Does cross-innervation occur after facial palsy?

W. TROJABORG

From the Laboratory of Clinical Neurophysiology, Rigshospitalet, University Hospital, Copenhagen, Denmark

SUMMARY When the unaffected facial nerve was stimulated in 30 patients with facial palsy, evoked action potentials could be recorded from the contralateral (paralysed) perioral muscles. Similarly, in four normal subjects responses were evoked contralateral to the stimulated facial nerve. The latency of these responses in the patients remained unchanged over several months, and they were conducted at a rate compatible with conduction along muscle fibres. The contralateral responses are suggested to be due to conduction along muscle fibres crossing the midline rather than to cross-innervation.

Reinnervation of perioral muscles from the contralateral facial nerve sufficient to produce visible movement, induced either voluntarily or by nerve stimulation, has been reported (Lefebvre and Lerique, 1964; Wochnik-Dyjas, 1964; Passerini et al., 1968; Fisch et al., 1968; Gambi and Tonali, 1973; Kilimov, 1975). Although the observed activity disappeared after anaesthesia of the non-affected facial nerve (Buchthal, 1957, 1965; Trojaborg and Siemssen, 1972) its basis has not been established with certainty.

The purpose of this study was to determine whether the activity evoked either by voluntary effort or by stimulation of the unaffected nerve was due to conduction along nerve fibres or muscle fibres crossing the midline.

Patients and method

Thirty patients with unilateral facial palsy, due in 12 to Bell's palsy, in 13 to removal of a malignant tumour of the parotid gland or an acoustic neuroma, and in five to trauma, were examined. Four subjects without history, signs, or symptoms of neuromuscular disorders were also included.

The electromyogram during full effort was recorded photographically with a three channel electromyograph (DISA). Records were obtained simultaneously from paralytic and non-paralytic sides using concentric or bifilar needle electrodes or both placed in the orbicularis oris muscle. The concentric needle electrode (DISA, type 13K54) had an outer diameter of 0.45 mm and a leading-off area of 0.07 mm². The bifilar electrode (DISA, type 13K80) had an outer diameter of 0.45 mm and consisted of two inner cores of platinum with a leading-off area of 0.015 mm².

The facial nerve was stimulated by needle electrodes with a bare tip of 3 mm. The near nerve electrode was placed at the stylomastoid foramen. Recording electrodes were placed symmetrically at either side of the midline. Muscle potentials were evoked by stimuli (0.2 ms duration) 4.5 times the threshold (T50=0.9±0.1 mA, n=54*) delivered via a double-screened transformer (DISA minstim, type 14E01).

An attempt was made to measure the propagation velocity of the muscle action potential along fibres of the perioral muscle. For this purpose muscle action potentials were recorded along the long axis of the orbicularis oris at different distances (10–60 mm) from the stimulating bifilar electrode placed near the corner of the mouth (see inset in Fig. 1). The action potentials recorded at threshold stimulation were mainly triphasic of 1.5–3 ms duration with a steep positive to negative deflection. With an increase in stimulus strength, the response increased in amplitude and new spikes appeared indicating activation of additional muscle fibres (Fig. 2, below, left).

* Throughout the paper ± refers to the standard error of the mean.
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Results

ELECTROMYOGRAPHY
In about one-third of the patients activity could be recorded from the paralysed orbicularis oris muscle on voluntary effort although stimulation of its nerve failed to evoke responses. The activity was present even though the recording electrode was bifilar and placed as far lateral as possible from the non-affected muscle. As no activity could be evoked in the paralysed facial muscle by stimulation of its nerve, the voluntarily evoked activity might represent pick-up from muscle fibres innervated by the contralateral nerve and crossing the midline. In a previous study (Trojaborg and Siemssen, 1972), it was shown that this type of activity disappeared when the uninvolved facial nerve was paralysed by procaine infiltration. This was not performed in the present study as it would not help to determine whether the activity was due to cross-innervation or to conduction along muscle fibres.

NERVE STIMULATION
Stimulation of the non-affected facial nerve evoked responses both in the normal and in the paralysed orbicularis oris muscle.

Ipsilateral response
This had a latency of $3.5 \pm 0.09$ ms, ($n=36$) when a concentric needle electrode was used for recording (distance $108 \pm 3.0$ mm) and $3.9 \pm 0.08$ ms.
Evoked action potentials from the orbicularis oris muscle (recorded with bifilar electrodes) by stimulating the contralateral facial nerve (upper half), and by direct stimulation of the contralateral muscle fibres (lower half).

(a) From a 64 year old woman with a right sided facial paralysis after parotidectomy eight years previously. Distance from point of stimulation of left facial nerve (stylomastoid foramen) to right orbicularis oris was 165 mm. Distance between stimulating and recording muscle electrodes was 48 mm.

(b) From a 58 year old woman with a right sided facial palsy after removal of an acoustic neuroma eight months previously. Distance from left stylomastoid foramen to right orbicularis oris 180 mm, between stimulating and recording muscle electrodes 56 mm. Numbers above each potential denote conduction time from site of stimulation.

(n=43), when bifilar electrodes were used for recording (distance 117±2.0 mm). The latency increased with the distance between sites of stimulation and recording whether concentric or bipolar electrodes were used for recording (Fig. 3). A difference in latency of 0.22 ms per 10 mm difference in distance corresponded to a conduction velocity of 46 m/s similar to the 44 m/s obtained when stimulating the facial nerve at two sites along its course and to the 43 m/s found by Olsen (1975).

Contralateral response

Bifilar electrodes were used for recording to avoid pick-up of spread of activity from the ipsilateral side. It was sometimes necessary to adjust the position of the recording electrode in the muscle to pick up a response. When two electrodes were adjusted in the paralysed perioral muscle 5–30 mm apart, the difference in latency between the evoked responses was 5.2±0.7 ms (Table, a) and increased with increasing distance (3.5 ms per 10 mm) corresponding to a conduction velocity of 3 m/s (r=0.66, Fig. 4, crosses). When the responses were recorded with one bifilar electrode, the latency of the action potential was 13.6±0.4 ms (n=54). The amplitude of the response averaged 530±70 µV. The latency increased 0.9 ms per 10 mm corresponding to a conduction velocity of 11 m/s (r=0.31), but this latency included conduction also along a normal facial nerve.

MUSCLE FIBRE STIMULATION

When a response was present over the contra-
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The potentials evoked by stimulation of nerve and of muscle had a similar shape and amplitude (Fig. 2) and the difference in latency between these responses was of the same order as the latency of the ipsilateral response evoked by nerve stimulation. Thus, the difference in latency between the contralateral and ipsilateral responses evoked by nerve stimulation was similar to the latency of the response evoked by muscle stimulation, and was, therefore, a fair estimation of the conduction time from the left to the right side or vice versa. When the segment of conduction along the facial nerve was excluded the latency of the contralateral response averaged 9.9 ± 0.4 ms (Table, c) and increased 1.63 ms per 10 mm increase in distance, corresponding to a conduction velocity of 6 m/s (r=0.52), slightly higher than that obtained when using two electrodes for pick-up, but similar to that obtained for muscle fibres.

**NORMAL SUBJECTS**

The latency of the action potentials recorded from the perioral muscle contralateral to the side of facial nerve stimulation was 11.8 ± 0.5 ms (distance 157 ± 3 mm, n = 8). In these subjects also, as in the patients, the latency increased 1.0 ms per 10 mm increase in distance between stimulating and recording electrodes.

In one subject the conduction along perioral muscle fibres was determined to 4.0 ± 0.4 m/s (n = 5).

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**Table** Latency of responses from orbicularis oris muscle evoked by direct or indirect stimulation

<table>
<thead>
<tr>
<th></th>
<th>Latency (ms) mean ± ME</th>
<th>Distance (mm) mean ± ME</th>
<th>ms/10 mm</th>
<th>CV m/s</th>
<th>Correlation coefficient</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ipsilateral response*</td>
<td>3.7 ± 0.06</td>
<td>113 ± 2</td>
<td>0.22</td>
<td>46</td>
<td>0.58</td>
<td>79</td>
</tr>
<tr>
<td>Contralateral response*</td>
<td>13.6 ± 0.4</td>
<td>172 ± 2</td>
<td>0.9</td>
<td>11</td>
<td>0.31</td>
<td>54</td>
</tr>
<tr>
<td>(a) Contralateral response†</td>
<td>5.2 ± 0.7</td>
<td>13 ± 1</td>
<td>3.5</td>
<td>3</td>
<td>0.66</td>
<td>19</td>
</tr>
<tr>
<td>(b) Muscle fibres</td>
<td>8.6 ± 1.0</td>
<td>33 ± 4</td>
<td>2.13</td>
<td>5</td>
<td>0.82</td>
<td>28</td>
</tr>
<tr>
<td>(c) Contralateral direct response</td>
<td>9.9 ± 0.4</td>
<td>55 ± 4</td>
<td>1.63</td>
<td>6</td>
<td>0.52</td>
<td>54</td>
</tr>
<tr>
<td>a + b + c</td>
<td>8.7 ± 0.4</td>
<td>41 ± 2</td>
<td>1.42</td>
<td>7</td>
<td>0.71</td>
<td>101</td>
</tr>
</tbody>
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* Stimulation of ipsilateral facial nerve.
† Difference in latency between two recording electrodes placed contralateral to side of nerve stimulation.
CV = motor conduction velocity.
FOLLOW-UP

To investigate whether the latency of the contralateral response decreased during recovery, 14 patients were reexamined two or more times.

At the first examination, less than one month and as early as seven days after the onset of the facial palsy, the latency was 11.1 ± 1.0 ms for a conduction distance of 170 ± 3 mm (n = 14). At the second investigation, two to four months after onset, the latency averaged 13.6 ± 0.7 ms (173 ± 2 mm distance, n = 24), and at the third examination, six to 31 months after onset, it was 12.4 ± 0.5 ms (distance 173 ± 2 mm, n = 10). Moreover, a comparison of the difference in latency in individual patients from one examination to another did not show any consistent reduction. In fact, there was rather a tendency towards the opposite.

Discussion

The question raised in this study was whether the activity evoked either voluntarily or by nerve stimulation could be accounted for by assuming the presence of cross-innervation, or whether the activity could be due to the anatomical arrangement of the muscle fibres.

Recording of the voluntarily evoked electromyogram did not solve the problem as activity may be present on the paralysed side and nerve block will abolish it in any case. When electrodes are placed symmetrically on the perioral muscles of both sides in patients with a unilateral peripheral facial palsy, voluntarily evoked activity may be recorded on the paralysed side even though the recording electrode is placed as far lateral as possible from the non-affected muscle. When using bifilar needle electrodes the activity is much reduced but not completely abolished (Trojaborg and Siemssen, 1972).

The absence of a contralateral response to nerve stimulation in patients with central involvement of the facial nerve and in normal subjects (Wochnik-Dyjas, 1964; Passerini et al., 1968) but the existence of a response in patients with a peripheral facial palsy would favour the assumption of cross-innervation. Similarly, the finding of activity

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Fig. 4 Relation between latency of action potentials and conduction distance. (a) Differences in latency between two electrodes placed in the orbicularis oris opposite to the side of facial nerve stimulation (X). (b) Action potentials elicited by muscle fibre stimulation (○), (c) differences in latency between muscle response recorded contralaterally and that from ipsilateral site (○). Triangles represent values from normal subjects. The full line is the regression line for values obtained from patients.
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in other paralysed facial muscles such as the corrugator glabellae (Kilimov, 1975), levator nasi and levator labii (Lefebvre and Lerique, 1964; Fisch et al., 1968), points to a neurogenic mechanism.

On the other hand the observation of Gambi and Tonali (1973) would support a 'myogenic' explanation. In four normal subjects they recorded action potentials in perioral muscles 20–25 mm from the midline contralateral to the stimulated facial nerve. They interpreted their findings as indicating extension of the motor endplates across the midline. However, with the electrodes spaced 10 mm apart they found a difference in latency of the evoked responses of 3.3–3.8 ms corresponding to a velocity of about 3 m/s. This is compatible with my findings and with the rate of conduction along skeletal muscle fibres (Buchthal et al., 1955). Moreover, the mean latencies of their ipsilateral and contralateral responses were similar to those reported here, (though conduction distances were not given). Passerini et al. (1968) found a mean latency of 8.4±0.7 ms (n=10), compatible with my findings when corrected for the difference in conduction distance (150 mm versus 172 mm).

When the latency of the contralateral response evoked by nerve stimulation was corrected for the conduction time along the nerve it was the same as the latency of the response evoked by stimulating muscle fibres with the recording electrodes left in place—that is, the same rate of conduction of the action potential, whether evoked by direct or indirect stimulation. A similar calculation of the data presented by Passerini et al. (1968) and Gambi and Tonali (1973) is in agreement with my findings and in keeping with the anatomical arrangement of the perioral muscle fibres. It is a sphincter with intermingling of muscle fibres on both sides of the midline (Nairn, 1975).

Responses contralateral to the stimulated facial nerve were found in all three patients with a facial palsy of less than one month duration (Passerini et al., 1968), in all my 14 patients examined in the acute stage, and as early as seven days after the onset of palsy. If the responses were conducted along newly formed nerve fibres one would expect changes in latency with time. However, the mean latency of the contralateral responses was the same in patients examined two to four months (n=24) after onset of palsy as in those examined six to 31 months (n=10) after the onset. A similar result was obtained by Passerini et al. (1968) and by Gambi and Tonali (1973). These findings favour the assumption that the responses are conducted along muscle fibres traversing the midline.

Although the histological location and extension of the endplate region in the perioral muscles is unknown, electrophysiological findings suggest that it is placed about 25 mm from the midline in the upper lip (Olsen, 1975). If cross-innervation occurs the peripheral sprouts must travel 25 mm or more to innervate the contralateral denervated muscle fibres. The establishment of sufficient innervation to account for the electrophysiological findings could be expected within a month or so, but was observed as early as one week after the onset of paralysis. Moreover, if cross-innervation occurs one would expect a much better result clinically with time than in fact occurs.

References


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