Effects of sympathetic nerve activity on acute mobility of the rabbit incisor tooth

Harald Aars
Department of Physiology and Biochemistry, Dental Faculty, University of Oslo, Oslo, Norway

Aars, H. Effects of sympathetic nerve activity on acute mobility of the rabbit incisor tooth. *Acta Odontol. Scand.* 1983, 41, 287–292. Oslo. ISSN 0001–6357.

The contribution of local blood flow to the buffering properties of the periodontal ligament was examined in 15 anesthetized rabbits. The position of the tooth was recorded by an ultrasonic transit time technique, and local vascular pressures and volumes were altered by electrical stimulation of the cervical sympathetic nerves. The unloaded tooth was shifted into a slightly intruded position by nerve stimulation, and from this position, intrusion movements evoked by 5- to 10-g loads were found to be 10-15% smaller than before the stimulation. Tooth mobility was unaffected by 20–30 mm Hg reductions in mean arterial pressure, but similar decreases in mobility as evoked by nerve stimulation were observed when the pressure was brought down to 10–25 mm Hg. This procedure also produced about the same intrusive shift of the unloaded tooth. Most likely, the sympathetic nerve activity decreased tooth mobility by causing a fall in periodontal vascular pressures, thereby moving the tooth into a position where it was more resistant to further, load-induced intrusion. □ *Load-induced intrusion; periodontal blood flow; periodontal ligament; tooth position*

Harald Aars, Department of Physiology and Biochemistry, Dental Faculty, University of Oslo, Box 1052, Blindern, Oslo 3, Norway

An abrupt, force-induced movement of a tooth must by necessity affect the distribution of blood in the periodontal ligament. Depending on the balance between the external force and the vascular pressures and on the resistance against the drainage of these vessels, the periodontal vasculature may contribute substantially to the resistance of the tooth against changes in position. Such suppositions have been made previously, based on observations of increased (3, 4) or decreased (13) displacements of the tooth in response to loading after exsanguination. Others, however, have found tooth mobility to be the same after exsanguination and shortly after death of the animal (7–9). Of probably greater physiological relevance, the axial mobility of the tooth has been observed to increase with a sudden rise in arterial pressure (10) and to remain largely unaffected by moderate changes in pressure (9). In short, while suggestions are plentiful, the evidence for a role of the systemic arterial pressure in the damping properties of the periodontal ligament is not convincing.

There are indications, however, that local vascular pressures take part. Thus, the axial mobility of incisor teeth is decreased after injection of noradrenaline or angiotensin into the periodontal ligament in monkeys (12, 13), and sympathetic nerve stimulation leads to alterations in the transverse mobility of the canine tooth in cats (2).

In the rabbit stimulation of the cervical sympathetic nerves causes intrusion of the incisor (1), presumably mediated by changes in vascular pressure and volume in the periodontal ligament. It is the object of the present paper to examine whether sympathetic nerve activity can also influence the load-induced displacements of the incisor—that is, its mobility.

Materials and methods

Fifteen rabbits (body weights, 2.4–3.4 kg) were used in this study. They were anesthetized by a mixture of chloralose (0.03 g/kg) and urethane (0.75 g/kg), half intravenously

and half intraperitoneally. Urethane was supplemented when necessary. The animals were allowed to breathe spontaneously, through a tracheostomy tube. Arterial blood pressure and, in most animals, the venous pressure were recorded with Statham P23Dc transducers connected to catheters placed in the central vessels of the left ear. Body temperature was maintained by covers and a heating lamp.

The animals were placed on their backs, with the head fixed to the table. The right and left cervical sympathetic nerves were placed on pairs of silver electrodes and covered by warm liquid paraffin. Bilateral stimulation of the cranial ends of the cut nerves was performed with square-wave pulses from a Grass SD9 stimulator, set at 6–8 V, 1–1.4 msec, and 0.5–10 Hz.

Instantaneous changes in axial position of the left upper incisor were recorded with an ultrasonic transit time technique (1). One piezo-electric crystal was fastened to the tooth, the other to the pre-maxilla, and the two crystals were brought together in front of the tooth. Changes in inter-crystal distance, which reflected movements of the tooth, were detected as changes in transit time of the ultrasonic pulses and were read on the recorder with a sensitivity of 0.5–2.0 μm/mm paper. Owing to the curved shape of the tooth and the radial distance between the tooth and the crystals, actual movements of the tooth were only 30–50% of those recorded for the crystals. Consequently, whenever absolute measures are given in this text, they have been calculated as 40% of the movement of the crystals (1).

The crown of the tooth was loaded by 5–10 g in a direction tangential to the tooth curvature. The electromagnetic, servo-controlled force generator could be operated manually or automatically, delivering pulses of 0.4 sec or more, with a rise time of about 0.1 sec. The actual load produced by the generator was measured by strain gauges mounted on the loading arm and was recorded together with blood pressure and tooth position on a Beckman R411 Dynograph.

The teeth were subjected to loading periods of 2–22 sec (single loadings) or 0.4–0.8 sec (single or sets of 3–14 loadings spaced 1 sec apart), before and during sympathetic nerve stimulation. Tooth responses to single loadings were read as the maximum amplitude of induced movements, while sets of loadings were represented by the amplitude of the movement evoked by the last loading in each set. Intrusion responses during stimulation were calculated in percentage of the average of two or three immediately preceding (of many) control loadings.

Unless otherwise specified, the data presented in this paper were obtained when mean arterial pressure was between 70 and 94 mm Hg. Average results are given as means ± SD. The significance of differences between groups were evaluated by Student's *t* test for unpaired data.

Results

The tooth moved into its socket during applications of axially directed loads of 5–10 g and quickly returned to control position when the loads were removed. These load-induced intrusive movements were 20% increased in one rabbit and unchanged in four after sectioning of the cervical sympathetic nerves, when re-studied 15–18 min after the initial observations. The time interval had probably little effect in itself, since, in a series of eight other rabbits, the intrusive movements in response to loading were unchanged when repeated 10–70 min later (102.9 ± 5.4% of control, 14 comparisons). The nerve sectioning evoked an extrusive shift in the unloaded position of the tooth in 13 of 15 animals (mean, 6 ± 5 μm).

Electrical stimulation of the cervical sympathetic nerves caused intrusion of the tooth, and when loaded during sympathetic stimulation, the tooth showed smaller responses to loading than before the stimulation (Figs. 1 and 2). The nerve-induced intrusion increased with the duration and frequency of the stimulation, and the greater the intrusion, the greater was usually the reduction in load-induced mobility (Fig. 3).

Average effects of the nerve stimulation on tooth mobility have been split in two groups, depending on whether the intrusion

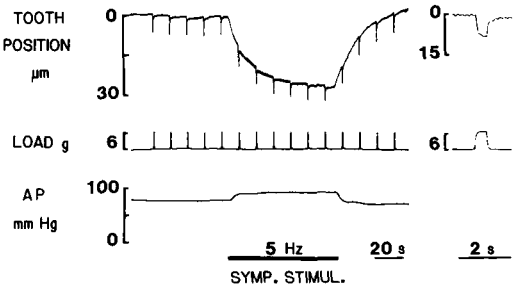

Fig. 1. Effects of sympathetic nerve stimulation (5 Hz, 6 V, 1 msec) on the unloaded position of the incisor and on its axial movements in response to brief loadings with 5 g. Mean blood pressure measured in the ear artery. *Inset:* Load and tooth movement recorded with higher paper speed.

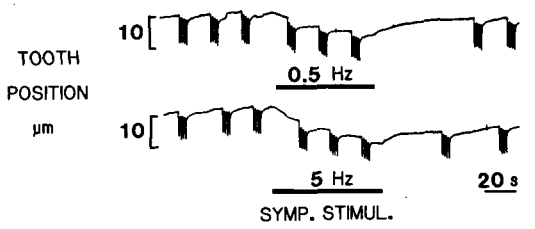

Fig. 3. Records from one animal showing frequency-related (0.5 and 5 Hz) effects of sympathetic nerve stimulations on tooth position and movements induced by 5-g loads.

responses to stimulation were greater or smaller than 6 µm. At nerve-induced intrusions of 6–60 µm, obtained by stimulations with 0.5- to 10-Hz pulses, tooth movements in response to different patterns of loading 15–180 sec after the start of stimulation were reduced to 84.8 ± 9.4% of control ($p < 0.001$, 103 loadings). Average decreases in mobility were the same for loading periods of 0.4–0.8 sec (10 rabbits) and 2–22 sec (9 rabbits), but the standard deviation was larger in the latter group (13.5% versus 7.3%). The standard deviation of 105 control observations was 3.6%. Related to a 'normal' value of 100% ± 2SD of control observations, the mobility of the tooth was unaffected in 1 and reduced in 12 rabbits when studied at resting tooth positions 6–60 µm intruded by sympathetic nerve stimulation. Ear artery pressure increased 1–15 mm Hg during these sympathetic nerve stimulations, while the venous pressure decreased 1–6 cm H_2O.

Sympathetic nerve stimulations that intruded the tooth less than 6 µm had smaller effects on tooth mobility. Thus, at nerve-induced intrusions of 2–5 µm, mobility was reduced in four of eight rabbits and unchanged in the rest. On an average, mobility was reduced to 90.4 ± 8.0% of control ($p < 0.001$, 34 loadings). The mobility was less reduced at nerve-induced intrusions of 2–5 µm than 6–60 µm ($p < 0.005$). Stimulation of the sympathetic nerves never led to an increase in the mobility of the tooth.

When in one animal intrusions similar to those produced by nerve stimulation were induced by a steady mechanical pre-loading of the tooth, the responses of the tooth to sets of brief loadings were reduced to the same degree as during the nerve stimulations.

The displacement of the tooth during brief loading periods was immediate and monophasic, but loads of longer duration in addition produced a secondary, much slower intrusion. This secondary movement during loadings of 10–22 sec and the corresponding slow intrusive shifts in unloaded position during sets of brief loading periods (as in Fig. 2, lower part, and Fig. 3) showed considerable variations during control loadings (SD = 12% for both). Hence, although sympathetic stimulation increased the average steepness of the secondary phases to 128% and 108% of control ($p < 0.05$) in the respec-

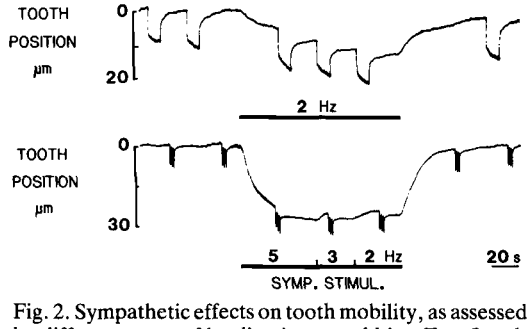

Fig. 2. Sympathetic effects on tooth mobility, as assessed by different types of loading in two rabbits. *Top:* Loading periods of 10 sec, 5 g; 2 Hz sympathetic nerve stimulation. *Bottom:* Sets of three 0.5-sec, 5-g thrusts; 5 to 2 Hz stimulation.

tive groups, the steepness in relation to the control ±2 SD was increased in only 2 of 6 rabbits in the 10- to 22-sec series and in none of 10 rabbits exposed to sets of brief loadings. The two animals showing a 50–60% increase in steepness of the secondary phase of the intrusion were the ones with the smallest sympathetic effects on maximum intrusion evoked by load applications of 10-sec duration (in the 6- to 60-μm group).

Since the effects of sympathetic nerve stimulation on tooth mobility presumably were mediated by vasoconstriction and reduced vascular pressures and volumes in the periodontal ligament, it was considered of interest to examine the effects on the tooth of changes in systemic vascular pressures. Reductions in mean arterial pressure of 20–30 mm Hg, produced in three rabbits by bleeding and within the same time span as used for sympathetic nerve stimulations, caused less intrusion than the nerve stimulations and no changes in tooth responses to brief loading tests. However, when in two other animals intrusions equivalent to those evoked by the nerve stimulation were brought about by arterial sectioning, the mobility at mean arterial pressures of 10–25 mm Hg was reduced to the same degree as during the preceding nerve stimulations (Fig. 4). Conversely, abrupt elevations of arterial or venous pressures, by means of aortic balloon inflation or occlusion of the jugular veins, extruded the tooth and usually caused a slight increase in its mobility (Fig. 5).

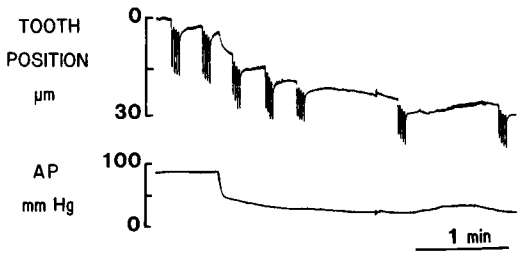

Fig. 4. Effects on position and mobility of the tooth of an abrupt fall in mean arterial pressure (recorded in the ear artery), caused by sectioning of the left femoral artery. Loads of 5 g. Same animal as in Fig. 3.

Discussion

As in a previous paper (1), the position of the rabbit incisor tooth was found to be very sensitive to changes in sympathetic nerve activity. The mobility of the tooth was also affected by sympathetic nerve activity, but a slightly higher discharge rate was required to induce measurable changes in this variable. Above threshold, at nerve-induced intrusions exceeding approximately 6 μm, mobility was clearly reduced by sympathetic nerve stimulation. It should be pointed out that the reduction in tooth mobility was demonstrated within a physiological range of sympathetic vasoconstrictor activity (6) and, except for the very lowest frequency, also within the range of the 0.1- to 6-Hz sympathetic stimulation found to cause a frequency-dependent reduction in periodontal ligament blood flow in the dog (5).

The evoked sympathetic discharge presumably acted mainly on precapillary resistance vessels inside or leading to the periodontal ligament, thereby greatly reducing capillary pressure and volume. A strong action on postcapillary vessels was unlikely, since this would have opposed the fall in capillary pressure and hence the intrusion of the tooth. The intrusive responses to sympathetic nerve activity were of about the same magnitudes as those resulting from reductions in mean arterial pressure to less than 30 mm Hg. The intrusion evoked by the sympathetic nerve stimulation can therefore probably be seen as the net result of the

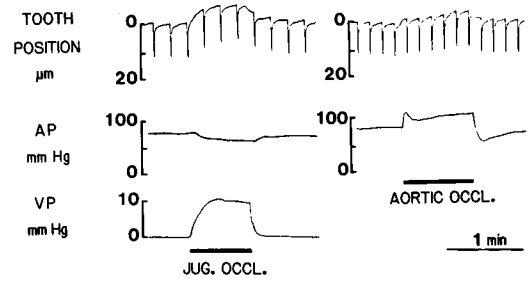

Fig. 5. Responses of the tooth and its axial, intrusive mobility to rises in venous (bilateral occlusion of jugular veins) or arterial pressures (inflation of intra-aortic balloon). Blood pressures recorded in ear vessels.

disturbed balance between a force tending to extrude the tooth and the connective tissue fibers resisting this movement. When pressure is reduced, the tooth will be moved into its socket by the elasticity of the periodontal fibers.

The changes in periodontal microcirculation could have contributed to the decrease in tooth mobility by two different, although connected, mechanisms: either directly, by altering the buffering properties of the periodontal blood vessels, or indirectly, by moving the tooth into a position where the fibers resisting inward movements of the tooth were already somewhat stretched, or uncoiled. The arguments for and against one or the other are hampered by the lack of precise knowledge of how the vascular compartment and the collagen tissue interact during loading of the tooth. The present experiments showed, however, that the sympathetic nerve stimulation caused equal reductions in tooth movements during brief and more long-lasting loading periods, usually without affecting the secondary intrusion. The stimulation had accordingly changed the abrupt initial phase of the movement, not the secondary phase. These two phases probably correspond to, respectively, the spring-like action of collagen fibers and the more viscoelastic properties of fluid and other cellular systems (for a review, see Ref. 11). The present results therefore indicate that the decrease in mobility was related to the nerve-induced intrusive shift in tooth position rather than the changes in the periodontal blood supply as such. This presumption agrees with the observations that intrusions similar to those evoked by nerve stimulations led to equal reductions in mobility when effected by sudden and marked reductions in arterial blood pressure (Fig. 4) or mechanical pre-loading of the tooth. The conclusion is also supported by the finding of reduced tooth mobility after injection of vasoconstrictor agents into the periodontal ligament in monkeys (12, 13).

That the tooth, when moved in one direction by sympathetic nerve stimulation, shows increased resistance to forces acting in the same direction is essentially the same argument as used by Aars & Linden (2) to explain how sympathetic nerve stimulation had increased the transverse mobility of the cat canine tooth: the tooth was more mobile in the palatal direction because the stimulation had tilted the tooth in the opposite direction. In this context, it is interesting to see how, in the present experiments, procedures extruding the tooth, like a sharp rise in arterial or venous pressure (Fig. 5), caused temporary increases in the movements induced by intrusive forces.

In conclusion, the present work has shown that, when tested by application of 5- to 10-g loads in periods of 0.4–22 sec, the axial mobility of the rabbit incisor tooth was reduced by stimulation of the cervical sympathetic nerves. Whether this sympathetic influence on tooth mobility is of importance for the sensory input from the periodontium, and for the jaw reflexes, remains to be seen.

References

1. Aars, H. The influence of sympathetic nerve activity on axial position of the rabbit incisor tooth. Acta Physiol. Scand. 1982, 116, 417–421
2. Aars, H. & Linden, R. W. A. The effects of sympathetic trunk stimulation on the position and mobility of the canine tooth of the cat. Arch. Oral Biol. 1982, 27, 399–404
3. Bien, S.M. Hydrodynamic damping of tooth movement. J. Dent. Res. 1966, 45, 907–914
4. Bien, S. M. & Ayers H. D. Responses of rat maxillary incisors to loads. J. Dent. Res. 1965, 44, 517–520
5. Edwall, L. & Kindlova, M. The effect of sympathetic nerve stimulation on the rate of disappearance of tracers from various oral tissues. Acta Odontol. Scand. 1971, 29, 387–400
6. Mellander, S. & Johansson, B. Control of resistance, exchange, and capacitance functions in the peripheral circulation. Pharmacol. Rev. 1968, 20, 117–196
7. Moxham, B. J. & Berkovitz, B. K. B. The effects of axially-directed extrusive loads on movements of the mandibular incisor of the rabbit. Arch. Oral Biol. 1979, 24, 759–763
8. Mühlemann, H. R. Tooth mobility. Tooth mobility changes through artificial alterations of the periodontium. J. Periodontol. 1954, 25, 198–202
9. Myhre, L., Preus, H. R. & Aars, H. Influences of axial load and blood pressure on the position of the rabbit's incisor tooth. Acta Odontol. Scand. 1979, 37, 153–159
10. Parfitt, G. J. The physical analysis of the tooth-supporting structures. In: The Mechanisms of Tooth Support. Bristol, John Wright & Sons Ltd. 1967, pp. 154–156

11. Picton, D. C. A., Johns, R. B., Wills, D. J. & Davies, W. I. R. The relationship between the mechanisms of tooth and implant support. Oral Sci. Rev. 1974, 5, 3–22
12. Slatter, J. M. & Picton, D. C. A. The effect on intrusive tooth mobility of noradrenaline injected locally in monkeys. J. Peridont. Res. 1972, 7, 144–150
13. Wills, D. J., Picton, D. C. A. & Davies, W. I. R. A study of the fluid systems of the periodontium in macaque monkeys. Arch. Oral Biol. 1976, 21, 175–185

Received for publication 26 May 1982