Blood flow and vascular pressure in the dental pulp

Summary

KARIN J. HEYERAAS TØNDER

Institute of Physiology, University of Bergen, Bergen, Norway

Tonder, K.J.H. Blood flow and vascular pressure in the dental pulp. *Acta Odontol. Scand.* 1980, 38, in press

The present summary is a review and a discussion of the following papers (I – V), submitted as partial fulfillment of the requirements for the degree of Doctor Odontologiae at the University of Bergen.

I. Blood flow in the dental pulp in dogs measured by local H_2 gas desaturation technique. Arch.Oral Biol. 1975, 20, 73 – 79. Co-author: K. Aukland
II. The effect of variations in arterial blood pressure and baroreceptor reflexes on pulpal blood flow in dogs. Arch. Oral Biol. 1975, 20, 345 – 349
III. Effect of vasodilating drugs on external carotid and pulpal blood flow in dogs: «Stealing» of dental perfusion pressure. Acta Physiol. Scand. 1976, 97, 75 – 87
IV. Nervous control of blood flow in the dental pulp in dogs. Acta Physiol. Scand. 1978, 104, 13 – 23. Co-author: G. Næss
V. Microvascular pressure in the dental pulp and gingiva in cats. Acta Odontol.Scand. 1979, 37, 161 – 168. Co-author: G. Næss

Other papers are referred to in the conventional manner.

Key-words: Pulpal circulation; blood vessels; vascular resistance

Karin J.H. Tonder, Institute of Physiology, Årstadveien 19, 5000 Bergen, Norway

Modern endodontic therapy shows a high success rate (16), but a nonvital tooth is brittle and thus prone to fractures. Every precaution should therefore be taken to preserve the vitality of the pulp. The vitality of the dental pulp depends on its blood supply. To understand the pathological changes in the pulpal circulation and to treat them, the normal magnitude and regulation of pulpal blood flow (PBF) should be known.

A large number of studies has described the morphology and histology of the pulpal vessels (5, 6, 11, 18, 30, 31). The pulp is abundantly vascularized and its vessels arise from and drain into the same vessels as in the periodontium and jaw bone marrow (4). No arteries, only terminal arterioles, enter the pulp and the collateral circulation is distinctly limited. The walls of arterioles and venules within the pulp are particularly thin in relation to the diameter of the lumen (21).

The pulpal circulation has been studied by a number of techniques, including local isotope clearance (8, 13), isotope clearance as a fraction of cardiac output (20, 28) and isotope labelled microspheres (20, 28), giving resting flow rates in the dental pulp of dogs and cats varying from 0.2 to more than 2 ml/min per gram tissue. Qualitative variations in

Received for publication, November 14, 1979

PBF have also been estimated from photoplethysmography (2) and from changes in linear velocity of erythrocytes in exposed pulpal vessels (29).

The results of previous studies on the regulating mechanisms of PBF are in part controversial (19, 23, 34, 37, 38, 39). The discrepancies may partly be due to problems in interpreting the results from experiments where different parameters are measured. Thus, changes in hydrostatic pulp pressure (19, 37, 38) are impossible to evaluate in terms of PBF, since either a fall in precapillary resistance or a rise in postcapillary resistance may give the same changes in pulp pressure, but opposite effects on PBF. Conflicting findings concerning the effect of vasoactive drugs on PBF may be due to whether the drugs are applied intravenously, by arterial infusion or locally. Intravenous drug infusion (22) may effect the systemic circulation and secondarily cause changes in PBF. On the other hand, intra-arterial infusion (III, 9, 35) can change blood flow in the tooth's associated structures and thereby alter the pulp perfusion pressure. Furthermore, local application of drugs to the exposed pulp (24) may give widely varying drug concentrations and also mechanically traumatize the pulp and change the PBF.

Studies of the magnitude and regulation of PBF by the use of quantitative flow measurements are rare, partly due to methodological difficulties caused by the pulp's good protection within dentinal walls and also because of the small dimensions of the tissue. The applicability of the local H_2 gas desaturation method for measurements of PBF was therefore tested in the present study (I). The aim was to adapt this method for studies of the regulating mechanisms of PBF.

The greatest vascular resistance, and thereby the most powerful control mechanisms of blood flow, is generally located in precapillary vessels. In the pulp, however, a relatively high tissue pressure (3, 33) indicates that a considerable part of pulpal vascular resistance is located in postcapillary vessels. In order to localize the segmental vessel resistance, a direct method for measurements of blood pressure in the different pulpal vessel segments was used (40).

The specific aims of the study can be summarized as follows:

1. To adapt the local H_2 gas clearance method for repeated quantitative measurements of blood flow in the dental pulp, with simultaneous measurements in several teeth with or without anesthesia.
2. To study the local, nervous and humoral regulating mechanisms of PBF.
3. To examine the segmental vascular resistance in the pulpal blood vessels.

MATERIALS AND METHODS

The methods used have been described in the different papers and only a brief summary will be presented here. All experiments were conducted on canine teeth. For the studies reported in papers I–IV a total of 61 mongrel dogs were used as experimental animals. In paper V, 36 cats were used. The animals were anesthetized with sodium pentobarbital with supplements when necessary. In the dogs, a tracheal cannula was inserted, and rectal temperature was maintained at about 38 °C. An estimation of local arterial pressure was obtained by cannulating a small artery close to the experimental tooth, usually the lateral nasal artery (II, III). Blood flow in the common or external carotid artery was measured by electromagnetic flowmeter (III, IV).

Measurements of blood flow in the dental pulp (I–IV)

The uptake or removal of an inert substance solely by blood flow is given by the Fick principle (I). Assuming diffusion

equilibrium between tissue and venous blood, the regional blood flow can be measured by recording the arterio-tissue concentration difference of an inert substance during saturation or desaturation (17).

The present measurements of local blood flow in the dental pulp were made by continuous recording of tissue hydrogen gas concentration during desaturation (1). H_2 gas concentration in the dental pulp was recorded polarographically by platinum electrodes implanted in the pulp. A potential of +0.2V versus a Ag/AgCl reference electrode was applied to the platinum electrode. The electrode current generated by oxidation of molecular hydrogen to hydrogen ions at this potential is proportional to hydrogen concentration. H_2 gas was supplied by inhalation; when the inhalation was stopped, the hydrogen concentration of arterial blood fell rapidly to zero as hydrogen was removed by the lungs. Blood flow was calculated from the rate constant of the H_2 desaturation curve. The rate constant was obtained by plotting the electrode current caused by oxidation of H_2 on a logarithmic scale against a linear time scale (I).

Pulpal blood pressure measurements (V)

Blood pressure in microvessels in the exposed coronal or apical pulp of upper canine teeth was measured directly with a modified Wiederhielm (40) servo-controlled counter-pressure technique (15). The pressure transducer consisted of a glass micropipette with a sharpened tip of 1–4 μm diameter which was inserted into the vessels with a Leitz-Wetzlar micromanipulator. Exposure of the pulp and micropuncture of the vessels were performed under a Wild M5 stereo microscope, illuminated by a two-armed fiber-optic lamp.

Drug administration (III, IV)

In general, the lingual artery was cannulated in the retrograde direction for drug infusion. In the study reported in paper IV, atropine and phenoxybenzamine were injected intravenously. All drugs were diluted in 0.9 % saline and infused at a constant rate of 0.5 or 1.0 ml/min.

Nerve stimulation (IV)

The cervical sympathetic, the mandibular and inferior alveolar nerves were cut and stimulated in the efferent direction using a bipolar silver gap electrode with monophasic square wave pulses. The impulse duration was 0.5–6.0 msec, intensity 4–30 V, and the frequency was varied between 1–20 Hz. The stimulation periods were of 4 to 12 min duration.

RESULTS AND DISCUSSION

Resting blood flow

Measurements of PBF during Nembutal anesthesia averaged 0.15–0.17 ml/min · g (I–IV) which is in the same range as blood flow in cerebral white matter and four times as high as in resting skeletal muscles. Any overstimate of resting PBF by H_2 gas seemed unlikely because loss of H_2 gas from the pulp by routes other than blood flow was negligible (I) and inflammatory reactions caused by the electrode were not found by histological examination 5 and 10 days after implantation (I). An underestimate of PBF due to diffusion of H_2 gas from tissue and outflowing blood into the arterioles in the root canal, so called counter-current exchange, seemed more probable. The three to six times higher pulpal flow rates measured by microspheres (28, Tønder unpublished) and from the uptake of ^{86}Rb (20) might suggest a marked counter-current exchange of H_2 in the pulp. However, a possible counter-current exchange would involve all diffusible substances, including oxygen. The «effective nutritive» blood flow might therefore be closer to the H_2 desaturation rate than to absolute blood flow.

The pulp is a loose connective tissue and is basically similar to connective tissue elsewhere in the body (21). The present resting flow values of 0.15–0.17 ml/min · g therefore seemed relatively high when compared to resting blood flow in more cellular tissues, suggesting the PBF to be in excess of the pulpal metabolic requirements. Due to the small tissue volume it was considered unlikely that the pulpal circulation plays an important role in the thermal homeostasis of the body as a whole. However, the pulp has the same embryologic origin as the skin and the possibility of its participation in thermoregulatory reflexes cannot be completely ignored. Measurements of PBF in conscious dogs during ambient variations in temperature large enough to cause panting or shivering did not, however, give decisive evidence for a thermoregulatory control of the pulpal circulation (Tønder, unpublished observations).

In general, the pulpal circulation seemed to have much in common with skin circulation. The pulp may survive fairly prolonged severe reductions of blood flow without permanent damage. Thus, Olgart & Gazelius (25) have found that the pulp in cats recovered normal sensory nerve activity and blood flow in spite of several hours' severe reduction of PBF caused by adrenaline. Furthermore, it has been claimed that replanted teeth in young dogs may survive, even when parts of the pulp were without blood supply for days (32).

The primary task of the pulp is dentin formation and the profuse circulation is, no doubt, valuable in the repair of local insults or trauma to which the pulp is frequently exposed. But it appears unlikely that the slow formation of secondary dentin in healthy pulps, which reduces the size of the pulp chamber by some mm throughout life, should need resting flow rates of 0.15–0.17 ml/min · g. The excitability of the sensory units in the tooth is shown to be strongly modulated by changes in pulpal circulation (10), thus decreased PBF produced a fall in excitability. This seems to be the only known pulpal function which is influenced by a transient fall in resting blood flow. Hence, the metabolic or functional requirements of high resting blood flow in the pulp remain partly unexplained.

Control of pulpal blood flow

Since cardiovascular mechanisms tend to keep the systemic arterial pressure fairly constant, the blood flow in any tissue is mainly regulated by changes in vascular resistance, according to Poiseuille's law. Vascular resistance is mainly determined by the radius of the blood vessels. Changes in vessel radius are therefore of decisive importance for the tissue blood supply. Vessel radius may be changed in two ways: passively, due to altered transmural pressure across the walls of the vessels, and actively by changed activity in the smooth muscles in the vessel wall. The latter is the principal regulator of vascular resistance. An inherent activity in the smooth muscles is responsible for a basal vascular tone which keeps the vessels in a state of partial constriction. The vascular tone is under control of local, nervous or humoral mechanisms.

The finding that papaverine infusion nearly doubled PBF (III) showed that the pulpal resistance vessels did have a resting smooth-muscle tone. In other words, the pulp had the ability to actively increase its blood flow. On the other hand, due to the fairly high pulpal tissue pressure, which has been claimed to vary in proportion to arterial pressure (3), passive changes in vessel radius might also be of importance.

Intrapulpal vascular pressure

Arteriolar blood pressure both in apical and coronal pulp was found to be relatively low, on an average 43 mm Hg (V). Blood pressure in the pulpal venules

averaged 19 mm Hg (V), which was a relatively high venous pressure when compared to the 3–5 mm Hg pressure in the veins which drain most other tissues. Capillary pressure averaged 35 mm Hg (V). The pressure drop from the arterioles entering the dental pulp to the venules leaving was only one fifth of the total arterio-venous pressure difference. Approximately 65 % of the total arteriovenous pressure difference was located on the arterial side before the vessels entered the pulp, and about 15 % was located in the draining veins after leaving the pulp (V) (Fig. 1) The pulpal perfusion pressure was therefore low, indicating that the vascular resistance within the pulp itself is small, and that a considerable arterial as well as venous resistance was located extrapulpally. Accordingly, PBF may also be altered by changes in extrapulpal resistance, either pre- or postpulpally. At unchanged intrapulpal resistance, every fall in extrapulpal resistance in the arteries directly feeding or in the veins directly draining the pulp (series coupled vessels) will increase PBF, and every rise will decrease PBF. However, changes in vascular resistance in the vessels of neighbouring tissues which are supplied by the same arteries as the pulp, so called parallel coupled vessels, may also change PBF. Changes in parallel coupled resistance may alter PBF since the pressure gradient from the central arteries to the arterial ramification of the parallel coupled vessels will always be proportionate to the flow proximal to the ramification. Thus, a fall in parallel coupled vascular resistance will increase flow in the main common proximal artery and decrease pulpal feeding pressure and thereby cause a fall in PBF (III), in spite of unchanged intrapulpal resistance. A rise in parallel coupled resistance would have the opposite effect on pulpal feeding pressure and increase PBF. To what extent such changes in resistance will alter PBF depends on the ratio of the parallel

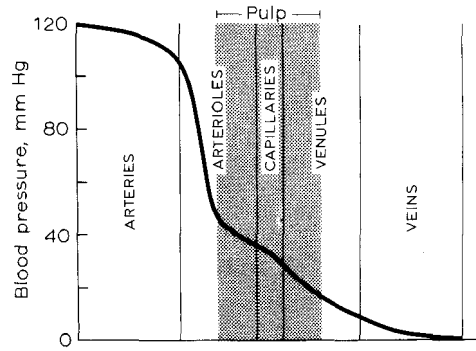

Fig. 1. Blood pressure profile along the extrapulpal and intrapulpal vessels.

coupled resistance (R_2) to intrapulpal resistance (R_p) and on the relative magnitude of the common series coupled resistance (R_1) as exemplified in Fig. 2. Obviously, the schematic analogue in Fig. 2 is extremely simplified as only one resistance is coupled in parallel (R_2) and one in series (R_1) to the intrapulpal resistance (R_p). However, the illustration shows that by unchanged R_p, a fall in R_2 decreases PBF and the greater the common series coupled resistance (R_1), the more the PBF will fall. It seems reasonable to assume that only larger parallel coupled vessels with a relatively high perfusion pressure will play any significance in «stealing» pressure, while every flow increase will lower the perfusion pressure of the «stealing» tissue as well.

Inter-individual differences in systemic arterial pressure in the range of 90 to 150 mm Hg were not associated with consistent changes in pulpal arteriolar or venular pressure (V). Hence, the pulpal perfusion pressure seemed to be maintained in the face of arterial pressure variations above 90 mm Hg. While no simultaneous flow measurements were made, these findings suggested a fall in prepulpal resistance relative to intra- and

postpulpal resistance at reduced arterial pressure. Depending on whether intrapulpal resistance was increased or remained unchanged, PBF may fall or remain unchanged.

The high tissue pressure (33) and the low arteriolar pressure (V) indicate an arteriolar transmural pressure of about 25 mm Hg. Even a moderate increase in pulp tissue pressure or fall in arteriolar blood pressure might therefore lead to a temporary «shut-off» of the arterioles (12). Apparently the pulpal arterioles may to some extent be protected against a luminal reduction due to passive elastic recoil of outer smooth muscle sheath by autoregulation of the feeding arteriolar pressure. The particularly small wall/radius ratio of the pulpal arterioles (21) would tend to reinforce this protection (12).

A rise in local tissue blood flow is generally due to a fall in precapillary resistance promoting increased capillary pressure and filtration. Because of the rigid encasement of the pulp, a low compliance, or low «distensibility» of the interstitial space, must be assumed. Hence, precapillary pulpal vasodilation with increased capillary pressure and filtration may cause a rise in pulpal interstitial pressure. A rise in tissue pressure would tend to compress the softwalled venules and thereby counteract a flow increase. However, the findings of the relatively constant pulpal arteriolar and venular pressure and the approximate equal distribution of intrapulpal pre- and postcapillary resistance (V) (Fig. 1) suggest that pulpal capillary pressure may stay approximately constant in spite of intrapulpal vasodilation or vasoconstriction. A proportional fall or rise in pre- and postcapillary resistances would leave capillary pressure and filtration unchanged. A selective fall in postcapillary resistance would thus increase flow and tend to reduce capillary pressure and favour fluid absorption. It might be speculated that

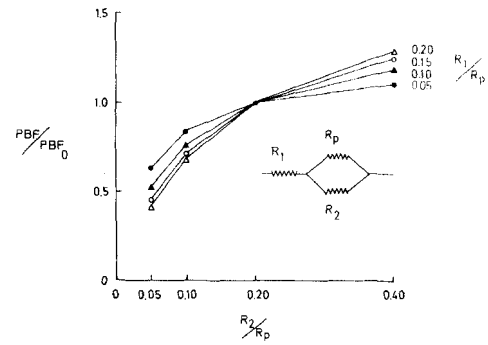

Fig. 2. A schematic, simplified analogue of the effect of variations in extrapulpal series-coupled (R_1) and parallel coupled vascular resistance (R_2) on pulpal blood flow (PBF) at constant intrapulpal resistance (R_P). Pulpal blood flow (PBF_0) at an assumed R_2/R_P of 0.2 is defined as 1.0.

the physiological significance of the high tissue pressure in bone marrow and teeth would be to prevent fluid accumulation during local inflammatory postcapillary vasodilation with increased capillary permeability.

Local control

Observations of a marked postobstructive hyperemia (I) suggest that local metabolites may influence vascular tone and possibly participate in pulpal flow regulation. Pulpal vasodilation induced by warming of the enamel surface (9) supports this view. However, PBF was not autoregulated when local arterial pressure was reduced (II). On the contrary, PBF was reduced proportionally more than local arterial pressure indicating increased pulpal vascular resistance. The increased resistance was not caused by nervous reflexes, as denervated teeth showed similar responses (II). Since there is no valid reason to suspect an increased vascular muscle tone, it seems most likely that the increased resistance is caused by lowered transmural pressure and passive reduction of the vessels' radius. PBF fell proportionately more than pressure in 15 out of 18 measurements, the average rise in resistance was 25% compared to the controls (II). It might be objected that

local arterial pressure was reduced below the autoregulatory range (II). However, while this might lead to a fall in PBF the calculated resistance should in any case be lower than the controls if autoregulation did exist. The measurements (II) did not rule out the possibility of autoregulation of PBF at local arterial pressures above 120 mm Hg.

Nervous control

The vasomotor nerves exert a remote control of blood flow in most tissues, securing an optimal distribution and adjustment of the circulating blood. The most important participants in this respect are the sympathetic adrenergic nerves.

Cutting of the cervical sympathetic nerve caused no change in PBF (IV) suggesting that the basal tones in the resting pulp were primarily due to inherent myogenic activity (III) and did not reflect sympathetic vasoconstrictor influence. Any pulpal vasodilation caused by reflex inhibition of resting vasoconstrictor discharge would therefore not be possible. A marked fall in PBF was observed during electrical stimulation of the cervical sympathetic nerve (IV) The pronounced vasoconstriction was ascribed to activation of α-receptors (IV,8) which were also shown to be activated through the carotid baroreceptor reflex (II). A vasodilation caused by activation of pulpal adrenergic β-receptors was not found, neither by sympathetic stimulation (IV) nor by close intra-arterial infusion of isoprenaline (III). On the contrary, infusion of isoprenaline gave a reduction in PBF, due to reduction of local arterial pressure (III).

However, as shown by micropuncture (V) the pressure fall from aorta to the pulpal feeding arterioles during control conditions, was much larger than previously assumed. The possibility exists, therefore, that the pressure drop in the pulpal feeding arterioles during isoprenaline infusion was on an average greater than 19 % of the controls, which was the average fall in PBF (IV). If this should be the case, isoprenaline must have reduced intrapulpal or postpulpal resistance correspondingly. The present measurements therefore seem inadequate to definitely exclude β-receptors in the pulpal vessels.

Atropine had no effect on PBF during stimulation of the cervical sympathetic nerve, thus providing no evidence of cholinergic sympathetic vasodilator fibers in the pulp (IV). In contrast, atropine consistently reduced the pulpal vasoconstrictor response caused by stimulation of the inferior alveolar nerve, indicating a cholinergic mechanism in the pulp, though not of sympathetic origin. If cholinergic vasodilation was induced in the extrapulpal tissues, atropine would cause decreased blood flow in these tissues, and a reversed «steal» effect could explain the rise in PBF. However, if the increased PBF should be caused by an inhibition of extrapulpal vasodilation, a near doubling of pulpal perfusion pressure compared to pressure during stimulation without atropine would be required (IV).

Antidromic stimulation of the trigeminal mandibular nerve caused pulpal vasodilation (IV) suggesting release of a vasodilatory substance at the sensory nerve endings in the pulp. This finding is in agreement with results recently reported by Olgart, Gazelius & Brodin (27). They found that inferior alveolar nerve stimulation after α-adreno-receptor blockade increased PBF, regardless of pretreatment with propranolol, atropine or mepyramine. An axon reflex, similar to that involved in the triple response in skin, would therefore seem possible also in the pulp, and might be activated following local insults to the pulp, as suggested by Edwall (7).

Remarkably high concentration of Substance P-Like Immunoreactivity (SPLI) has been found in dental pulps

from dog, cat and man (14) and antidromic stimulation of the inferior alveolar nerve has been shown to increase the concentration of SPLI in superfusate of the dental pulp (26). These findings may indicate that SPLI is involved in the pulpal vasodilation associated with sensory nerve stimulation.

Humoral control

Humoral control of blood flow is exerted through blood borne vasodilator and vasoconstrictor substances. In the dental pulp of dogs, close intra-arterial infusion of noradrenaline (NA) (IV) caused a marked vasoconstriction. The vasoconstrictor response of NA was shown to be clearly dosedependent (IV): Doses of 5 μg/min completely stopped the H_2 washout rate, while does lower than 0.01 μg/min gave no measurable effect on PBF. The vasoconstriction was partly bloked by α-blockers but was never replaced by a vasodilator response. Thus, the vasoconstriction was caused by occupying the α-receptors and NA infusion gave no evidence of pulpal β-receptors (IV). Adrenal medullary hormones, therefore, would be expected to decrease PBF. However, the quantitative importance of the humoral control of PBF seemed to be insignificant in relation to the sympathetic nervous control. The lower doses (0.01 to 0.5 μg/min), corresponding well to the catecholamine secretion during extreme asphyxia in cats (4a), gave only trivial pulpal vasoconstriction compared with that evoked by sympathetic nerve stimulation. In comparison, Celander (4a) observed that the vasoconstriction in cutaneous blood vessels obtained by direct sympathetic innervation was roughly 10 to 20 times greater than those elicited by the adrenal medullary output.

Vasodilator agents such as bradykinin (III) or histamine (9) have been shown to have little effect on PBF. This lack of dilating effect might possibly be explained by a reduction of pulpal feeding arteriolar pressure caused by increased flow to the tooth's neighbouring tissues, or by increased intrapulpal resistance caused by a rise in pulpal tissue pressure due to increased capillary permeability and increased capillary filtration (36).

CONCLUSIONS

a) The hydrogen gas clearance method seems to be a reproducible indicator of blood flow in the dental pulp. The main advantages of the method are that repeated measurements can be performed, and that PBF can be measured simultaneously in several teeth. H_2 desaturation rate may not equal absolute blood flow in the pulp but rather indicate effective flow with respect to diffusible substances.

b) The pressure drop between incoming arterioles and draining venules within the pulp is only 20% of the total systemic arteriovenous pressure difference. Since the resting pulpal blood flow is relatively high, the low pulpal perfusion pressure indicates a low vascular resistance within the pulp itself. A considerable arterial as well as venous vascular resistance to pulpal blood flow exists extrapulpally, and changes in extrapulpal resistance may also alter PBF.

c) Regulation of blood flow in the dental pulp seems to be dominated by nervous control. Activation of sympathetic adrenergic fibers causes vasoconstriction. Any pulpal vasodilation caused by sympathetic activation of β-receptors or reflex inhibition of resting sympathetic vasoconstrictor discharge was not found. Antidromic stimulation of the sensory fibers causes pulpal vasodilation. An axon reflex, similar to that involved in the triple response in skin therefore seems possible also in the pulp. Except for a postischemic reactive hyperaemia, there is little evidence for local control of vascular resistance in the dental pulp.

REFERENCES

1. Aukland, K., Bower, B.F. & Berliner, R.W. Measurement of local blood flow with hydrogen gas. Circ. Res. 1964, 14, 164 – 187
2. Beer, G., Negari, H. & Samueloff, S. Feline dental pulp photoplethysmography during stimulation of vasomotor nerve supply. Arch. Oral Biol. 1974, 19, 81 – 86
3. Brown, A.C. & Beveridge, E.E. The relation between tooth pulp pressure and systemic arterial pressure. Arch. Oral Biol. 1966, 11, 1181 – 1193
4. Castelli, W. Vascular architecture of the human adult mandible. J. Dent. Res. 1963, 42, 786 – 792

4a. Celander, O. The range of control exercised by the 'Sympathico-Adrenal System'. Acta Physiol. Scand, 1954, 32, Suppl. 116, 1 – 132

5. Corpron, R.E., Avery, J.K. & Lee, S.D. Ultrastructure of terminal pulpal blood vessels in mouse molars. Anat. Rec. 1974, 179, 527 – 541
6. Dahl, E. & Mjör, I.A. The fine structure of the vessels in the human dental pulp. Acta Odontol. Scand. 1973, 31, 223 – 230
7. Edwall, L. Some effects of sympathetic nerve activation in oral tissues as studied by tracer disappearance. Thesis, Department of Pharmacology, Karolinska Institutet, Stockholm, Sweden, 1971
8. Edwall, L. & Kindlová, 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
9. Edwall, L., Olgart, L. & Haegerstam, G. Influence of vasodilator substances on pulpal blood flow in the cat. Acta Odontol. Scand. 1973, 31, 289 – 296
10. Edwall, L. & Scott, D. jr. Influence of changes in microcirculation on the excitability of the sensory unit in the tooth of the cat. Acta Physiol. Scand. 1971, 82, 555 – 566
11. Eifinger, F.F. Die Mikromorphologie der menschlichen Zahnpulpa. Carl Hanser Verlag, München 1970
12. Folkow, B. & Neil, E. Circulation. Oxford University Press, New York. 1971, pp 53 – 55
13. Fontenelle, Ã., Meyer, E., Laliberté, H. & Desaulniers, G. Monoexponential analysis of Krypton-85 clearance curves obtained from canine dental pulp by means of a semiconductor probe. Int. J. Nucl. Med. Biol. 1973, 1, 101 – 103
14. Gazelius, B., Brodin, E. & Olgart, L. Release and degradation of substance P-like immunoreactivity (SPLI) in the dental pulp. Scandinavian Division of IADR (NOF). Abstract 1979, No. 40
15. Intaglietta, M., Pawula, R.F. & Tompkins, W.R. Pressure measurements in the mammalian microvasculature. Microvasc. Res. 1970, 2, 212 – 220
16. Kerekes, K. & Tronstad, L. Long-term results of endodontic treatment performed with a standardized technique. J. Endod. 1979, 5, 83 – 90
17. Kety, S.S. The theory and applications of the exchange of inert gas at the lungs and tissues. Pharmacol. Rev. 1951, 3, 1 – 41
18. Kramer, I.R.H. The distribution of blood vessels in the human dental pulp. In: Finn, S. B. ed. Biology of the Dental Pulp Organ: A symposium. Univ. of Alabama Press 1968. pp 369 – 379
19. Kroeger, D.C. Possible role of neurohumoral substance in the pulp. In: Finn, S.B. ed. Biology of the Dental Pulp Organ: A symposium. Univ. of Alabama Press 1968, pp 333 – 352
20. Meyer, M.W. Distribution of cardiac output to oral tissues in dogs. J. Dent. Res. 1970, 49, 787 – 794
21. Mjör, I.A. & Pindborg, J.J. Histology of the human tooth. Munksgaard, Copenhagen 1973, pp 54 – 61
22. Neidle, E.A. & Liebman, F.M. Effects of vasoactive drugs and nerve stimulation on blood flow in the tooth pulp and allied structures of the cat. J. Dent. Res. 1964, 43, 412 – 422
23. Neidle, E.A., Mauss, E.A. & Liebman, F.M. Effect of cranial nerve stimulation of orofacial vascular beds. J. Dent. Res. 1965, 44, 574 – 581
24. Ogilvie, R.W. Direct observations of the cat dental pulp microvascular response to electrical and drug stimuli. Anat. Record 1967, 157, 379
25. Olgart, L. & Gazelius, B. Effects of adrenaline and felypressin (octapressin) on blood flow and sensory nerve activity in the tooth. Acta Odontol. Scand. 1977, 35, 69 – 75
26. Olgart, L., Gazelius, B., Brodin, E. & Nilsson, G. Release of substance P-like immunoreactivity from the dental pulp. Acta Physiol. Scand. 1977, 101, 510 – 512
27. Olgart, L., Gazelius, B. & Brodin, E. Nerve mediated vasodilation in the cat dental pulp by sensory fibres? Scandinavian Division of IADR (NOF). Abstract 1979, No. 41
28. Path, M.G. & Meyer, M.W. Quantification of pulpal blood flow in developing teeth of dogs. J. Dent. Res. 1977, 56, 1245 – 1254
29. Pohto, M. & Scheinin, A. Microscopic observations on living dental pulp. I. Method for intravital study of circulation in rat incisor pulp. Acta Odontol. Scand. 1958, 16, 303 – 314
30. Provenza, D.V. Comparative morphology of the pulp vascular system. In: Finn, S.B. ed. Biology of the Dental Pulp Organ: A symposium. Univ. of Alabama Press 1968, pp 353 – 368
31. Seltzer, S. & Bender, I.B. The dental pulp. J.B. Lippincott Company, Philadelphia and Toronto. Second ed., 1975, pp 98 – 112
32. Skoglund, A., Tronstad, L. & Wallenius, K. A microangiographic study of vascular changes in replanted and autotransplanted teeth of young dogs. Oral Surg. 1978, 45, 17 – 28
33. Stenvik, A., Iversen, J. & Mjör, I.A. Tissue pressure and histology of normal and inflamed tooth pulps in macaque monkeys. Arch. Oral Biol. 1972, 17, 1501 – 1511
34. Sticht, F.D. Some factors influencing blood pressure of tooth pulp. Am. J. Physiol. 1967, 212, 1421 – 1428

35. Sicht, F.D., Woodbury, R.A. & Cox, B.A. The effects of certain drugs on the blood pressure within the tooth pulp. J. Oral. Thera. Pharmacol. 1966, 2, 421 – 435
36. Tonder, K.H. Effect of intraarterial infusion of bradykinin on pulpal blood flow. J. Dent. Res. Abstract 1975, 54, L561
37. Van Hassel, H.J. Physiology of the human dental pulp. Oral Surg. 1971, 32, 126 – 134
38. Van Hassel, H.J. & Brown, A.C. Effect of temperature changes on intrapulpal pressure and hydraulic permeablity in dogs. Arch. Oral Biol. 1969, 14, 301 – 315
39. Weiss, R.C., Tansy, M.F., Chaffee, R.B. jr. & Kendall, F.M. Functional control of intrapulpal vasculature: II. Physiologic evidence of a sympathetic cholinergic vasoactive system. J. Dent. Res. 1972, 51, 1350 – 1358
40. Wiederhielm, C.A., Woodbury, J.W., Kirk, S. & Rushmer, R.F. Pulsatile pressures in the microcirculation of frog's mesentery. Am. J. Physiol. 1964, 207, 173 – 176