Effects of the Proprioceptive Afferents from Masseteric Nerve on the Reflex Mechanism Controlling Hypoglossal Motoneuron Activity in the Cat.*

by
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INTRODUCTION

A functional relationship between the functions of the jaw muscles has somewhat difference from the limb muscles which are controlled by the spinal nerves. Especially, the mandibule has a bilateral joint and the movement of their muscles which are attached to the bone is different from the antagonistic and synergetic muscles of the limbs.

On the regulation of the jaw movement, relaxation and the muscle tonus, the roles of the muscle spindles and the tendon organs were being clearly studied by electrophysiology recently (1, 2, 3, 4, 5, 6, 7, 8, 9). In conformity with these new information, to analyze the function of the jaw movement is indispensable to the physiological study on the mechanism of the jaw movement.

In previous data, we knew that the afferent fibres from muscle spindles of the masseter have excitatory synaptic linkage with masseteric motoneurons (10, 11, 12, 13).

In the movements between the tongue and the mandibule, the hypoglossal nuclei of the medulla oblongata has very important function. But the afferent impulses from the masseteric muscle effecting on the activity of the hypoglossal motoneurons have ever been cleared. In this study, we have tried to find the roles of proprioceptive afferents from the masseter in regulation of the outflow of efferent impulse to tongue from the hypoglossal nucleus.

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METHODS

GENERAL

Eighteen cats were used under the anaesthesia with pentobarbital sodium (30 mg/kg i. p.). Tracheotomy and cannulation into the left femoral vein were performed. 5 mg/kg of the pentobarbital sodium was added intravenously when necessary. The cerebellum was removed from all cats.

During the recoding period, the animal was artificially ventilated by using gallamine triethiodide (Flaxedil) as a muscle relaxant, and pneumothorax was made bilaterally.

Animals were immobilized by repeated injection of gallamine triethiodide throughout the experiment. Body temperature and temperature of the paraffin pool covering the exposed cerebral cortex and brain stem were maintained at 36-38°C with a heating pad and an infrared lamp.

PREPARATION OF NERVES

The left hypoglossal nerve was exposed by ventral approaching and isolated from the surrounding tissue.

The mandibular skin was cut longitudinally at middle line to make a mineral oil pool for this nerve. The head of the cat was then fixed on a stereotaxic apparatus. After the left zygomatic bone and the temporal muscle were removed, the masseteric nerve was prepared from the temporal fossa about 15-20 mm. All these nerves were ligated at the peripheral end and sectioned.

Except the above the left parietal bone was holled (1×1cm diameter) by rongeur at the A 7.0 of the atlas of brain map (14) for recording the action potential in semilunar ganglion by unipolar electrode.

STIMULATION and RECORDING

The stereotaxic apparatus was tilted down 80° from horizontal plane on left side and two pools were made of the skin flaps of mandibular and temporal regions; one for the hypoglossal nerve, the other for the masseteric nerve. The peripheral ends of these nerves were placed on bipolar silver wire electrodes (polar distance, 3-4 mm); the whole isolated nerves were then immersed in mineral oil and the stimulation or recording was made. (Fig. 1). Concentric needle electrodes (inner core; 0.2 mm, sheath; 0.5 mm, polar distance; 0.5 mm) were inserted stereotaxically into the left trigeminal mesencephalic nucleus and left hypoglossal nucleus. Their tips were fixed at the optimal point for evoking direct responses in the masseteric and hypoglossal nerves respectively.

The other same type of electrode was inserted into the left minor part of semilunar ganglion to monitor the incoming volleys from the masseteric nerve or the volleys elicited by stimulation of the mesencephalic nucleus of the trigeminal nerve. The potential was recorded monopolarly between the core and the neck muscle.

Electrical pulse generated by electronic stimulater (NIHON KHODEN MSE 3 and 20) was used for stimulation through the isolation transformers.

Pulses of 0.01-0.02 msec in wideth were applied to the masseteric nerve and hypoglossal nerve, and 0.05-0.1 msec in wideth were applied to the mesencephalic and hypoglossal nucleus. Responses were recorded on a cathod ray oscilloscope (NIHON KHODEN VC 40 MR) by superimposed 10 responses.

The amplitudes of conditioned antidromic spikes were divided by those of control responses expressing the control value as 100%.

After the experiment was finished animals were sacrificed with i. v. injection of overdose of pentobarbital sodium and the brain was taken out and fixed in 10% formaline, and checked the extent of lession histologically.

The brain was stained with Klüver-Barrera method.

RESULTS

A. Antidromic evoked potentials in hypoglossal nucleus

Stimulation of the hypoglossal nerve evoked in the ipsilateral hypoglossal nucleus a directly evoked antidromic spike (DEAS) potential. The amplitude of this spike continued to increase almost directly proportionally with the stimulus up to 3-4 times the threshold (xT), reaching the maximal amplitude at 4-5 xT (Fig. 2). The latency of its onset and peak was 2.1±0.3 msec (mean±S. D. n=12) and 3.2±0.4 msec (n=12), respectively.

B. Effect of masseteric nerve stimulation on the antidromic responses in the hypoglossal nucleus.

The stimulation intensity applied to the hypoglossal nerve was selected voltage of

that the DEAS potential has changed linerly.

Conditioning stimulation of the masseteric nerve induced a suppressed of DEAS in the hypoglossal nucleus. The stimulation of the masseteric nerve induced the decrease in excitability of the hypoglossal depolarization.

The suppressed DEAS potential was never detected by masseteric nerve stimulation if the intensity was less than 2 xT (Fig. 3) Fig. 3 shows an example of the relationship between the conditioning nerve volley and the DEAS potential effect. A slight decrease of DEAS amplitude was detected at 2 xT, and the amplitude decreased 5% of the control at 2.5 xT where the amplitude of the nerve potential reached a quasi-plateau. At 4 xT of the masseteric stimulation, the decreased DEAS reached a quasi-plateau.

The relation between the conditioning volley to the masseteric nerve and the hypoglossal DEAS potential was studied by monitoring that the evoked potential in the masseteric nerve at the ipsilateral semilunar ganglion near the motor root (Fig. 3 Aa). At 6 xT of the conditioning intensity, the suppressed DEAS amplitude reached to 57%. This decrease continued down to 8 xT, which was the highest intensity applied to the masseteric nerve during this study.

The time course of conditiong effect was performed in 6 cats. (Fig. 4).

The decrease of the amplitude of the DEAS potential began at the conditioning-test interval of 8-10 msec and reached at a peak in 15-20 msec, after this peak the conditioning effect gradually decreased, reaching the control level at 80-100 msec. At conditioning-test intervals of longer than 100 msec, no effect of conditioning stimulus were appeared.

Except the above, the amplitude was increased at a short interval 2-6 msec, but this tendency was reversed after 10 msec.

C. Blocking effect of masseteric nerve on DEAS

Stimulation of one cutaneous branch is known to induce effectiveness on other cutaneous branch (15, 16, 17, 18). In order to exclude the possibility of spread of stimulating current to any of other cutaneous branches, the conduction of the masseteric nerve was blooked at a portion proximal to the stimulating electrode either by local application of procaine to the nerve (4 cats) or by mechanical use of forceps (4 cats). The 5 xT and 10 xT of the stimulation intensity to the masseteric nerve were compared before and after the conduction block.

The blocking effect was cheked by recording the potential in the semilunar ganglion, evoked by masseteric nerve stimulation.

After the block, the effect of conditioning stimulus was disappeared completely, but when the conduction had recovered from procainization, the conditioning effect of masseteric nerve stimulation was appeared again.

D. Effects of semi-transection at the obex level on the DEAS.

Semi-transection of brain stem at the level of the obex or 2 mm rostral to it including the trigeminal spinal nucleus and tract which caused the conditioning effect of masseteric nerve stimulation of DEAS, was abolished completely (Fig. 5).

E. Effect of stimulation of the trigeminal mesencephalic nucleus upon the antidromic potential in the hypoglossal nucleus.

Stimulation of the trigeminal mesencephalic nucleus evoked in the ipsilateral masseteric nerve a monosynaptic masseteric reflex (19).

In our study, the amplitude of the

antidromic spike recorded in the masseteric nerve increased with stimulus intensity up to 6 xT, and the orthodromic potential was delivered from 1. 2 xT and increased largely at 3 xT when the stimulus intensity was more increased (Fig. 6).

Afferent fibers from both primary and secondary endings of muscle spindles of the masseteric muscle are reported to have their cell bodies in the trigeminal mesencephalic nucleus (13). If group Ia or II from the masseteric muscle takes part in the hypoglossal nucleus excitation, stimulation of the mesencephalic nucleus should evoke a similar effect to that evoked by masseteric nerve stimulation.

In fact, stimulation of the mesencephalic nucleus (6 cats) evoked the similar effect in the ipsilateral hypoglossal nucleus with almost the same time course as that evoked by stimulation of the masseteric nerve (Fig. 7).

There was no increased amplitude at a short conditioning-test interval when the mesencephalic nucleus was stimulated.

F. Drug effect on the DEAS in the hypoglossal nucleus induced by the masseteric nerve stimulation

The effects of pentobarbital sodium on the activity of hypoglossal nucleus were studied in 2 cats. Initial increasing effect of the conditioning stimulus on antidromic response of hypoglossal nucleus became prominent, and subsequent decreasing effect was suppressed after pentobarbital sodium 10 mg/kg i. v. injection. (Fig. 8 squares). Following i. v. injection of pentobarbital sodium 10 mg/kg, strychnine nitrate 0.08-0.1mg/kg was injected intravenously in 2 cats. The strychnine markedly enhanced the increasing DEAS, and decreasing effect was suppressed. (Fig. 8. triangles).

DISCUSSION

The effects of the masseteric nerve stimulation upon the excitability of the hypoglossal nucleus were tested, and they were found that a diminution of the amplitude of DEAS was produced by masseteric nerve stimulation.

Blom (20) and Green et al (21) reported that the amplitude of DEAS potential at the hypoglossal nucleus which was induced by hypoglossal nerve stimulation was changed by the time interval of the conditioning stimulus which was applied to the lingual nerve. And when the conditioning-test stimulus interval was less than 10 msec, the augmented spike potential (facilitated phase) was obtained, but after the facilitated phase, the amplitude of DEAS potential was decreased at 15-25 msec, and lasted for a long time.

In our experiment, although the conditioning stimulus was given to the masseteric nerve, but their time courses were very similar to the fact found in another trigeminal nerve stimulation.

In the spinal cord, the amplitude of DEAS potential recorded on the reflex arc was examined as critical excitability of motoneurons (22, 23, 24), but in this experiment, the amplitude of DEAS potential was considered as a measure of excitability. Because the monosynaptic reflex circuit had not yet been discovered between the hypoglossal nuclei.

Masseteric nerve stimulation evoked a triphasic potential in ipsilateral semilunar ganglion. The amplitude reached a quasi-plateau at stimulating intensity of 2.5-3.0 xT. Comparing with our previous papers (25), this triphasic response is assumed to be produced by activation of group I fibres of the masseteric nerve. The lowest intensity necessary for inducing the effect on the hypoglossal nucleus excitation was 2.5 xT. This suggests

the lowest threshold afferents from the masseteric muscle, i.e. group Ia and Ib, not contribute appreciably to effectiveness, and even if it was possible that group Ia and Ib afferent contribute to this effect as a whole was not marked owing to the small number of group I fibres in the masseteric nerve(26).

The hypoglossal nucleus was not affected when the stimulating intensity given to masseteric nerve was not raised to 2.0-2.5 xT which was the maximal intensity for all the group I fibres. The DEAS potential was diminished with the increase of stimulus intensity within the supra-maximal range for group I fibres. It is clearly assumed that the participation of high threshold muscle afferent i. e. group II and group III.

According to Windle (27), the nerves to masseteric muscle was contributed by 57% of large fibres (9μ or more in diameter), 30% of medium (5-9μ in diameter), 13% of small ones (5μ or less in diameter) and a few unmyelinated nerve fibres. Scince the large part of the masseteric nerve fibres were contributed by group I and group II by Lloyd's classification (28).

As the above we described, the stimulation of hypoglossal nerve induced the DEAS potential in hypoglossal nucleus. It changed with the conditioning stimulus given to masseteric nerve or mesencephalic nucleus.

With the pharmacological study of application of pentobarbital sodium or strychnine (29, 30, 31), and referred to Porter's research (32, 33), the diminished DEAS, in time course, might be assumed that the inhibitory postsynaptic potential (IPSP) produced in the hypoglossal nucleus due to the conditional stimulation of masseteric nerve.

The diminished phase induced by stimulation of masseteric nerve or mesencephalic nucleus might be as follows: 1) IPSP induced by conditioning stimulation may block the antidromic impulse spreading from initial segment (IS) to soma and dendrite of neuron (SD); 2) the antidromic impulse was blocked by IPSP spread from myelinesheath (M) to IS; 3)SD became smaller for IPSP effect (33); and 4) when the orthodromic spike potential through down along with hypoglossal nerve, the antidromic spike may cancel each other.

In cranial motor axon, because it had no collaterals (34), we neglect the feed-back inhibition by Renshaw cell.

In time course, the facilitatory phase in 10 msec may be thought that it is caused by the flexor reflex afferent (FRA) impulse as in spinal reflex, but the more detailed experimental research is necessary.

According to Olszewski (35), Darian-Smith (37) and Stewart et al (38), the obex lies on the border between nuclei interpolaris and caudalis (of trigeminal spinal nucleus). The fact that the abolition of diminishing of conditioning effect induced by semi-section of obex and/or 2 mm rostral is elucidated that the nucleus caudalis of trigeminal spinal nucleus is necessary for conditioning stimulation.

SUMMARY

1. The effects of the masseteric nerve stimulation upon the excitability of the hypoglossal nucleus were studied.
2. Stimulation of the masseteric nerve with intensities of more than 2.5 times threshold induced a suppression in amplitude of antidromic spikes directly evoked by stimulation of hypoglossal nerve. This suppression in excitability of hypoglossal nucleus started around 10 msec after conditioning stimulation of the masseteric nerve reached its peak after 15-20 msec, and returned to the control level after 80-

100 msec. The increased excitability was occurred in 2-8 msec. The effect was increased by raising the stimulus intensity in the supramaximal range for low threshold fibres in the masseteric nerve.

3. Semi-transection of the brain stem at the obex level, including the trigeminal nucleus and tract, reduced or abolished the suppression of the antidromic spike.
4. It is concluded that group II and III afferents in the masseteric nerve induce suppressed hypoglossal depolarization when the masseteric musle excited, and the movement of tongue was inhibited.

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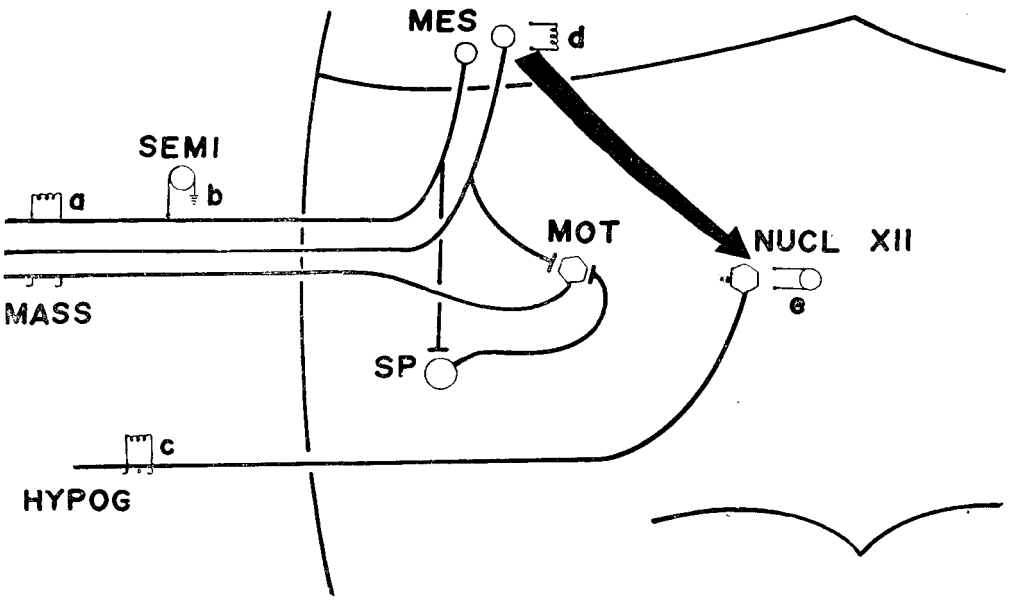

Fig 1. Experimental setup. a, Bipolar silver wire electrode used either for conditioning stimulation of massetric nerve or for recording response to mesencephalic stimulation. b, monopolar electrode for recording evoked volleys in semilunar ganglion. c, bipolar silver wire electrode for test stimulation of hypoglossal nerve. d, concentric bipolar stimulating electrode in trigeminal mesencephalic nucleus, e, concentric bipolar recording electrode in hypoglossal nucleus. Abbreviations; MASS: masseteric nerve. SEMI: semilunar ganglion. HYPOG: hypoglossal nerve. MES: trigeminal mesencephalic nucleus. SP: trigeminal spinal nucleus. MOT: trigeminal motor nucleus. NUCL XII: hypoglossal nucleus.

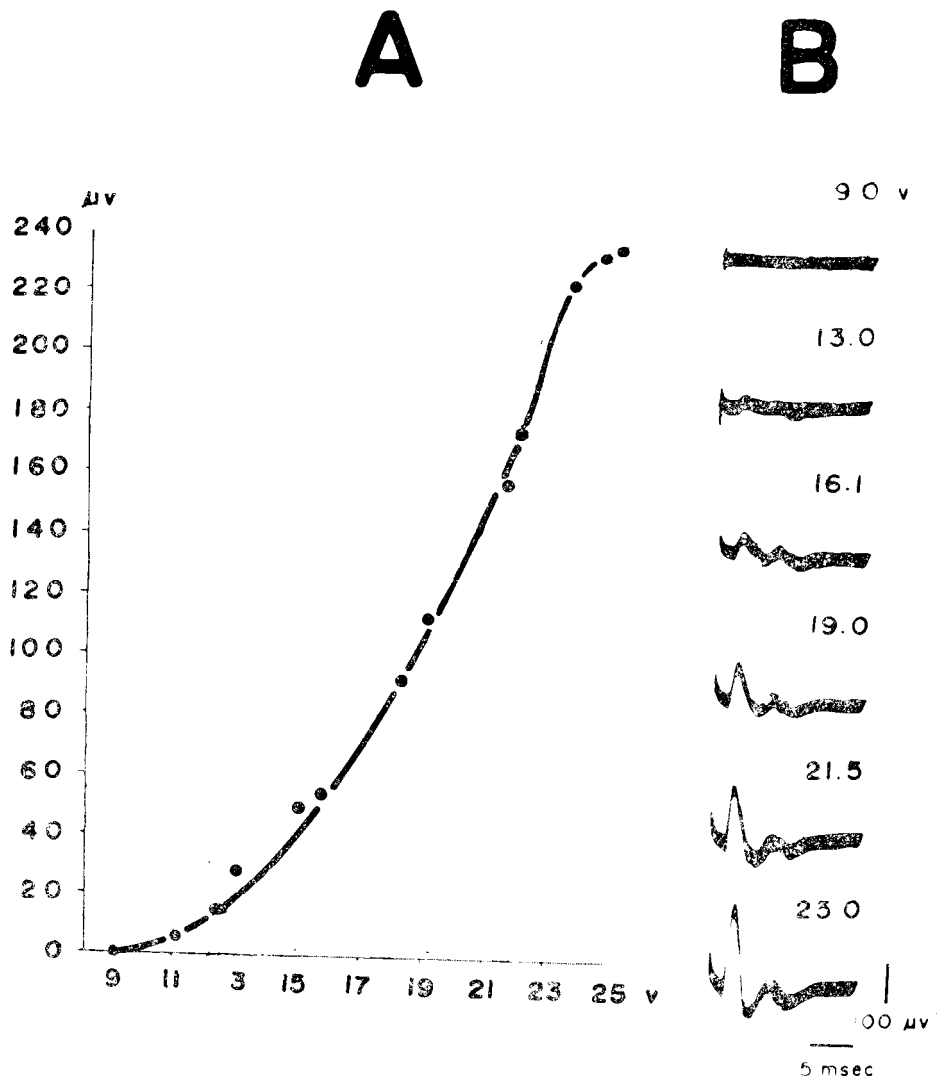

Fig. 2. Antidromic responeses in the hypoglossal nucleus evoked by stimulation of ipsilateral hypoglossal nerve. A, directly evoked antidromic spike (DEAS) in hypoglossal nucleus in relation to intensity of stimulation of hypoglossal nerve. Abscissa: intensity of stimulation (1 c/s, 0.1 msec) in volts. Ordinate: amplitude of DEAS (circles). B, DEAS, Stimulation: left hypoglossal nerve (1 c/s, 0.1 msec), applied intensities expressed at right upside. Record: left hypoglossal nucleus, 10 sweeps superimposed. Time base; 5 msec. Calibration; 100μV.

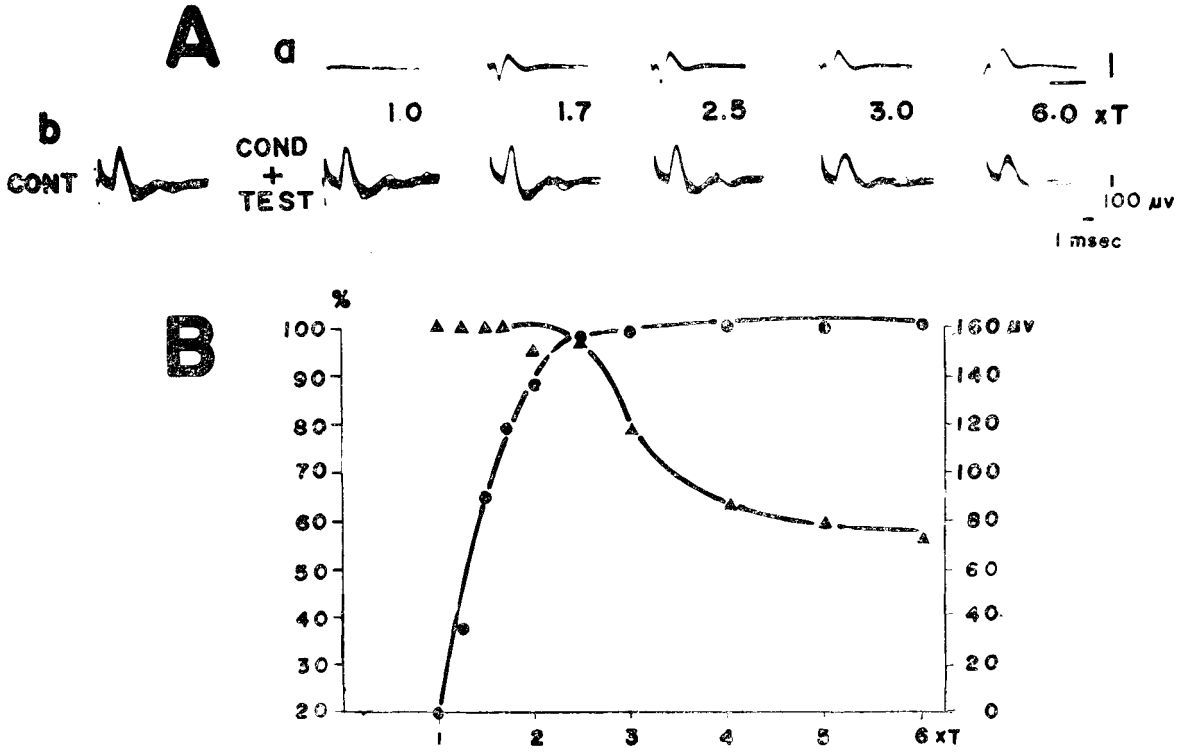

Fig. 3. Hypoglossal DEAS in relation to intensity of masseteric nerve stimulation. Aa row: responses in semilunar ganglion to masseteric nerve stimulation. Stimulation; left masseteric nerve (1 c/s, 0.02 msec), applied intensities expressed by numerals representing multiples of nerve threshold (xT) in each record. Record: left semilunar ganglion, monopolar, upward deflection, negative. Ab row; hypoglossal DEAS induced by hypoglossal nerve stimulation. CONT: control response 1 c/s, 0.1 msec, 20 V. COND+TEST: conditioned response. Conditioning stimulus; left masseteric nerve (1 c/s, 0.02 msec), applied intensities expressed by numerals representing multiples of masseteric nerve threshold in Aa. Test stimulus; left hypoglossal nerve stimulation. 1 c/s, 0.1 msec, 20 V. Conditioning-test interval; 20 msec. Record; left hypoglossal nucleus. 10 sweeps superimposed. B, effects of masseteric nerve stimulation on hypoglossal DEAS and on responses in semilunar ganglion. Circles: peak to peak amplitude of responses in semilunar ganglion to masseteric nerve stimulation. Triangles: amplitude of conditioned DEAS in hypoglossal nucleus in percentage (control DEAS: 100%). Abscissa: intensities of masseteric nerve stimulation represented as multiples of nerve threshold. Ordinates: left applies to triangles, right to circles. A and B obtained from a curarized cat.

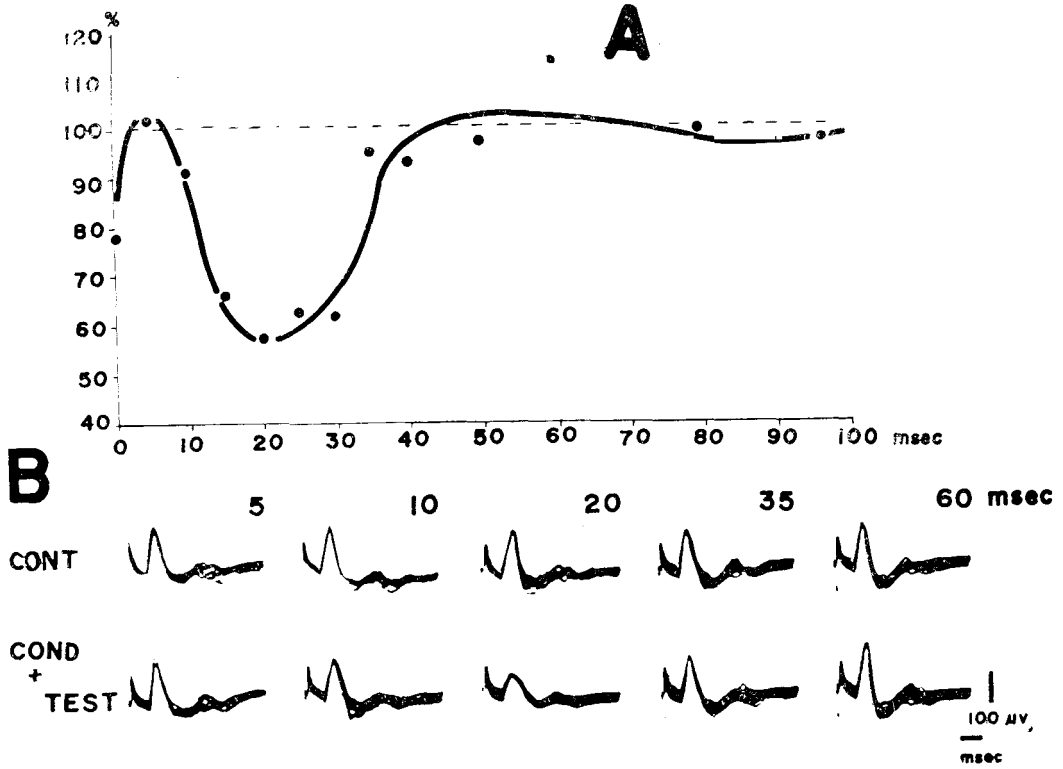

Fig. 4. Time course of hypoglossal depolarization induced by stimulation of masseteric nerve.

A: time course of hypoglossal depolarization. Abscissa: conditioning-test intervals in msec. Ordinate: amplitudes of conditioned DEAS in per cent (control 100%). B, sample records of hypoglossal depolarization produced by stimulation of masseteric nerves. Test stimulus: left hypoglossal nerve (1 c/s, 0.1msec, 23V). Conditiong stimulus: left masseteric nerve (0.02 msec, 3V).Record: left hypoglossal nucleus. Upper row; control responses in left hypoglossal nucleus to stimulation of left hypoglossal nerve. Lower row: responses conditioned by left masseteric nerve stimulation. All are 10 sweeps superimposed. Numerals represent conditioning-test intervals in msec.

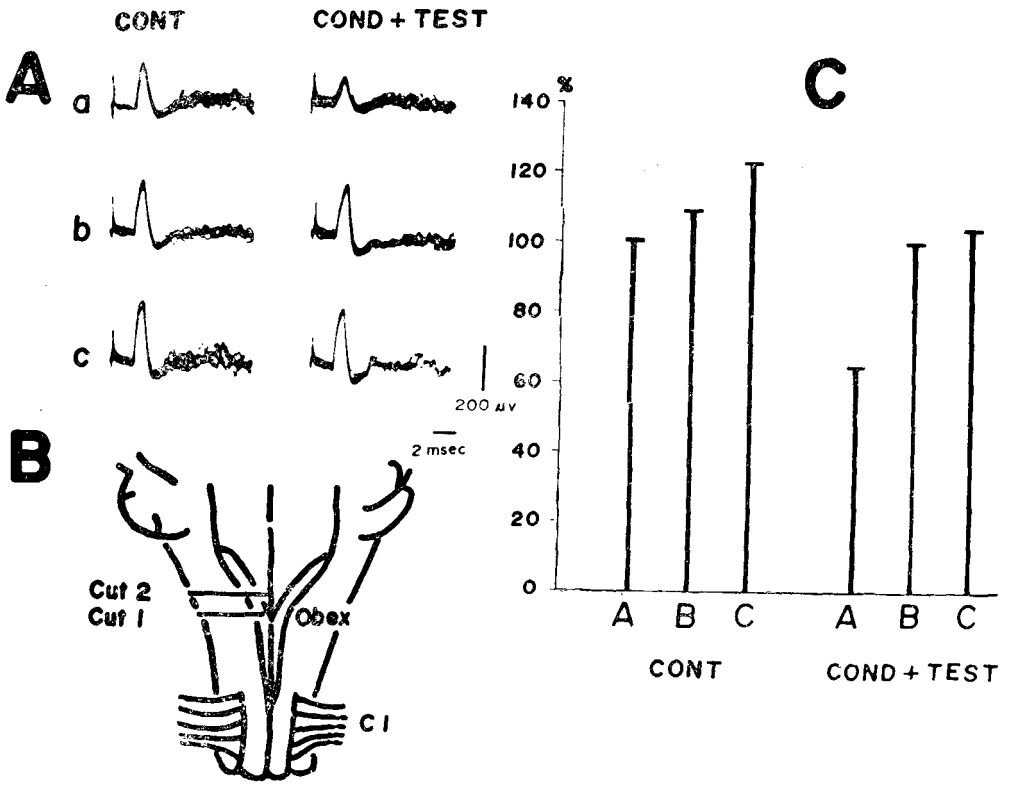

Fig. 5. Effects of the semi-transection at the obex level on the DEAS.

A: masseteric conditioning effect on DEAS in the hypoglossal nucleus before and after ipsilateral semi-transection. a row: before transection. b row: after semitransection at the obex level (1st semi-transection) c row: after 2 mm rostral semi-transection following the 1st semi-transection. CONT: control response. COND+TEST: responses conditioned by ipsilateral masseteric nerve stimulation. Test stimulation: left hypoglossal nerve, (1 c/s, 0.1 msec, 19 V). Conditioning stimulus: left masseteric nerve (0.02 msec, 5 V). Record: left hypoglossal nucleus. All are 10 superimposed. Conditioning-test interval: 20 msec. B: schematic representation of semi-transection. Cut 1: semi-transection at the obex level. Cut 2: 2 mm rostral semi-transection after the obex level transection. C: compare the amplitude of DEAS before and after the semi-transection. They are obtained from Fig. 5. Aa, b, and c rows. Abscissa: amplitude of DEAS in per cent (control DEAS: 100% in Fig. 5Aa). CONT. A,B,C: The testing DEAS amplitude ratios of former (Aa), 1st semi-transection (Ab), and 2nd semi-transection (Ac) to Aa of the test stimulus. COND+TEST. A,B,C: amplitude ratios of conditioned DEAS in per cent in each Aa, Ab and Ac row.

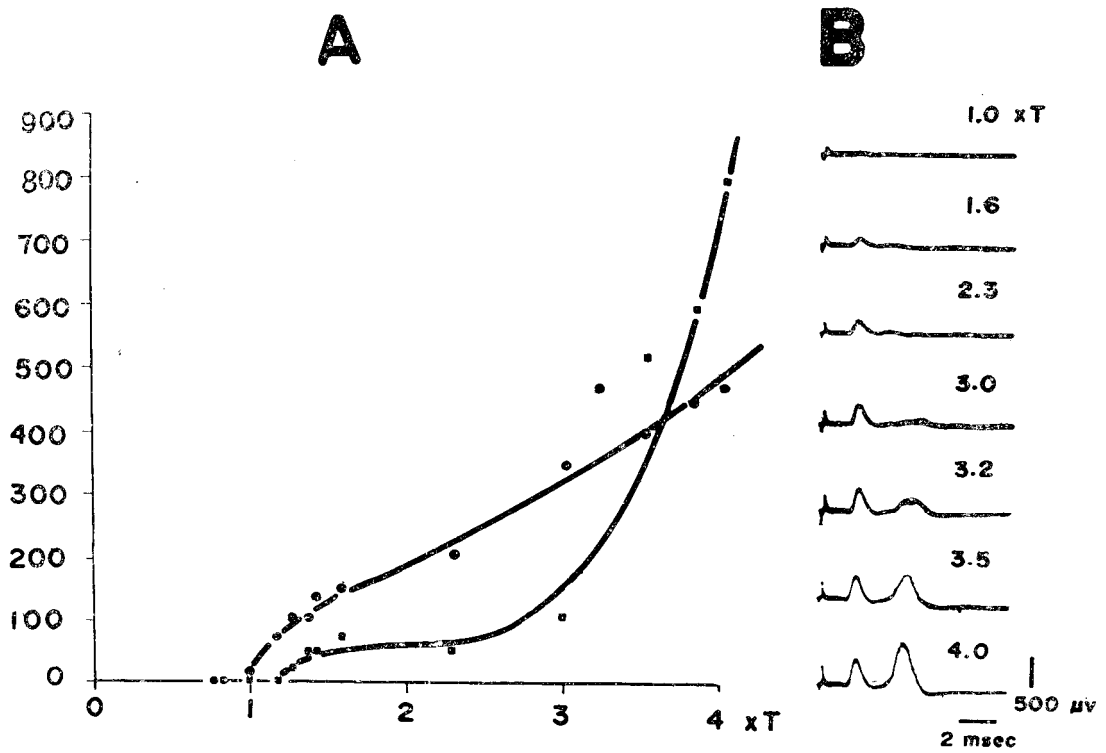

Fig. 6. Masseteric monosynaptic reflex responses in the masseteric nerve evoked by stimulation of ipsilateral mesencephalic nucleus.

A: antidromic (circles) and orthodromic (squares) spike potentials in masseteric nerve in relation to intensities of stimulation of trigeminal mesencephalic nucleus. Abscissa: intensity of stimulation (1 c/s, 0.1 msec), applied intensities expressed by numerals repressenting multipls of nucleus threshold (xT). Ordinate: amplitude of antidromic and orthodromic spike potential. B. directly evoked antidromic spike and monosynaptically evoked orthodromic responses. Stimulation: left trigeminal mesencephalic nucleus (1 c/s, 0.1 msec), apllied intensities expressed by numerals representing multiples of nucleus threshold (xT) in each record. Record: left masseteric nerve. 10 sweeps superimposed.

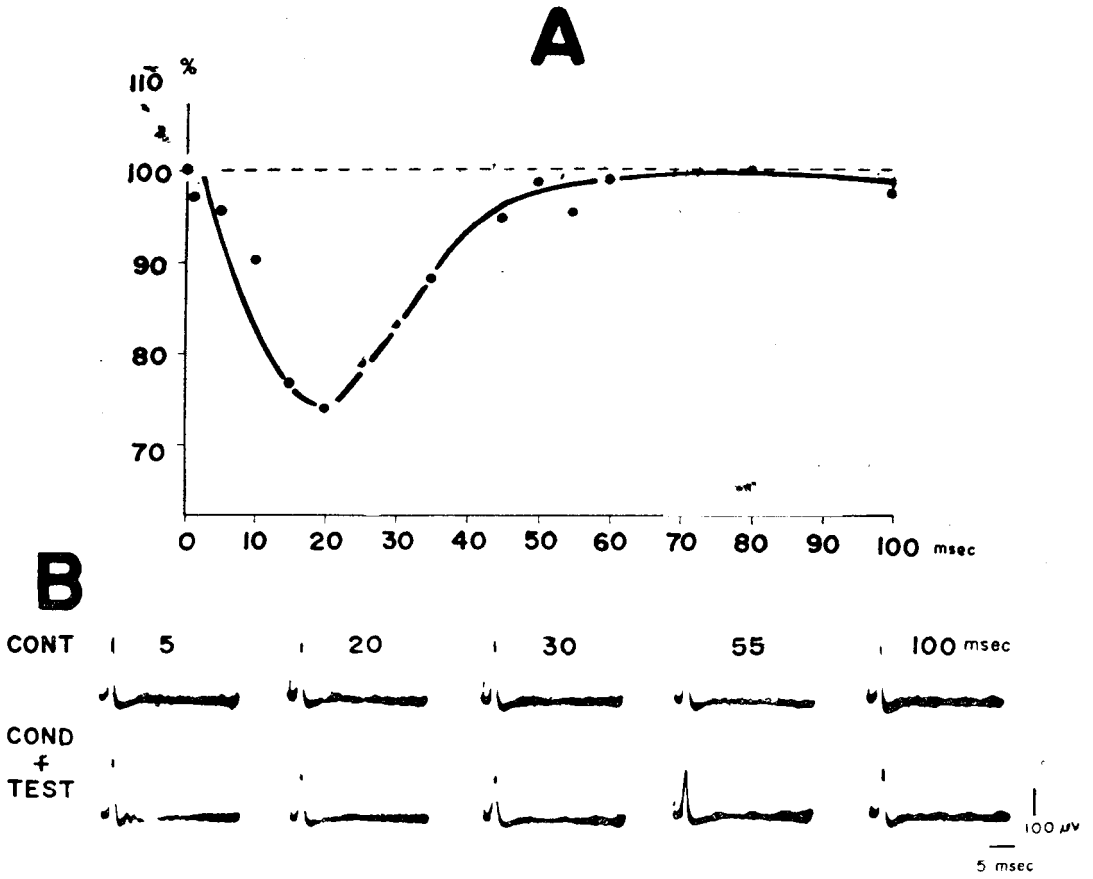

Fig. 7. Time course of hypoglossal depolarization induced by stimulation of trigeminal mesencephalic nucleus.

A: time course of hypoglossal depolarization. Abscissa: conditioning-test intervals in msec. Ordinate: amplitudes of conditioned DEAS in per cent (control 100%). B: sample records of hypoglossal depolarization produced by stimulation of mesencephalic nucleus. Test stimulus: left hypoglossal nerve (1 c/s 0.1 msec, 25V). Conditioning stimulus: left trigeminal mesencephalic nucleus (0.05 msec, 6V). Record: left hypoglossal nucleus. Upper row; control responses in left hypoglossal nucleus to stimulation of left hypoglossal nerve. Lower row; responses conditioned by left trigeminal mesencephalic nucleus stimulation. All are 10 sweeps superimposed, numerals represent conditioning-test intervals in msec.

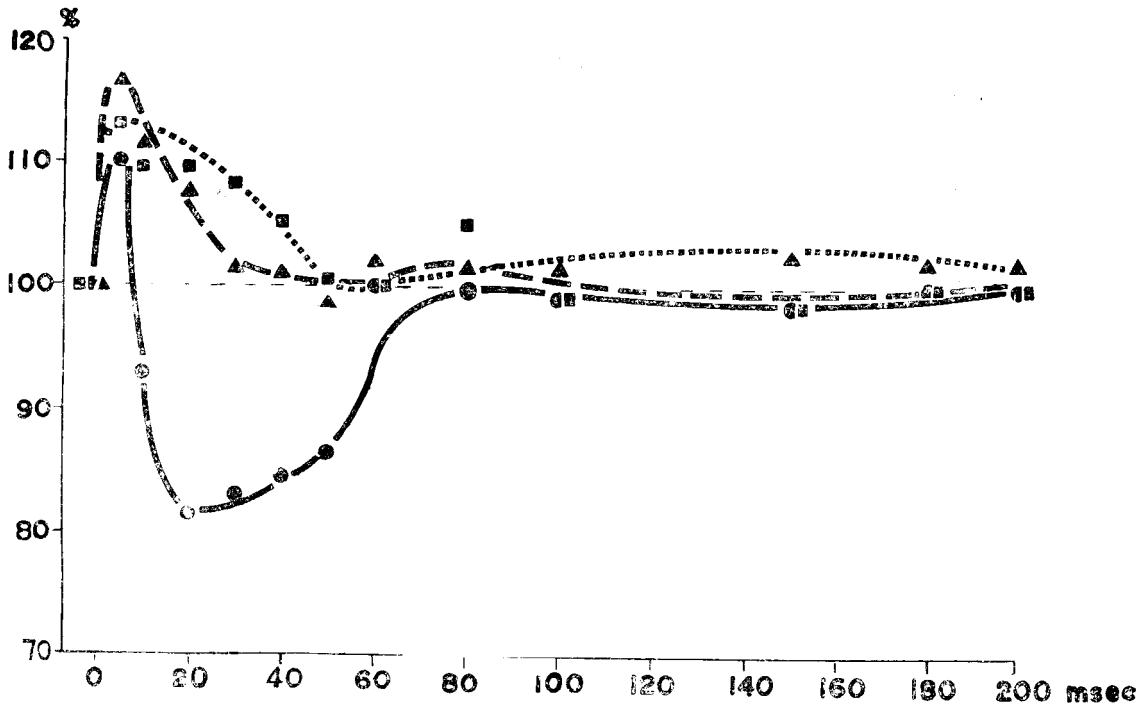

Fig. 8. Time course of masseterically induced hypoglossal depolarization and effect of sodium pentobarbital and strychnine. Abscissa: Conditioning-test intervals in msec. Ordinate: amplitude of conditioned DEAS in per cent (control 100%). Circles: before application of drugs. Squares: after i.v. injection of pentobarbital sodium, 10 mg/kg. Triangles: after i.v. injection of strychnine 0.09 mg/kg. applied 40 min after injection of pentobarbital sodium at 10 mg/kg. They are obtained from a curarized cat.

中 文 摘 要

咬肌固有受容性衝動對調整舌下神經核細胞活動之反射機序的影響

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高等動物在養成咀嚼運動時，顎及舌二者之間，要有極密切的協同作用才能完滿達成其運動。惟其運動方式，因其功用特殊，與其他四肢之運動方式稍有不同。

吾人欲闡明其運動機序，茲以猫之咬肌爲對象，使之與舌研究其固有受容器所發出之向心性衝動對於舌運動之影響，以期將來能在臨床醫學上，神經生理學上，或行動科學上，有所貢獻。實驗結果如次：

給與咬肌神經之刺激強度，如果超過該神經興奮閾值之2.5倍以上時，則對於同側舌下神經刺激，而在腦幹舌下神經運動核中誘發的興奮，有抑制作用。

測定咬肌神經興奮閾值之高低，及參照Eccles, Lloyd 及 Windle 等的電氣生理學及解剖學上的業績來分類，則對於舌下神經運動核有抑制作用的咬肌神經纖維，其第二、及第三神經纖維皆在興奮時對運動核與奮性有抑制作用。至於第一a及b群纖維，則沒有抑制作用。

該抑制作用之時間經過 (Time Course) 是咬肌神經一舌下神經刺激間隔爲 10msec 以上至80～100msec之間有抑制效果。其中15～20msec時，其效果達到最高。至於 100msec 以上時，即無效果之發現。又 2～8msec 以內，有臨時性的促進作用。

給與咬肌神經的刺激愈大，其產生的抑制效果亦隨之增加。

又，由因腦幹門部之半橫切（同側），引起抑制作用之消失，可認爲同側三叉神經脊髓路核爲其抑制經路。