Intracranial stimulation of the trigeminal nerve in man

I. Direct motor responses

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SUMMARY Direct electrical stimulation of the intracranial portion of the trigeminal nerve was performed in 23 subjects undergoing retrogasserian thermocoagulation for the treatment of idiopathic trigeminal neuralgia. In 16 subjects, who were having the operation for the first time, neurological examination was normal, as was neurophysiological testing of trigeminal function. Seven subjects were being operated for the second time, owing to a recurrence of symptoms. In all the subjects being operated for the first time, direct motor responses were obtained from ipsilateral temporalis, masseter and anterior belly of the digastric. The motor conduction velocity was equal for the fibres directed to all three muscles. This was estimated to be 54 m/s in the masseteric nerve and 55–68 m/s in the intracranial portion of the trigeminal nerve. Patients who had undergone previous thermocoagulation had a considerably slower conduction velocity. It is supposed that myelin sheaths had been damaged at the first operation.

The trigeminal nerve is a mixed nerve subserving sensation of the face and the muscles of mastication. The motor and sensory fibres, however, run in separate nerves for most of their course, partially merging only in the retrogasserian rootlets and in a short portion of the mandibular nerve.¹ While the cutaneous nerves, through the supraorbital, infraorbital and mental foramina, emerge on the surface of the face, the motor nerves lie in the depth of the facial structures and their terminal branches enter the muscle bellies on their deep surface. This makes the stimulation of trigeminal efferent and afferent alpha fibres difficult. When compared with the main limb nerves, electrophysiology has provided little information about the human trigeminal nerve. The conduction velocity of the motor fibres and the afferent sensory fibre groups have not been directly measured, the cortical evoked potentials are far from being certainly identified^{2,3} and even the more widely used trigeminal reflexes are obscure in many details.^{4,5}

The operation of percutaneous trigeminal rhizotomy provides a unique opportunity to reach the intracranial portion of the trigeminal nerve and to

stimulate motor and sensory fibres, thus allowing the recording of both direct and reflex responses. This paper describes the motor responses in the masticatory muscles from stimulation of the retrogasserian rootlets and the mandibular nerve, in subjects without neurological defects who were having the operation for the first time, and in subjects who had been already submitted to a surgical retrogasserian lesion, either by “controlled differential thermocoagulation”^{6,7} or by “partial root section”.^{8,9} Both techniques are supposed to spare the motor fibres.

Methods

The study has been carried out in 23 subjects, who gave informed consent, undergoing retrogasserian rhizotomy by radiofrequency thermocoagulation⁶ for the treatment of idiopathic neuralgia in the third (V3) or second (V2) division. There were 11 men and 12 women. The right side of the face was affected in 14 cases and the left in nine cases.

Sixteen cases were diagnosed as idiopathic trigeminal neuralgia, eight in V3 and eight in V2. Their ages ranged from 43 to 67 years, the mean age being 56 years. These subjects were selected among those without abnormal neurological or electrodiagnostic findings using known electrophysiological techniques: blink reflex and jaw jerk, masticatory EMG.^{10–14} In particular, the supraorbital nerve was stimulated transcutaneously at the supraorbital groove and the infraorbital nerve via a pair of fine needle-electrodes inserted into the homonymous foramen. In both cases the right and left side were stimulated with square wave 0.1–0.3 ms pulses of adequate intensity and surface recordings were

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Table 1 Direct responses in the masticatory muscles (mean $\pm$ standard deviation of negative components)

	Masseter			Temporalis			Digastric		
	Onset latency (ms)	Amplitude (μ V)	Duration (ms)	Onset latency (ms)	Amplitude (μ V)	Duration (ms)	Onset latency (ms)	Amplitude (μ V)	Duration (ms)
Stimulation at foramen ovale (16 subjects)	2.2 $\pm$ 0.19	3100 $\pm$ 2300	3.9 $\pm$ 0.7	2.2 $\pm$ 0.36	4300 $\pm$ 4000	5.3 $\pm$ 0.9	2.94 $\pm$ 0.33	4400 $\pm$ 3700	4.3 $\pm$ 0.5
Stimulation at clivus (paired differences with foramen ovale in 8 V3 subjects)	+0.26 $\pm$ 0.14	-900 $\pm$ 1800	0.1 $\pm$ 0.12	0.25 $\pm$ 0.19	-1200 $\pm$ 3000	+0.14 $\pm$ 0.44	+0.24 $\pm$ 0.09	-1100 $\pm$ 2000	+0.03 $\pm$ 0.23
	p < 0.01	NS	p < 0.05	p < 0.01	NS	NS	p < 0.01	NS	NS
Stimulation at zygomatic arch (paired differences with foramen ovale in 8 V3 subjects)	-0.55 $\pm$ 0.21	+1200 $\pm$ 1500	-0.5 $\pm$ 0.6						
	p < 0.01	NS	p < 0.05						

taken from the inferior orbicularis oculi muscle bilaterally. The jaw jerk was evoked by tapping the chin with a triggered hammer and recorded from the masseter and temporal muscles bilaterally.

Seven cases had previously undergone an operation for the treatment of facial pain which had been diagnosed as idiopathic trigeminal neuralgia (table 2). Five cases were having the same operation, that is radiofrequency thermocoagulation, for the second time, and two cases had had surgical partial section of the root. In two cases there had been no pain relief and in the others the neuralgia had recurred. All presented with sensory deficits to a variable extent or neurophysiological abnormalities. Most patients were receiving anticonvulsants (mainly carbamazepine) up to 24 hours before surgery. During the operation the subjects were fully conscious, while they were always fully anaesthetised during the coagulation, by means of a short-acting barbiturate (methohexitone IV).

The percutaneous method employed for the lesion of the sensory root has been described in detail by several authors.^{6,7,15} The ganglion was approached by passing a cannula through the foramen ovale, guided by means of x rays and image intensification with a TV monitor. The intra-operative recordings were first performed when radiographs in lateral projection showed that the tip of the cannula had reached the foramen ovale and then when the level of the clivus was attained. In the first position the needle was presumed to be in the mandibular nerve just distal to the ganglion, and in the second within the trigeminal root.^{1,7} The stimulating electrode consisted of an insulated stylet with a bare tip of 2 mm which protruded by 1 mm from the teflon-coated surgical needle, whose tip was bared for 5 mm. The stylet was connected to the cathode and the cannula to the anode of the stimulator. Square-wave negative pulses (0.5–50 mA, 0.1–0.5 ms) were delivered manually at a low rate or repetitively at 1 Hz.

Table 2 Masseter responses in re-operated subjects

Case, sex & age (yr)	Previous operation	M-wave latency	M-wave amplitude	Intracranial velocity	Jaw reflex	Clinical-EMG function	Comment
Case 4 F 61	Thermocoagulation for left V2 2 years before	2.9 ms (abnormal)	2.1 mV	28–34 m/s (abnormal)	Delayed	Normal	Demyelination
Case 5 M 63	Thermocoagulation for right V2–V3 3 months before	2.2 ms	0.3 mV (abnormal)	35–40 m/s (abnormal)	Absent	Abnormal	Demyelination & fibre loss
Case 11 M 68	Thermocoagulation for left V3 10 months before	2.1 ms	1.2 mV	60–73 m/s	Normal	Normal	Normal
Case 13 F 46	Thermocoagulation for right V3 1 month before	2.4 ms	2.2 mV	30–32 m/s (abnormal)	Delayed	Normal	Demyelination
Case 14 M 78	Partial right root section 20 years before	Absent	Absent	Non evaluable	Absent	Abnormal	Massive fibre loss
Case 19 F 66	Partial left root section 12 years before	2.2 ms	1.8 mV	48–56 m/s	Normal	Normal	Normal
Case 22 M 70	Thermocoagulation for left V2 5 years before	2.6 ms (borderline)	2.4 mV	40–49 m/s (borderline)	Normal	Normal	Doubtful

Signals were recorded by surface electrodes from the ipsilateral temporal, masseter and anterior belly of the digastric muscles, and by needle-electrodes from the orbicularis oculi. In the first cases, recording from the contralateral facial and masticatory muscles was also performed, as well as recordings by concentric EMG needles inserted into the muscle bellies. Signals were amplified, filtered (8–3200 Hz) and displayed by means of standard electromyography, and recorded on tape. Off-line processing included rectification and averaging when adequate, A/D conversion and storage on floppy-disks for computer analysis. Statistical significance was evaluated with the Student's *t* test.

The day after the operation, eight cases were submitted to stimulation of the masseteric nerve by means of two fine needles inserted immediately below the zygomatic arch and just anterior to the T-M joint to a depth of 10–15 mm, according to the technique described by Godaux and Desmedt.¹⁶ By measurements on skulls and a topographic atlas,¹⁷ this position was evaluated to be 30 mm from the foramen ovale and 40 mm from the masseter motor point in the lower third of the muscle; the overall distance from the foramen ovale was approximately 70 mm to the masseter and temporalis and 112 mm to the anterior belly of the digastric.

Results

Neurophysiological testing of trigeminal function

The three trigeminal divisions were investigated by means of the blink reflex to supraorbital stimulation (V1), the blink reflex to infraorbital stimulation (V2) and the jaw jerk to taps to the chin (V3).

Latency, duration and amplitude of all reflexes fell in the normal range.^{5 11 13 18} However, because of the relatively wide interindividual range of normal values, the intraindividual latency difference between the right and left side was taken as a further discriminant for all reflexes. Latency differences of more than 1.2 ms, 8 ms and 0.5 ms, for the early and late components of the blink reflex and for the jaw jerk respectively, were considered to be pathological findings. Three subjects had at least one delayed response on the same side as the neuralgia and consequently did not enter the study. Among the 16 selected cases, the early component of the blink reflex to infraorbital stimulation was bilaterally absent in four, while the late component and supraorbital blink were normal. This was not considered as a sign of trigeminal malfunctioning, since it occurs in a large proportion of normal subjects.¹⁸

Responses to mandibular nerve stimulation

When stimulating the mandibular nerve at the level of the foramen ovale, direct motor responses (M-waves) were readily obtained from the ipsilateral temporal, masseter and anterior belly of the digastric muscle, in all the 16 subjects with no neurological abnormalities

(fig 1). Concentric needle recordings demonstrated that the potentials actually originated in these muscles. The current threshold was 0.5–4 mA, without significant differences among the three muscles. Responses of maximum voltage, starting with a negative component were recorded with the electrode disposition shown in fig 1. The average features of the M-waves to supramaximal stimulation are given in table 1. The onset latency showed little inter-subjects variation and was the same for the temporal and masseter muscles, 2.2 ms. The latency to the digastric was longer, 2.9 ms, and the difference (0.72 ± 0.23 SD) was statistically significant ($p < 0.001$).

The negative component of the temporalis M-wave was longer in duration and usually bifid (fig 1). This was possibly due to the anatomical features of the muscle itself, which is divided into anterior and posterior portions and is peripherally innervated by three separate nerve branches.¹⁹

Mylohyoid contraction could correspond to the M-wave recorded via surface electrodes overlying the digastric but its direct activation was not proved owing to its thin and expanded shape which made it difficult to determine the correct positioning of a concentric needle.

The facial muscles and the contralateral masticatory muscles were not directly activated. A small amplitude potential, with onset latency of 3–4 ms and peak latency of 5–6 ms, usually preceded the ipsilateral orbicularis oculi reflex,²⁰ though it was absent in the recordings via concentric needle, as if originating elsewhere than the orbicularis oculi. No movement of the eye-ball was clinically detected. Potentials with similar features could be picked up by surface electrodes or non insulated needles also from the contralateral facial and masticatory muscles (fig 2). It was possible to exclude a reflex origin for these potentials,

Fig 1 Direct motor responses from stimulation of the mandibular nerve. Left: schematic drawing showing disposition of the stimulating and recording electrodes; letters indicate active recording electrodes; black dots indicate reference electrodes. Right: temporal (A), masseter (B) and digastric (C) M-waves. Arrow indicates stimulus onset. Calibration: 2 ms, 2 mV.

since their latency and morphology were quite stable. Moreover, when the stimulating electrode was pushed centralward in the retrogasserian position, the latency of these early waves, if still present, was slightly longer as if the distance had increased, which would be the case for a direct response. Since the potential was always accompanied by the presence of M-waves in the ipsilateral masticatory muscles, it was thought to be a far-field potential from some masticatory muscle.

Responses to retrogasserian stimulation

When stimulating the trigeminal rootlets at the level of the clivus, the elicitation of M-waves required higher stimulus intensities in general and also depended on whether the tip of the electrode was closer to the third or the second division. The masticatory muscles were activated in all eight cases undergoing a lesion of V3, at a current threshold of 1.5–7 mA.

In three out of the eight subjects undergoing a lesion of V2, no direct motor responses were obtained with stimuli up to 50 mA. In the others high stimulus intensities were required: 5–20 mA. In two cases the M-waves were far smaller than the respective responses elicited from the mandibular nerve, as if only some of the relevant motor nerve fibres had been activated.

When high intensity stimuli evoked painful sensations, supramaximal responses were recorded just before the coagulation, when the patient was anaesthetised.

Latencies to retrogasserian stimulation were always longer, to a small but very constant extent, than to foramen ovale stimulation. In the eight cases with V3 stimulation, the average latency difference was $0.25 \text{ ms} \pm 0.19 \text{ SD}$ for the temporal, $0.26 \pm 0.14 \text{ SD}$

for the masseter and $0.24 \pm 0.09 \text{ SD}$ for the digastric responses (table 1). The average depth of the tip of the cannula, as measured from the surface of the skin, was 65 mm to the first position of stimulation at the level of foramen ovale and a further 14 mm to the second, retrogasserian, position. A difference between the two sites of 17 mm was however computed by measuring the distance on radiographs. The resulting conduction velocity was then 55 m/s–68 m/s, according to the method of measuring the distance.

The M-waves were not followed by any response at either site of stimulation. In particular no F-like waves were identified with supramaximal stimuli.

Responses to masseteric nerve stimulation

When stimulating the masseteric nerve below the zygomatic arch, the masseter M-waves could be always obtained, even though particular care had to be taken in placing the stimulating electrodes close to the nerve in order to minimise the stimulus artifact (fig 3). M-waves had shorter duration (table 1) than the respective responses obtained from intracranial stimulation. The average latency was 1.65 ms (1.4–1.8), allowing for a conduction velocity of 54 m/s in the portion foramen ovale-zygomatic arch.

Findings in patients having a second operation

Though all had some defect of sensation in the face, only two out of the seven patients who had already undergone trigeminal surgery had clear signs of motor impairment.

Intracranial stimulation revealed definite abnormalities of motor conduction in four cases and borderline values in one. The results for the masseter muscle, which are similar to those for the other masticatory muscles, are summarized in table 2. An example of temporal muscle responses is given in fig 4.

Fig 2 Recordings from facial muscles. (A) ipsilateral orbicularis oculi. (B) contralateral orbicularis oculi. (C) ipsilateral orbicularis oris.

Retrogasserian V2 stimulation.

Two successive sweeps are superimposed.

Calibration: 8 ms/div, 0.2 mV/div.

Early far-field potentials (FF), R1- and R2-like responses can be easily identified. Note that early potentials are stable in all derivations; R1-like response is present only in the ipsilateral orbicularis oculi and shows a small jitter; R2-like responses are even more unstable.

Fig 3 Direct motor responses from different sites of stimulation. M-waves recorded from ipsilateral masseter (R) following stimulation of masseteric nerve (1), mandibular nerve (2) and retrogasserian root (3). Onset latencies are indicated by vertical lines.

Calibration: 1 ms, 2 mV. Note latency increase from 1 to 3.

Fig 4 Abnormalities in re-operated subject. Recordings from temporal muscles. (A) example of normal M-wave from stimulation at the level of foramen ovale in normal subject. (B, C) M-waves from stimulation at the level of foramen ovale and of the clivus in re-operated subject. Onset latencies are indicated by vertical lines. Calibration 2 ms, 2 mV. D, E: jaw-reflex from the normal (D) and operated side (E) in re-operated subject. Calibration: 5 ms, 0.5 mV. Note latency increase of M-waves and jaw-reflex in the operated side.

In one patient who had surgical partial root section (case 14), it was impossible to evoke any M-waves with stimuli up to 50 mA apart from a small response (3.6 ms, 100 μ V) in the digastric muscle. In three patients who had radiofrequency thermocoagulation (cases 4, 5, 13), there was a marked slowing of conduction velocity: 28–40 m/s. The distal latencies were increased in case 4 (2.9 ms latency to the masseter and 3 ms to the temporalis) and in case 13 (2.7 ms to the temporalis). Conduction times were equivocal in case 22. The amplitude of the M-waves was abnormally small in case 5.

Discussion

Are the subjects normal?

The experiments were carried out in subjects who may not be regarded as suitable for a study mainly aimed at assessing normal nerve function.

Sub-clinical nerve lesions have been suggested

among the possible causes of trigeminal neuralgia, the "idiopathic" label being misleading, particularly by those authors espousing the notion of peripheral aetiology resulting from mechanical factors, such as the pulsatile contact between the internal carotid artery and the ganglion or impingement on the root by vascular loops in the posterior fossa.^{21 22} In fact, strong evidence in favour of pathological changes in primary neurons has been provided following the studies of Beaver *et al*²³ and Kerr.²⁴ In addition, the patients were being treated with anticonvulsants which can decrease the conduction velocity in the distal portion of limb nerves.²⁵ However, our subjects did not show any neurophysiological abnormality in the three trigeminal divisions, as assessed by the blink reflex to supraorbital and infraorbital stimulation and by the jaw jerk. These facial reflexes have been shown to be normal in patients suffering from "idiopathic" trigeminal neuralgia and who were taking anti-convulsants, and are considered the best means of differentiating essential from symptomatic trigeminal neuralgia.^{11 13}

We agree with the hypothesis of a peripheral and mechanical aetiology for the neuralgia, but the common observation of no neurological deficit in the great majority of cases implies that either trigeminal function is protected by a large biological reserve or, as Kerr himself stated,²¹ it is possible that only a few fibres are involved.

Saunders *et al*¹² found fibrillation potentials in the temporal or masseter EMG in seven out of 18 cases of trigeminal neuralgia. However, two of these patients were suspected of lues and multiple sclerosis and the others ranged in age between 74 and 79 years. The finding of denervation potentials never occurred in the present study.

The average age in our case material was 56 years. Changes in the myelin sheath which occur with ageing could only affect nerve conduction to a very small extent in this age group.²⁶ The only studies which provide information about a trigeminal motor response at older ages are those measuring the latency of the jaw jerk by Goodwill¹⁰ and Ongerboer de Visser.¹³ In Goodwill's group the latency of the jaw jerk was the same in subjects younger and older than 40 years, while Ongerboer de Visser and Goor found a 11–12% increase of latency in subjects aged 50–80 years in comparison with the 20–50 age groups. It is impossible to assess the separate contribution of the afferent, central and efferent delays to this latency.

What seems most worth reporting is the fact that the latency of the masseter M-wave to stimulation of the masseteric nerve found in the present study did not exceed the values (1.3–1.9 ms) reported by Godaux and Desmedt¹⁶ in a group of normal volunteers in their twenties.

Findings in "normal" subjects

As expected, stimulation of the mandibular division at the level of the foramen ovale activated all the ipsilateral masticatory muscles from which the recordings were taken. There were no noteworthy differences in the motor fibres directed to the temporal, masseter and digastric muscles, as shown by their threshold and conduction velocity. The M-wave of the anterior belly of the digastric muscle has been recorded for the first time in man. It was similar to the direct responses of the short hand muscles or of the masseter, the only significant difference from the latter being the longer latency, ascribable to the longer conduction distance. We have no direct evidence for the activation of the pterygoids and the mylohyoid, though they could be the generators of the small potentials picked up from the facial derivations. It is possible to exclude activation of the contralateral muscles. This was reported as the result of the stimulation of the trigeminal "dorsal root" in one case of root section by the posterior approach.²⁷ These authors attempted intracranial stimulation in that one case only and gave no explanation of the event.

Retrogasserian stimulation also evoked fully developed direct motor responses in most cases, though the needle-electrode was not aimed at the motor root, being presumed to be within the so-called "portio major". Actually, throughout the interval from pons to ganglion the sensory and motor fibres run in a number of rootlets with several anastomoses among the sensory divisions and between sensory and motor rootlets.¹ Our findings by no means imply that afferent and efferent fibres run intermingled in the trigeminal root. Although the stimulus should remain confined to a relatively small area, owing to the electrode's characteristics, its possible spread cannot be determined precisely. One cannot rely completely upon the subject's report either. The fact that motor threshold progressively increased at the mandibular nerve, V3 and V2 stimulation sites seems to indicate that the motor fibres were progressively more distant from the three structures.

Conduction velocity of trigeminal motor fibres

The intracranial motor conduction velocity was estimated to be 55–68 m/s. The short distance and the difficulty of accurate measurement might cast some doubts on the reliability of the velocity values. There were differences of a few millimetres only between the measurements taken from the cannula and from the radiographs, which may nevertheless result in large errors in calculating velocity. On the other hand, the velocity of 54 m/s found in the masseteric nerve was slightly lower than the intracranial velocity, which is reasonable. The estimated conduction velocity in the whole portion between clivus and zygomatic arch was

55–60 m/s.

Indirect support comes from the latency difference between masseter and digastric M-waves. The distance from the foramen to the anterior belly of the digastric is approximately 40 mm longer than to the masseter, which results in a 58 m/s conduction velocity, the delay being 0.72 ms. We think that this value should be taken as the most reliable, being computed over the longest distance.

McIntyre and Robinson,²⁸ proposed a conduction velocity of 100 m/s, on the basis of the fibre size they measured with light microscopy in the motor root of one necropsy specimen. The largest diameter being 17 μ m, a conduction speed of 100 m/s was calculated by multiplying by a factor of six, according to the method of Hursh.²⁹ However, in 1979 Young and Stevens,³⁰ by using electron microscopy, were able to determine the overall fibre size spectrum of the human trigeminal motor root. Myelinated fibres presented two main peaks, the larger fibres falling in the 8–14 μ m range. The ratio between conduction velocity and fibre diameter has not been established in humans, and normative morphological data for muscle nerves are not available in man.³¹ However, a comparison can be drawn with the few data existing on the other human cranial nerves: the diameters of the largest peaks of the nervi accessorius, hypoglossus and facialis are 8–18 μ m,³² 11–13 μ m³³ and 12–15 μ m³² respectively, the corresponding conduction velocities being 71 m/s,³⁴ 57 m/s³⁴ and 45–65 m/s.^{35 36}

The occurrence of an F-like response^{37 38} to stimulation of the masseteric nerve has been reported by Fujii³⁹ in one third of cases, but was not seen in the present series.

The absence of F-responses to intracranial stimulation could be due to the short conduction distance. Since the site of stimulation could be as far from the motor nucleus as 45 mm,¹⁷ it would take only 1.5 ms to an antidromic motor volley travelling at 58 m/s to get to the centre and come back. The time needed for the generation of the recurrent action potentials in the axon hillock is not known, though a value of 1 ms is usually accepted.³⁸ If this delay does not exceed 1.5 ms, an F-wave would reach the muscle during the absolute refractory period of muscle fibre, which can last 3 ms.⁴⁰

Findings in re-operated subjects

The findings in the two patients who had their roots sectioned were clear and anticipated by clinical examination: in one the motor root was spared and in the other it was cut.

Surprisingly, while a masticatory impairment had been noticed in one, there was a remarkable slowing of the intracranial conduction velocity to 28–40 m/s in three of the patients who had already undergone

percutaneous thermocoagulation, while the amplitude was significantly reduced in only one case (see table 2). Thus the neurophysiological data seem to indicate that the myelin sheath was far more affected than the axons. Among the possible causes, the heat produced by the radiofrequency lesion should be taken into account.

The Sweet and Wepsic technique,⁶ is said to be differential in damaging the smaller fibres while sparing the larger ones. Despite the precautions taken in adjusting the position of the coagulating needle and the graded control of temperature, the occurrence of defects of tactile sensation is almost inevitable, while a partial motor root weakness is a transient side effect in 22%–43% of cases.^{6,7}

We found a subclinical impairment of the motor root up to 2 years after the lesion. This means that a stable defect could pass unnoticed in the common evaluation of the side effects of percutaneous radiofrequency thermocoagulation.

A larger number of observations are of course needed. Ideally, the same patients on whom the original recordings were performed should be employed, in the event that a relapse occurs and a further operation is performed.

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