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MODULATION OF THE JAW JERK REFLEX BY THE SYMPATHETIC NERVOUS SYSTEM

C. GRASSI[1], F. DERIU[2], E. ARTUSIO[2] AND M. PASSATORE[2]

[1] *Istituto di Fisiologia Umana, Università Cattolica del Sacro Cuore, Largo F. Vito 1, 00168 Roma and* [2] *Dipartimento di Anatomia e Fisiologia Umana, Università di Torino, Corso Raffaello 30, 10125 Torino, Italy*

INTRODUCTION

The jaw jerk reflex (JJR) is the stretch reflex elicited in the jaw closing muscles by a tap delivered to the mandible. Unlike limb monosynaptic reflexes, the JJR exhibits a large variability in magnitude among individuals and within the same subject (8). This indicates that motoneurons innervating masticatory muscles receive more fluctuating inputs, in comparison with motoneurons supplying limb muscles.

Several morphological and functional data have been reported in the literature which could contribute to identifying the factors responsible for the variability of this reflex. These findings show that input arrangement to jaw-elevator motoneurons, and in general the neuronal circuitry involved in jaw movements, significantly differ from that of limb muscles for numerous significant characteristics (15, 16, 26, 34). In particular, individual muscle afferents have been reported to project only to a «small proportion» of the total population of jaw-elevator motoneurons (4) and it has been recently confirmed that no equivalent of Renshaw inhibitory pathway exists in this territory (47). In addition it should be mentioned that reticular neurons heavily impinge on the soma of both the sensory and the motor nuclei involved in the JJR and, with regard to the motor command, many projections of the sensorimotor cortex reach the masticatory motoneurons through reticular neurons (24, 25, 31, 46). Then, somatic and visceral sensory inputs should be able to modulate this reflex by modifying the level of activity of the reticular formation. At variance with spinal reflexes, such a modulation can be exerted not only at the motoneuron level but also on the soma of the afferent neurons (11, 13, 14, 28-30, 32, 36, 43). Furthermore, central mechanisms capable of controlling the afferents from peripheral receptors have been reported in this territory, in analogy to what happens for other sensory inputs. In fact the spindle afferent activity from jaw elevator muscles is modulated by corticofugal signals from the cerebral masticatory area, through an action exerted on the mesencephalic trigeminal nucleus (27, 36).

Finally, another modulatory influence on reflexes has to be considered, that is the one exerted by the noradrenergic neurons. There is ample evidence that the monoaminergic pathways originating in the locus coeruleus, subcoeruleus and nucleus parabrachialis can affect the motor function by acting on different central

nervous system structures responsible for, or involved in, motor control (45, ref. in 3, 20). In addition, data are accumulating in the literature indicating that the sympathetic nervous system can influence the skeletal muscle function through an action exerted at the peripheral level. In particular, activation of the sympathetic command has been reported to modify the afferent information from muscle spindles (21, 38, 41). Some changes in the tonic vibration reflex and in the phasic stretch reflex have been also reported in cat hindlimb muscles following both catecholamine administration and lumbar sympathetic chain stimulation (18, 42). With regard to the trigeminal district we have shown that activation of the sympathetic system, at frequencies within the physiological range, can influence both the afferent information from neuromuscular spindles located in the jaw elevator muscles and the contraction of the same muscles (38-41).

We are currently trying to evaluate how these two effects produced by sympathetic activation do integrate in a simple motor action. In this context data have been reported showing that sympathetic stimulation depresses the tonic vibration reflex in the rabbit masseter muscle (19); in the present study we investigated the effects produced by the same stimulation on the jaw jerk reflex.

METHODS

The experiments were performed on 20 rabbits (weight 2.5-3.5 kg) anesthetized with urethane, ketamine and xylazine (0.4 g/kg, 5 mg/kg and 1.5 mg/kg respectively, i.v.). This initial dose was supplemented by continuous infusion of adequate doses of the last two drugs, in the same above reported proportions, through a cannulated femoral vein (Terumo Syringe Pump, STC-S21). Anesthesia was discontinued after the animals underwent precollicular decerebration. However, since in most occasions spontaneous movements occurred and/or the variability of the reflex was high, small doses of either ketamine or of ketamine and xylazine were administered by continuous infusion.

The animal's skull was fixed in a stereotaxic frame by using screws distributed over the frontal bone. The cervical sympathetic nerve (CSN) was bilaterally isolated and sectioned; its peripheral stump was placed on platinum stimulating electrodes mounted 2 mm apart inside a 2 cm long polyethylene cylinder filled with mineral oil and surrounded by blotting paper embedded with oil for further insulation. Then the muscles and skin of the neck were sewn. Stimulation of the CSN was performed at 10/s (0.5 ms pulse duration, 4-8 V). In 3 experiments the CSN was also inserted into a small T-shaped polyethylene catheter, positioned about 2 cm peripherally to the stimulating electrodes; thus the conduction of impulses along the nerve could be blocked by injecting 2% lidocaine solution through the vertical tube. In all experiments pupillary dilatation was evaluated to have information on the effectiveness of the CSN stimulation.

The jaw jerk was elicited by a downward mandibular displacement of 1-2 mm (at a speed of 0.1-0.2 mm/ms) maintained for 0.5-1 s, repeated every 4-6 s. This mechanical stimulus was delivered through an electromagnetic puller (Ling 200 driven by a function generator Noselab, Milan) rigidly connected with the lower jaw through a screw fixed to the mandibular symphysis with cold-curing resin. A feed-back control system which utilized the length signal of jaw position (inductive proximity sensor Selet B18/5) allowed the jaw lowering movement to be constantly reproduced. Mechanical stimuli of trapezoidal shape were chosen, rather than brief pulses simulating a chin-tap, in order to avoid that

the raising phase of the mandible occurred during the development of the reflex contraction, thus making its evaluation difficult. In addition, the stimulus employed allowed us to detect the tonic component of the reflex which took place during the holding phase of the movement. Force developed by jaw muscles of both sides was measured through an isometric transducer (subminiature load cell M 11, R.D.P. Electronic Ltd.) placed in series with the puller. This signal was low-pass filtered (25-50 Hz) in order to cancel the initial transient force oscillation produced by the short-range stiffness of the muscle and by the mass of the mandible, during the imposed jaw movement. This filtering was useful to avoid that the tension development of reflex origin, due to its short latency, were deformed by the above mentioned force oscillation. The reflexly induced force was however inscribed on the passive tension changes induced by the jaw displacement, which could be evidenced after both the tonic and the phasic JJRs had been suppressed by rapid injection of anesthetic (Fig. 1). Such evaluation of passive tension agreed well with the one which was obtained at the end of each experiment after the animal had been curarized (Tubarine, Wellcome, 0.3 mg/kg, i.v.). The amount of the active tension developed by the stretch reflex for a given jaw lowering was determined by subtracting the passive tension from the total recorded force. After the above mentioned anesthetic administration, 20 to 30 min were allowed before performing the sympathetic stimulation trials.

EMG activity from the masseter muscle and occasionally from temporalis and digastric muscles was bilaterally recorded through gross «belly-tendon» copper leads insulated except for the tip. In most experiments a bipolar coaxial electrode was also inserted into the masseter muscle and slowly moved to find the activity of few or single motor units (amplifier bandpass 10 Hz-3 kHz).

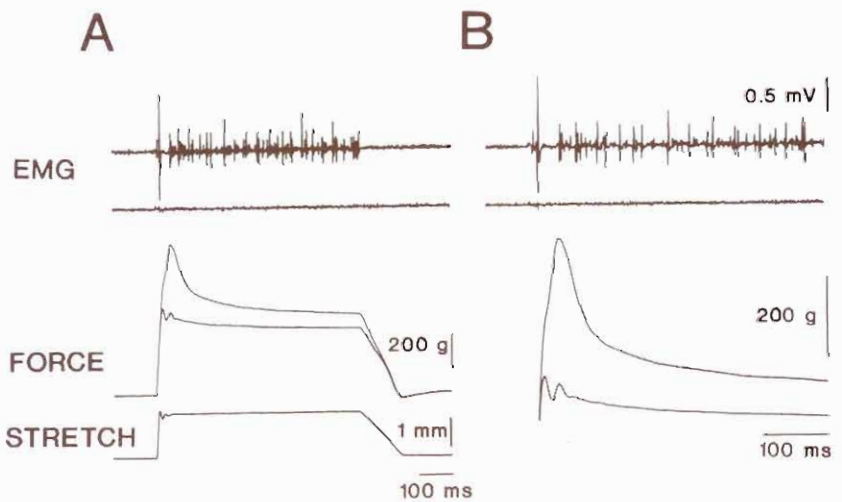

Fig. 1 – *Method used to elicit and to evaluate the JJR, in a decerebrate rabbit.*

Comparison between the effects induced by the downward mandibular movement before and after anesthetic administration (ketamine 1 mg/kg plus xylazine 0.3 mg/kg, i.v.) which abolished the reflex response to stretch. In A and B, from top to bottom: EMG activity from the right masseter muscle before and after administration of the anesthetic, superimposed traces of jaw muscle force before and after anesthesia, stretch of the jaw elevator muscles (upward deflections correspond to increase in force and jaw lowering, respectively). Force recorded after having suppressed the reflex response gives a good indication of the passive tension developed during the jaw lowering. Therefore the difference between the two superimposed tension records allows to evaluate the magnitude of both the phasic and the tonic components of the reflex (see also Methods). In B, identical traces shown in A are displayed with different calibrations. The tension signals are low-pass filtered at 25 Hz.

These trials were routinely carried out at mandibular positions which corresponded to an interincisal distance of 2-4 mm, occasionally wider openings up to 12 mm were utilized. Some trials were performed before the rabbits underwent precollicular decerebration; under this condition the above mentioned stretch parameters were able to elicit a JJR, provided the animal was lightly anesthetized.

In another series of experiments, performed on 5 rabbits, the neuromuscular junctions were blocked by administration of tubocurarine (Tubarine, Wellcome, 0.3 mg/kg, i.v. repeated as needed); these animals were then artificially-ventilated so as to maintain the end-tidal CO_2 to the precurarization level (capnometer Engström Eliza Duo). The masseter muscle was electrically stimulated with single pulses through a pair of Ag-AgCl electrodes sewed to the muscle (0.1/s, 0.5 ms pulse duration, 10-20 V) and the force developed by the contraction was recorded as described above. Stimulation of the CSN was performed as in the previous series of experiments.

In 3 rabbits a plastic snare was placed around each common carotid artery so that they could be temporarily occluded. In 5 animals an α-adrenergic blocking agent (phentolamine, Regitin, Ciba, 1.6-3.5 mg/Kg i.v.) was administered.

In all experiments heart rate was routinely monitored and chest movements recorded through an inductive proximity sensor (Selet B18/5) to have information on whether cardiac and respiratory rhythmicity changed or movements occurred, which often happens in decerebrate rabbits. The results reported below refer to trials in which these parameters did not exhibit significant changes. The animal body temperature was maintained at 38°C through an electric blanket regulated by feedback from a rectal probe (Harvard).

All signals were recorded on a polygraph (Gould ES1000 equipped with SP 400, SP 100 and SP 110A preamplifiers) and stored on a magnetic tape for further analysis. The EMG and the force signals are presented either as single sweeps or as averages of 5 to 15 sweeps (Tektronix 2230 digital storage oscilloscope, plotted on Tektronix HC100).

RESULTS

A rapid downward displacement of the mandible reflexly induced EMG activation in the jaw closing muscles (minimum latency of 5 ms) associated with a force development having the general appearance of a muscle twitch. Under our experimental conditions, a tonic stretch reflex was often observed following the phasic response, which consisted of both EMG activation and tension development lasting as long as the holding phase of the imposed movement. A silent period, i.e. transitory interruption of the EMG activation, could be observed between the tonic and the phasic components of the reflex.

The amplitude of the JJR increased by enhancing the speed and the size of the imposed movement. When stimuli just above threshold were employed, the developed reflex exhibited a low spontaneous variability, while high-speed and large-magnitude stimuli elicited large but unstable JJRs. The movement parameters which evoked the largest magnitude reflexes with an acceptable stability were usually selected. Under these experimental conditions, the tension developed by the phasic and the tonic stretch reflexes respectively ranged from 20 g to 400 g and from a few grams up to 210 g, depending on the stretch parameters and on the arousal level of the animal.

Unilateral stimulation of the peripheral stump of the CSN at 10/s (trains lasting

2-4 min) consistently caused a marked decrease or disappearance of the ipsilateral EMG signals together with a 30%-40% reduction of the reflexly developed muscle tension (Figs. 2, 3). When electrical activity was recorded from a few motor units, the compound potential progressively decreased within the first minute of sympathetic stimulation, due to the asynchronous dropping of the different units composing such potential (Fig. 2). The latency of the reflex was not affected by the CSN stimulation. EMG activity of the masseter muscle contralateral to the stimulated CSN, which was used as control, did not show significant changes (Fig. 3).

Bilateral sympathetic stimulation markedly reduced or suppressed the EMG activity in the masseter muscles of both sides. Correspondently the force developed by the reflex was markedly reduced, reaching values which ranged between 12.5 and 37.0% of controls, with an average of 28.9% ± 8.9 S.D. (Figs. 3 and 4).

The sympathetically-induced decrease in the JJR appeared with a latency of 20 to 40 s from the stimulus onset in the different preparations and it was often preceded by a modest transitory enhancement. Both EMG and tension returned to control values 1 to 2 min after CSN stimulation was discontinued, a faster recovery usually occurring when short-lasting sympathetic stimulations had been employed. A transient increase of the JJR usually preceded the return to control values, as shown by a small enhancement of the developed tension and recruitment

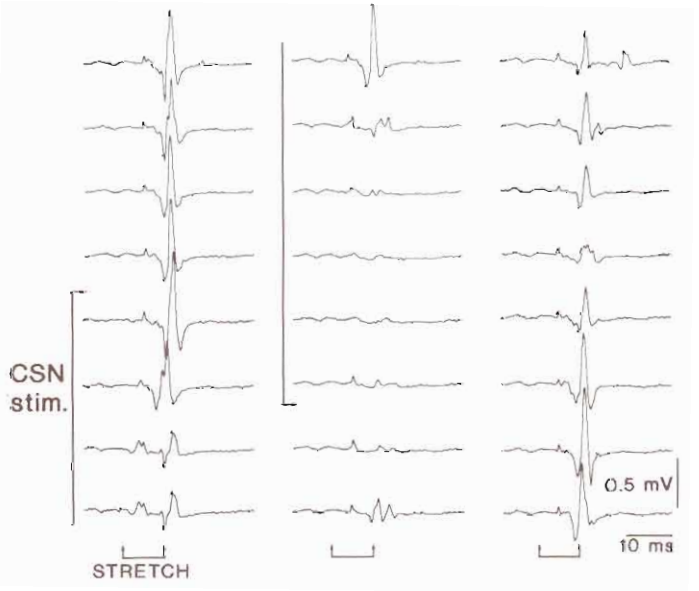

Fig. 2 – *Effect of CSN stimulation on the phasic component of the jaw jerk reflex induced by stretch of the jaw elevator muscles.*

Compound potential due to synchronous discharge of a few motor units is recorded from the masseter muscle; single sweeps are sampled every 10 s. Stimulation of the CSN at 10/s (0.5 ms pulse duration, 6 V_n signalled with bars) produces the progressive disappearance of the different units composing the action potential.

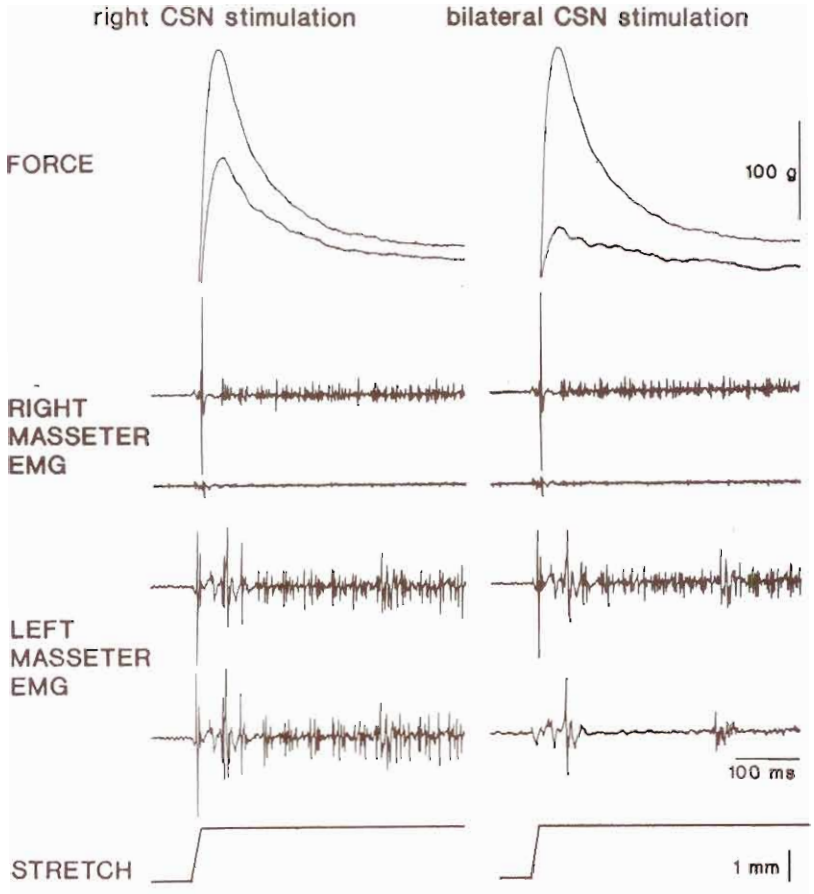

Fig. 3 – *Effect of unilateral and bilateral CSN stimulations on both the phasic and the tonic components of the JJR.*

In unilateral stimulation trials, the EMG contralateral to the stimulated side is used as control. The upper portion of the jaw muscle force records is shown as in Fig. 1B; control and CSN stimulation trials superimposed. In each couple of EMG signals data collected before (upper traces) and during sympathetic stimulation (lower traces) are shown. Each trace is the average of 7 sweeps. In the right CSN stimulation trial, the decrease in the reflexly developed force is accompanied by the disappearance of ipsilateral EMG activity while EMG signals in the contralateral masseter muscle are unaffected. Bilateral CSN stimulation markedly reduces jaw muscle force as well as EMG activity on both sides.

of new motor units. Similar data have been also obtained when sympathetic stimulations lasting 5-10 min were employed. In these trials the maximum reduction of the reflex response was reached within 1-2 min from the stimulus onset, no further decrease occurring in the next minutes of stimulation.

The sympathetically-induced effects on both EMG and tension were also observed when the stretch reflex was elicited in the non-decerebrate animal, the tension values being lower in this last condition.

It was then checked whether the vasoconstriction induced in the jaw muscles by CSN stimulation could be responsible for the observed reduction of the JJR.

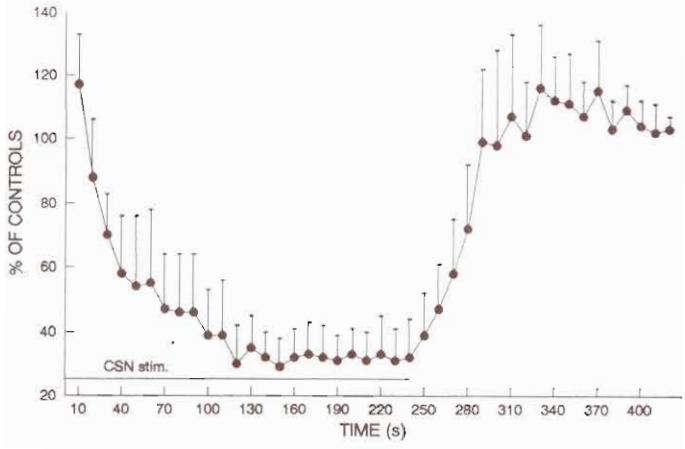

Fig. 4 – *Time course of the effect of bilateral CSN stimulation on tension developed by the JJR.* Data collected in 10 experiments are plotted as mean values and standard deviations.

In order to test this possibility, we evoked the reflex while a large sudden reduction or the interruption of the blood supply to the muscles was respectively produced by unilateral and bilateral occlusion of the common carotid artery. The carotid occlusion was maintained for 3 to 5 minutes while the reflex was being elicited at the usual rate; no decrease in the JJR was detected in these trials.

We also checked whether the sympathetically-induced reduction in the JJR could be partly due to a simultaneous activation of the jaw lowering muscles. In order to define this, the effect of CSN stimulation on the JJR was also tested in 3 rabbits in which the central tendon of the digastric muscle, i.e. the main jaw lowering muscle, had been bilaterally sectioned. The comparison between these data and those obtained in animals with all jaw muscles intact did not show significant differences.

In 2 experiments, after having checked that sympathetic stimulation was effective, i.e. it induced the usual depression of the JJR as well as pupillary dilatation, the impulse conduction along the CSN was blocked by local administration of lidocaine (see Methods). Under this condition the CSN stimulation failed to elicit any change in the JJR as well as in the pupillary diameter.

Since in these experiments the sympathetic stimulation produced a very marked reduction of the reflex, we verified whether an action exerted by the adrenergic mediator on the muscular contraction could concur in decreasing the developed tension. For this reason the effect of sympathetic stimulation was tested on the masseter muscle contraction induced by direct electrical stimulation in both curarized and non-curarized animals.

Single-pulse stimulation of the masseter muscle induced a twitch response whose amplitude was stable over time, particularly in curarized animals. Stimulation of the CSN at 10/s consistently elicited an increase in both amplitude and duration

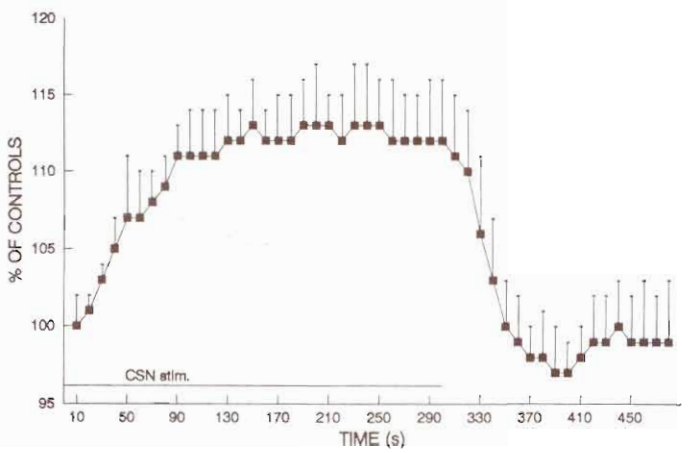

Fig. 5 – *Effect of CSN stimulation on the force developed by twitch contractions of the directly stimulated masseter muscle.*

Data are plotted as mean values and standard deviations.

of the muscular response. This effect started 10-20 s from stimulus onset and, within the next 1.5-3 min, reached a maximum increment of 10%-15% of the control values, which was maintained as long as the stimulation was continued (Fig. 5).

Administration of an α-adrenergic blocking agent (phentolamine, 1.6-3.5 mg/kg, i.v.) almost completely abolished the effect of sympathetic stimulation described above, i.e. both the depressant action exerted on the JJR and the activatory action on the masseter muscle contraction elicited by direct stimulation of the muscle.

DISCUSSION

Bilateral stimulation of the CSN, at frequencies within the physiological range, markedly depressed the jaw jerk reflex whose amplitude reached a mean value of 28.9±8.9 S.D.% of controls. The sympathetic origin of such an effect is shown by its disappearance after having interrupted the impulse conduction along the stimulated CSN, as well as after the blockade of α-adrenergic receptors. In unilateral sympathetic stimulation trials, the EMG activity of the jaw closing muscles contralateral to the stimulated CSN was not significantly modified. This finding proves that the depression of the reflex is neither due to uncontrolled changes in the mechanical stimulus, nor to aspecific events occurring during sympathetic activation. Moreover, this effect is not secondary to the vasomotor changes occurring during sympathetic stimulation since bilateral occlusion of the common carotid artery, a manoeuvre which in the decerebrate animal interrupts the blood flow to the jaw muscles, fails to elicit a reduction in the JJR.

Therefore, we considered the possibility that the marked depression of the reflex

response was due to an action directly exerted by the adrenergic mediator on the spindle receptor or/and on the muscular effector. The latter possibility was discarded on the basis of both data collected in the present work and literature reports. In fact, CSN activation caused a modest enhancement in both amplitude and duration of the twitch contraction elicited by electrical stimulation of the masseter muscle. This finding is also consistent with recent data showing a sympathetically-induced increase of the contractile force in the rabbit digastric muscle (40). Such an effect was observed in contractions elicited by stimulating either the muscle directly or discrete areas of the mesencephalic trigeminal nucleus, containing the afferent neurons of periodontal mechanoreceptors, whose stimulation reflexly activates the muscle. On the other hand, a sympathetically-induced decrease in the contractile force was not to be expected in the jaw muscles which are mainly composed of fast-contracting fibers (50). It has been shown in fact that administration of catecholamines, at doses up or above the physiological limits, elicits a reduction of the twitch tension only in slow-contracting limb muscles, whilst an increase has been observed in fast-contracting muscles (ref. in 9).

Therefore, since the adrenergic mediator does not reduce the contractile force of the jaw muscles, the cause of the JJR decrease should be sought in a sympathetically-induced modification of the spindle afferent information. Functional data have been reported in the literature suggesting that the sympathetic system exerts a direct action on neuromuscular spindles in several muscle territories (18, 21, 22, 38, 41, 42). In masticatory muscles, sympathetic stimulation, at frequencies within the physiological range, has been shown to induce a global increase in the firing rate of spindle afferents. Preliminary data suggest that CSN stimulation also produces, in the same territory, a marked decrease in the spindle sensitivity to muscle length changes, evaluated under both static and dynamic conditions (38, 39). Such a decrease in spindle sensitivity has been considered responsible for the marked reduction of the tonic vibration reflex produced by CSN stimulation in the jaw elevator muscles (19). In cat hindlimb muscles, sympathetically-induced threshold changes of spindle receptors to stretch have also been reported, consisting of a modest transitory reduction, followed by a marked increase lasting throughout the stimulation (21). A direct action of the sympathetic system on muscle spindles is also suggested by morphological data indicating the presence of noradrenergic terminals, reportedly unrelated to blood vessels, within the spindle capsule and also in «neuroeffective association» with both bag and chain intrafusal fibers, in cat hindlimb muscles (6). In the rat masseter muscle, 35% of muscle spindles has been found to receive sympathetic innervation (5).

All these data suggest that an action directly exerted by the adrenergic mediator on muscle spindles is likely to be responsible for the above described marked decrease in the stretch reflex. However, the discrepancy between the number of spindles reported to receive adrenergic innervation and the marked reduction of the JJR remains to be explained, assuming that masseter muscle of rabbits and rats have a similar proportion of sympathetically-innervated spindles. In addition, the same morphological studies report an abundant adrenergic innervation of ex-

trafusal fibers, which could be responsible for the small potentiation of the muscle contraction elicited by sympathetic stimulation.

Even though the above commented data point to the responsibility of spindle afferents in the sympathetically-induced reduction of the JJR, it should be considered that other afferent inputs, modulated by the sympathetic activation (39, ref. in 2), may contribute to such reduction. The receptors that are more liable than others to reduce the JJR are those whose activation elicits a reflex jaw opening. Contrasting data have been reported concerning the sympathetic influence on the activity of single neurons innervating periodontal receptors (10, 37), while a marked inhibitory action has been described on the jaw opening reflex elicited by tooth-tapping (1). Our experimental protocol did not allow us to evaluate whether CSN stimulation reduced this reflex; however, we could exclude that a significant activation of the jaw opening reflex, able to partially counteract the JJR, occurred in our experimental conditions since the magnitude of the sympathetically-induced depression of the JJR was not modified by the bilateral section of the digastric muscle.

To summarize, the above reported data show that activation of the sympathetic system produces a marked depression of the JJR, which is likely to be due to the previously reported decrease in spindle sensitivity to muscle length changes. This means that, during the execution of a movement, activation of sympathetic outflow should reduce the feed-back control of muscle length. In addition, the data reported here and in previous work evidence that sympathetic activation can potentiate muscle contraction through: i) an action exerted directly on the contractile mechanism; ii) an increase of spindle afferent discharge which should reflexly provide an additional drive to α-motoneurons. Then the global modulatory influence exerted, at peripheral level, by the sympathetic system on the skeletal muscle function would depend on the relative weight of the central command versus the peripheral afferent input in a given motor action.

Furthermore, as already mentioned in the Introduction, central monoaminergic pathways can modulate the stretch reflexes. In particular, stimulation of locus coeruleus has been shown to produce a noradrenaline-mediated enhancement of monosynaptic spinal reflexes. Noradrenergic projections are reported to facilitate both extensor and flexor monosynaptic reflexes (12, 49) as well as to depress the transmission from group II muscle afferents to spinal interneurons intercalated in the reflex pathways to motoneurons (23). In addition, microinfusion of noradrenaline into the trigeminal motor nucleus produced a dose-dependent increase of the masseteric reflex elicited by electrical stimulation of the mesencephalic trigeminal nucleus (48).

All the above commented data suggest that noradrenergic neurons may play a modulatory role on the stretch reflex by acting at all levels of the reflex loop. Such actions would confer on the sympathetic nervous system the ability of both potentiating and inhibiting the reflex response, depending on the site of action. On the basis of data showing a differential activation of the various sections of the sympathetic nervous system, as well as of the little information available concerning the central monoaminergic pathways (7, 17, 33, 35, 44, ref. in 20),

it may be hypothesized that numerous conditions, such as emotional states, level of arousal, exercise etc., create differential modifications of the outflow in the various catecholaminergic pathways. Consequently, a change of the sympathetic command associated with the above mentioned physiological conditions could be expected to produce a wide range of modulatory actions on the motor function.

SUMMARY

The effect of sympathetic stimulation on the jaw jerk reflex has been studied in precollicular decerebrate rabbits. This reflex was elicited by a downward mandibular movement applied to the lower jaw through a servo controlled puller.

Unilateral stimulation of the cervical sympathetic nerve at 10/s consistently induced a decrease in the JJR, i.e. a marked reduction of the EMG activity in the ipsilateral masseter muscle, accompanied by a 30-40% decrease in the reflexly developed force. In these trials EMG of the contralateral muscle, recorded as control, was not significantly affected. Bilateral stimulation of cervical sympathetic nerve strongly reduced or suppressed the EMG activity in both sides and produced a parallel decrease in the developed force which reached values ranging from 12.5% to 37.0% of controls (with an average of 28.9% ± 8.9, S.D.).

The effect of sympathetic stimulation was also tested on the contraction of the masseter muscle elicited by direct electrical stimulation. Sympathetic activation induced a modest increase in both amplitude and duration of muscle twitch, thus showing that the reduction in the reflex response can not be attributed to an action exerted by the adrenergic mediator on the muscular contraction.

All these effects were almost completely abolished by the blockade of α-adrenergic receptors. They were proved not to be secondary to the sympathetically-induced vasomotor changes.

Therefore the marked JJR reduction produced by activation of the sympathetic nervous system is suggested to be due to the sympathetically-induced decrease in neuromuscular spindle sensitivity to muscle length changes, previously reported.

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