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The Site of Impulse Generation in Transcranial Magnetic Stimulation of the Facial Nerve

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The facial nerve can be stimulated in its intracranial course through transcranial magnetic stimulation (TMS). We studied the site of impulse generation produced by TMS by comparing the latencies of the muscle evoked potentials (MEPs) elicited with TMS and intracranial electrical stimulation (IES) of the facial nerve during neurosurgical posterior fossa procedures. In a series of 25 patients, the mean latency of the TMS elicited MEPs, recorded in the orbicularis oris muscle, was 5.0 ms (SD 0.58). Also IES of the distal part of the facial nerve in the internal acoustic meatus showed a mean latency of 5.0 ms (SD 0.68). Proximal IES in the root entry zone of the facial nerve, and intermediate IES between root entry zone and meatus, produced MEPs with significantly longer latencies compared to TMS and distal IES ($p < 0.05$). The findings suggest that the TMS induced facial nerve activation, leading to a MEP response, takes place within the internal acoustic meatus. *Key words: location of facial nerve activation.*

INTRODUCTION

The intracranial part of the facial nerve can be stimulated through transcranial magnetic stimulation (TMS) (1). The method has been used for facial nerve (FN) function tests in Bell's palsy (2). However, clinical applications of the method require a more exact knowledge of the FN impulse generation site. TMS may also provide information on other cranial nerves (3, 4).

The latencies of muscle evoked potentials (MEPs) elicited with TMS are 1.0–1.2 ms longer than those elicited with electrical stimulation extracranially at the stylomastoid foramen (1, 5, 6). The impulse generation site can be calculated from this difference in latency, given that the FN conduction velocity is approximately 15–50 m/s (7, 8). The TMS site, with this method, may come close to the internal acoustic meatus.

In order to determine the TMS impulse generation site more precisely, we compared the MEPs elicited with TMS to those elicited with intracranial electrical stimulation (IES) of the facial nerve in 25 patients. The two methods were compared during posterior fossa surgery where low constant current electrical impulses can be given under visual control along the course of the nerve.

MATERIAL AND METHODS

Patients. The clinical details of the 25 patients operated on at the Helsinki University Central Hospital are presented in Table I.

In 8 patients, a single dose of 2 mg pancuronium, a muscle relaxant with a relatively long-lasting effect, was given at the beginning of anaesthesia. In patients 1 and 21, pancuronium was also given during the operation. Between the two neurophysiological examinations, no relaxants were given. In the rest of the patients, only short-lasting muscle relaxants were administered for endotracheal intubation (Table I).

With the patient placed in the lateral position we used a retrosigmoid approach, and debulked and dissected the tumor free from the cerebellum, the brain stem and the cranial nerves. We used IES to identify the FN. We opened the meatus with a high speed drill and completed the dissection alternatingly from the proximal and the distal direction. In some patients with FN tumor infiltration, some small pieces of tumor were left along the preserved FN.

Equipment. We used a commercially available four-channel Nihon Kohden Neuropack ENMG device for the electrical stimulation and for the recording of electrically as well as magnetically induced responses. The magnetic stimulation was performed with three different commercial devices (Table I).

Intracranial electrical stimulation (IES). We used a direct cathodal surface contact, using a small-tip electrode and a 0.2 mA electrical impulse with a duration of 0.1–0.2 ms. The anode was fixed to the scalp, making the stimulation practically monopolar. Repetitive 1 to 10 Hz frequency impulses were given.

The distal FN stimuli were given in the internal acoustic meatus and the proximal ones at the FN

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Table I. *Patients with a posterior fossa tumor*

Abbreviations: *pr* = propofol, *t* = thiopenthal, *f* = fentanyl, *s* = suxamethanon, *v* = vecuronium, *pa* = pancuronium, a single dose of 2 mg at intubation, *Pa* = pancuronium, a total dose of more than 2 mg was given during the operation, *I* = isoflurane, *N* = nitrous oxide. *ENoG asymmetry* = preoperative ENoG side-to-side amplitude difference (%). *CFP* = Cadwell MES 10 magnetic stimulator with a Focal Point coil ring. *Btf* = Butterfly, type of coil ring used with Magstim. *AN* = acoustic neurinoma

Code number	TMS device and coil	Age	Sex	Anaesthetics	Diagnosis	Size of tumor (cm)	Rectal temperature	Preop. ENOG asymmetry (%)
1	Dantec S 100	43	female	t + f + Pa + I + N	acoustic neurinoma	3		15
2	Dantec S 100	53	female	t + f + s + pa + I + N	meningeoma	1.5		7
3	Magstim High Power	50	male	t + f + v + I + N	acoustic neurinoma	2		0
4	Magstim High Power	56	female	t + f + v + s + I + N	acoustic neurinoma	1.5		35
5	Magstim High Power	40	male	t + f + v + I + N	acoustic neurinoma	1		15
6	Magstim High Power	50	female	t + pr + f + s + pa + I + N	acoustic neurinoma	1		4
7	Magstim High Power	59	male	t + f + v + I + N	acoustic neurinoma	3		11
8	Magstim High Power	18	male	pr + f + s + pa + N	acoustic neurinoma	3		2
9	Magstim High Power	54	male	pr + f + s + pa + N	acoustic neurinoma	3		17
10	Magstim High Power	44	male	t + f + s + N	acoustic neurinoma	2		20
11	Magstim High Power	36	male	pr + f + s + pa	acoustic neurinoma	5		13
12	Magstim High Power	44	male	pr + f + s + pa + I	acoustic neurinoma	1.5		25
13	Magstim High Power	51	female	pr + f + s + pa + I	acoustic neurinoma	1		3
14	Magstim High Power	49	female	pr + f + s + pa + I	acoustic neurinoma	2	34.2	24
15	Magstim High Power	45	male	pr + f + s + N	acoustic neurinoma	4		38
16	CFP & Magstim btf	52	female	pr + f + v + s + N	acoustic neurinoma	1	34.4	49
17	Magstim butterfly	23	male	pr + f + v + s + N	acoustic neurinoma	2.5	35.2	14
18	Magstim butterfly	73	female	pr + f + v + s	meningeoma	2	34.9	11
19	Magstim butterfly	42	female	pr + f + v + s	acoustic neurinoma	3	35.5	33
20	Magstim butterfly	65	female	pr + f + v + N	acoustic neurinoma	1		28
21	Magstim butterfly	52	male	pr + f + s + Pa + N	acoustic neurinoma	0.7		0
22	Magstim butterfly	42	female	t + f + s + pr + N	acoustic neurinoma	3	35.1	0
23	Magstim butterfly	29	female	pr + f + s + N	acoustic neurinoma	1.5		34
24	Magstim butterfly	37	male	pr + f + v + N	acoustic neurinoma	2	34	0
25	Magstim butterfly	30	male	pr + f + v + s + N	acoustic neurinoma	2.5	35.8	40

root entry zone near the brain stem (9). Cisternal stimuli proximal to the meatus but distal to the root entry zone were called intermediate stimuli.

Transcranial magnetic stimulation (TMS). The coil was placed tangentially on the head in the temporo-occipital region. The estimated distance between the plastic coat of the coil and the scalp varied between 0.5–1 cm. There was some intra- and interindividual variation in the location of the coil due to instrumentation on the operational area. Also the type and shape of the coil ring varied. Dantec S 100 coil was used with two patients, Magstim High Power coil ring with 13 patients, and Magstim butterfly coil ring with 10 patients. In one patient, Cadwell Focal Point coil ring was used in addition to butterfly coil (Table I). Impulses, 35% to 50% of the maximum device capacity were sufficient to evoke responses comparable to those with the distal IES.

Recording of responses. Muscle responses were recorded on the side of the stimuli. In patients 1, 2, 3, 15, and 20, a 5 mm surface electrode was placed on the nasolabial fold. The referential electrode was located ipsilaterally on the nose at the nasal bone level.

In patients 4 to 25, including 15 and 20, a standard 0.1 mm coaxial concentric needle electrode was used for recording the responses. The electrode was inserted in the superior orbicularis oris muscle close to the nasolabial fold. We found the needle electrode more reliable because of its constant impedance throughout long operations, and it also proved more sensitive for nerve monitoring during surgery. In cases with both surface and needle recordings, the responses recorded with needle electrode were used for statistical comparison.

The amplifier bandpass was set at 3 Hz (high pass) to 3000 Hz (low pass).

We measured the muscle response latency from the beginning of the deflection. The amplitude was the maximum peak-to-peak voltage difference.

The facial nerve was sensitive to saline flushing and manipulation. Anaesthetics and changes in body temperature (10) may also modify facial nerve and myoneural junction conductivity. Therefore in all IES and TMS measurements, we accepted the response with the shortest latency for statistical analysis, because it was probably least modified by external factors. The responses in Figs. 1 and 3 do not neccessarily show the responses with shortest latencies in each stimulation site but are rather time and phase related to surgery.

Statistical analysis. Student's t-test was used to compare the significance of differences.

RESULTS

Table II shows the individual responses. With TMS the mean minimum latency of responses was 5.0 ms (SD 0.58). With distal IES the corresponding latency was 5.0 ms (SD 0.68). With intermediate and proximal IES the mean latencies were 6.0 ms (SD 0.74) and 6.7 (SD 0.89), respectively, and they differed significantly from the responses elicited with TMS and distal IES ($p < 0.05$). The MEP responses were usually polyphasic, as shown in Fig. 1. The latency variation with the different IES sites and the TMS was evident also with surface recording (Fig. 2).

The continuity of the facial nerve was broken proximal to the internal acoustic meatus in one

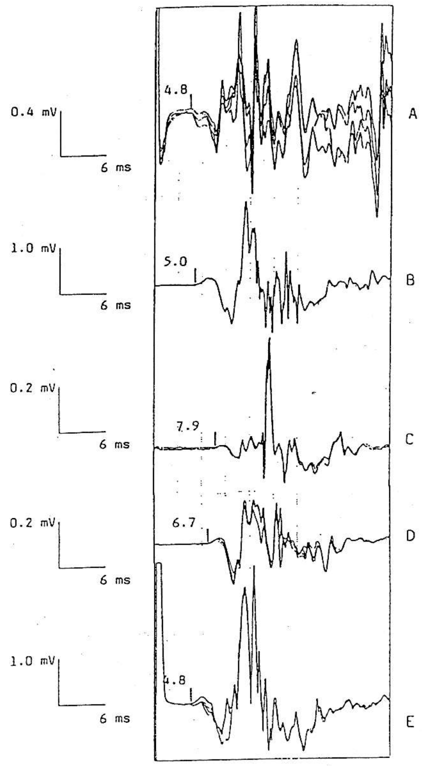

Fig. 1. Five series of four consecutive facial motor evoked potentials recorded with a needle electrode from the superior orbicularis oris muscle (Patient 11). Recordings were performed before and after facial nerve injury 1 cm distal from the pons. (A) Transcranial magnetic stimulation (TMS) before the injury, at 10.40 hours. (B) Intracranial electrical stimulation (IES) at the internal acoustic meatus before the lesion, at 16.55 hours. (C) IES at root entry zone at 20.00 hours, some minutes before the lesion. (D) IES on the distal side of nerve anastomosis after the injury. (E) TMS after the injury, at 21.37 hours.

Table II. *Latencies and amplitudes of the responses. Responses with minimum latencies for each stimulation site and method are shown*

Code number	Latencies of the responses (ms)				Latency difference between TMS and Distal IES	Amplitudes of the responses (mA)			
	TMS	Distal IES	Intermediate IES	Proximal IES		TMS	Distal IES	Intermediate IES	Proximal IES
1	4.9	4.9		6.4	0.0	1.2	1.0		0.6
2	5.1	5.0	6.0	6.0	0.1	1.0	1.0	0.4	1.0
3	5.0	4.8	6.0	6.6	0.2	1.6	1.4	0.6	0.9
4	5.0	5.0	5.7	6.7	0.0	0.7	0.7	0.8	0.4
5	5.0	4.5	5.7	6.5	0.5	1.0	0.8	0.4	0.4
6	5.4	5.5	6.3		−0.1	3.5	1.5	0.8	
7	6.0	6.0	8.0	9.0	0.0	3.5	1.6	0.5	0.8
8	4.0	4.0		6.0	0.0	3.5	3.5		1.1
9	5.6	5.6		7.6	0.0	1.3	1.0		0.5
10	4.4	4.8	6.0	7.0	−0.4	2.0	2.0	2.0	0.5
11	4.5	4.7	5.9	7.3	−0.2	0.8	1.8	1.0	1.0
12	5.7	5.3			0.4	0.6	2.0		
13	5.5	5.5	6.1	6.2	0.0	0.4	0,7	0.4	0.4
14	4.7	4.2		6.8	0.5	2.0	2.0		1.5
15	4.9	4.8	5.1	6.2	0.1	1.8	2.0	2.0	1.2
16	5.5	6.1		7.0	−0.6	2.0	2.0		1.4
17	4.0	4.0		5.0	0.0	1.0	1.2		1.6
18	6.0	6.1		7.6	−0.1	1.0	2.0		2.0
19	4.2	4.0	5.4	5.8	0.2	0.8	1.2	1.0	1.0
20	4.5	4.4		6.6	0.1	2.0	2.7		1.2
21	5.0	5.8		6.9	−0.8	2.0	1.4		2.0
22	4.6	4.7		8.0	−0.1	2.0	2.0		0.7
23	5.3	5.7		6.3	−0.4	0.9	0.8		0.7
24	5.7	5.8		8.0	−0.1	0.7	1.1		1.8
25	4.5	4.4		5.6	0.1	2.0	2.0		1.4
Number	25	25	11	23	25	25	25	11	23
Mean	5.0	5.0	6.0	6.7	0.0	1.6	1.6	0.9	1.0
SD	0.58	0.68	0.74	0.89	0.30	0.90	0.67	0.59	0.50
Min	4.0	4.0	5.1	5.0	−0.8	0.4	0.7	0.4	0.4
Max	6.0	6.1	8.0	9.0	0.5	3.5	3.5	2.0	2.0

IV

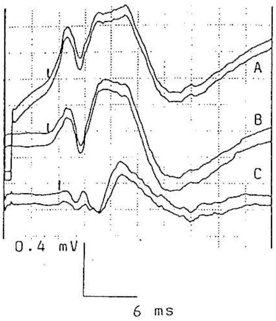

Fig. 2. Three series of two consecutive facial motor evoked potentials recorded with a surface electrode from the superior orbicularis oris muscle (Patient 20). (A) Transcranial magnetic stimulation (TMS), latency 4.5 ms. (B) Intracranial electrical stimulation (IES) at the internal acoustic meatus, latency 4.5 ms. (C) IES at the root entry zone, latency 6.0 ms.

patient (19) (Fig. 3). In two other patients (1, 22), with the preserved anatomical nerve continuity, the electrophysiological nerve conductivity was broken at the same level. MEPs could be elicited with TMS in each of these patients, and the latencies equalled to those registered before the nerve lesions.

In one patient (11), the nerve continuity was lost 1 cm distal from the pons. The intermediate IES latency was thereafter 6.7 ms. The TMS latencies were 4.8 ms before and after the injury (Fig. 1).

Furthermore, in patient 2 the intermediate IES latency increased to 9.0 ms while the nerve was being pulled. In comparison, the minimum latency for electric stimulation at the root entry zone before the conduction delay was 6.0 ms. In patient 7, the proximal IES latency was prolonged from the minimum value of 9.0 ms to 18.0 ms during the dissection of the tumor. Correspondingly, the amplitudes of the responses were decreased from the maximum of 1.1 mV to 0.02 mV. The reason for latency increases was most likely a partial nerve injury. The TMS responses remained essentially as before the lesion in both of the patients.

DISCUSSION

During posterior fossa operations the intracranial part of the facial nerve is accessible to direct electrical stimulation. The precise point of the stimulation is possible using a small-tip electrode and a low current. The site of impulse generated with TMS can be determined by comparing the latencies of TMS responses to those elicited with IES in predetermined parts of the facial nerve.

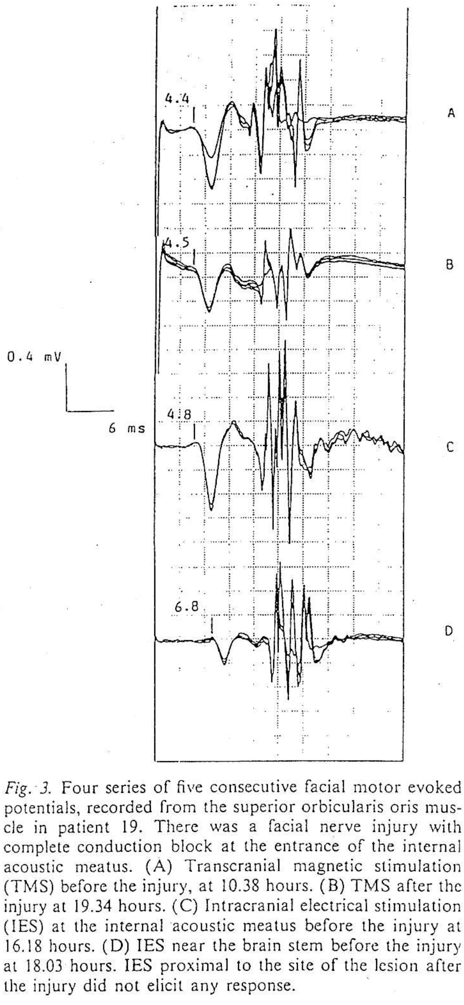

Fig. 3. Four series of five consecutive facial motor evoked potentials, recorded from the superior orbicularis oris muscle in patient 19. There was a facial nerve injury with complete conduction block at the entrance of the internal acoustic meatus. (A) Transcranial magnetic stimulation (TMS) before the injury, at 10.38 hours. (B) TMS after the injury at 19.34 hours. (C) Intracranial electrical stimulation (IES) at the internal acoustic meatus before the injury at 16.18 hours. (D) IES near the brain stem before the injury at 18.03 hours. IES proximal to the site of the lesion after the injury did not elicit any response.

Responses elicited with TMS had the latency difference range of -0.8 to 0.5 ms when compared with distal IES at the internal acoustic meatus, the mean latencies being practically the same, 5.0 ms, with both stimulation methods. Responses from intermediate and proximal stimulation invariably showed longer latencies ($p < 0.05$). The facial nerve continuity (2 patients) or conductivity (4 patients) was lost or impaired near the internal acoustic meatus or in the intermediate area in 6 cases. In each of these patients the TMS responses were intact after the injury, but the IES responses, proximal or at the site of the lesion, were absent or longer than those from TMS and distal IES. These findings suggest that TMS-induced facial nerve activation leading to MEP-response takes place near the internal acoustic meatus (11, 12). Based on the variation in the range of latency differences with TMS and distal IES, we consider that the site of impulse generation may also vary at some extent between the individuals.

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MEPs from intraoperative TMS have on an average 0.6 ms longer latencies compared to those in healthy volunteers (1). Facial nerve compression and tumor infiltration may be some reasons for this difference. The lower body temperature during the operation, and anaesthetics may also prolong the conduction times.

The shape of the coil may have an effect on the magnetic field stimulated (13). In this study, however, our comparison of round and butterfly coils showed no difference in the site of impulse generation. Probably because all magnetic stimulators generate a current widely distributed to the brain, the type of stimulator does not affect the responses.

The magnetic field induced by a magnetic coil is large (13) and covers the whole intracranial part of the facial nerve. Probably the magnetic field activates a longer segment of the nerve rather than an exact site in the nerve. Possibly a proximal activation, simultaneously with a more distal one, may be inhibited by the refractory period of the nerve. Therefore the depolarization of the distal nerve segment only, i.e. the segment near the internal acoustic meatus, probably generates the impulse initiating the MEP.

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