Intracranial stimulation of the trigeminal nerve in man

II. Reflex responses

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SUMMARY The reflex responses evoked by direct electrical stimulation of the intracranial portion of the trigeminal nerve have been studied in 16 subjects undergoing percutaneous retrogasserian thermocoagulation for the treatment of trigeminal neuralgia affecting the second or third division. In the orbicularis oculi muscle, early and late responses similar to the R1 and R2 components of the blink reflex were recorded. The former could be evoked only by stimulation of the second division and its latency was consistent with intermediately fast afferents. A late reflex (50–70 ms) was occasionally recorded from the anterior belly of the digastric muscle. The response was sometimes followed by a later activity and showed the features of a polysynaptic reflex. No response was obtained in the jaw elevators when fully relaxed. With the subject voluntarily clenching his teeth, both an early “H-like” response and two silent periods in the background EMG were obtained. The second silent period was similar in the muscles ipsi- and contralateral to intracranial stimulation, while the first silent period was longer in the ipsilateral muscles. Possible mechanisms contributing to the inhibition following stimulation of the mixed portion of the nerve are discussed.

Owing to difficulties in obtaining cortical evoked potentials devoid of myogenic contamination¹ and the lack of nerve bundles both superficial and long to allow measurements of conduction velocity,² neurophysiological testing of the trigeminal system has mainly developed on reflex responses.³

Most widely used are the trigeminofacial reflexes of the orbicularis oculi muscle: blink reflex to mechanical or electrical stimulation of the supraorbital or infraorbital territories^{4–7} and corneal reflex to mechanical or electrical stimulation of the corneal mucosa.^{3 8–10} The blink reflex consists of two separate responses, an early ipsilateral (R1) and a late bilateral (R2) component. The early component is transmitted through at least one interneuron^{7 11} in the pons, while the late component relays in the spinal trigeminal nucleus, and then ascends along a bilateral polysynaptic pathway, possibly through the lateral bulbar reticular formation.^{12 13} Efferent impulses of both components are transmitted by the same orbicularis oculi motoneurons.¹⁴ The corneal reflex is

similar to the late component of the blink reflex, but it is not mediated by the same interneurons¹⁰ and is never preceded by an early response.^{8 9} R2 is regarded as a nociceptive reflex^{3 15} though it is supposed to be mediated by low-threshold cutaneous receptors innervated by medium myelinated fibres;^{6 16} R1 is regarded by some authors as myotatic and proprioceptive^{4 17 18} and by others as cutaneous and exteroceptive.^{3 5 16}

Among the trigeminotrigeminal reflexes, the jaw jerk to taps to the chin (or jaw reflex or mandibular reflex) was the first to be introduced in routine clinical neurophysiology.^{3 4 19} Afferent fibres from spindles of the jaw-closing muscles have their cell bodies in the mesencephalic nucleus and relay monosynaptically with the ipsilateral masticatory motoneurons.^{3 19} Afferent fibres are thought to pass through the motor^{19–21} or the sensory root.^{3 22 23} Recently, an H-reflex of the masseter and temporal muscles has been described.^{24 25} Peripheral pathways and central connections should be the same as for the jaw jerk.

Inhibitory trigeminal reflexes can be demonstrated in man when the subject voluntarily activates his masticatory muscles.²⁶ A suppression of the background EMG activity, or silent period, appears in the jaw-closers bilaterally after mechanical or electrical stimulation of adequate intensity anywhere within the max-

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illary or mandibular territories. Taps usually evoke a pause preceded by a short excitation, that is probably a jaw jerk followed by a classical silent period due to unloading of spindles or activation of Golgi tendon organs²⁷⁻²⁹ while electrical stimuli evoke a double phase of silence (SP1 and SP2), which has been called cutaneous silent period in masseter muscles,³⁰ masseter silent period,³¹ masseter inhibitory reflex,³² or exteroceptive suppression³³ to emphasise its different origin from proprioceptive silent periods. Little has been ascertained about the anatomical and functional organisation of these reflex inhibitions in man. It has been proved that SP1 and SP2 are distinct phenomena, possibly mediated by oligo- and polysynaptic circuits³² in the pons.³⁰

Unlike animals, humans do not usually show any reflex excitation of the digastric muscle, though responses to loud sounds,³⁴ stretch³⁵ and electrical stimulation of lip mucosa³⁶ have been reported.

On the whole, the reflex organisation of the human trigeminal system appears fairly different from that of human limbs or animal trigeminal region. In order to further its understanding, we studied the reflex responses evoked by stimulation of the intracranial portion² of the trigeminal nerve in man. This was performed during the operation of percutaneous retrogasserian thermocoagulation³⁷ on subjects suffering from idiopathic trigeminal neuralgia.

Material and methods

This study was carried out on 16 subjects undergoing percutaneous rhizotomy by radiofrequency thermocoagulation for the treatment of trigeminal neuralgia.³⁷ Subjects were the same already examined for the preceding investigation.² All were diagnosed as idiopathic trigeminal neuralgia (V2 and V3 being affected in eight cases each), showed no neurological abnormalities and were having a trigeminal operation for the first time.

The stimulation and recording techniques are described in the preceding paper.² Briefly, the intracranial portion of the trigeminal nerve was stimulated at two different sites: first, at the level of the foramen ovale, and second, at the level of the clivus. In the first position the tip of the stimulating electrode was presumed to be in the mandibular nerve just distal to the ganglion, and in the second within the trigeminal root.³⁸ Identification of the proper division was achieved by the subject's report on the distribution of paresthesias evoked by delivering to the root a low intensity 100 Hz current.

Recordings were performed from the temporal, masseter, anterior belly of the digastric, orbicularis oculi et oris muscles. Electrodes of various kinds (surface, non insulated needles, insulated needles with bared tip, concentric needle) were used in order to clarify the origin of the signals.

For the evaluation of the digastric and H reflexes and of the R2 component of the blink reflex, a pseudorandom manual stimulation at a low rate (1 pulse per 3-10 s) was used.

For the evaluation of the H-reflex and of the SPs, intra-

cranial stimulation was performed during voluntary contraction of the jaw closing muscles. Previously instructed subjects were requested to clench their teeth at maximum strength and to relax their jaws after each stimulus.

In the period before the operation, the subjects were submitted to neurophysiological testing, which included the blink and jaw reflexes, using known electrodiagnostic techniques.²

By measurements on radiographs, skulls and topographic atlas,³⁹ the distance between the infraorbital foramen and the site of retrogasserian stimulation at clivus level was estimated to be 70-80 mm, while approximately 32 mm lay between the foramen ovale and the pons entry.

Results

Orbicularis oculi reflexes

In all but two of 16 subjects, a clinical blinking of the eyes could be elicited from any site of intracranial stimulation. The corresponding EMG activity (figs 1 and 2) consisted of a late polyphasic response similar to the R2 component of the blink reflex. It ranged widely in threshold (3-15 mA), latency (28-49 ms) and duration (20-61 ms), and was unstable in any given subject with repeated trials. It was immediately suppressed by repetitive stimulation and by the barbiturate which was administered before the lesion. The threshold current did not significantly change at the two stimulation sites, but while it was always higher than the motor threshold with stimulation of the mandibular nerve, it could be equal or even lower with retrogasserian stimulation. The average latency was $37.3 \text{ ms} \pm 6.7 \text{ (SD)}$ to stimuli at the level of the foramen ovale, and $34.9 \text{ ms} \pm 6.3 \text{ (SD)}$ to stimuli at the level of the clivus. The difference is not statistically significant and both latencies were longer than

Fig 1 Late component of the orbicularis oculi reflex (R2) and early far-field potential (FF). Stimulation of increasing intensity (from top to bottom) at foramen ovale. Recording from ipsilateral orbicularis oculi. Calibrations: 10 ms, 0.2 mV. Note that R2-like response shows a clear latency gain to stimuli of increasing intensity and a threshold higher than for the early far-field direct response.

Fig 2 Early (R1) and late (R2) responses of orbicularis oculi reflex. Stimuli of equal intensity at V2 retrogasserian position. Recording from ipsilateral orbicularis oculi. Calibrations: 10 ms, 0.2 mV. Note that both responses show morphological and latency variation with repeated trials, the late component to a greater extent.

the latency to infraorbital stimulation, $34 \text{ ms} \pm 3.1$ (SD), which was assessed before the operation.² Occasionally, this response was followed by a later activity constituted by short and irregular bursts between 90 and 170 ms (fig 3b).

When stimulating the trigeminal root at the level of the clivus, a R1-like response with threshold of 3–11 mA and latency of 7.8–11 ms was identified in seven subjects. The response appeared at the same intra-individual threshold as the R2-like response, was present in the ipsilateral orbicularis oculi only and endured repetitive stimulation at 1 Hz. Its reflex nature was ascertained by the morphological and latency variability. By examining sequential responses to stimuli of constant intensity, evident shape modifications and a small latency jitter became apparent (fig 2). Its occurrence was far more common

Fig 3 Digastric and orbicularis oculi late components. Stimuli of equal intensity at foramen ovale. Simultaneous recordings from anterior belly of digastric (a) and orbicularis oculi (b). Calibrations: 50 ms, 0.1 mV. Note the presence of two digastric reflex responses at the same time as the R2- and R3-like orbicularis oculi components.

when the stimulating needle was aimed at V2 (six out of eight cases) than at V3 (one out of eight cases). The two V2 cases in which there were no responses of this kind also showed bilateral absence of R1 to stimulation of the infraorbital nerve.

The response was stable enough to allow accurate measurements of the latency in the six V2 cases. In the V3 case the response was merging with the preceding early wave described in the preceding paper. The average latency to stimulation of the root was $9.6 \text{ ms} \pm 1.1$ (SD), while in the same subjects the average latency to infraorbital stimulation was $11.4 \text{ ms} \pm 0.8$ (SD). The difference is statistically significant ($p < 0.01$).

Fig 4 Stimulus intensity (a) and paired shock conditioning (b) effect on the H-like response. Stimulation at foramen ovale. Recordings from ipsilateral masseter muscle. (a) M- and H- responses to stimuli of increasing intensity (from 1 to 5). Calibration: 5 ms, 1 mV. Note progressive augmentation and then suppression of the H-like response. (b) two stimuli (S1 and S2) of equal intensity are delivered with 10 ms interval. Calibration: 2.5 ms/div, 0.5 mV/div. Note that the M-waves to conditioning (S1) and test (S2) stimuli are similar, while the H-like response is absent after S2.

Digastric reflex

With the subject normally relaxed, no reflex was evoked in the jaw closing or opening muscles by intracranial stimulation. However, when even a low level of spontaneous EMG activity was present, a reflex response could occasionally be recorded from the anterior belly of the digastric muscle (fig 3).

The reflex was constituted by a polyphasic response with a latency more commonly about 50–70 ms, but widely ranging from 40 to 100 ms. The duration varied from 20 to 80 ms. It could be evoked by stimuli of low intensity, but showed a great latency gain (up to 40 ms) to stronger stimuli above the pain threshold, at the same time increasing greatly its duration. It did not endure the repetitive stimulation or the barbiturate.

This response was sometimes followed after some further 20 ms, by a later response, shorter and more unstable (fig 3a).

In general, a certain correlation with the orbicularis oculi responses was noted. The first digastric response, when fully developed, was always accompanied by the presence in the orbicularis oculi of the R2-like response, and the second by the above mentioned late activity between 90 and 170 ms (fig 3b).

H-reflex

Stimulation of the mandibular nerve at the level of the foramen evoked a response similar to the H-reflex of the lower limbs in the ipsilateral jaw closing muscles of the five subjects who were instructed to clench their teeth. Under these conditions, low intensity stimuli (0.5–4 mA) elicited a small negative deflection of about 6 ms (5.4–7) peak latency. This response could appear either isolated or together with an earlier response, that is the liminary direct motor response (M-wave).² With stronger stimuli, while the amplitude of the M-wave progressively augmented up to a stable level, the late response first increased then decreased, but was not always abolished by supramaximal stimuli, thus not complying fully with the classical sequence of the triceps surae H-reflex. A complete sequence was observed in two cases, one of which is shown in fig 4a. However, the reflex nature of the response was proved by a small latency jitter (some 0.5 ms) and by its complete suppression when a double shock was delivered with an interstimulus interval of 10 ms (fig 4b).

There was no noteworthy difference between the masseter and temporalis recordings. Maximum amplitude ranged from 0.4 to 2 mV, that is the 15–50% of the corresponding supramaximal M-waves. The onset latency of responses at their maximum amplitude could often not be ascertained, since they were merging with the preceding M-waves. The

Fig 5 Bilateral silent periods of the jaw-closing muscles. Five superimposed trials to supramaximal stimuli. Stimulation at foramen ovale. Recordings from ipsi- (A) and contralateral (B) masseter. Calibration: 20 ms/div, 0.2 mV/div. In the ipsilateral muscle the direct response (M-wave) is followed by early (SP1) and late (SP2) phases of suppression of the background EMG activity. In the contralateral muscle there are two SPs without any direct response, SP2 is similar as in the ipsilateral muscle, while SP1 is less developed.

shortest latencies which could be detected in the six subjects ranged from 4.4 ms to 6 ms, with an average latency of 5.1 ms.

No response with these features was identified in the recordings from the digastric or the contralateral muscles.

Fig 6 Bilateral silent periods in the jaw-closing muscles. Responses to one shock of low intensity. Stimulation and recording as in fig 5. Calibration: 20 ms, 0.5 mV. Note absence of SP1 in the contralateral muscle.

Subjects were not requested to repeat the strong contraction of the jaw muscles when the tip of the stimulating needle was positioned into the retrogasserian root, in order to prevent possible movements of the needle, which might result in the need for further adjustment of position.

Inhibitory reflexes

While the subjects were clenching their teeth, intracranial stimulation evoked periods of electrical silence in the background EMG activity of the ipsi- and contralateral jaw closing muscles. The limited time allowed by the intraoperative conditions did not permit recording of a large number of trials. Hence, it was not possible to perform quantitative measurements through full wave rectification and adequate averaging.^{32 33} Nevertheless, the responses were clear and stable, thus allowing qualitative observations.

The contralateral inhibition (fig 5) presented with a double silent period, with the features of cutaneous silent periods. SP1 had a latency of 10–15 ms and a duration of 8–20 ms, SP2 began at 40–50 ms and lasted a further 30–50 ms. Notwithstanding difficulties in assessing the threshold for this kind of response, it was clear that stimuli above the motor and sensory thresholds were needed.

On the other hand, suppression of the ipsilateral background EMG seemed to appear with and then parallel the augmentation of the M-wave. To low intensity stimuli, the M-wave was followed by the “H-like” activity, a first pause, an intermediate recurrence of EMG at about 30–50 ms, a second pause and final reappearance of uninterrupted voluntary activity at 90–100 ms. When fully developed, SP1 merged with the preceding M-wave and lasted up to 30–40 ms; SP2 values did not differ significantly from those of the contralateral side. As shown in fig 5, the ipsilateral SP1 was always longer than the contralateral one. This began 8–10 ms after the onset of the M-wave and ceased 5–10 ms before the ipsilateral SP1. The difference was striking when, to moderately intense stimuli, the contralateral SP1 was small or absent (fig 6).

The general aspect of the inhibition was similar in temporal and masseter recordings.

Discussion

Although subjects were suffering from trigeminal neuralgia, we regarded them as “normals”, or at least suitable for a study of trigeminal function. In this regard, a full discussion is provided in the preceding paper.² It should be emphasised here that subjects were selected among those without neurological

deficits as assessed by clinical examination and neurophysiological testing using known electrophysiological techniques, such as the jaw and blink reflexes.^{40 41}

Orbicularis oculi reflexes

Intracranial stimulation evoked early and late orbicularis oculi responses with the features of the R1 and R2 components of the blink reflex (figs 1, 2). Occasionally there appeared also a later activity resembling the so-called R3 reported by Penders and Delwaide,⁵ though inconsistency of data prevents any interpretation of its actual nature.

The early response (7.8–11 ms) presented with a small latency jitter and stood repetitive stimulation. It was strictly confined to the ipsilateral orbicularis oculi and could be evoked only when stimulating V2. In one case it also occurred when the stimulating needle-electrode was aimed at V3, but this could be due to spread of stimulus current or to incorrect report by the patient on the distribution of paresthesias.² The fact that stimulation of the mandibular nerve was never able to evoke an R1-like response, however, confirms that its afferents were limited to V2 (no subject undergoing V1 lesion was examined).

Conversely, the late response was highly unstable, with large variability of latency and duration, both inter- and intraindividually, and habituated quickly, as expected for a polysynaptic reflex, mediated through the lateral reticular formation.¹³ It appeared in the orbicularis oculi bilaterally, and occasionally also in the orbicularis oris, after stimulation of the mandibular nerve or the retrogasserian rootlets, without difference.

These findings confirm the opinion that afferents for R1 are fewer and less dispersed than for R2, and give an explanation of the greater susceptibility of the former to extrinsic lesions.^{7 42} The two responses showed approximately the same threshold. A direct comparison between R1- and motor-threshold cannot be drawn since the motor fibres were stimulated from a distance when the needle was within V2.² However, since R2-threshold was definitely higher than the motor threshold when stimulating the mandibular nerve, where afferents and efferents are intermingled, it might be concluded that the threshold for both reflexes was higher than for motor fibres.

The latency of the early response was 1.8 ms shorter to intracranial than to infraorbital stimulation. Over a distance of approximately 70–80 mm between clivus level and infraorbital foramen, this results in an afferent conduction velocity of 39–44 m/s, which is lower than the conduction velocity of human trigeminal motor fibres² and falls in the range of the intermediate velocity, medium myelinated fibres.⁴³

Shahani¹⁶ obtained similar values by stimulating the supraorbital nerve at two points along its course on the forehead. His method was criticised and considered to yield exceedingly low values because of tapering and scattering of fibres in the forehead.¹¹ Distal tapering would directly result in a slowing of velocity in the afferent limb. Scattering of nerve branches outside the emergence point implied that distal stimuli (high on the forehead) activated fewer fibres than proximal stimuli (over the foramen), thus providing a smaller spatial summation on synaptic transmission. If synaptic transmission times are longer at distal than proximal stimulation, the latency difference would be further increased and the conduction time along the afferents mistakenly overestimated. The abovementioned pitfalls should weigh little with our data, which deal with the retrogasserian-infraorbital portion of V2.

These results are in favour of a cutaneous origin of R1,^{3,5,6} though they cannot be considered conclusive. The finding of a reflex threshold above that of motor fibres does not assure that the reflex afferents are really less excitable. As a matter of fact, a reflex threshold also depends on spatial summation of afferent input. The argument that afferent conduction velocity is low might be also questioned because of the difficulty in measuring accurately the conduction distance.

Digastric reflex

The failure to record a digastric reflex reported by most authors^{26,44} has been ascribed by Godaux and Desmedt³³ to the use of too high a rate of stimulation. These authors reported that a very low rate (1 pulse per 1–15 min) was necessary. In our case the reflex followed relatively high frequencies (1 per 3–10 s). However, it required a certain degree of facilitation, as shown by the presence of sparse spontaneous EMG activity. When absent, we did not wait several minutes between stimuli to check for reflex activity.

No short-latency response comparable to the H-reflex, to the R1 component of the blink reflex or to the jaw opening reflex commonly observed in animals, was obtained. The digastric response showed the features of a polysynaptic reflex: long latency and duration, large latency gain, habituation. It often presented with two components similar to the R2 and R3 of the orbicularis oculi reflex,⁵ with which it usually occurred synchronously (fig 3).

Similarly to the blink reflex, the digastric reflex could exhibit a defensive reaction, by actively opening the mouth. Its central circuits might not be activated in normal conditions, since in man interruption of the tonic contraction of the jaw elevators is sufficient to

promptly release the jaw. It is possible that the intracranial stimulus occasionally yielded the activation of a particular pattern of afferents, which could be more effective than cutaneous, mucous or dental stimulation.

H-reflex

The short-latency responses which have been recorded from the jaw-closing muscles strongly resembled the H-reflex previously reported to stimulation of the masseteric and deep temporal nerves.^{24,25} Equally, it appeared only during sustained voluntary contraction, could be elicited by stimuli below the motor threshold and was first augmented then suppressed as the intensity increased (fig 4a). The classical recruitment sequence of the limb H-reflex was not always completed up to the disappearance of the response to supramaximal stimuli. We ascribe this to the fact that the antidromic block was sometimes incomplete even at supramaximal intensity and then allowed the passage of a few reflex impulses, owing to two possible factors: some orthodromic impulses produced by the strong voluntary clenching could collide with the antidromic volley before the reflex impulses, or the intracranial stimulating electrode might be occasionally unable to activate all the motor fibres. In any case, the complete suppression of the test response to paired shocks (fig 4b) should prove its reflex nature. The only other kind of short-latency excitatory reflex of the human masticatory muscles is the so-called periodontal-masseteric reflex.²⁷ This should be mediated by oral mechanoreceptors and appear in the voluntary contracting muscles at a shorter latency than the jaw jerk. This possibility has been recently challenged and the reflex equated to a jaw jerk.⁴⁵

By considering the response as an H-reflex, afferent impulses would travel along Ia fibres. Their cell bodies form a column that extends from the level of the masticatory nuclei caudally to the posterior commissure rostrally.⁴⁶ These neurons are unipolar cells issuing axon collaterals directed to the ipsilateral trigeminal motor nucleus.⁴⁶ The extrinsic conduction velocity should be equal to or slightly higher than that of motor fibres.⁴⁷ The conduction velocity of the motor trigeminal fibres is approximately 58 m/s according to the best estimate we were able to give in the preceding investigation.² To travel 65 mm (twice the distance from the foramen ovale to the pons entry),^{39,48} 1.1 ms or slightly less is sufficient. This, added to the 2.2 ms of the M-wave's distal latency, gives a total of 3.3 ms for afferent and efferent conduction times and neuromuscular junction delay. The average of the shortest latencies of the "H-like" responses in our subjects being 5.1 ms, at least 1.8 ms are left for the central

delay, which includes both synaptic time and conduction time within the brainstem. It is not possible to determine the synaptic time, given the improbability that conduction velocity remains the same along extrinsic and intrinsic portion of the axons, and the difficulty in assessing the distance between unipolar neurons and motoneurons.

Both Godaux and Desmedt²⁴ and Fujii²⁵ regarded their H-like responses to masseteric nerve stimulation as a monosynaptic reflex. Fujii also gave an estimate of distances, conduction velocity, end plate delay and a not well defined "intermuscular delay", which allowed for 1 ms central delay. However, by using his own estimated values of distance (70 mm from the site of stimulation to the mesencephalic nucleus), velocity (69 m/s), M latency (2 ms) and H latency (6 ms), it can be easily computed that 2 ms were left for the central delay.

On the whole, these responses show some differences from the classic limb H-reflex, such as the need for the intense volitional activation of motoneurons, the facilitation exerted by vibration²⁴ and the relatively long central delay.

Inhibitory reflexes

When stimulating a mixed limb nerve, a number of factors have been suggested to explain the appearance of a silent period in the target muscles, namely antidromic block by impulses passing up the motor fibres⁴⁹ and recurrent inhibition by Renshaw cells to antidromic invasion of axon collaterals,^{50 51} unloading of the spindles^{49 52} and activation of Golgi tendon organs,⁵³ as well as sensory fibres.^{54 55}

Our particular experimental conditions offered the possibility of comparing the effect of a stimulus delivered to the mixed portion of the trigeminal nerve on the target muscle and on the contralateral one. In the latter, mechanical events due the twitch, as well as any antidromic effects, do not occur. Besides, strong evidence exists that stimulation of group I afferents from jaw-closing muscles does not produce any effect on contralateral motoneurons.⁵⁶ Actually, the inhibition of muscles contralateral to intracranial stimuli showed the same configuration, latencies and durations as the cutaneous silent period, or exteroceptive suppression, which appears bilaterally after stimulation of intra- and extraoral tissues or mental and infraorbital nerves.²⁹⁻³³

The inhibition in the ipsilateral muscles, in appearance, looked like the classic silent period in the hand muscles, since it began directly after the M-wave, was interrupted at 30-50 ms by a recurrence of EMG activity (though in this case there is no contribution by F-waves)² and ceased at 90-100 ms.

The final resumption of activity always occurring at

the same time on both sides, it could be assumed that the late phase of inhibition, or SP2, could be due to activation of sensory fibres, as believed by some^{54 55} for the limb silent period. It is more difficult to explain the mechanisms underlying the silent period in the first 30-40 ms after the stimulus. Owing to the short distance, it takes the antidromic volley less than one ms to reach the homonymous motoneurons. Besides, recurrent inhibition is unlikely to take place, since trigeminal motoneurons lack axon collaterals⁵⁷ and stimulation of the cat masseteric nerve fails to produce any disynaptic inhibition which could be ascribed to recurrent inhibition.⁵⁸ Exteroceptive fibres are definitely able to yield early inhibitory effects on the jaw elevators, but these are known to be strictly symmetrical on both sides,²⁹⁻³³ while a clear asymmetry was present between the ipsi- and contralateral SP1 in our recordings (fig 5, 6). The ipsilateral SP1 not only had a lower threshold, but also began earlier and lasted longer than contralaterally. Other factors must then be involved. Golgi tendon organs should probably be far more effective than spindle unloading in our high tension-isometric conditions.^{21 28 59} In any case, the mechanical twitch should begin about 10 ms after the arrival of the M-wave and further time would be needed before receptors start changing their activity and motoneurons are inhibited or disfacilitated.⁵⁵ Hence, proprioceptive effects on the jaw elevators could not begin before some 20 ms after the stimulus, thus contributing to SP1 only in its last part. In the first part, a sum of factors could act in sequence: refractory period of motoneurons fired by antidromic impulses, autogenic inhibition by Ib fibres activated by the stimulus and paucisynaptic inhibition exerted by other low threshold sensory fibres.

The technique of intracranial stimulation with the percutaneous approach to the trigeminal root seems to be able to provide useful information on human trigeminal organisation. Further and larger series of cases would be needed for a better quantitation of reflex responses. Other surgical procedures, such as the implantation of permanent electrodes in the Gasserian ganglion,⁶⁰ could avoid the restrictions of time and of the subject's collaboration, characteristic of the intraoperative recordings.

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