EFFECT OF LINGUAL NERVE AND ITS RELATED NUCLEUS ACTIVITY ON THE JAW-CLOSING REFLEX MECHANISM*

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INTRODUCTION

Oral reflexes were first extensively studied by Miller *et al.* (15) and Sherrington (20). In thier studies, the natural and electrical stimulation was applied to the decerebrate cat, and several reflex movements were induced. They reported that single electrical shock applied to the lingual nerve caused opening of the mouth via reflex arc of the masticatory muscles (15)

Anatomical studies have shown that the processes of trigeminal mesencephalic nucleus terminate in the spindles of masticatory muscles and give collaterals to the masseteric motoneuron (12, 25). Functionally, these collaterals make monsynaptic and excitatory linkage with masseteric motoneuron (11, 13, 17, 25).

Therefore, the masseteric reflex is not only caused by masseteric stretch but also elicited by stimulation of trigeminal mesencephalic nucleus (11). During the masticatory movement, some highly coordinated relation which underlies between tongue and jaw to prevent the injury from each other must exist. In previous data, we knew that activation of masseteric nerve induced a marked depression of linguo-hypoglossal reflex (18) and presynaptic inhibition of jaw-opening reflex (16).

Recently, Goldberg (10) reported that activation of lingual nerve induced a profound suppression on masseteric monosynaptic reflex. However, the role of trigeminal spinal nucleus which is the most important neural linkage in masticatory movement has not been clarified. In this paper, we will describe the effects of trigeminal spinal nucleus on the masseteric monosynaptic reflex elucidated by electrophysiological and pharmacological observation.

METHOD

General

The experiments were done on 40 cats. In earlier several animals, pentobarbital sodium (30 mg/kg i.p.) was used for anesthesia druing cannulation and surgical operation. However, the application of pen-

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tobarbital sodium gave a prolonged suppression effect on reflex potential. Thus, in later experiments ether inhalation for cannulation and then thiopental sodium for anesthetizing were used.

Tracheotomy and cannulation into the left femoral vein were performed under ether inhalation, then the ether inhalation discontinued and anesthesia was maintained by repeated injection of thiopental sodium (5mg/Kg i.v.) every 15 min throughout the period of surgical operation. After local application of procaine, the spinal cord was transected between C_2 and C_3 with an opthalmic scissors and artificial ventilation was immediately started.

The cerebellum was removed in all cats to expose the brain stem. At the beginning of recording which was 2 hours after the last injection of thiopental sodium, gallamine triethiodide (Flaxedil) was injected and pneumothorax was made bilaterally. Cats were immobilized by repeated injection of gallamine triethiodide whenever necessary. Rectal temperature, temperature of brain stem and temperature of the paraffin oil pool were kept at 36-38°C with a heating pad and two infrared lamps.

Preparation of nerves

The left lingual nerve was exposed by ventral approaching and isolated from the surrounding tissue. The mandibular skin was cut longitudinally to make a paraffin oil pool for this nerve. 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 with cotton thread and sectioned. The left parietal bone was holed (1 cm in diameter) by rongeur at the A. 8.0 of the atlas of brain map (22) for recording the action potential in Gasserian ganglion.

Stimulation and recording

The cat's head was fixed in the sterotaxic apparatus then the apparatus was tilted about 80°with the left side down. Two pools were made of the skin flaps of the mandibular and temporal regions, one for the lingual nerve, the other for the masseteric nerve. The peripheral end of these nerves were placed on bipolar silver wire electrodes (polar distance 3-4 mm). The whole prepared nerve was then immersed in paraffin oil and stimulation or recording was made (Fig 1).

According to the atlas of the brain map (22), the concentric bipolar needle electrodes insulated except for a 0.5-1.0 mm tip were obliquely inserted into the left mesencephalic nucleus and the left trigeminal spinal nucleus (subnucleus interpolaris) for recording or stimulation. The other same type of electrode was inserted perpendicularly through the previously drilled hole into left minor part of Gasserian ganglion to monitor the incoming volleys of the masseteric nerve. The incoming volleys were rocorded monopolarly between the core and the neck muscle.

Electrical train pulse or single shock generated by electronic stimulators (Nihon Khoden MSE 3R and 40) were used for stimulation through isolation transformers. The pulses of 0.01-0.1 msec in duration were applied to masseteric nerve, of 0.02-0.1 msec to lingual nerve, and of 0.1 msec to the mesencephalic nucleus and trigeminal spinal nucleus.

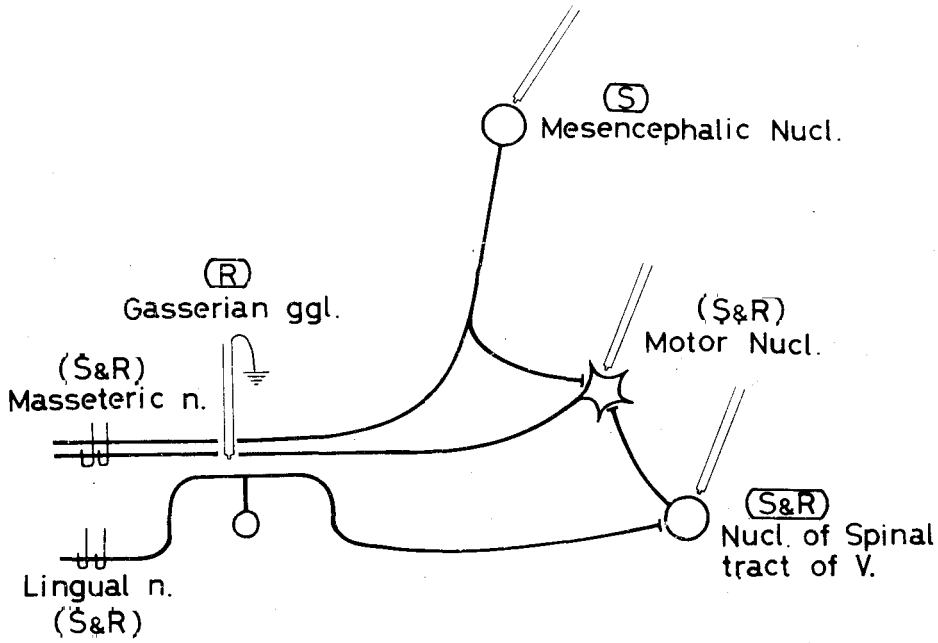

Fig 1. Schematic representation of experimental setup. The letters "S" and "R" in the circumscribed area represent stimulation and recording, respectively. The concentric bipolar needle electrodes and silver bipolar wire electrodes were used.

Train pulse was applied to the lingual nerve or trigeminal spinal nucleus as conditioning stimulus at an interval of 1.6 or 2 msec. The term "conditioning-test interval" means the intervals between the end of conditioning pulse and the beginning of test stimulus in our experiment. Responses were recorded on a cathode ray oscilloscope (Nihon Khoden VC 7) by superimposed 6-10 responses. The average amplitude of conditioned potentials were divided by those of control responses, expressing the control value as 100%.

RESULT

Responses in Gasserian ganglion to stimulation of masseteric nerve

Stimulation of masseteric nerve evoked a triphasic potential. The latency of the onset of the first negative peak was 0.16±0.04 msec(mean ±S.D. n=11).

With this latency and the length of nerve, the conduction velocity of fibers yielding the earliest potential was 91-96 m/sec. The amplitude from the first negative to the positive peak increased almost proportionally with the intensity of stimulus up to 2 times the threshold (xT) of the nerve and then reached a quasi-plateau until 4 xT (Fig 2) which was the highest intensity applied in our experiment.

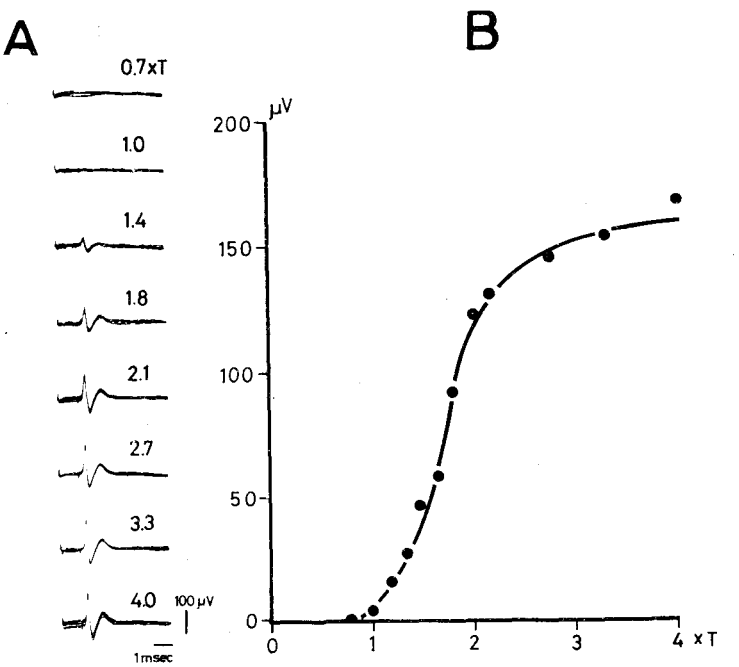

Fig 2. Responses in Gasserian ganglion to stimulation of ipsilateral masseteric nerve. A. Orthodromic responses evoked by stimulation of masseteric nerve. Stimulation: left masseteric nerve (1/sec, 0.01 msec); applied intensities expressed by numerals representing multiples of nerve threshold (xT) in each record. Record: left Gasserian ganglion monopolar, upward deflection, negative; average of 10 responses. B. Peak-to-peak amplitude of masseteric nerve potential in relation to intensity of stimulation. Abscissa: intensities of masseteric nerve stimulation expressed by multiples of nerve threshold. Ordinate: amplitude of masseteric nerve potential.

Masseteric monosynaptic reflex (masseteric MSR) evoked by stimulation of ipsilateral trigeminal mesencephalic nucleus

Stimulation of the trigeminal mesencephalic nucleus elicited, in the masseteric nerve, a monosynaptic reflex (2, 11). Our observation was identical to the previous reports. The evoked potential consisted of 2 spikes, an initial small spike and a following large spike (Fig 3Aa). The latency of the onset of the first spike and second spike was 0.74 ± 0.06 msec (n=9) and 1.59±0.17 msec (n=9), respectively. The second spike was susceptible to repetitive stimulation. With the increase in stimulation frequency, the second spike was gradually suppressed (Fig 3B). Stimulation frequency of 5/sec completely abolished the spike. However, the first spike was not altered even after changes in stimulation frequency up to 100/sec. Therefore, the first spike which has a briefer latency must have been due to impulses propagated antidromically down the masseteric nerve. However, the second spike with longer latency and susceptible to changes in stimulation frequency was an orthodromically evoked,

monosynaptic reflex via the motor nucleus (1, 6, 7, 11, 25). The orthodromic spike increased almost linearly with the stimulus intensity up to 10 times the threshold (xT) of mesencephalic nucleus excitation, but the antidromic potential reached a maximum at 2.5 xT (Fig 3Ab).

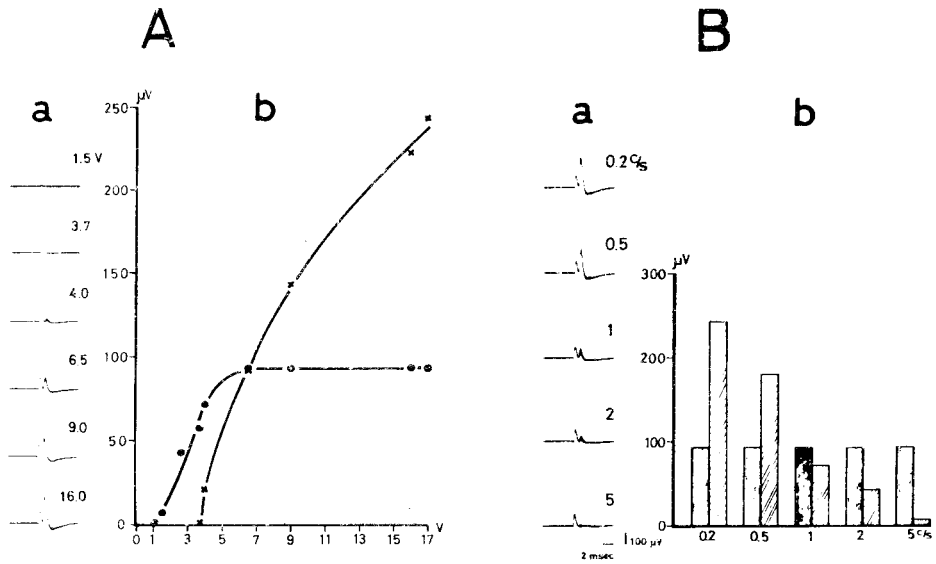

Fig 3. Masseteric monosynaptic reflex evoked by stimulation of ipsilateral mesencephalic nucleus. Aa. Directly evoked antidromic spike and monosynaptically evoked orthodromic responses. Stimulation: left trigeminal mesencephalic nucleus (0.2/sec 0.1 msec); applied intensities expressed at right upside in each record. Record: left masseteric nerve, 10 sweeps superimposed. Ab. Antidromic (filled circles) and orthodromic (crosses) spike potentials in masseteric nerve in relation to intensities of stimulation of trigeminal mesencephalic nucleus. Abscissa: intensities of stimulus (0.2/sec, 0.1 msec) in volts. Ordinate: amplitude of antidromic and orthodromic spike potentials. B. Effect of repetitive stimulation on masseteric monosynaptic reflex. Ba: Responses in the masseteric nerve evoked by various frequencies of stimulation. Stimulation: left trigeminal spinal nucleus (0.2/sec, 0.1 msec); applied frequencies expressed at right upside in each record. Record: left masseteric nerve, 10 sweeps superimposed. Bb: Amplitude of antidromic (shadow column) and orthodromic (oblique line column) spike potential in relation to frequencies of stimulation. Abscissa: frequencies of stimulation expressed by cycles per second (c/s). Ordinate: amplitude of antidromic and orthodromic spike potential.

Antidromically and/or orthodromically evoked potentials in trigeminal spinal nucleus and lingual nerve

Activation of the trigeminal spinal nucleus evoked in the ipsilateral lingual nerve a spike with a brief latency and a group of rhythmic

potential (Fig. 4Aa).

The latency of the spike and the first peak of the rhythmic potential was 0.81±0.17 msec (n=4) and 4.90±0.52 msec (n=4), respectively. The amplitude of the spike continued to increase almost proportionally with the intensity up to 5 times the threshold (xT) of the spinal nucleus excitation. The rhythmic potential appeared at 2-2.3 xT of evoking the spike and reached the maximum at 3.3 xT. The rhythmic potential was very liable to changes in the frequency of stimulation.

It was completely suppressed with a stimulation frequency of 5/sec, but the spike was consistently evoked.

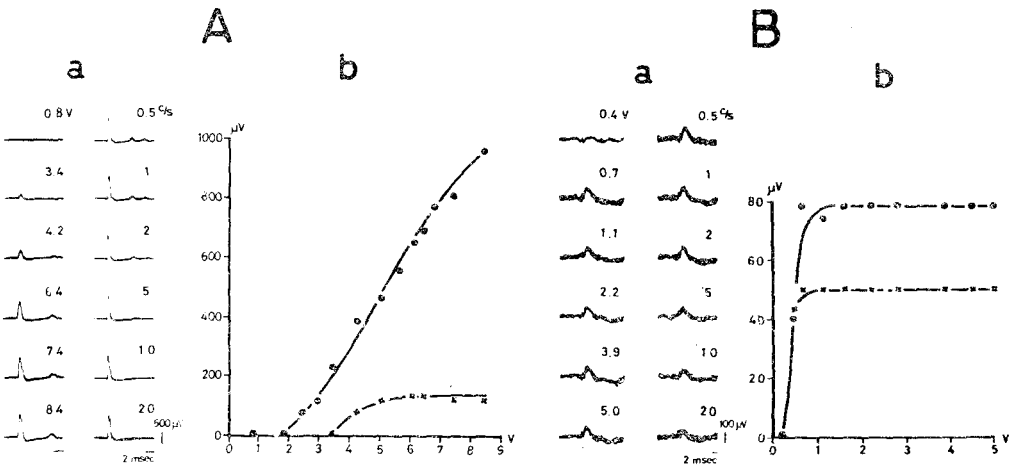

Fig 4. Antidromically and/or orthodromically evoked potentials in trigeminal spinal nucleus and lingual nerve. A. Antidromic responses in the lingual nerve evoked by stimulation of ipsilateral trigeminal nucleus. Aa: left column—the numerals represent the intensity of stimulation in volts. right column—the numerals represent the frequency of stimulation. Stimulation: left trigeminal spinal nucleus (1/sec, 0.1 msec). Record: left lingual nerve; 10 sweeps superimposed. Ab: the antidromic spike (filled circles) and the rhythmic potential (crosses) in lingual nerve in relation to intensity of stimulation. Abscissa: intensities of stimulation (1/sec, 0.1 msec) in volts. Ordinate: amplitude of the antidromic spike and the first peak of the rhythmic potential. B. Orthodromic responses in the trigeminal spinal nucleus evoked by stimulation of ipsilateral lingual nerve. Ba: Left column—the numerals represent the intensity of stimulation in volts. Right column—the numerals represent the frequency of stimulation. Bb: amplitude of presynaptic component (crosses) and postsynaptic component (filled circles) in relation to intensities of stimulation of trigeminal spinal nucleus. Abscissa: intensities of stimulation in volts. Ordinate: amplitude of presynaptic and postsynaptic component.

Stimulation of the lingual nerve evoked orthodromically, in the ipsilateral trigeminal spinal nucleus, a brief-latency positive-negative potential which was followed by a negative complex. The latency of the positive peak of the positive-negative potential was 0.8 msec (n=2)

and the latency of the onset of the negative potential was 1.6 msec (n=2). The amplitude of the positive-negative potential was stable and stimulation frequency of 20/sec did not suppress the potential appreciably. On the other hand, the negative complex was variable in shape and an increase of stimulation frequency up to 20/sec induced a reduction of 60% of the amplitude compared with that evoked by 0.5/sec stimulation. Thus, the brief-latency potential and the following negative complex represented the activities of the presynaptic and postsynaptic components in the trigeminal spinal nucleus, respectively, as reported by Erickson *et al.* (8) (Fig. 4Bab).

Effect of lingual nerve stimulation on masseteric MSR

The masseteric MSR was evoked by stimulation of the trigeminal mesencephalic nucleus, recording in the masseteric nerve. The threshold conditioning stimuli were applied to the ipsilateral lingual nerve. There were two phases of suppression. The first completely suppressed phase began at conditioning-test interval of 3 msec, and the masseteric MSR was completely suppressed at 8-15 msec. A period characterized by rapid recovery in reflex amplitude followed the first suppression phase.

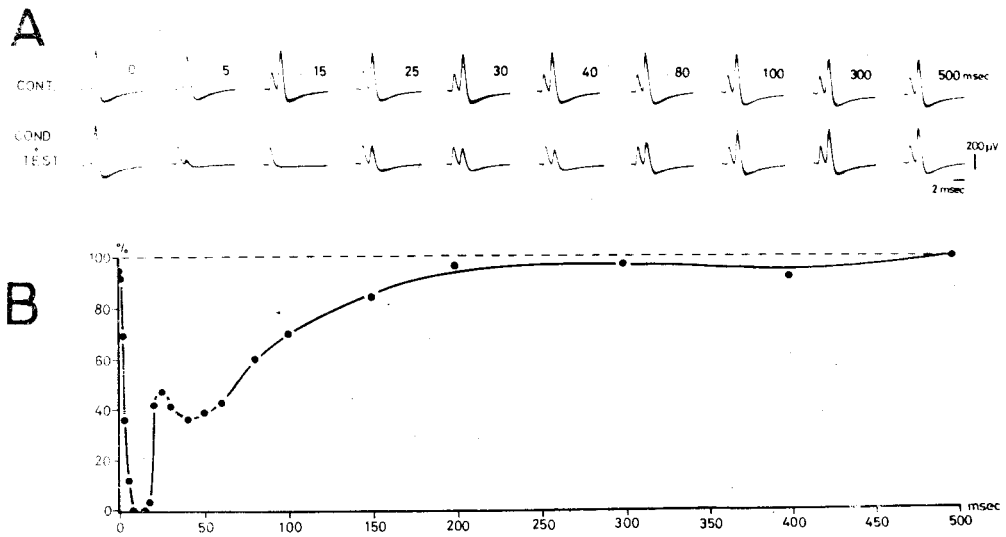

Fig. 5. Time course of suppression of the masseteric MSR induced by stimulation of ipsilateral lingual nerve. A. Sample records of suppression of masseteric MSR induced by stimulation of ipsilateral lingual nerve. Test stimulus: left trigeminal mesencephalic nucleus (0.5/sec, 0.01 msec, 25 V). Conditioning stimulus: left lingual nerve (0.5/sec, 0.02 msec, 1.65 V, single shock). Record: left masseteric nerve. Upper row: control responses in left masseteric nerve to stimulation of ipsilateral trigeminal mesencephalic nucleus. Lower row: responses conditioned by ipsilateral lingual nerve. All are 10 sweeps superimposed; numerals represent conditioning-test intervals in msec. B. Time course of suppression of masseteric MSR. Abscissa: conditioning-test intervals in msec. Ordinate: amplitude of conditioned masseteric MSR in per cent (control: 100%).

This increase of reflex amplitude reached a peak at 25 msec, and then there began a second phase of suppression, less effective but more prolonged than the first phase. The second suppression phase was most profound at conditioning-test interval of 40 msec, and was slowly decreased. The time course of suppression induced by lingual nerve last about 200 msec. Fig 5 illustrates an example of the time course of suppression of the masseteric MSR induced by ipsilateral lingual nerve stimulation.

Effect of strychnine on the masseteric MSR suppression induced by lingual nerve

Strychnine nitrate was applied to observe the effect of masseteric MSR. In case of large dose, the early period of the suppression was reversed. Fig 6 illustrates an example of the effect of strychnine (0.1 mg/Kg, i.v.) on the masseteric MSR suppression. After 65 min of the application, the early phase of the suppression was reversed to become greatly facilitatory. This facilitation became most evident at

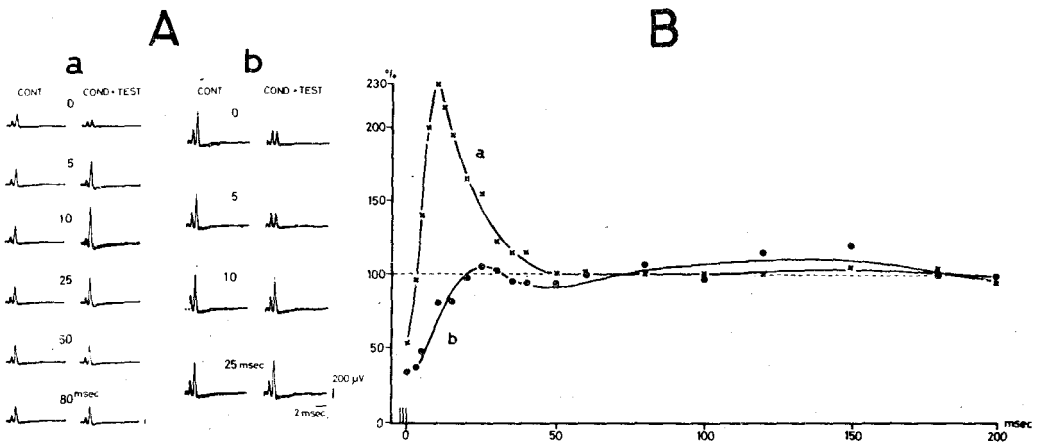

Fig 6. Effect of strychnine nitrate on the suppression of masseteric MSR induced by ipsilateral lingual nerve. A. Samples taken beginning 65 min (Aa) and 160 min (Ab) after intravenous injection of strychnine (0.1 mg/Kg). Both in Aa and Ab, left column: control responses in left masseteric nerve to stimulation of left trigeminal mesencephalic nucleus. right column: responses conditioned by stimulation of ipsilateral lingual nerve. All are 6 sweeps superimposed; numerals represent conditioning-test intervals in msec. Time base: 2 msec. Calibration: 200 μV for all records. B. Effect of strychnine on time course of suppression of masseteric MSR induced by ipsilateral lingual nerve. Curve a: beginning 65 min after strychnine application. Curve b: beginning 160 min after strychnine application. Abscissa: conditioning-test intervals in msec. Ordinate: amplitude of conditioned masseteric MSR in per cent (control: 100%).

conditioning-test interval of 5 msec and then gradually reduced to control value at 50 msec. The late period of the time course did not change significantly. After 160 min of the application, the facilitatory phase disappeared. The time coures recovered toward that before application and two phases of suppression reappeared.

Blocking effect of lingnal nerve on suppression of masseteric MSR

The conduction of lingual nerve was blocked at a portion proximal to the stimulation electrode by crush with a forceps. The stimulation inten-

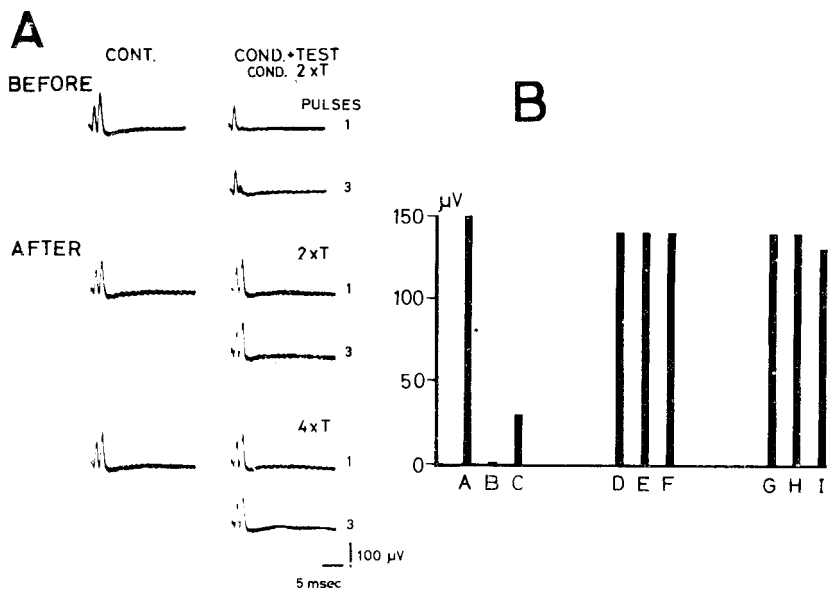

Fig 7. Effect of crush of the lingual nerve on the lingually induced suppression of masseteric MSR. A. Samples taken before and after crushing the lingual nerve. BEFORE: representing before crushing the lingual nerve, 2 times the threshold (xT) of lingual nerve delivered as conditioning stimulus, pulse of conditioning stimulus expressed by numerals on the right side of each record. AFTER: representing after crushing lingual nerve, 2 or 4 times the threshold of lingual nerve delivered as conditioning stimulus, pulse of conditioning stimulus expressed by numerals on right side of each record. CONT.: control responses. COND + TEST: conditioned responses. Test stimulus: left trigeminal mesencephalic nucleus (0.5/sec, 0.1 msec, 3 pulses or single pulse); applied intensities expressed as multiples of nerve threshold (xT). Conditioning — test intervals are all 10 msec. B. Histogram comparing the amplitude of conditioned responses before and after crushing. A- control before crush. B- conditioned response before crush (2 xT, 1 pulse). C- conditioned response before crush (2 xT, 3 pulses). D- control response after crush. E- conditioned response after crush(2 xT, 1 pulse). F- conditioned response after crush (2 xT, 3 pulses). G- control response after crush. H- conditioned response after crush (4 xT, 1 pulse). I- conditioned response after crush (4 xT, 3 pulses).

sity of 2 xT and 4 xT of the lingual nerve were compared before and after crush. The conditioning-test intervals are all 10 msec. After the crush, the effect of single pulse or 3 pulses of conditioning stimuli was completely abolished. The masseteric MSR was not suppressed by stimulation of lingual nerve (Fig 7).

Suppression of masseteric MSR induced by stimulation of ipsilateral trigeminal spinal nucleus

The masseteric MSR was suppressed by stimulation of lingual nerve.

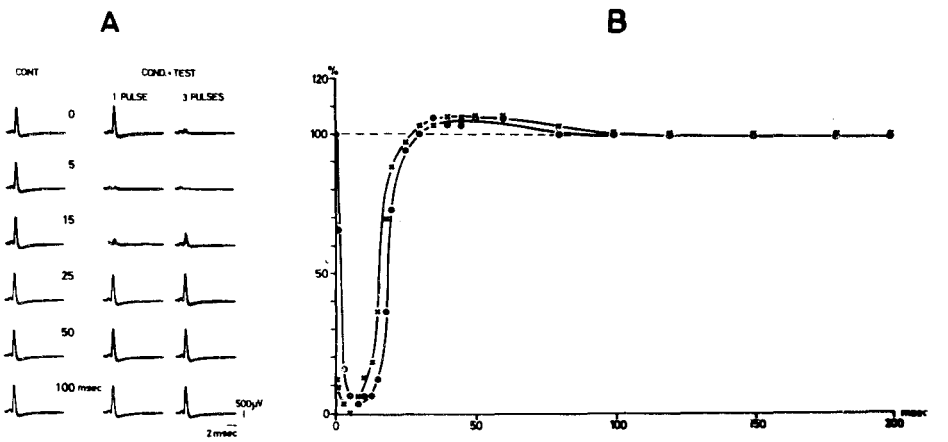

Fig 8. Suppression of masseteric MSR induced by stimulation of trigeminal spinal nucleus. A. Sample records of masseteric MSR induced by stimulation of spinal nucleus. Test stimulus: left trigeminal mesencephalic nucleus (0.5/sec, 0.1 msec, 25 V). Conditioning stimulus: left trigeminal spinal nucleus (0.5/sec, 0.01 msec, 10 V, single shock or 3 pulses, 1.6 msec interval). Record: left masseteric nerve. CONT. : control responses. COND+TEST: 1 pulse—responses conditioned by single pulse of conditioning stimulus; 3 pulses—responses conditioned by 3 pulses of conditioning stimulus. Numerals represent conditioning-test intervals in msec. Time base: 2 msec. Calibration: 500 μV for all records B. Time course of suppression of masseteric MSR induced by stimulation of ipsilateral trigeminal spinal nucleus. x x x: conditioned by 3 pulses; . . . : conditioned by single shock. Abscissa: conditioning-test intervals in msec. Ordinate: amplitude of conditioned masseteric MSR in per cent (control: 100%).

The trigeminal spinal nucleus as a relay nucleus of lingual nerve, we consider that it must be involved in this suppression. Either single shock or 3 shocks of conditioning stimulus were delivered to the ipsilateral trigeminal spinal nucleus. Fig 8 illustrates the time course of the masseteric MSR suppression induced by conditional stimulus of spinal nucleus. There was only one phase of suppression. 3 shocks of conditioning stimulus suppressed the masseteric MSR earlier and more effectively than single shock. With 3 shocks of conditioning stimulus the suppression began at conditioning-test interval of 0 msec and the reflex was completely

suppressed at 5 msec, then the amplitude of the reflex rapidly recovered to control value at 25 msec. While suppression induced by single shock of conditioning stimulus began at conditioning-test interval of 3 msec, reached the maximum at 8 msec then the amplitude of the reflex rapidly recovered to control value at 30 msec.

Drug effects on the masseteric MSR suppression induced by stimulation of ipsilateral trigeminal spinal nucleus

The effect of pentobarbital sodium on the suppression of masseteric MSR was studied in unanesthetized cats. After intravenous injection of pentobarbital

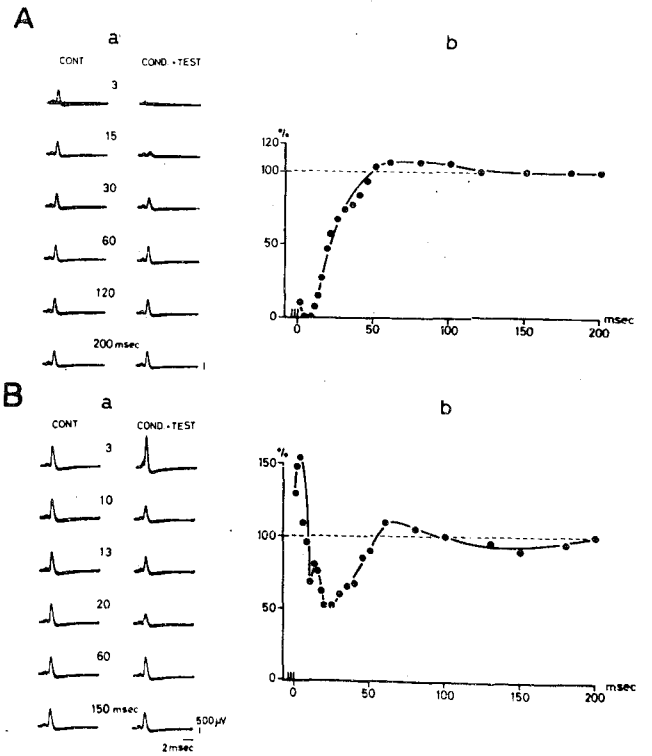

Fig 9. Drug effects on suppression of masseteric MSR induced by stimulation of ipsilateral trigeminal spinal nucleus. A. Samples taken beginning 5 min after intravenous injection of pentobarbital sodium (10 mg/kg). Test stimulus: left trigeminal mesencephalic nucleus (0.5/sec, 0.1 msec, 25 V). Conditioning stimulus: left trigeminal spinal nucleus (0.5/sec, 0.1 msec, 10 V, 3 shocks, 1.6 msec interval). Record: left masseteric nerve. Aa. CONT.: control responses. COND+TEST: conditioned responses. Numerals represent conditioning-test intervals in msec. Ab. Action of pentobarbital sodium on suppression of masseteric MSR induced by stimulation of trigeminal spinal nucleus from Aa. Abscissa: conditioning-test intervals in msec. Ordinate: amplitude of conditioned responses after application of pentobarbital sodium in per cent (control: 100%). B. Action of strychnine on suppression of masseteric MSR induced by stimulation of ipsilateral trigeminal spinal nucleus. Ba. Samples taken beginning 5 min after strychnine (0.1 mg/kg i.v.) following 3 hours after injection of pentobarbital sodium (10 mg/kg i.v.). CONT.: control responses. COND+TEST: conditioned responses. Time base and calibration: 2 msec and 500 μV in all records. Bb. Action of strychnine (0.1 mg/kg i.v.) on suppression of masseteric MSR induced by trigeminal spinal nucleus from Ba. Both A and B obtained from unanesthetized cat.

sodium (10 mg/kg) the suppression was enhanced and the recovery period last for a longer duration. The masseteric MSR was completely suppressed at conditioning-test intervals of 3-8 msec then the amplitude of the reflex gradually recovered to control value at 50 msec (Fig 9 Ba).

Strychnine nitrate (0.1 mg/kg) was intravenously injected 3 hours after administration of pentobarbital sodium (10 mg/kg i.v.). After this application, the early period of the suppression phase was altered significantly. The period at conditioning-test intervals of 0-5 msec was reversed to become facilitatory. Following the facilitatory period, a peak which characterized by slight recovery in the amplitude of the reflex appeared at 10-20 msec. However, the late period of the time course was not altered (Fig 9Bb).

DISCUSSION

Stimulation of massetreic nerve evoked a triphasic potential in the ipsilateral Gasserian ganglion. The amplitude from the first negative to the positive peak increased almost proportionally with the intensity of stimulus, then reached a quasi-plateau at intensity of 2 xT of the nerve. The most fast conduction velocity of masseteric nerve was 91-96 m/sec and this value was coincident with the other previous report (16). The masseteric nerve carries both muscle spindle afferents and axons of masseteric motoneuron (14). The threshold of these afferent fibers is lower than that of axons of masseteric motoneuron (13, 17). On the basis of its fast conduction velocity and low threshold, we suggested that the fibers responsible for yielding the earliest potential were muslce spindle afferents.

Stimulation of trigeminal spinal nucleus induced in the ipsilateral lingual nerve a spike with a brief latency followed by a rhythmic potential consisting of 3-4 peaks. The rhythmic potential which had longer latency was very susceptible to repetitive stimulation. However, the spike was consistently induced. Our observation was coincident with the other reports (16, 19, 23). On this basis, we affirmed that the spike with brief latency represented antidromically evoked activity, i.e. directly evoked antidromic spike (DEAS), and the rhythmic potential which had long latency and was unable to follow repetitive stimulation represented the trigminal dorsal root reflex (TDRR).

The masseteric MSR evoked by trigeminal mesencephalic nucleus was tested by lingual nerve stimulation. It was found that a single threshold shock delivered to ipsilateral lingual nerve induced a profound suppression of the reflex. There were two phases of suppression, an earlier phase in which the reflex was completely suppressed at conditioning-test intervals of 8-15 msec and the second phase which had long duration and was most profound at 40 msec. The whole time course of suppression last about 200 msec. It was the same as the other reports (10, 13). There were also two phases of masseteric MSR suppression induced by inferior dental nerve (13). The profound inhibition and short duration of the first phase suggested that postsynaptic inhibition may be involved. The long duration of the second phase suggested that presynaptic inhibition may be involved (23). Goldberg (10) used picrotoxin (0.6

mg/kg i.v.) which has it's specific effect on blocking presynaptic inhibition (3, 7, 16, 21) to reveal the mechanism of the suppression. As the result, there was no change in the postsynaptic inhibitory component —first phase of suppression. While the effectiveness of the presynaptic inhibitory component — second phase was significantly decreased. In our experiment, strychnine which has been reported to abolish postsynaptic inhibition and to leave unaltered any presynaptic inhibitory mechanism (3, 7, 16, 21) was applied. We found that strychnine (0.1mg/kg i.v.) greatly altered the postsynaptic inhibitory phase and early period of presynaptic inhibitory phase but did not change the late period of the presynaptic inhibitory phase significantly.

Form this strychnine application, we assumed that postsynaptic inhibition may be involved in the early period of the second phase and some overlapping of presynaptic and postsynaptic inhibition may consist in the early period of second phase of this reflex suppression. Simultaneously, the strychnine experiments also showed that the mechanism of the first suppression phase was postsynaptic inhibition in nature. The report by Goldberg (9) has shown that electrical stimulation of lingual nerve caused the induction of hyperpolarizing potentials, i.e. IPSP in masseteric motoneuron. Thus, activation of lingual nerve induced intense postsynaptic inhibition on masseteric MSR via motoneuron. After application of strychnine the intense postsynaptic inhibition was released, this releasing led the early phase of masseteric MSR suppression to become facilitatory (19). This was proved by the observation that when strychnine action gradually faded away the two phases of suppression reappeared. In this study the threshold stimulation was delivered to the lingual nerve and obvious suppression of the masseteric MSR was observed. From the report by Windle (27), there were 30% of large myelinated nerve fibers (9 μ or more in diameter) in lingual nerve and these fibers were recognized to have the low threshold excitability. It is to say that the excitation of low threshold lingual nerve lead to the suppression of masseteric MSR.

Activation of one nerve branch is known to induce excitability change in other passive nerves (1, 4, 5, 6,). In order to check that the suppression of masseteric MSR really induced by lingual nerve, the conduction of the lingual nerve was blocked at a portion proximal to the stimulation electrode by crush with a forceps. After the blocking the effect of conditioning stimulus was abolished completely. This experiment confirmed that the effect of the conditioning stimulus was really induced by lingual nerve but not by the excitability change of other nerves or by electrical spreading to other nerves.

The masseteric MSR evoked by stimulation of trigeminal mesencephalic nucleus was also tested by stimulation of ipslateral trigeminal spinal nucleus. Either single shock or 3 shocks of conditioning stimulus were delivered to the trigeminal spinal nucleus. The report by Nakamura *et al.* (17) suggested that with application of 3-5 cortical shocks, the massteteric MSR was suppressed more profoundly. In our experiment, with application of 3 shocks to trigeminal spinal nucleus the masseteric MSR was suppressed

more profoundly than single shock. The time course of masseteric MSR suppression induced by conditional stimulation of spinal nucleus made some difference to that induced by lingual nerve stimulation. There was only one phase of suppression. From the time course and the aspect of suppression, it was assumed that this suppression phase was postsynaptic inhibition in nature. According to the physiological and anatomical aspect (11, 25), published reports (9, 10, 17), and the study added in our experiment, we suggest that in the neuronal relation among the lingual nerve, spinal nucleus and motor nucleus of trigeminal nerve, the first phase is related with spinal nucleus and motor nucleus, but the second phase is caused by spinal nucleus, motor nucleus and lingual nerve. Our pharmacological observation in masseteric MSR greatly supported our assumption. However, the more detailed experimental research is necessary.

SUMMARY

The effects of ipsilateral lingual nerve and trigeminal spinal nucleus stimulation on the masseteric monosynaptic reflex were studied.

The stimulation of mesencephalic nucleus in ipsilateral masseteric nerve evoked two peaks of potential having a latency of 0.74 and 1.59 msec, respectively. The latter is the monosynaptic reflex potential which was suppressed by repeated mesencephalic stimulation at 2-5/sec, and the former is the antidromic potential which was not altered even in the situation of the stimulation frequency up to 100/sec.

The post- and presynaptic inhibition appeared when the low threshold stimulus was applied to the lingual nerve, however, the postsynaptic inhibition was revealed at the stimulation of trigeminal spinal nucleus.

Intravenous injection of strychnine caused the disappearrence of postsynaptic inhibition.

REFERENCE

1. BROCK, L. G., COOMBS, J. M., and ECCLES, J.C., The recording of potentials from motoneurones with an intracellular electrode, *J. Physiol.*, 117 (1952) 431-460.
2. DALE-SMITH, R., MARCARIAN, H. Q., and NIEMER, W. T., Direct projection from the masseteric nerve to the mesencephalic nucleus, *J. Comp. Neurol.*, 133 (1969) 495-502.
3. ECCLES, J. C., SCHMIDT, R., and WILLIS, W. D., Pharmacological studies on presynaptic inhibition, *J. Physiol.*, 168 (1963) 500-530.
4. ECCLES, J. C., KOSTYUK, P. G., and SCHMIDT, R. F., Central pathways responsible for depolarization of primary afferent fibers, *J. Physiol.*, 161 (1962) 237-257.
5. ECCLES, R. M., and LUNDBERG, A., Integrative pattern of Ia synaptic actions on motoneurones of hip and knee muscles, *J. Physiol.*, 144 (1958) 271-298.
6. ECCLES, J. C., ECCLES, R. M., and LUNDBERG, A., The convergence of monosynaptic excitatory afferents on to many different species of alpha motoneurones, *J. Physiol.*, 137 (1957) 22-50.
7. ECCLES, J. C., *The Physiology of Synapses.*, Berlin: Springer, 1964.

8. ERICKSON, R. P., KING, R. L., and PFAFFMAN, C., Some characteristics of transmission through spinal trigeminal nucleus of rat, *J. Neurophysiol.*, 24 (1961) 621-632.
9. GOLDBERG, L. J., Some evidence for the reciprocal nature of lingual nerve afferent influences on trigeminal and hypoglossal motoneurons, *Anat. Rec.*, 157 (1967) 248.
10. GOLDBERG, L. J., Excitatory and inhibitory effects of lingual nerve stimulation on reflexes controlling the activity of masseteric motoneurons, *Brain Research.*, 39 (1972) 95-108.
11. HUGELIN, A., ET BONVALLET, M., E'tude e'lectrophysiologique d'un re'flexe monosynaptique trige'minal, *C. R. Soc. Biol. (Paris)*, 150 (1956) 2067-2071.
12. JERGE, C. R., Organization and function of the trigeminal mesencephalic nucleus. *J. Neurophysiol.*, 26 (1963) 379-393.
13. KIDOKORO, Y., KUBOTA, K., SHUTO, S., and SUMINO, R., Reflex organization of cat masticatory muscles, *J. Neurophysiol.*, 31 (1968) 695-708.
14. McINTYRE, A. K., Afferent limb of the myotatic of reflex arc, *Nature (Lond)*, 168 (1951) 168-169.
15. MILLER, F. R., and SHERRINGTON, C. S., Some observations on the buccopharyngeal stage of reflex deglutition in the cat, *Quart. J. of Exp. Physiol.*, 9 (1915) 147-186.
16. NAKAMURA, Y., and WU, C. Y., Presynaptic inhibition of jaw-opening reflex by high threshold afferents from the masseter muscle of the cat, *Brain Research.*, 23 (1970) 193-211.
17. NAKAMURA, Y., and GLODBERG, L. J., and CLEMENTE, C. D., Nature of suppression of the masseteric monosynaptic reflex induced by stimulation of the orbital gyrus of the cat, *Brain Research.*, 5 (1967) 184-198.
18. SAUERLAND, E. K. and MIZUNO, N., Effect of trigeminal proprioceptive input on the linguo-hypoglossal reflex, *Anat. Rec.*, 163 (1969) 322-323.
19. SCIBETTA, C. J., and KING, R. B., Hyperpolarizing influence of trigeminal nucleus caudalis on primary afferent preterminals in trigeminal nucleus oralis, *J. Neurophysiol.*, 32 (1969) 229-238.
20. SHERRINGTON, C. S., Reflexes elicitable in the cat from pinna vibrissae and jaw, *J. Physiol.*, 51 (1917) 404-431.
21. SHMIDT, R. F., The pharmacology of presynaptic inhibition, *Progress in Brain Research*, Vol. 12, Elsevier, Amsterdam, 1964, pp. 119-131.
22. SNIDER, R. S., and NIEMER, W. T., A stereotaxic atlas of the cat brain, The Univ. of Chicago Press. 1961.
23. STEWART, D. H., JR., SCIBETTA, C. J., and KING, R. B., Presynaptic inhibition in the trigeminal relay nuclei, *J. Neurophysiol.*, 30 (1967) 135-153.
24. STWART, D. H., JR., and KING, R. B., Effect of conditioning stimuli upon evoked potentials in the trigeminal complex *J. Neurophysiol.*, 29 (1966) 442-455.
25. SZENTA'GOTHAI, J., Anatomical consideration of monosynaptic reflex arcs, *J. Neurophysiol.*, 11 (1948) 445-454.

26. WALL, P. D., Excitability changes in afferent fiber terminations and their relation to slow potentials, *J. Physiol.*, 142 (1958) 1-21.
27. WINDLE, W. F., The distribution and probable significance of unmyelinated nerve fibers in the trigeminal nerve of the cat, *J. Comp. Neurol.*, 41 (1926) 453-477.

中 文 摘 要

舌神經及三叉神經脊髓核對閉口反射機構之作用

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高等動物在咀嚼運動時，舌及顎二者之間，需有極密切的協同作用才能圓滿達成。今以猫的舌神經及三叉神經脊髓核爲對象，研究其興奮時對閉口反射的作用。

刺激三叉神經中腦核，在咬肌神經可紀錄到雙峯的動作電位，其潛伏期（latency）分別爲0.74 及1.59 msec. 第二峯電位爲通過單突觸的咬肌神經反射（masseteric monosynaptic reflex），此反射可被2-5次/秒的反複刺激所抑制。而第一峯電位爲逆行性電位（antidromic potential），此電位並不因刺激頻率增加到100次/秒而有所改變。

給于舌神經低閾（low threshold）的刺激對咬肌神經反射產生突觸前（presynaptic）及突觸後（postsynaptic）抑制作用。而三叉神經脊髓核的興奮對咬肌神經反射只產生突觸後抑制作用。

靜脈注射番木鼈素（strychnine）後，突觸後抑制作用消失。