PES of the hypoglossal nerve with simultaneous recordings from either side of the tongue to assess the degree of 'cross-over' activity by comparing the CMAP amplitudes. However, methodological problems in the assessment of CMAP amplitudes following PES include the deep location of the hypoglossal nerve and its variable trajectory, making supramaximal stimulation difficult (Campos et al., 1995). Therefore, high stimulation intensities are required, inducing co-contractions of mouthfloor muscles which lead to gross tongue movements with the risk of displacement of the recording device (Redmond and Di Benedetto, 1988). Gross tongue movements, however, were never observed or experienced following TMS. In view of the intra-individual trial-to-trial variability of the amplitudes of responses to both PES (Redmond and Di Benedetto, 1988) and TMS (Urban et al., 1994) we therefore do not believe that the proposal made by the authors, to use the relative amplitudes after PES and TMS as an estimate of the pattern of crossed and uncrossed cortico-hypoglossal projections, is appropriate.

Drs. Muellbacher and Mamoli now report the presence of CMAPs in the contralateral half of the tongue after PES in 23 out of 40 control subjects. Because further details of the recording device were not reported (Muellbacher et al., 1991; 1994), we assume that the observed 'cross-over' activity might either be due to a too narrow distance between both pairs of recording electrodes or to the technical problems associated with the electrical stimulation technique described above. With a view to these factors, we do not consider that PES of the hypoglossal nerve can be regarded as a valid parameter in the assessment of the amount of 'cross-over' activity. Rather, we recommend improvement of the recording device (e.g. by enlarging the distance between the recording electrodes) to avoid the registration of volume conducted activity and to demonstrate its absence in patients with unilateral hypoglossal nerve sections following TMS, as performed in our study (Urban et al., 1994).

However, we agree with Muellbacher et al. and Maloney et al., that before examining ipsi- and contralateral cortico-hypoglossal projections, selective recordings from either half of the tongue should be performed.

We also appreciate the communication from Maloney et al., who address the question of ipsi- and contralateral cortico-hypoglossal fibre distribution. Human post-mortem degeneration studies have described bilateral projections from each hemisphere to both hypoglossal nuclei (Hoche, 1898). In most cases, there was a more pronounced degeneration contralaterally, showing, however, inter-individual differences with regard to the volume of corticofugal connections to the ipsilateral hypoglossal nucleus (Kuypers, 1958; Schoen, 1969). The bilateral fibre distribution has been suggested to explain the absence or only transitory nature of supranuclear tongue paresis following unilateral lesion of the primary motor cortex or of the internal capsule (Brodal, 1981; Willoughby and Anderson, 1984). Corresponding to the histopathological data, our electrophysiological results demonstrated bilateral cortico-hypoglossal projections from either hemisphere (Urban et al., 1994). As reported previously, a relationship between the bilateral responses observed after unilateral cortical stimulation using a circular coil and unintentional co-stimulation of the contralateral hemisphere could not be established in our study, because similar results were obtained following stimulation with a figure-of-eight coil (Urban et al., 1994). Furthermore, in many patients with unilateral hemispheric or brainstem stroke, stimulation of the affected hemisphere with a circular coil demonstrated absent responses to either half of the tongue (Urban et al., 1995, 1996a, b, 1997). This observation further excludes a co-stimulation of the contralateral hemisphere, which may be due to the lateral coil position (4–6 cm lateral from the vertex). In case of unintentional bihemispheric stimulation, responses should occur following stimulation of the affected hemisphere.

In a recent study, investigating a total of 43 healthy subjects (21 female, 22 male, aged 30 ± 10.1 years) we obtained bilateral responses in each case (Table 1). A circular coil (mean diameter 9 cm) was used for cortical stimulation and in the majority of subjects (n = 25) as well as a figure-of-eight coil (Magstim, Novametrix, Medical Systems Inc., Wallingford, Conn., USA). The centre of the circular coil was positioned tangentially, 4–6 cm lateral from the vertex. On stimulation of the left (right) hemisphere, side A (B) was viewed from above. During slight preinnervation, stimulation strength was increased stepwise until stable latencies were

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findings were recently confirmed by Meyer et al. Nevertheless, our results suggest a predominance of the amplitudes observed following stimulation of the right hemisphere. However, the difference was statistically significant (Wilcoxon test, \( P < 0.05 \)) only for the amplitudes observed following stimulation of the right hemisphere. Nevertheless, our results suggest a predominance of the contralateral cortico-hypoglossal projections. These findings were recently confirmed by Meyer et al. (1997), who demonstrated bilateral tongue muscle responses following focal cortical stimulation in all their subjects (\( n = 21 \)).

Maloney et al. have reported obtaining only contralateral tongue muscle responses after focal cortical stimulation and present the figures of three subjects. This observation, however, is in contrast to all other investigators and to the clinical experience mentioned above, that supranuclear tongue paresis is absent or only transitory following stroke. In the presence of strictly contralateral cortico-hypoglossal projections, as often observed by the authors, persistent tongue paresis following stroke should therefore occur frequently. This is, however, not the case. We thus assume that the results reported by Maloney et al. might be due to an inadequate stimulation or recording technique. However, with a view to the sparse information on the stimulation and recording technique, we can only speculate on the reasons for the reported absence of ipsilateral tongue muscle responses. The authors have not provided any information regarding the coil position, stimulation intensity, number of investigated subjects, and CMAP latencies and amplitudes in control subjects (Maloney et al., 1996).

In their description of the recording technique, Maloney et al. state that custom EMG surface recording electrodes were mounted onto intraoral mandibular splints positioned under the tongue. It therefore appears possible, that the authors recorded activity primarily from muscles of the mouth floor (mylohyoid and venter anterior from the digastric muscle), which are innervated by a motor branch of the trigeminal nerve. However, in most subjects, in muscles innervated by the trigeminal nerve only contralateral responses have been obtained following cortical stimulation (Cruccu et al., 1989). This possibility can be excluded in our studies, because tongue muscle activity is recorded from the back of the tongue.

With respect to the stimulation technique, the results obtained by these authors might, in part, be due to the application of the figure-of-eight coil. In a small number of the subjects we observed, that the position of the figure-of-eight coil required to elicit a contralateral and ipsilateral tongue muscle response varied slightly (up to 2 cm, primarily in the anterior-posterior direction), but when using the circular coil, both, contra- and ipsilateral responses occurred at the same coil position. This observation suggests that the cortical representation areas of the contra- and ipsilateral projections are not identical in these subjects and can thus be activated separately with the figure-of-eight coil. Therefore, the use of the figure-of-eight coil might result in the detection of only one of the two cortico-hypoglossal projections unless the coil position is systematically varied. In contrast, the circular coil activates both areas, perhaps because of the larger electric field associated with this coil. Meyer et al. (1997) performed cortical mappings in five subjects using the figure-of-eight coil. Confirming our observations, they report that ‘in the individual subjects maximal ipsilateral tongue response could be elicited from the same point or from a neighbouring point on the surface grid as contralateral responses’. Yet, a detailed mapping of the tongue motor cortex in a larger number of subjects has not been performed so far.

### Table 1

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<tr>
<th></th>
<th>TCT (ms)</th>
<th>PCT (proximal XII nerve)</th>
<th>DML (distal XII nerve)</th>
<th>CCT (ms)</th>
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<td>L cortex</td>
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<td>latency</td>
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<td>3.7 ± 0.6†</td>
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<td>latency</td>
<td>8.9 ± 0.8</td>
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<td>amplitude</td>
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- \( \text{L} \) lateral
- \( \text{R} \) right
- \( \text{TCT} \) total conduction time
- \( \text{PCT} \) proximal conduction time
- \( \text{DML} \) distal motor latency
- \( \text{CCT} \) central conduction time

Total conduction times (TCT) were measured following cortical stimulation with the circular coil. The peripheral conduction time (PCT) was calculable in 34 (right tongue) and 31 (left tongue) subjects and the side-to-side difference in 28 subjects only. Distal motor latency (DML) was obtained by supramaximal electrical stimulation of the distal hypoglossal nerve at the angle of the jaw.
References


