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IDENTIFICATION AND CHARACTERIZATION OF AFFERENT PERIODONTAL Aδ FIBRES IN THE CAT

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SUMMARY

1. The presence and responsiveness of afferent periodontal Aδ fibres was studied in pentobarbitone-anaesthetized adult cats.

2. Extracellular single fibre recordings were made from fine nerve filaments split from the proximally cut end of the inferior alveolar nerve. Periodontal nerve fibres were identified by constant current stimulus pulses applied via platinum wire electrodes inserted into the periodontal space of the lower canine tooth.

3. Of a total of 252 periodontal nerve fibres, 97 (37 %) were classified as Aδ fibres according to their conduction velocities (CV) ($> 2{\cdot}5$ m s^{-1}, < 30 m s^{-1}) as determined by electrical stimulation of the periodontal ligament. The mean (±S.D.) conduction velocity was $11{\cdot}0 \pm 7{\cdot}7$ m s^{-1} ($n = 97$; range: 2·6–28·2 m s^{-1}).

4. A good exponential correlation ($r = 0{\cdot}85$) was found between the electrical thresholds of the Aδ fibres and their conduction velocities.

5. For four Aδ fibres a complete stimulus–duration curve was determined. It followed rather well the $I = I_0/(1-e^{t/\tau})$ law, where I represents the stimulus amplitude, t the stimulus duration, I_0 the rheobasic current and τ the time constant.

6. In the intact tooth none of the identified periodontal Aδ fibres showed any ongoing activity in the absence of intentional stimulation.

7. The responses of sixteen electrically identified periodontal Aδ fibres were tested by mechanical, thermal and chemical stimuli applied to the periodontal space. Seven of nine periodontal Aδ fibres tested responded to mechanical forces applied to the tooth from different directions of which none could be activated by slight touch. A rudimentary directional sensitivity was seen. When a human tooth was stimulated by a mechanical stimulus of similar strength the sensation evoked was described as a dull, poorly localized pain.

8. Six periodontal Aδ fibres were activated by heat and/or cold and/or chemical stimulation. Two of eight periodontal Aδ fibres tested responded to heat and four of six Aδ fibres tested responded to cold stimuli applied to the alveolar bone overlying the periodontal ligament; none of them responded to both types of thermal stimuli. Two of seven periodontal Aδ fibres tested were activated by a saturated solution of potassium chloride applied locally to the periodontal ligament; two of these responded also to cold.

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9. The response behaviour of periodontal afferent Aδ fibres observed in this study suggests that they may play a role in periodontal nociception.

INTRODUCTION

The periodontal ligament is innervated by both thick and thin myelinated as well as unmyelinated nerve fibres (Hannam, 1982; Byers, 1985). In electrophysiological studies the thick myelinated (Aβ) fibres have been characterized as mechanoreceptors (Karita & Tabata, 1985; Tabata & Karita, 1986; for reviews cf. Anderson, Hannam & Matthews, 1970; Hannam, 1982; Linden, 1984; 1990). Unmyelinated (C) periodontal afferent nerve fibres have been shown to be polymodal nociceptors (Mengel, Jyväsjärvi & Kniffki, 1992). Nearly nothing is known about the functional role of small myelinated (Aδ) nerve fibres. There is only one symposium report in which recordings of some trigeminal neurons with periodontal Aδ fibre input were described (Mei, Hartmann & Aubert, 1977). Therefore, the purpose of the present study was to identify single afferent periodontal Aδ fibres and to study their functional characteristics.

METHODS

The experimental design has been described previously (Mengel *et al.* 1992) and therefore will be described only briefly here.

General procedures

The experiments were carried out on thirty-eight young adult cats of either sex (body weight: 2·0–4·0 kg) with intact fully developed permanent teeth. The animals received an initial intraperitoneal sodium pentobarbitone injection (40 mg kg^{-1}) and subsequently were kept in deep anaesthesia by continuous intravenous infusion of sodium pentobarbitone (3·1 mg kg^{-1} h^{-1}) throughout the experiments. The depth of anaesthesia was considered adequate, when the flexor reflex to pinching an intraphalangeal skin fold could not be elicited and the blood pressure, as monitored intra-arterially, was not elevated by this procedure. Spontaneous ventilation was facilitated by a tracheostomy. The cats were placed in a recording frame; the head and the mandible were fixed with the mouth in an open position, the lower margin of the mandible showing upwards. Arterial blood pressure and body core temperature were kept within normal physiological limits.

Preparation of tooth, periodontal ligament and nerve

The lower margin of the left mandible was removed to open the mandibular canal. The inferior alveolar nerve was carefully exposed and cut as far proximally as possible. A pool for microdissection of the nerve and recording was formed by skin flaps and filled with paraffin oil at 34 °C. The alveolar bone around the left lower canine tooth was exposed with all gingival tissues removed. Four holes were carefully drilled through the bone until the periodontal ligament had been reached and thin platinum wire electrodes were inserted (E1–E4; Fig. 1*A* and *B*). Another electrode was inserted into the cementum of the crown of the canine tooth for monopolar electrical stimulation of the dental pulp (E5; Fig. 1*A*). The periodontal ligament was stimulated bipolarly by any two of the four electrodes.

Identification of nerve fibres and stimulation protocol

The cut end of the inferior alveolar nerve was dissected until the neuronal activity of functional single fibres could be recorded. Single periodontal and pulpal nerve fibres were identified according to their all-or-none response evoked by a square wave constant current pulse applied to the periodontal ligament or to the tooth pulp using a constant current stimulator (amplitude: 0–200 μA; duration: 0·2–50 ms). Conduction velocities were calculated from the shortest latency of

the response evoked by a suprathreshold stimulus and the distance between the cathodal stimulating and recording electrodes. Fibres responding to electrical stimulation of both the periodontal ligament and the dental pulp were considered to branch and excluded from this study.

The responses of periodontal Aδ fibres to non-electrical stimulation were studied by applying mechanical forces to the crown of the tooth from different directions. Strong pressure was applied to the tooth with a glass rod for about 10 s. The intensities of these stimuli were only coarsely

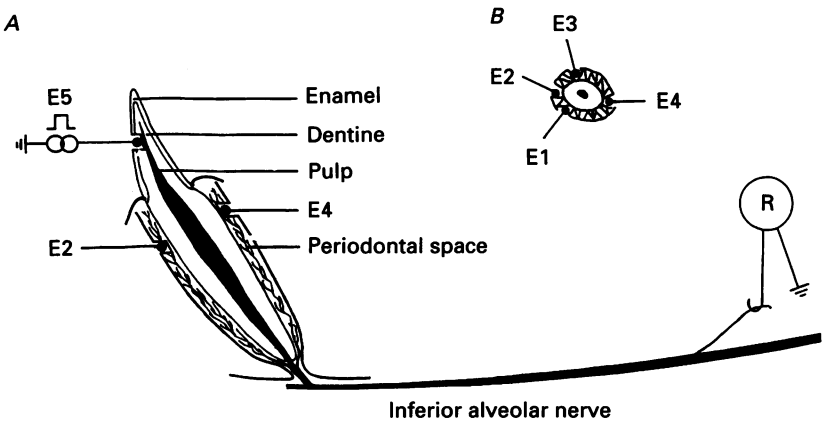

Fig. 1. Schematic diagrams showing the experimental set-up (*A* and *B*) for identification of single nerve fibres of the inferior alveolar nerve supplying the periodontal ligament of the cat's lower canine tooth. R symbolizes the recording devices. For identification of periodontal nerve fibres, electrical stimuli were applied to any two of the four electrodes (E1–E4; *A* and *B*) using a constant current stimulator. Monopolar stimulation of the tooth via electrode E5 was used to distinguish nerve fibres innervating the periodontal ligament only from branching ones also innervating the dental pulp.

differentiated into weak (i.e. tapping the tooth as required for activation of the low threshold mechanoreceptors supplied by Aβ fibres) and strong (i.e. strong pressure causing dull, poorly localized pain when applied to a human tooth). The responses to heating or cooling were studied by placing a thermode (contact temperature +80 °C) or a piece of dry ice (contact temperature −80 °C) onto the alveolar bone overlying the periodontal ligament for 10–20 s. These stimuli caused a temperature change of about 1 °C s^{-1} in the periodontal space as measured with a thermistor in a nearby electrode hole. For chemical stimulation, cotton pellets soaked with potassium chloride solution were placed in the electrode holes. After 3 min, the pellets were removed and the electrode holes rinsed with Tyrode solution.

RESULTS

Electrical stimulation

A total of 260 single periodontal nerve fibres were identified by electrical stimuli applied to the periodontal ligament. Of these fibres 97 (37%) were classified as Aδ fibres (2·5 < CV (conduction velocity) ⩽ 30 m s^{-1}), 142 (55%) as C fibres (CV ⩽ 2·5 m s^{-1}), and 21 (8%) as Aβ fibres (CV > 30 m s^{-1}). Due to the searching protocol, there is a bias in the total sample towards the fine fibres. The results on C fibres have been reported elsewhere (Mengel *et al.* 1992). None of the Aδ fibres showed any on-going activity without intentional stimulation.

Registrations of action potentials of four of the Aδ fibres from different experiments are shown in Fig. 2. The corresponding conduction velocities are indicated. As

demonstrated by displaying several successive traces separately (Fig. 2*A*) or superimposed (Fig. 2*C* and *D*), respectively, the latencies to electrical stimulation (repetition rate: 1 Hz) were very stable with suprathreshold stimuli. This was observed in all Aδ fibres studied.

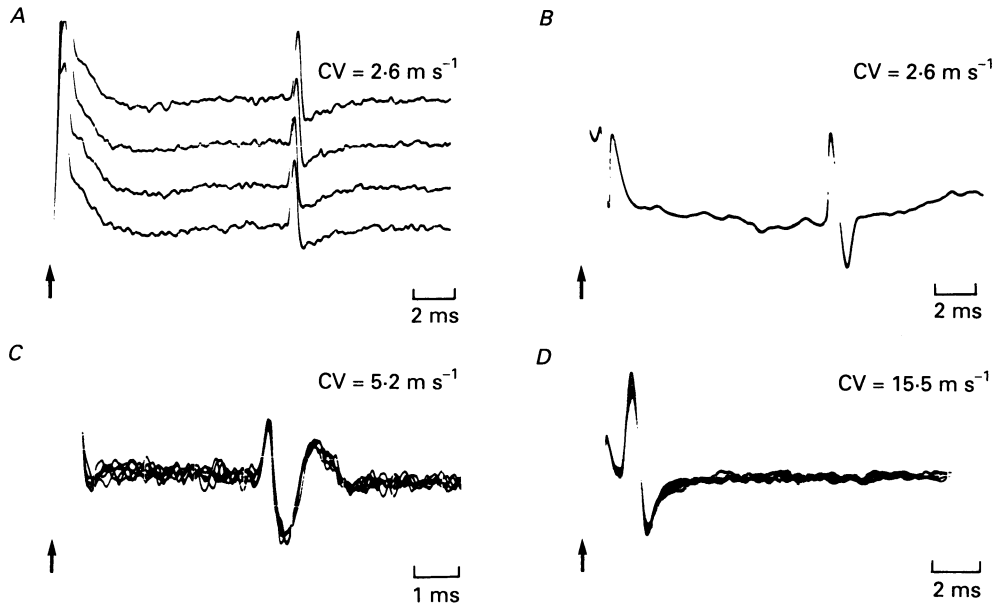

Fig. 2. Responses of four single periodontal Aδ fibres to suprathreshold stimulus pulses applied to the periodontal ligament of the cat's lower canine tooth. The arrows indicate the onset of electrical stimulation. Conduction velocities (CV) are given at the upper right corner. In *A* four traces are displayed separately; in *C* and *D* five traces are superimposed (repetition rate: 1 Hz).

The distribution of the conduction velocities of the ninety-seven recorded periodontal Aδ fibres using a class width of 1 m s^{-1} is shown in Fig. 3. It is asymmetrical with a high percentage of slowly conducting and a lower percentage of medium and fast conducting fibres: 34% of the identified periodontal Aδ fibres had conduction velocities below 5 m s^{-1} and more than half of them below 10 m s^{-1}. No sharp border between the distribution of the conduction velocities of C and Aδ fibres was found. This is demonstrated also by the inset in Fig. 3 showing the slowly conducting Aδ fibres ($\leqslant$ 7·5 m s^{-1}; filled bars) together with the C fibres (open bars) with the same class width of 0·5 m s^{-1}: a smooth transition between both classes of fibres can be seen. The mean conduction velocity ($\pm$S.D.) of the Aδ fibres was 11·0$\pm$7·7 m s^{-1} (n = 97; range: 2·6–28·2 m s^{-1}).

The thresholds of the nerve fibres to electrical stimulation of the periodontal ligament were determined as the lowest current intensity, using a 0·2 ms stimulus pulse, between any two of the four electrodes at a given pulse duration necessary to elicit an action potential. According to their activation thresholds, two groups of fibres could be distinguished. The average thresholds for periodontal Aδ fibres of the

first group were $134{\cdot}7 \pm 49{\cdot}0\ \mu A$ ($n = 52$; range: 8–200 μA). The second group, including forty-five Aδ fibres, had electrical thresholds exceeding the maximum output current of the stimulator (200 μA) using the 0·2 ms stimulus duration and are therefore given as stimulus duration at maximum current intensity: $1{\cdot}35 \pm 0{\cdot}72$ ms (range: 0·5–4 ms).

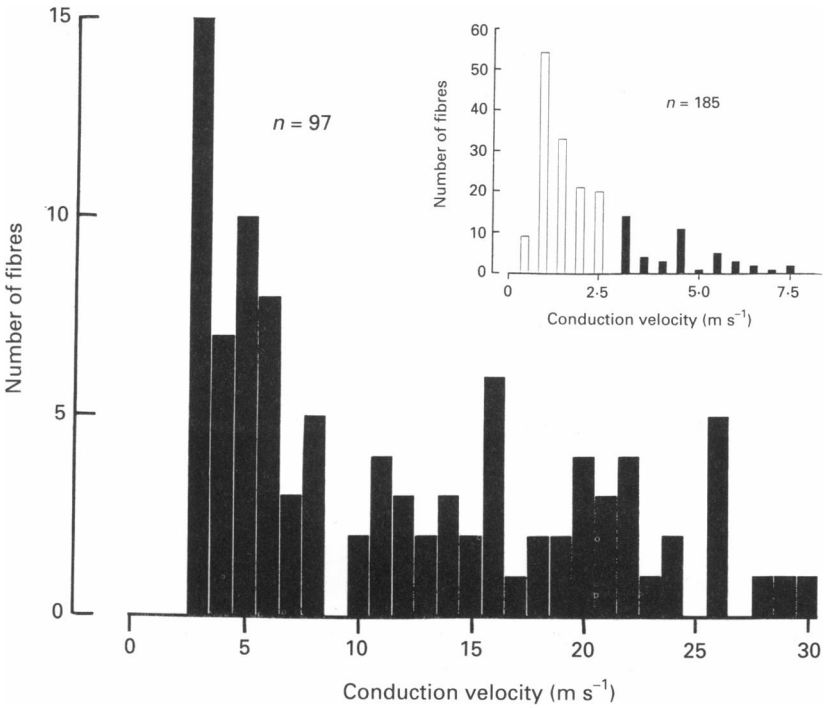

Fig. 3. Distribution of the conduction velocities, determined by electrical stimulation of the periodontal ligament for the ninety-seven identified periodontal Aδ fibres at a class width of 1 m s^{-1}. The inset shows the conduction velocities of slow conducting Aδ fibres up to 7·5 m s^{-1} (filled bars) compared to C fibres (open bars) at the same class width of 0·5 m s^{-1}.

The thresholds of periodontal Aδ fibres to electrical stimulation were inversely correlated with their conduction velocities, that is, the higher the threshold of a single fibre, the lower was its conduction velocity. While for the periodontal Aδ fibres with lower thresholds (that is, below 200 μA using 0·2 ms stimuli) the fitted function $y = 194{\cdot}6x^{-0{\cdot}2} + 8$ showed only a weak correlation ($r = 0{\cdot}36$), for the periodontal Aδ fibres with high electrical thresholds the function $y = 4{\cdot}88x^{-1{\cdot}42} + 0{\cdot}2$ ($r = 0{\cdot}97$) could be well fitted through these data. The periodontal C fibres, for comparison, had properties similar to that of the high-threshold group of Aδ fibres; their function is $y = 2{\cdot}14x^{-0{\cdot}66} + 0{\cdot}2$ ($r = 0{\cdot}86$). The equation derived from data of both periodontal high-threshold Aδ and C fibres is $y = 2{\cdot}54x^{-0{\cdot}61} + 0{\cdot}2$ ($r = 0{\cdot}85$), where y represents the mean electrical threshold and x the mean conduction velocity. This is shown in Fig. 4.

In Fig. 4*A* the electrical thresholds of Aδ (■) and C (●) fibres, expressed as the stimulus duration of a 200 μA stimulus pulse, are plotted against their conduction velocities. Figure 4*B* shows a portion of the data containing C and slow conducting Aδ fibres, the error bars indicating the S.E.M.

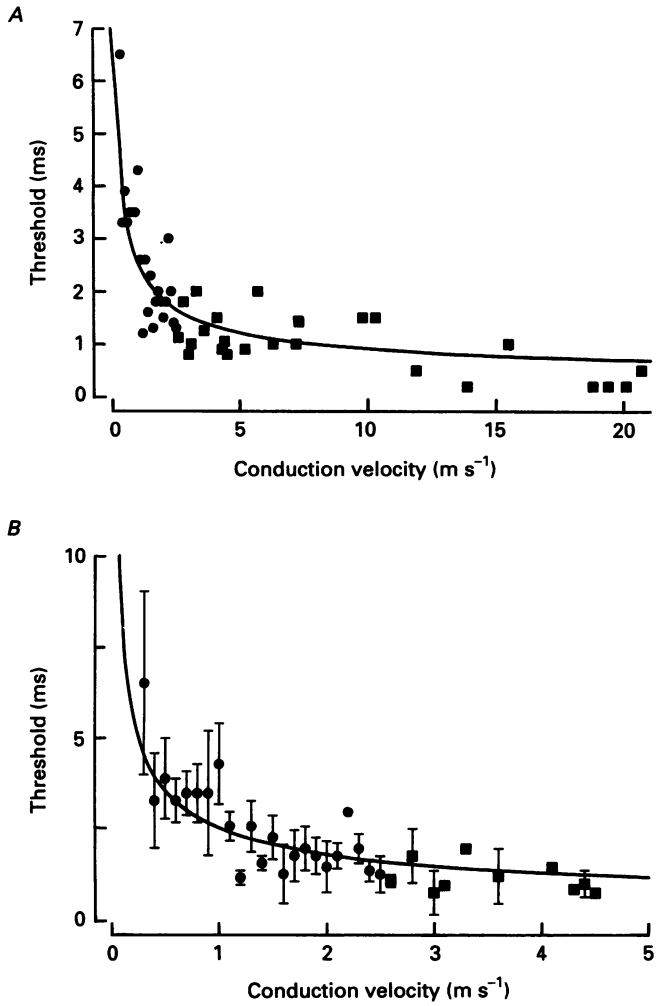

Fig. 4. Mean electrical thresholds of periodontal Aδ and C fibres plotted against their conduction velocities. The electrical thresholds are given as the stimulus duration of a 200 μA constant current pulse. *A*, electrical thresholds of Aδ fibres (■) and C fibres (●) plotted against the conduction velocity. To improve clarity, no error bars are shown. *B*, portion of the data shown in *A*, including C and slowly conducting Aδ fibres. The error bars indicate the S.E.M. The line fitted through the data points is given by the function $y = 2{\cdot}54x^{-0{\cdot}61} + 0{\cdot}2$, with the correlation coefficient $r = 0{\cdot}85$.

In four high-threshold Aδ fibres for different stimulus durations the threshold currents were determined. For the averages the Lapicque function $I = I_0/(1-e^{t/\tau})$ was fitted using the method of least squares (Fig. 5*A*), where I represents the mean

stimulus amplitude and t the mean stimulus duration. The time constant, τ, was determined as 0·65 ms. The rheobasic current I_0 was calculated by averaging the stimulus amplitudes for 5 to 10 s stimulus duration and was found to be 122 μA. For the chronaxie a value of 0·9 ms was determined using the fitted equation. The inset

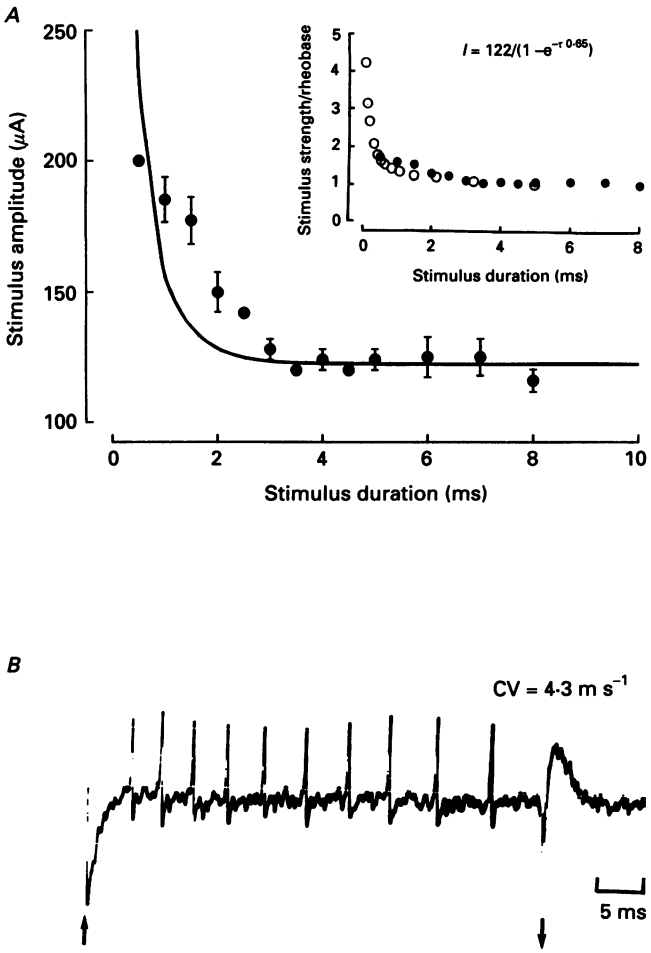

Fig. 5. *A*, mean stimulus – duration curve of four periodontal Aδ fibres. The line fitted through the data points is given by the function $I = 122/(1-e^{-t/0·65})$ where I is stimulus amplitude and t is stimulus duration. The error bars indicate the S.E.M. The inset figure compares our data (●) with those of Tasaki (1939; ○). The data points have been normalized by dividing them by the rheobase. *B*, repetitive response of an Aδ fibres to a 48 ms stimulus applied to the periodontal ligament. The arrows indicate the onset and the offset of the stimulus. The conduction velocity (CV) is given above the recording.

in Fig. 5*A* shows our results (●) in comparison with data obtained in classical experiments in the toad's nerve (Tasaki, 1939; ○), demonstrating a striking similarity. Data points of both studies have been normalized by dividing each value by the rheobase.

Though rarely tested with pulses of very long duration, repetitive discharges were found in three Aδ fibres. One of them is shown in Fig. 5*B*. During the 48 ms stimulus duration (maximum output of the stimulator), the fibre discharged ten times with increasing interspike intervals. Based on observations in voltage clamp experiments

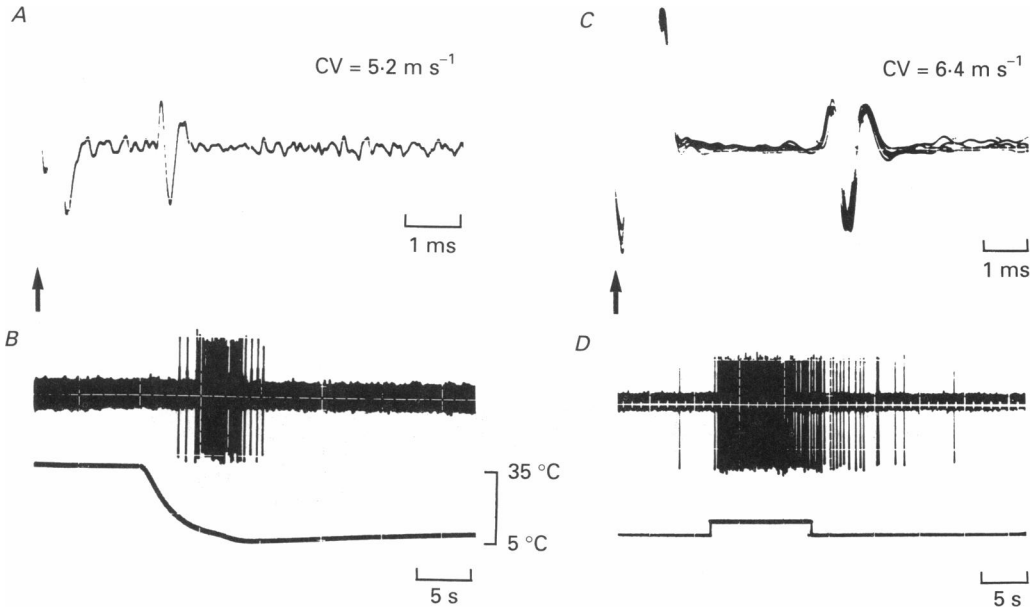

Fig. 6. Responses of two periodontal Aδ fibres to stimulation of the periodontal ligament with cold. *A* and *C*, electrical identification of the fibres. The arrows indicate the stimulus onset. The conduction velocity (CV) is given above the recordings. *B* and *D*, response of the fibres evoked by the application of a cold stimulus (dry ice) to the alveolar bone. The temperature trace in *B* indicates the time course of the temperature decay as measured on the surface of the alveolar bone near the stimulation site. The bar in *D* indicates the stimulus duration.

on the node of Ranvier in myelinated nerve fibres, repetitive discharges to steady current applications were interpreted as a hint of an afferent character of a fibre (Honerjäger, 1968).

Non-electrical stimulation

The stability of the recordings, and the amplitude of the recorded action potentials with respect to the noise level were adequate for studying the responses of sixteen of the ninety-seven identified periodontal Aδ fibres to non-electrical stimulation of the periodontal ligament.

The mechanical sensitivity of nine periodontal Aδ fibres was tested by applying strong mechanical force with a glass rod to the lower canine tooth from different directions. Application of a weak force activated none of the fibres. Seven of the fibres tested responded to strong forces. Although the forces were not quantified, much higher intensities were required for activation of Aδ fibres compared to the

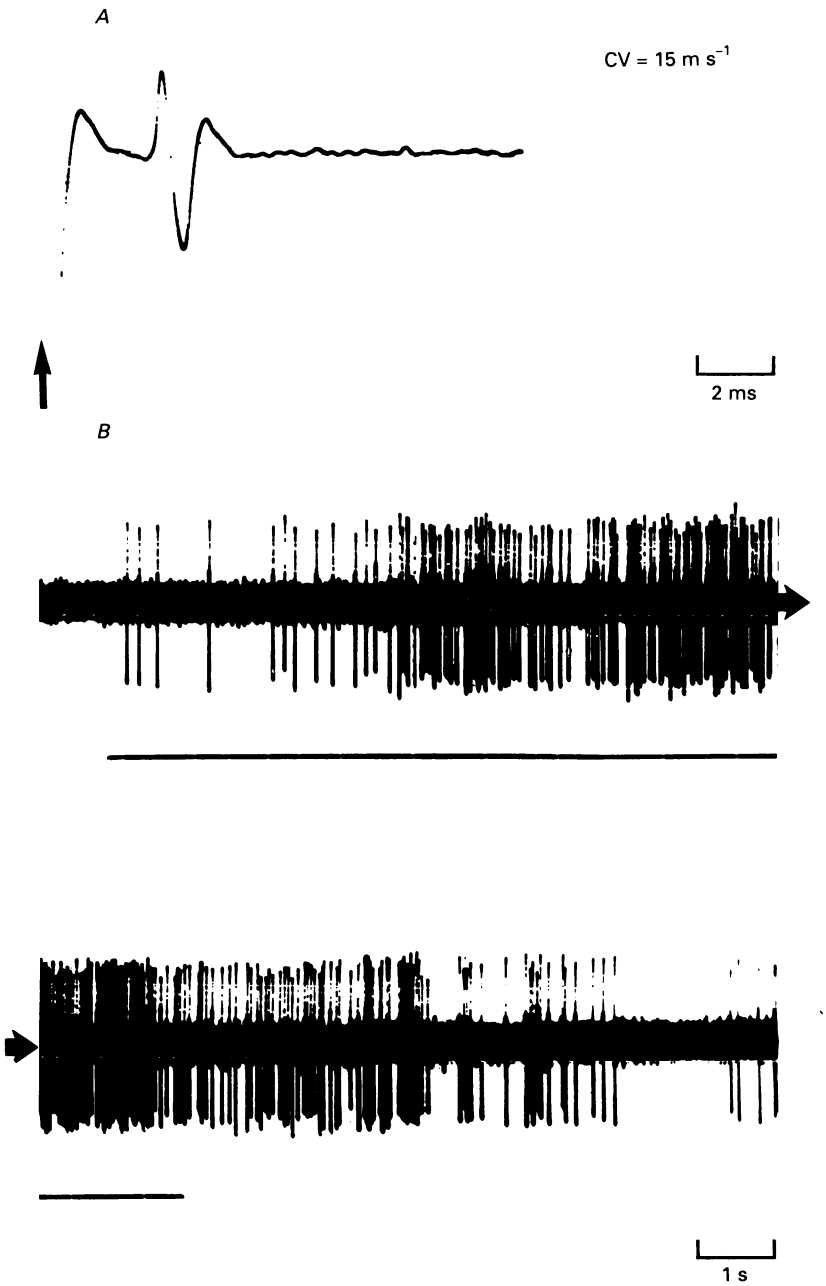

Fig. 7. Response of a periodontal Aδ fibre to stimulation of the periodontal ligament with heat. *A*, electrical identification of the fibre. The arrow indicates the stimulus onset. The conduction velocity (CV) is given above the recording. *B*, response of the fibre to heat applied to the alveolar bone. The bar indicates the stimulus duration. The recorded temperature in the adjacent electrode hole at the onset of the response was about 37 °C and increased to 49 °C at the offset of the stimulus. The amplitude of the spike was changed because of a movement of the filament on the recording electrode during the 15 min that elapsed between recordings *A* and *B*.

weak forces necessary to activate Aβ mechanoreceptors which were also observed but not studied in detail. The Aδ fibres responded strongest when stimulated from one direction but responded also to pressure from different directions with a lower frequency. The responses started at the onset of the force application and stopped at the offset showing practically no adaptation during the 10 s application time.

The effect of cold stimulation of the periodontal ligament was investigated in six periodontal Aδ fibres. Of these, four responded to this kind of stimulation. Two examples of the responses are given in Fig. 6. The electrical identification of these two fibres is shown in Fig. 6*A* and *C*; the responses to application of dry ice to the alveolar bone are shown in Fig. 6*B* and *D*. As shown in the lower traces of Fig. 6*B*, a response started after a latency of about 2 s, increased in frequency during temperature decay at the bone surface and stopped after the temperature reached a steady state.

The response latency to cold stimulation ranged from less than 1 s to 35 s. Of the four cold-sensitive periodontal Aδ fibres one was also activated by a strong mechanical force applied to the tooth crown and two fibres were activated by heat stimulation of the periodontal ligament as well. One fibre was activated by mechanical and both kinds of thermal stimuli.

Of the eight periodontal Aδ fibres tested, two responded to heat application to the alveolar bone overlaying the periodontal ligament. In Fig. 7 an example of such a heat-evoked response of a periodontal Aδ fibre is shown. The firing started after a latency of about 1 s when the temperature measured in the adjacent electrode hole had increased to about 37 °C. The stimulus was removed when the temperature had reached 49 °C. The discharge increased during temperature increase, was irregular and bursting, and the firing continued for nearly half a minute after the heat stimulus had been removed.

The responses of periodontal Aδ fibres to application of a saturated solution of potassium chloride into the periodontal space via one of the holes drilled for the stimulating electrodes were investigated in seven fibres. Two of them were activated by this type of stimulation. Only few discharges could be observed in these fibres. One of them could be activated also by strong mechanical forces and cold.

All of the fibres tested either by thermal and/or chemical stimuli were also tested by applying strong mechanical pressure to the exposed surface of the alveolar bone overlying the root of the tooth. None of the fibres could be activated by this mechanical pressure application.

DISCUSSION

The results of the present electrophysiological study, in agreement with recent morphological studies (Cash & Linden, 1982; Byers, 1985) prove the presence of periodontal Aδ fibres. The responses of the identified single periodontal Aδ fibres to natural stimulation of the periodontal ligament suggest that some of them may be polymodal nociceptors.

The 260 periodontal nerve fibres recorded were classified according to their conduction velocities determined by bipolar stimulation of the periodontal ligament. This type of stimulation could be shown to produce a reliable classification of periodontal nerve fibres (Mengel *et al.* 1992). Of these fibres, ninety-seven were Aδ

fibres, the majority of them slow conducting (below 10 m s^{-1}). In the distribution of the conduction velocities, a smooth transition between C and Aδ fibres was found. Despite our bias to slowly conducting fibres it seems that the Aδ fibres are not distributed according to a bell-shaped Gaussian distribution. Fibre spectrum analyses of the inferior alveolar nerve have shown that more than 50% of the fibres in this nerve are unmyelinated (Holland, 1978; Hoffmeister & Schendel, 1986).

According to their activation thresholds, two groups of periodontal Aδ fibres could be distinguished. While the first group had thresholds in the same range as intradental Aδ fibres (M. K. C. Mengel, E. Jyväsjärvi & K.-D. Kniffki, unpublished observation) and reasonably higher than periodontal Aβ fibres, the second group had thresholds similar to those of periodontal C fibres (Mengel *et al.* 1992).

Relatively high current intensities were needed for activation of periodontal high-threshold Aδ fibres compared to the current intensities required for activation of intradental fibres (Virtanen, Närhi, Huopaniemi & Hirvonen, 1983; Jyväsjärvi & Kniffki, 1989). On the other hand, the relationship between the thresholds of Aδ and C fibres was reasonable as demonstrated by the smooth transition between Aδ and C fibres when thresholds are plotted against conduction velocities (see Fig. 5). The relatively high thresholds might have been caused by differences in the arrangement of the conducting and isolating tissues within the periodontal space and the dental pulp. This may have led to a different distribution of current densities. The stimulation strength–duration curve, however, follows the classical law $I = I_0/(1-e^{t/\tau})$ with a time constant $\tau = 0{\cdot}65$ ms. For this value it has to be taken into consideration that the stimulus strength–duration relation derived here is based on data obtained by stimulation of the periodontal ligament and not the axons themselves. Thus, the time constant of the connective tissues within the periodontal space is also reflected in the value of τ. When the strength–duration curves are normalized by dividing each value by the rheobase and compared to strength–duration curves obtained by other authors even in different species (e.g. Tasaki, 1939) and normalized the same way, a striking similarity is found.

The thresholds of periodontal Aδ (and C) fibres were inversely correlated with their conduction velocities. This is in accordance with findings showing a correlation between conduction velocity and diameter and an inverse correlation of electrical threshold and fibre diameter, hence an inverse correlation between threshold and conduction velocity. In unmyelinated fibres, the electrical threshold is inversely proportional to the square root of the diameter, and the conduction velocity directly proportional to the square root of the diameter. Hence in unmyelinated fibres the electrical threshold is inversely proportional to the conduction velocity. For references on this topic see Jack, Noble & Tsien (1983).

These experiments provide no quantitative description of the response characteristics of the recorded Aδ fibres to non-electrical stimuli. This is because the accurate location of the receptive areas of the fibres could not be determined prior to thermal and chemical stimulation without excessive removal of the alveolar bone around the tooth. This in turn might have damaged the periodontal ligament and altered the excitability of the nerve fibres. Therefore, the exact intensity of the thermal and chemical stimuli *in situ* remained unknown. It cannot be ruled out that the stimuli used in the present study might have excited the axons directly (see Matthews, 1977). However, the constant responses to repeated applications of

thermal stimuli suggest that receptive structures were excited. Like the periodontal C fibres described previously (Mengel *et al.* 1992), none of the Aδ fibres showed any on-going activity without intentional stimulation, but some developed a long-lasting activity after a noxious stimulus. The response behaviour seems to be different from that described in skin afferents (Bessou & Perl, 1969) e.g., there were no low-threshold mechanoreceptors in our sample of Aδ fibres. On the other hand, the response characteristics of periodontal Aδ fibres seem to be similar to those of pulpal Aδ fibres (Jyväsjärvi & Kniffki, 1987).

The periodontal Aδ fibres unresponsive to non-electrical stimulation might have had their receptive fields too far away from the stimulation sites – that is, the alveolar bone between the electrode holes for thermal and the electrode holes themselves for chemical stimulation. They also might have been located outside the periodontal ligament and might have been electrically activated by spreading of the stimulating current: some nerve fibres pass the periodontal ligament to innervate the marginal gingiva, which had been removed in the preparation. Fibres that could be activated by electrical stimulation of both dental pulp and periodontal ligament and therefore were most probably branching, were excluded from the present study.

The response behaviour to cold stimulation of periodontal Aδ fibres was different from that of periodontal C fibres described earlier (Mengel *et al.* 1992) and similar to that described for intradental Aδ fibres (Jyväsjärvi & Kniffki, 1987): the fibres seemed to respond to the temperature gradient rather than to the final temperature level.

Some differences were found from the responses of periodontal Aδ fibres recorded by Mei *et al.* (1977) in the Gasserian ganglion: they could always record responses to potassium chloride. Since they injected the substance into the periodontal ligament, it is conceivable that the responses were elicited by mechanical stimulation of the receptors by the rather large amount injected (0·1–0·5 ml). On the other hand, as mentioned above, our stimulation site might have been too far away from the receptor. Mei *et al.* (1977) never found cooling responses, which might have been due to the stimulation method of cooling a needle inserted in the periodontium with a spray of local anaesthetic. This might have resulted in temperature gradients insufficient for evoking Aδ fibre responses. We agree with Mei *et al.* (1977) that Aδ fibre receptors (termed by them as type II receptors) are insensitive to weak mechanical forces known to activate periodontal mechanoreceptors and respond only to strong stimuli which will give pain sensations if applied to human teeth. This behaviour is very similar to that of nociceptive periodontal C fibres. The response characteristics of periodontal afferent Aδ fibres suggest that they might be nociceptors rather than mechanoreceptors.

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