J. Physiol. (1982), **329**, *pp.* 451–463
With 6 text-figures
Printed in Great Britain

EFFECTS OF SYMPATHETIC NERVE STIMULATION ON INTRA-ORAL MECHANORECEPTOR ACTIVITY IN THE CAT

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(*Received 3 February 1982*)

SUMMARY

1. Micro-electrode recordings were made from intra-oral mechanoreceptor neurones in the trigeminal ganglion and mesencephalic nucleus of the fifth nerve in the cat.

2. The effect of cervical sympathetic trunk stimulation on the discharge of the mechanoreceptors to a controlled force was observed.

3. Almost half of the mechanoreceptor neurones studied were modulated by sympathetic stimulation. Sympathetic stimulation both decreased the impulse frequency to a controlled force application and raised the threshold in these receptors. The remainder were unaffected by sympathetic stimulation.

4. In previous studies involving recordings from either the trigeminal ganglion or mesencephalic nucleus no spontaneously active slowly adapting intra-oral mechanoreceptors have been observed, yet in peripheral alveolar nerve studies many workers have reported spontaneously discharging neurones. In the present study spontaneous activity has been recorded from intra-oral mechanoreceptors both in the trigeminal ganglion and in the mesencephalic nucleus after cutting the cervical sympathetic trunk. The spontaneous activity could be abolished in all cases but one, by sympathetic stimulation. It is suggested that reported spontaneous activity from intra-oral mechanoreceptors could be due to cutting the peripheral nerve and therefore the sympathetic supply, thus removing an inhibitory influence from the sensory unit. The means by which the sympathetic supply inhibits the sensory unit has not been resolved.

INTRODUCTION

Activity from periodontal mechanoreceptors has been recorded in functionally single axons dissected from peripheral nerves in a number of studies (Pfaffmann, 1939; Ness, 1954; Wagers & Smith, 1960; Suzuki, 1963; Matthews, 1965; Hannam, 1969*a*, *b*, 1970; Sakada & Onodera, 1974). From the results of these studies, periodontal mechanoreceptors have been divided into three groups: rapidly adapting, slowly adapting and spontaneously discharging. Spontaneously discharging neurones exhibit all the characteristics of slowly adapting periodontal mechanoreceptors but they also produce a spontaneous discharge in the absence of any overt stimulation to the teeth.

Cell bodies of primary afferent periodontal mechanoreceptor neurones are found

in both the trigeminal ganglion and the mesencephalic nucleus of the fifth nerve. Micro-electrode recordings from these two areas have identified rapidly adapting and slowly adapting neurones but have failed to find spontaneously discharging neurones (Jerge, 1963; Kerr & Lysak, 1964; Beaudreau & Jerge, 1968; Linden, 1978).

It has been suggested either that the cell bodies of the spontaneous neurones are somewhere other than the trigeminal ganglion or mesencephalic nucleus, or that the spontaneous activity recorded peripherally is an experimental artifact, due to some technical or physiological differences in the experimental conditions between central and peripheral studies (Linden, 1978).

The technique for recording from functionally single axons dissected from the relevant peripheral nerve has, in all the studies quoted above, involved cutting the main nerve bundle, and this means that the sympathetic supply to the teeth is also cut (Ogilvie, 1969; Matthews, 1976; Anderson & Linden, 1977; Matthews & Robinson, 1980). It has been reported that in a group of ten intra-oral mechanoreceptor neurones the response to a force applied to the teeth was reduced following sympathetic stimulation (Anderson & Linden, 1977).

The purpose of this investigation was to examine more fully the modulation of periodontal mechanoreceptor activity by sympathetic stimulation.

METHODS

Twenty-nine adult cats 2·0–3·5 kg in weight, anaesthetized with sodium pentobarbitone (initial dose not exceeding 45 mg kg^{-1} I.P.; maintenance dose of 3 mg kg^{-1} I.V.) were used. The cats were maintained at a light anaesthetic level, at which the flexion withdrawal reflex could just be elicited. The body core temperature was maintained thermostatically at $37 \pm 0{\cdot}2$ °C with an electric blanket using feed-back from a thermistor probe inserted into the peritoneal cavity.

The trachea was cannulated and all animals were artificially ventilated with 40% oxygen. Respiratory minute volume was adjusted using a Starling Ideal pump and the end-tidal CO_2 was monitored and maintained between 3·5 and 4·5%. The femoral artery was cannulated in all the experiments and the lingual artery cannulated in some of the experiments. Blood pressure was monitored constantly and the experiments were terminated if the mean arterial pressure fell below 10 kPa (75 mmHg). The animal's head was held in a stereotaxic frame in such a manner as to facilitate access to intra-oral structures (Linden, 1978). The sympathetic nerves were dissected out in the neck using a dorsal approach. Both the right and left cervical sympathetic chains were cut. The peripheral end of the left sympathetic chain was placed on platinum stimulating electrodes in a liquid paraffin pool. An isolated constant voltage stimulator (Digitimer) was used to provide square wave shock stimuli to the peripheral end of the sympathetic trunk (1–5 V, 1 ms duration, at frequencies between 1 and 20 Hz). Loops of thread were placed around both common carotid arteries, in order to study the effects of carotid occlusion.

A flap of skin overlying the left side of the cranium was reflected; the temporalis muscle separated from the skull and reflected laterally. A 1–2 cm^2 craniotomy was performed using a dental drill. All the cut surfaces of the bone were sealed with bone wax to stop bleeding and prevent air emboli.

Access was made to the left trigeminal ganglion by gently aspirating the left cerebral hemisphere through the craniotomy hole to expose the ganglion and then carefully removing the covering meninges. Recordings were made using glass insulated tungsten micro-electrodes driven into the ganglion under direct vision.

Recordings from the left mesencephalic nucleus were made using the same micro-electrodes and Horsley–Clark co-ordinates (A 4·0–P 4·0; 2·3 mm lateral to the mid line (Berman, 1969), as previously reported (Linden, 1978)).

In order to differentiate between periodontal and 'type P' (see Linden, 1978) mechanoreceptors in the mesencephalic nucleus recordings, the recording electrodes were stereotactically placed while stimulating electrically the inferior alveolar and palatine nerves at a frequency of 1 Hz (2–5 V,

200 μs duration). Inferior alveolar nerve stimulation was achieved with bipolar silver ball electrodes placed in contact with the left inferior alveolar nerve in the lower border of the left mandible. Two small bur holes (size $\frac{1}{2}$ round) were drilled through the lateral aspect of the mandible about 5 mm apart just distal to the molar teeth into the inferior alveolar canal. The ball electrodes were placed into these holes and held in place using dental acrylic. Similarly two small holes were drilled about 3 mm apart on the left side of the palate distal to the posterior palatine foramen and two silver ball electrodes were placed in contact with the left palatine nerve.

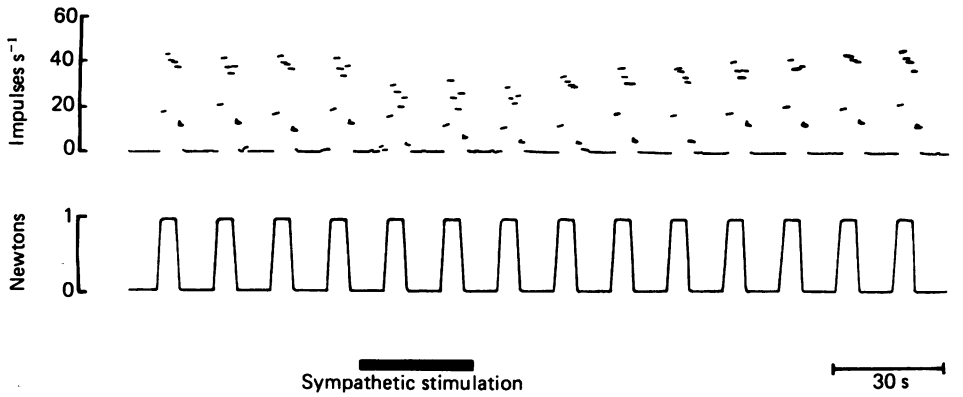

Fig. 1. Results from a modulated neurone recorded in the trigeminal ganglion. Lower record shows forces of 1 N being applied to the tooth once every 15 s. Upper record shows the number of impulses per second. Sympathetic stimulation was applied during the bar (5 V, 10 Hz, 1 ms duration).

Identification of periodontal mechanoreceptor neurones in the trigeminal ganglion was made by visually advancing the micro-electrodes into either the mandibular or maxillary division of the ganglion whilst manually stimulating the left mandibular or maxillary canine tooth.

Mechanical stimulation of the teeth was applied with an electromechanical force generator (Ling 100, 3 Ω) incorporating strain gauges to record the applied force. The force generator was attached to the tooth with a small amount of dental sticky wax so that it could be used to either push or pull on the tooth, and it was powered by a constant current Dynamometer 1189 (designed by System 696 Ltd., specifically for this laboratory) driven by a programmer (Digitimer). With this apparatus, constant ramp and plateau forces could be applied to the tooth with a fixed rate of rise, amplitude, duration, and rate of fall and these were monitored on a pen recorder and tape recorded. Usually forces of 1 N were applied to the tooth with a rise time of 1 s, held for 4 s and removed over a further 1 s. This sequence was repeated once every 15 s. Forces were always applied to the tooth in the direction of maximal sensitivity of the particular neurone being studied at the time. This was determined by applying controlled forces to the crown of the tooth until a maximum discharge of the receptor was achieved.

RESULTS

Trigeminal ganglion

A total of sixty-four periodontal mechanoreceptor neurones have been recorded from the trigeminal ganglion of eighteen cats. In thirty-two of these the response to a controlled force application fell either during or following a period of cervical sympathetic nerve stimulation. The remaining thirty-two neurones showed no detectable change in their rate of discharge.

In the total of sixty-four neurones, seven were spontaneously discharging perio-

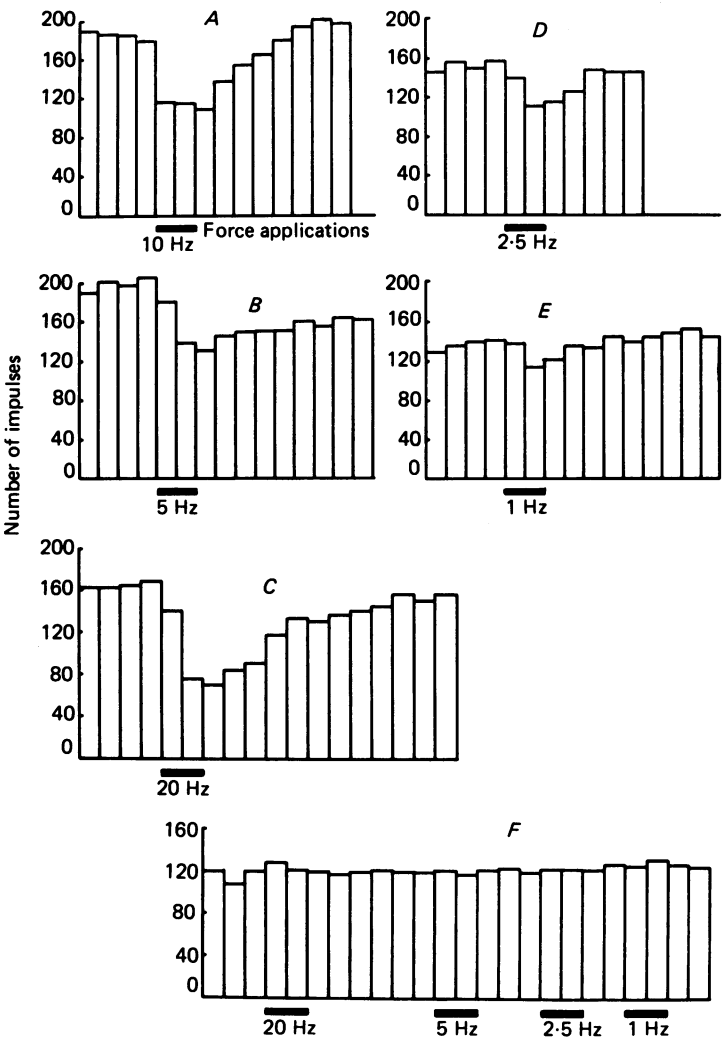

Fig. 2. The results when one modulated unit, recorded in the trigeminal ganglion, was subjected to sympathetic stimulation at different frequencies. Frequencies of sympathetic stimulation were applied randomly in the order: *A*, 10 Hz; *B*, 5 Hz; *C*, 20 Hz; *D*, 1·5 Hz; *E*, 1 Hz. All force applications were identical. Forces were applied to the teeth once every 15 s. Number of impulses shown by the histogram-like blocks represents the total number of action potentials recorded over the entire 6 s period during which the force was being applied to the tooth. Sympathetic stimulation was for 30 s and was applied during the solid bars (5 V, 1 ms duration). Phentolamine (4 mg kg^{-1} I.V.) abolished the effects of sympathetic stimulation (*F*).

dontal mechanoreceptor neurones. Sympathetic stimulation at frequencies of 10 Hz or less abolished the spontaneous activity in all seven neurones.

Fig. 1 is an example of a neurone modulated by sympathetic nerve stimulation recorded from the maxillary division of the trigeminal ganglion. In this example we were able to record long enough to stimulate the sympathetics at a range of frequencies and return to control values between stimulations (Fig. 2*A–E*).

When the animal was given phentolamine (4 mg kg^{-1} I.V.) the effect of sympathetic stimulation was completely abolished (see Fig. 2*F*).

Fig. 2 suggests that the reduction in mechanoreceptor discharge depended on the frequency at which the sympathetic supply was stimulated and in most of the other neurone studies for which at least two stimulation frequencies were used, the higher frequencies always produced the greater effect on mechanoreceptor activity.

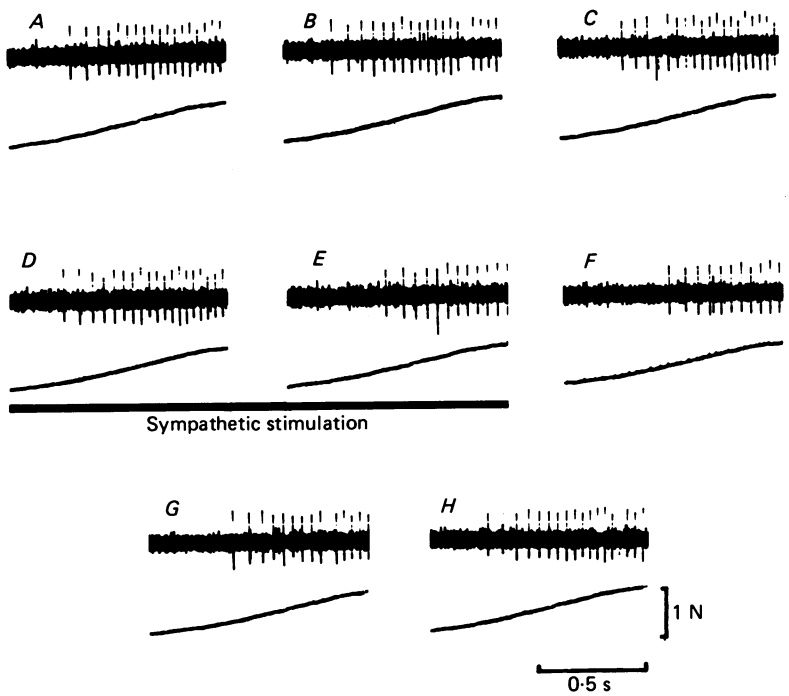

Fig. 3. Records of eight consecutive force applications to the tooth and the accompanying action potentials recorded from the trigeminal ganglion. As in Figs. 1 and 2, forces are being applied to the tooth once every 15 s. Only the time during which the force is building in a ramp-like fashion is shown (1 s). Records *A*, *B* and *C* are before sympathetic stimulation, *D* and *E* during the 30 s of sympathetic stimulation (5 V, 1 msec duration, 2·5 Hz), and *F*, *G* and *H* following sympathetic stimulation. The force at which the first impulse was evoked was raised during the latter half of the period of sympathetic stimulation and thereafter for 45 s.

Changes in threshold of the mechanoreceptors were studied using the procedure illustrated in Fig. 3. The force was applied to the tooth in a ramp like fashion at a rate of 1 N/s. By comparing how far up the ramp the force had proceeded before the first spike was seen it was possible to measure any change in threshold of the units, assuming that there was no significant change in conduction velocity of the nerve fibre following sympathetic stimulation.

Fig. 3 shows the force exerted and the response of the neurone to eight consecutive force applications each 15 s apart: *A*, *B* and *C* before sympathetic stimulation; *D* and *E* during sympathetic stimulation (2·5 Hz, 5 V, 1 ms); *F*, *G* and *H* after

sympathetic stimulation. The thresholds of the same neurone as in Fig. 3 are shown in Fig. 4, for 2·5, 10 and 20 Hz sympathetic nerve stimulation.

All neurones whose discharge frequency was reduced by sympathetic nerve stimulation showed a rise in threshold similar to that in Figs. 3 and 4. Also, just as the reduction in impulse frequency was greater with increasing frequencies of sympathetic stimulation, so too were the changes in thresholds. In addition, the

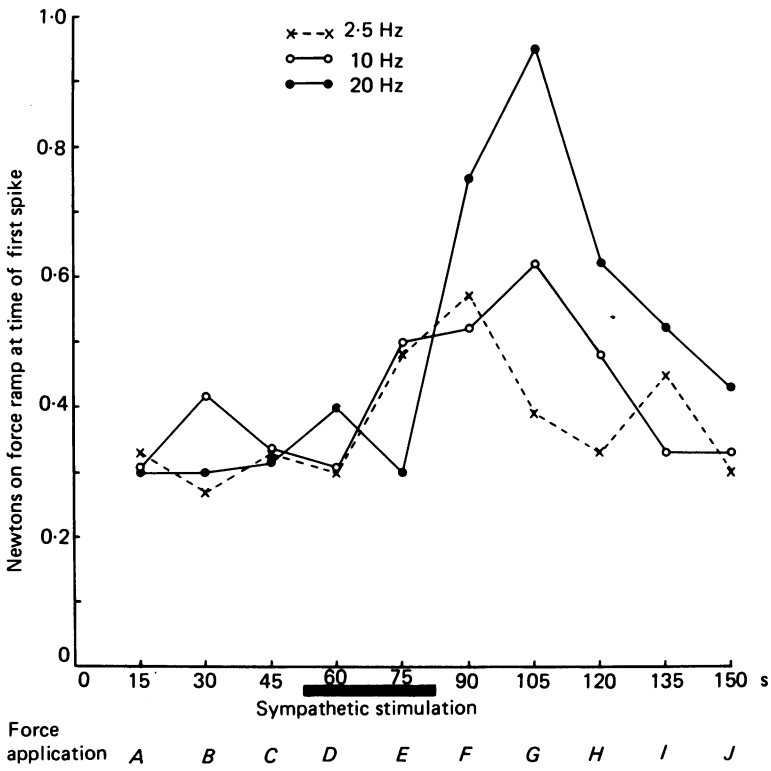

Fig. 4. Time in seconds and the timing of the force applications *A*, *B*, *C* etc. as in Fig. 3 are shown on the abscissa. On the ordinate is shown the force in Newtons being applied to the tooth at the time the first spike reaches the recording electrodes.Thresholds following sympathetic stimulation at 20, 10 and 2·5 Hz (5 V, 1 ms duration) are shown.

recovery to pre-sympathetic stimulation thresholds was slower with increasing frequencies of sympathetic stimulation. In none of the non-modulated neurones did sympathetic stimulation affect thresholds. No measure of threshold changes in spontaneously active neurones could be made using this method.

In nine cats the left lingual artery was cannulated and the lingual artery blood pressure monitored. With bilateral carotid occlusion the lingual artery blood pressure fell to below 5 kPa, but never below 3 kPa; presumably because the cat has a large vertebral artery supply to the head region.

Occlusion of the carotids for 30 s (i.e. the same length of time the sympathetic nerves were usually stimulated) produced no effect on either modulated or non-modulated mechanoreceptor neurones.

Mesencephalic nucleus

A total of thirty-seven intra-oral mechanoreceptor neurones have been recorded from the mesencephalic nucleus in eleven cats. Twenty-one neurones were periodontal mechanoreceptor neurones and sixteen were 'type P' neurones as defined by Linden (1978). Of the twenty-one periodontal neurones only two were modulated by sympathetic stimulation. Neither of these two neurones discharged spontaneously. There was only one spontaneously discharging periodontal mechanoreceptor neurone

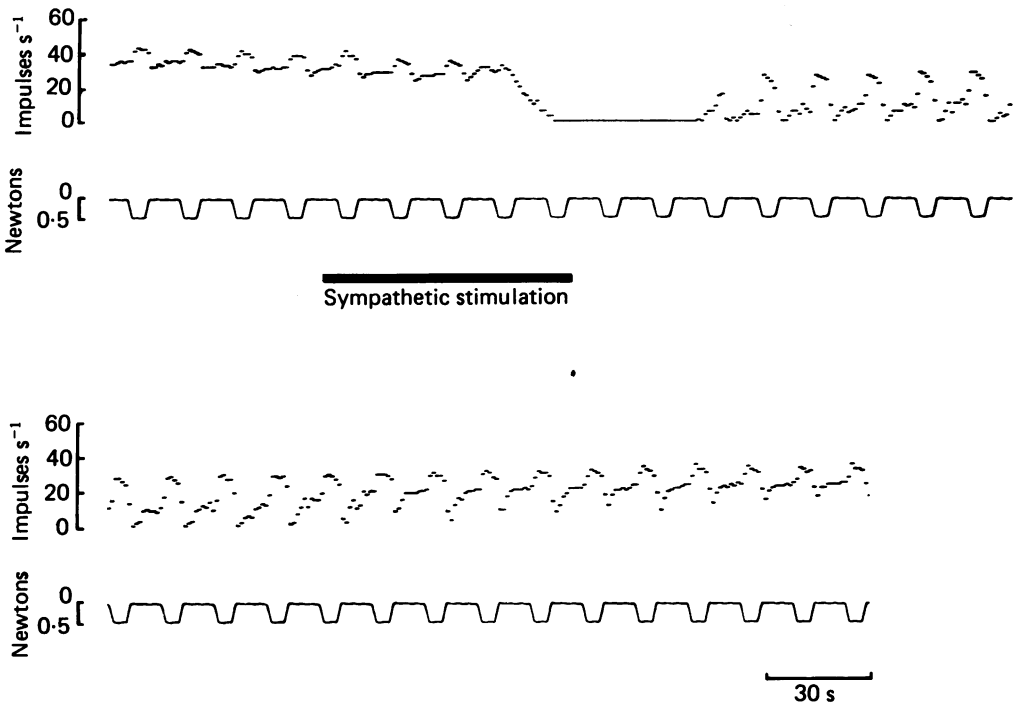

Fig. 5. Results from a spontaneously discharging 'type P' neurone recorded in the mesencephalic nucleus of V. Lower trace, as in Fig. 1, is the record of forces applied to the tooth. The force applications were applied once every 15 s but in this case a 'pulling' rather than a 'pushing' force was being applied. The upper record shows the number of impulses per second. There is spontaneous activity of the neurone when no force is being applied to the tooth, and an increase in the rate of discharge of the neurone when forces are applied to the tooth. Following sympathetic stimulation (5 Hz, 5 V, 1 ms) all activity of the neurone ceased for over 30 s. Both spontaneous activity and the response to a 0·5 N force returned gradually back to control values.

recorded in the mesencephalic nucleus and this neurone was not modulated. This was the only spontaneously discharging neurone in the entire study whose spontaneous activity could not be abolished completely by sympathetic nerve stimulation.

Of the sixteen 'type P' neurones twelve were modulated and four were not modulated by sympathetic stimulation. In the total there were six spontaneously discharging 'type P' neurones, all of which were modulated, and in all cases the spontaneous discharge was abolished by sympathetic stimulation.

Fig. 5 is an example of a spontaneously discharging 'type P' neurone recorded from the mesencephalic nucleus. The spontaneous impulse frequency was 35 Hz between force applications rising to 48 Hz during the application of the force and falling to 35 Hz when the force was removed.

During and after sympathetic stimulation the spontaneous activity was reduced and eventually abolished, and the response to the controlled force was also reduced and abolished. All responses gradually returned to control values over a period of a

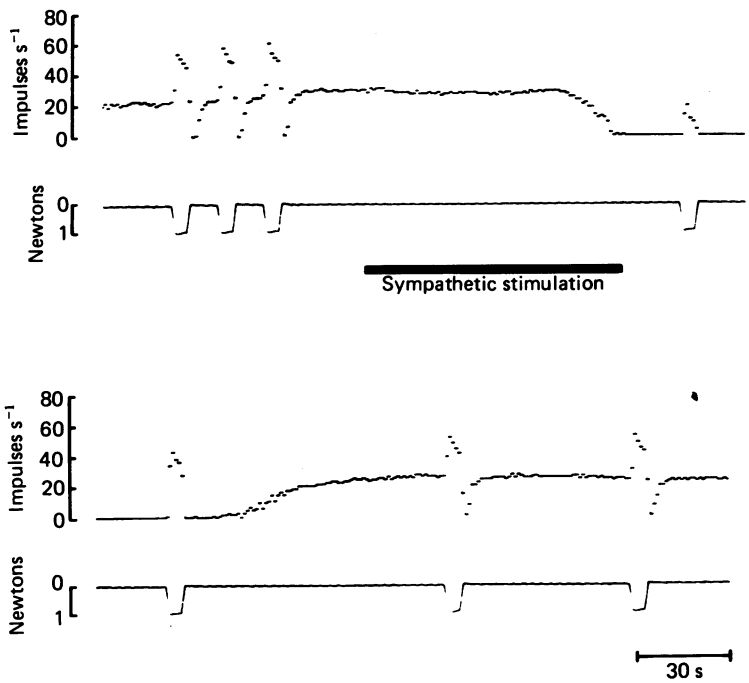

Fig. 6. Records from the modulated 'type P' neurone of Fig. 5. Forces are no longer being applied once every 15 s but at irregular intervals as can be seen by the lower trace monitoring force applications. 'Pulling' forces of 1 N are being used. Upper trace shows the level of ongoing spontaneous activity and response of the neurone to force applications. Sympathetic stimulation (5 V, 1 ms, 5 Hz) abolishes the spontaneous activity for up to 80 s. The response of the neurone to a force applied to the tooth is also reduced. There is a gradual return to control values.

few minutes. Fig. 6 is a trace from the neurone shown in Fig. 5. In this example the forces are not being applied to the tooth in a regularly repeating manner but at irregular intervals as can be seen from the force record. Following sympathetic stimulation spontaneous activity is abolished for up to 80 s. Force applications during this time produced a smaller response than the same force before sympathetic stimulation. As in Fig. 5, both spontaneous activity and response to the force returned to control values within a few minutes after sympathetic stimulation ceased.

In the mesencephalic nucleus as in the trigeminal ganglion, bilateral carotid occlusion had no effect on either the modulated or non-modulated neurones.

DISCUSSION

Claude Bernard in 1851 first suggested that sensory input could be modulated by activity of the sympathetic nervous system. Sympathetic stimulation has been shown to alter afferent input from a variety of receptors: muscle spindles (Eldred, Schnitzlein & Buchwald, 1960; Hunt, 1960), cutaneous mechanoreceptors in frogs (Loewenstein, 1956; Chernetski, 1964; Calof, Jones & Roberts, 1981), cutaneous cold receptors in frogs (Spray, 1974), cutaneous mechanoreceptors and guard hair receptors in cats (Pierce & Roberts, 1981), and cat's vibrissae (Nilsson, 1972). Sympathetic stimulation affects peripheral gustatory activity (Chernetski, 1964) and can alter impulse frequency in nerves from tooth pulp (Edwall & Scott, 1971; Matthews, 1976). Sympathomimetic agents and/or adrenergic blocking agents also modulate afferent input from a variety of receptor types (Loewenstein, 1956; Loewenstein & Altamirano-Orrego, 1956; Hunt, 1960; Calma & Kidd, 1962; Paintal, 1959, 1964; Bhoola, Diete-Spiff & Webster, 1962; Nilsson, 1972; Schiff, 1974).

In this study it has been found that almost half of the mechanoreceptor neurones from which recordings were made were modulated by sympathetic stimulation. Stimulation of the sympathetic nerves both decreased the impulse frequency to a controlled force application, and raised the threshold, even at frequencies considered to be within the physiological range (1–10 Hz). In the majority of the studies quoted above, sympathetic stimulation has been found to increase afferent impulse frequency for a given stimulus. Sympathetic stimulation has been found to decrease afferent input in a few studies (Nilsson, 1972; Calof *et al.* 1981; Pierce & Roberts, 1981) and to decrease afferent input following an initial transient increase (Hunt, 1960; Edwall & Scott, 1971). In this study we have found only a decrease in the response of oral mechanosensitive neurones with sympathetic stimulation.

Also, as stated previously, no spontaneously active intra-oral mechanoreceptor neurones have been reported in studies where recordings have been made centrally with peripheral nerves intact, i.e. recordings from the mesencephalic nucleus or trigeminal ganglion. On the other hand, spontaneous activity has been found in most of the peripheral nerve studies. It has been suggested that in recordings made from the mesencephalic nucleus or the trigeminal ganglion, efferent pathways remain intact, whereas in the more peripheral recordings the sympathetic nerves have been cut along with the peripheral nerve bundle (Linden, 1978). In the present study we recorded from both the mesencephalic nucleus and the trigeminal ganglion; but unlike all previous central studies the cervical sympathetic nerves were cut. In this study for the first time, spontaneously active intra-oral mechanoreceptor neurones were recorded with micro-electrodes in the trigeminal ganglion and the mesencephalic nucleus.

Hannam (1969*b*) simulated the activity of spontaneous neurones by subjecting non-spontaneously active neurones to low-grade mechanical stimulation of the teeth. So similar was the behaviour of the simulated neurones and that of the truly spontaneously active neurones that Hannam suggested that spontaneous activity might be due to sustained tension in the periodontal ligament.

We should like to extend this hypothesis and suggest that some of the low-threshold, very slowly adapting mechanoreceptors are made spontaneously active by the

removal of sympathetic tone which would normally inhibit the receptor either directly or indirectly. In this study all the spontaneously active neurones except one were silenced by sympathetic stimulation at frequencies considered to be within the physiological range (1–10 Hz).

Two papers are worthy of special consideration since they report spontaneous activity in intact peripheral nerves. Kizior, Cuozzo & Bowman (1968) did not cut the inferior alveolar nerve. They made whole nerve recordings and showed some spontaneous activity in the absence of any overt stimulation to intra-oral structures. The inferior alveolar nerve contains pulpal and gingival afferents as well as periodontal and the authors could not assume that the spontaneous activity came from periodontal mechanoreceptors alone.

Johansson & Olsson (1976) recorded from the inferior alveolar nerve in man and did not cut the nerve in their study. They recorded from only two dental mechanoreceptors, one of which was intermittently spontaneous. The fact that this unit was intermittently spontaneous suggests that it might have been a low-threshold unit which was being intermittently stimulated.

Thus we feel that none of the studies in which spontaneously active intra-oral mechanoreceptors have been reported offers any major evidence which would cause us to reject the hypothesis that spontaneous activity is an experimental artifact.

The cutting of the sympathetic supply, however, is not the only difference between our study and others. Most other workers who have found spontaneously discharging units have worked on mandibular teeth and have therefore needed to fix the mandible in some way. It has been shown that forces on one part of the jaw can cause deformation at some distance from the site of the force (Picton, 1962) and it is possible that fixing the mandible might subject low-threshold slowly adapting mechanoreceptors to low-grade mechanical stimulation. It is our impression that when we fixed the mandible to the maxilla we recorded more spontaneous activity.

When sympathetic stimulation has been found to modulate afferent activity the effects have sometimes been mimicked by injection of adrenaline and/or noradrenaline (Loewenstein, 1956; Eldred, *et al.* 1960; Hunt, 1960; Matthews, 1976). Nilsson (1972) was mostly unsuccessful in mimicking his effects with I.V. noradrenaline but could block the effects of sympathetic stimulation with an α-blocker as could Eldred *et al.* (1960) and Matthews (1976). In this study intravenous phentolamine prevented sympathetic modulation of previously modulated receptors.

The question arises as to how the efferent sympathetic activity might affect the discharge of the receptor. Is it a direct effect of neurotransmitter released from sympathetic endings on the receptor, or is it a secondary effect caused by blood flow or temperature changes? Nilsson (1972), working on vibrissal receptors in cats, measured intracutaneous temperature and observed a slight lowering of temperature which did not exceed 1 °C. It seems unlikely that this small variation would result in significant receptor depression. The sensitivity to temperature changes of the vibrissal receptors on which Nilsson was working is unknown as is that of intra-oral mechanoreceptors, but similar slowly adapting skin receptors in the cat are only slightly affected by such small temperature changes (Tapper, 1965). A fall in temperature of 1–2 °C in the periodontal ligament following sympathetic stimulation has been recorded (Anderson & Linden, 1977).

Vascular occlusion and blood flow changes have altered receptor response in a number of studies in a similar way to that produced by sympathetic stimulation or sympathomimetic drugs (Matthews, 1933; Paintal, 1959; Eldred *et al.* 1960; Bhoola *et al.* 1962; Calma & Kidd, 1962; Edwall & Scott, 1971; Matthews, 1976). On the other hand vascular occlusion has failed to mimic or effect the response to sympathetic stimulation in a number of studies (Hunt, 1960; Nilsson, 1972; Calof *et al.* 1981; Pierce & Roberts, 1981) and Paintal (1959) argues that not all his responses could be explained by vasoconstriction. Nilsson (1972) clamped the ipsilateral carotid and produced a similar fall in temperature to that produced by sympathetic stimulation but he found no change in response of the sensory unit. We occluded both carotid arteries in our experiments and recorded blood pressure in the lingual artery which fell to below 5 kPa. By occluding both carotids for 30 s, i.e. the same length of time for which we usually stimulated the sympathetic nerves, we were never able to affect mechanoreceptor response. However, the blood pressure in the lingual artery never fell to zero, presumably because the cat has a substantial vertebral arterial supply to the head; and even if blood pressure in the arteries supplying the mechanoreceptors falls to below 5 kPa this does not necessarily mimic the reduction of blood flow to the mechanoreceptor brought about by sympathetic stimulation. Nevertheless, with bilateral carotid occlusion, which caused a marked fall in arterial blood pressure distal to the occlusion, we were unable to alter mechanoreceptor response to our standard stimulus. It seems unlikely that the sympathetic modulation reported here is produced primarily by blood flow changes.

It is possible that the intra-oral mechanoreceptors that can be modulated are lying in tissue very close to a blood vessel and that the position of the receptor relative to the tooth is altered during sympathetic stimulation due to constriction of the blood vessel and subsequent movement of the receptor. Until the exact distribution of the receptors and their spatial relationship to the tooth, bone and blood vessels is known this possibility remains speculative.

In conclusion, this study has shown another receptor type whose output to a controlled stimulus can be altered by sympathetic nerve stimulation. Stimulating the sympathetic supply increases the threshold and decreases the discharge from a significant proportion of intra-oral mechanoreceptor neurones even with frequencies of sympathetic stimulation within the physiological range. In peripheral nerve studies on the neurones, the sympathetic supply has almost always been cut along with the afferent nerves, thereby removing an inhibitory influence from some of the mechanoreceptors. Spontaneous activity has not been found in previous central recording studies where the sympathetic nerves are left intact. In this study we recorded centrally but did cut the sympathetic supply and found spontaneous neurones.

It is suggested that reported spontaneous activity from intra-oral mechanoreceptors is brought on by a combination of at least two factors: (1) cutting the peripheral nerves and in so doing cutting the sympathetic supply and thus removing an inhibitory influence from the sensory unit; and (2) fixing the jaws and thereby subjecting the mechanoreceptors concerned to low-grade mechanical stimulation. The means by which the sympathetic efferents produce this inhibitory influence on the sensory unit has not been resolved.

This work was supported by a grant from the Medical Research Council to R.W.A.L. We are indebted to Professor Declan Anderson for critical reading of the manuscript and to Mr Charles Rickards for technical assistance.

REFERENCES

ANDERSON, D. J. & LINDEN, R. W. A. (1977). Sympathetic modulation of intraoral mechanoreceptor activity. *J. dent. Res.* **56**, D125.

BEAUDREAU, D. E. & JERGE, C. R. (1968). Somatotopic representation in the Gasserian ganglion of tactile peripheral fields in the cat. *Archs oral Biol.* **13**, 247–256.

BERMAN, A. L. (1969). *The Brain Stem of the Cat; a Cytoarchitectonic Atlas with Stereotaxic Coordinates.* Madison, Milwaukee and London: University of Wisconsin Press.

BERNARD, C. (1851). Influence du grand sympathique sur la sensibilité et sur la calorification. *C.r. Séanc. Soc. Biol.* **3**, 163–164.

BHOOLA, K. D., DIETE-SPIFF, K. & WEBSTER, R. A. (1962). The effect of adrenaline on mammalian muscle spindles. *J. Physiol.* **164**, 16–17P.

CALMA, I. & KIDD, G. L. (1962). The effect of adrenaline on muscle spindles in cat. *Archs ital. Biol.* **100**, 381–393.

CALOF, A. L., JONES, R. B. & ROBERTS, W. J. (1981). Sympathetic modulation of mechanoreceptor sensitivity in frog skin. *J. Physiol.* **310**, 481–499.

CHERNETSKI, K. E. (1964). Sympathetic enhancement of peripheral sensory input in the frog. *J. Neurophysiol.* **27**, 493–515.

EDWALL, L. & SCOTT, D. (1971). Influence of changes in microcirculation on the excitability of the sensory unit in the tooth of the cat. *Acta physiol. scand.* **82**, 555–566.

ELDRED, E., SCHNITZLEIN, N. H. & BUCHWALD, J. (1960). Response of muscle spindles of stimulation to the sympathetic trunk. *Expl Neurol.* **2**, 13–25.

HANNAM, A. G. (1969*a*). The response of periodontal mechanoreceptors in the dog to controlled loading of the teeth. *Archs oral Biol.* **14**, 781–791.

HANNAM, A. G. (1969*b*). Spontaneous activity in dental mechanosensitive units in the dog. *Archs oral Biol.* **14**, 793–801.

HANNAM, A. G. (1970). Receptor fields of periodontal mechanosensitive units in the dog. *Archs oral Biol.* **15**, 971–978.

HUNT, C. C. (1960). The effect of sympathetic stimulation on mammalian muscle spindles. *J. Physiol.* **151**, 332–341.

JERGE, C. R. (1963). Organization and function of the trigeminal mesencephalic nucleus. *J. Neurophysiol.* **26**, 379–392.

JOHANSSON, R. A. & OLSSON, K. Å. (1976). Microelectrode recordings from human oral mechanoreceptors. *Brain Res.* **118**, 307–311.

KERR, F. W. L. & LYSAK, W. R. (1964). Somatotopic organization of trigeminal–ganglion neurones. *Archs Neurol., Chicago* **11**, 593–602.

KIZIOR, K. E., CUOZZO, J. W. & BOWMAN, D. C. (1968). Functional and histologic assessment of the sensory innervation of the periodontal ligament of the cat. *J. dent. Res.* **47**, 59–64.

LINDEN, R. W. A. (1978). Properties of intraoral mechanoreceptors represented in the mesencephalic nucleus of the fifth nerve in the cat. *J. Physiol.* **279**, 395–408.

LOEWENSTEIN, W. R. (1956). Modulation of cutaneous mechanoreceptors by sympathetic stimulation. *J. Physiol.* **132**, 40–60.

LOEWENSTEIN, W. R. & ALTAMIRANO-ORREGO, R. (1956). Enhancement of activity in a Pacinian corpuscle by sympathomimetic agents. *Nature, Lond.* **178**, 1292–1293.

MATTHEWS, B. (1965). Action potentials from dental mechanoreceptors in the dog. *J. dent. Res.* **44**, 1167.

MATTHEWS, B. (1976). Effects of sympathetic stimulation on the response of intradental nerves to chemical stimulation of dentin. In *Advances in Pain Research and Therapy*, vol. I., ed. BONICA, J. J. & ALBE-FESSARD, D. New York: Raven.

MATTHEWS, B. & ROBINSON, P. P. (1980). The course of post-ganglionic sympathetic fibres distributed with the trigeminal nerve in the cat. *J. Physiol.* **303**, 391–401.

MATTHEWS, B. H. C. (1933). Nerve endings in mammalian muscle. *J. Physiol.* **78**, 1–53.

NESS, A. R. (1954). The mechanoreceptors of the rabbit mandibular incisor. *J. Physiol.* **126**, 475–493.

NILSSON, B. Y. (1972). Effects of sympathetic on mechanoreceptors of cat vibrissae. *Acta physiol. scand.* **85**, 390–397.

OGILVIE, R. W. (1969). The vasomotor innervation of the cat's lower right canine tooth pulp. *Anat. Rec.* **163**, 237.

PAINTAL, A. S. (1959). Facilitation and depression of muscle stretch receptors by repetitive antidromic stimulation, adrenaline and asphyxia. *J. Physiol.* **148**, 252–266.

PAINTAL, A. S. (1964). Effects of drug on vertebrate mechanoreceptors. *Pharmac. Rev.* **16**, 341–380.

PFAFFMANN, C. (1939). Afferent impulses from the teeth due to pressure and noxious stimulation. *J. Physiol.* **97**, 207–219.

PICTON, D. C. A. (1962). Distortion of jaws during biting. *Archs oral Biol.* **7**, 573–580.

PIERCE, J. P. & ROBERTS, W. J. (1981). Sympathetically induced changes in the response of guard hair and type II receptors in the cat. *J. Physiol.* **314**, 411–428.

SAKADA, S. & ONODERA, K. (1974). On the specificity of spontaneously discharging units in the cat inferior alveolar nerve. *Bull. Tokyo dent. Coll.* **15**, 7–22.

SCHIFF, J. D. (1974). Role of the sympathetic innervation of the Pacinian corpuscle. *J. gen. Physiol.* **63**, 601–608.

SPRAY, D. C. (1974). Characteristics, specificity and efferent control of frog cutaneous cold receptors. *J. Physiol.* **237**, 15–38.

SUZUKI, H. (1963). Responses of the mechanoreceptors in the periodontal membrane to vibratory stimulus on the canine of the cat. *J. Physiol. Soc. Japan* **25**, 415–551.

TAPPER, D. N. (1965). Stimulus–response relationship in the cutaneous slowly-adapting mechanoreceptor in hairy skin of the cat. *Expl Neurol.* **13**, 364–385.

WAGERS, P. W. & SMITH, C. M. (1960). Responses in dental nerves of dogs to tooth stimulation, and the effects of systemically administered procaine, lidocaine and morphine. *J. Pharmac. exp. Ther.* **130**, 89–105.