"How Can Sensitive Dentin Become Hypersensitive?"

2

David H. Pashley

Abstract

When dentinal tubules first become exposed, patients note that those areas become more sensitive to tactile, evaporative, and osmotic stimuli. However, over time, especially with poor plaque control, those areas become progressively truly hypersensitive. There are a number of mechanisms responsible for hypersensitivity including localized pulpal inflammation, sprouting of pulpal nerves, and expression of "inflammatory" sodium channels. Often such hypersensitivity spontaneously disappears. These protective mechanisms will be reviewed. The problem arises for patients whose exposed dentin becomes hypersensitive and whose endogenous protective mechanism fails to correct the hypersensitivity.

2.1 Introduction

Brännström's (1962, 1992) hydrodynamic theory of dentin sensitivity proposed that hydrodynamic stimuli (hot or cold, tactile, evaporative or osmotic) caused sudden minute shifts of dentinal fluid that activate pulpal mechanoreceptors (Fig. 2.1) to cause sharp, well-localized tooth pain, thought to be due to A-delta sensory nerves (Narhi et al., 1992). A corollary of the hydrodynamic theory of pain is that anything that reduces dentin hydraulic conductance should decrease dentin sensitivity. Conversely, anything that increases dentin hydraulic conductance should increase dentin sensitivity. In this model, dentin hypersensitivity is equated with hyperconductance of dentinal fluid through tubules. In such a simple model, all things staying constant, increases in fluid flow should cause increases in dentin sensitivity. Unfortunately, all things do not stay constant.

2.2 Fluid Flow Models of Sensitivity

Our group was the first to report dentinal fluid flow across dog dentin in vivo (Pashley et al. 1981). In that same paper, we reported pulpal tissue pressures in dogs of 15–47 cm H_2O and that dentinal fluid flow was driven by pulpal tissue pressure (Fig. 2.2). This was later confirmed in humans in vivo (Ciucchi et al. 1995). In that paper, we reported pulpal tissue pressures of

D.H. Pashley, DMD, PhD
Department of Oral Biology,
College of Dental Medicine, Georgia Regents University,
1120 15th Street, CL-2112, Augusta, GA 30912, USA
e-mail: dpashley@gru.edu

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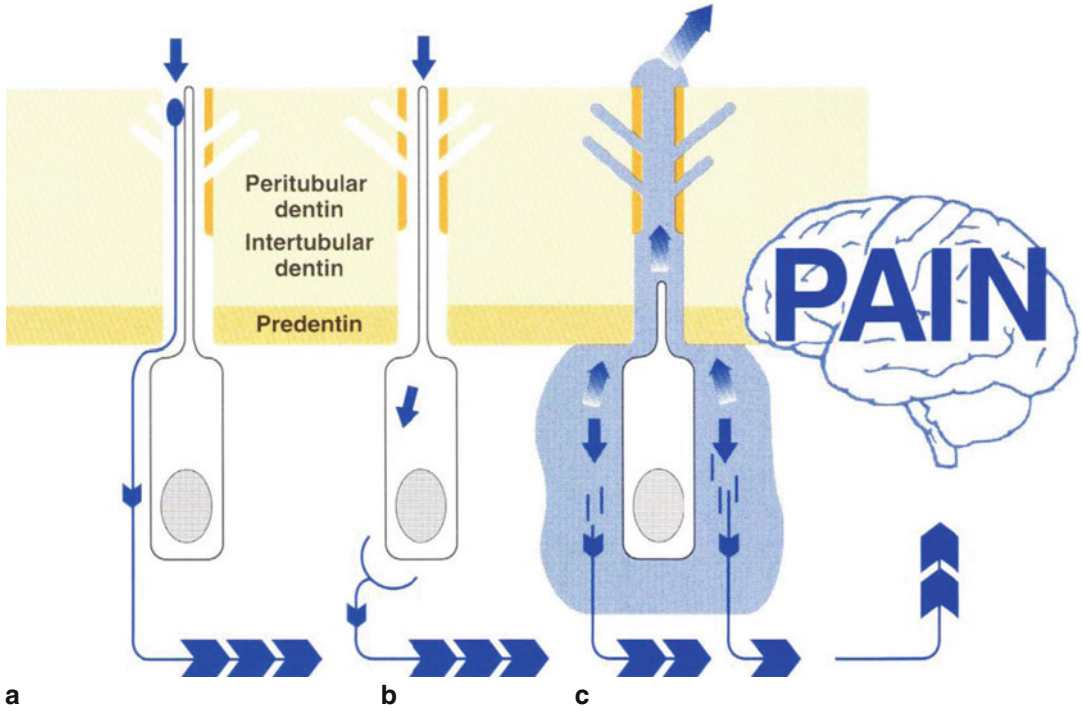

Fig. 2.1 Theories of dental pain. (**a**) Old theory that pulpal nerves extended out to the DEJ and made the DEJ sensitive. However, careful histology failed to demonstrate nerves at the DEJ. (**b**) In the 1950s, scientists thought that pulpal nerves synapsed with odontoblasts and that odontoblast processes extended to the DEJ. Careful histology revealed that odontoblast processes do not extend to the DEJ. (**c**) The hydrodynamic theory of dentin pain is that movements of dentinal fluid in tubules in either direction activate mechanoreceptor nerves in the pulp to cause dentin sensitivity (From Ten Cate (1998), p. 191 with permission)

Fig. 2.2 Flattened occlusal surface of a tooth in vitro filled with blue dye that slowly seeps across open dentinal tubules under a pulpal tissue pressure of 20 cm H_2O. The tubules over pulp horns are shorter and are located closer together, making these tubules very sensitive (From Pashley and Tay (2012), with permission)

14–15 cm of water that was similar to pulpal tissue pressure in cats of 16 cm H_2O reported by Vongsavan and Matthews (1992). Vongsavan and Matthews actually calculated the velocity of outward movement of dentinal fluid in cat dentin at 1.4 μm s^{-1}. This outward seepage of dentinal fluid flow occurs because the pulpal tissue pressure is 16 cm H_2O greater than atmospheric pressure in normal pulps. This slow outward fluid flow is too slow to activate pulpal nerves. Using radioactive ^{125}I, Pashley and Matthews (1993) confirmed that outward directed convective fluid flow could significantly restrict the inward diffusion of small molecules. After Nissan et al. (1995) showed that bacterial endotoxin can diffuse through human dentin, Puapichartdumcong et al. (2005) reported that outwardly directed fluid flow under 15 cm H_2O pressure could significantly lower the flux of endotoxin across dentin. Thus, although outward fluid flow reduces inward diffusion, it does not eliminate it.

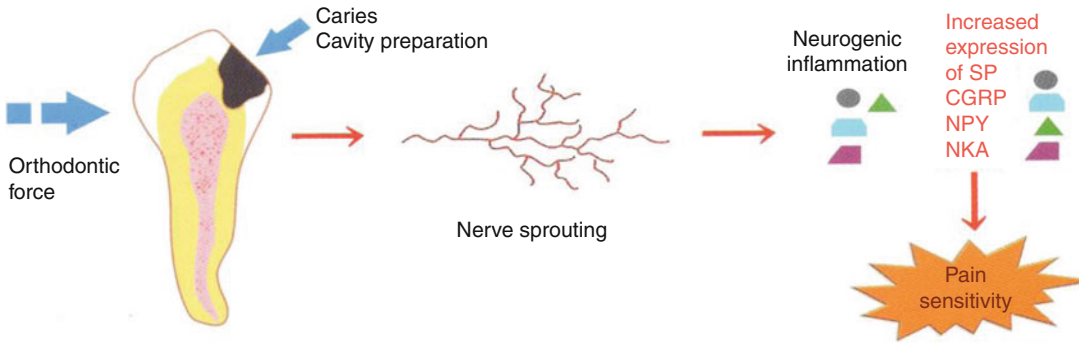

Fig. 2.3 Although this figure shows neuronal responses of the pulpodentin complex to caries, cavity preparation, or orthodontic tooth movement, many authorities believe that hypersensitive pulps undergo the same responses to inflammatory mediators (From Fouad (2012), with permission)

2.3 Effects of Inflammation of the Pulp

During pulpal inflammation, a large number of chemical mediators of inflammation are released, including histamine and serotonin, complement activation, and bradykinin release (Fouad 2012). Many of the mediators make pulpal nerves more sensitive than normal and cause the surrounding fibroblasts to divide more rapidly than normal. Fibroblasts release more nerve growth factor (NGF) that, in turn, causes nerve terminals to sprout and increase their neuropeptide content. This increase in nerve density is thought to be associated with the development of dentin hypersensitivity (Fig. 2.3). A receptive field in dentin is all of the dentinal tubules innervated by a single nerve and its branches. An increased size of receptive fields in inflamed pulps has been reported (Byers and Närhi 1999). Most of the nerves that sprout contain calcitonin gene-related product (CGRP) and substance P (SP). Both neuropeptides are known to vasodilate pulpal blood vessels and increase capillary permeability. These reactions lead to increases in tissue pressure (Heyeraas and Kvinnsland 1992; Berggren and Heyeraas 1999) and in outward fluid flow. This increase in outward fluid flow occurs not because dentin is more hyperconductive, but because of increases in local tissue pressure, the driving force for dentin fluid flow (Pashley 1992). As this fluid flows through narrow tissue spaces between pulpal nerves and dentin, the local shear forces on mechanoreceptors may bring the resting membrane potential of pulpal nerves closer to threshold, making them "hypersensitive" (Fig. 2.4). Working in cats in vivo, Matthews and Vongsavan (1994) reported that application of a negative pressure of −300 mmHg caused outward fluid flow to increase from 1.4 to 25 μm s^{-1} to fire pulpal mechanoreceptors. They calculated that fluid flow rates >1.5 nl s^{-1} mm^{-2} were required to activate intradental nerves in anesthetized cats.

2.4 Effects of Inflammation on Sodium Channels in Nerves

Inflammation also modifies the types of sodium channels that are distributed in sensory nerves. Sodium channels can be classified as tetrodotoxin sensitive or tetrodotoxin (TTX) resistant. When nerves are surrounded by inflammatory mediators, they not only sprout, they also upregulate new sodium channels (Na_v 1.8 and 1.9) that seem to be more easily activated (Fig. 2.5) than is seen in the absence of inflammation (Renton et al. 2005; Wells et al. 2007; Luo et al. 2008; Warren et al. 2008; Henry et al. 2009). Thus, in clinical cases of dentin hypersensitivity, if patients cannot successfully remove bacterial plaque on exposed hypersensitive dentin, that dentin may remain hypersensitive until some tubule occluding agent can reduce in inward flux of plaque products. These bacterial products sustain the localized pulpal inflammation, which

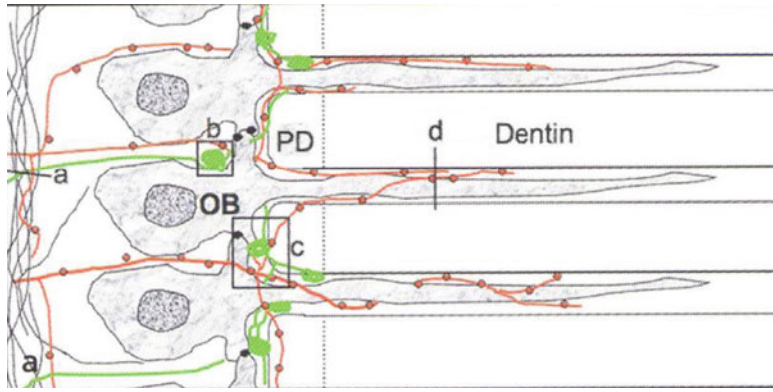

Fig. 2.4 Schematic of odontoblasts extending cellular processes out into deep dentin. Nerves in *red* (C-fibers) contain peptide neurotransmitters, substance P, and calcitonin gene-related peptide (CGRP). The larger *green* nerves are A-**δ** sensory nerves that carry sharp, well-localized pain of dentin sensitivity. *OB* designates the cell body of an odontoblast; *PD* designates the presence of predentin interposed between the odontoblast layer on the *left*, and mineralized dentin on the *right* (From Byers et al. (2012), p. 136)

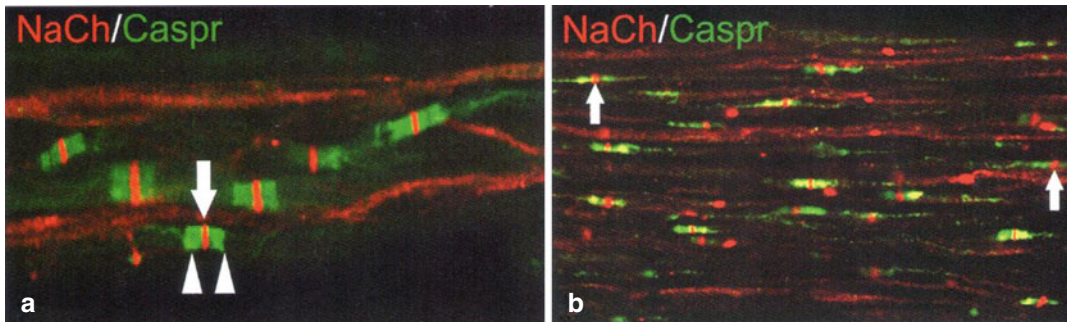

Fig. 2.5 (**a**) Confocal micrographs of sodium channels (*red*) Na_v 1.7 and Na_v 1.8 in pulpal axons and Caspr (*green*) that identify nodes of Ranvier in mylinated nerves in normal pulp. (**b**) On the *right* panel are shown those same neural proteins in inflammed pulps. Note the presence of more nerves in painful pulps (From Byers et al. (2012), p. 124)

makes pulpal nerves hypersensitive to hydrodynamic stimuli that might not normally cause pain.

Partial or complete tubule occlusion is an important part of treating dentin hypersensitivity, but plaque control is also very important. Tubule occluding agents may be more effective clinically if they include antibacterial agents that can control plaque.

When dentin is freshly exposed, it is very sensitive, even when the underlying pulp is healthy. However, if the freshly exposed dentin becomes covered with bacterial plaque, the tooth becomes hypersensitive when the pulps show signs of local inflammation beneath the exposed dentinal tubules (Lundy and Stanley 1969). What is in bacterial plaque that causes exposed dentin to become hypersensitive?

2.5 Effects of Bacterial Products on the Pulp

In 1962, Brännström recruited teenaged children whose premolars were scheduled for extraction for their orthodontic treatment. Under local anesthesia, he ground through the enamel of those premolars into the mid-coronal dentin to expose 5–10 mm^2 of dentin covered by smear layer

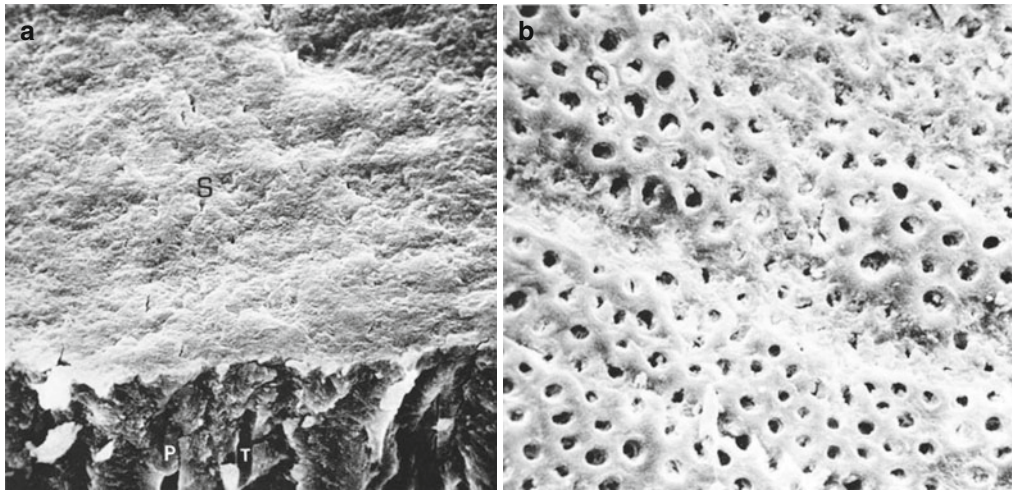

Fig. 2.6 (**a**, **b**) Brännström prepared shallow cavities in orthodontic premolars scheduled for extraction. The teeth were not restored but were left open to the oral cavity for up to 7 days. Sensitivity was scored immediately and after 1 week. The teeth were extracted and examined histologically and by SEM. On the left, the freshly prepared cavity dentin was covered with a smear layer. On the right, the smear layer dissolved from the exposed dentin after 1 week. Note how open are the tubules (From Figs. 14 and 15, Brännström (1981), with permission)

(Fig. 2.6a). He tested the initial sensitivity of the exposed dentin to air blasts and tactile probing and recorded their pain responses. He left the drilled cavities unfilled and exposed to saliva and oral bacteria. One week later, he had them return for rescoring of dentin sensitivity (Fig. 2.6b). He found that sensitivity had increased a great deal due, in part, to the loss of the smear layer. When the teeth were extracted for orthodontic treatment, and the pulps were examined histologically, he found that the pulpal surface of cut, exposed tubules that were allowed to dry in air, showed aspiration of some odontoblast nuclei into tubules. When dentin was exposed to saliva for a week, the pulps became infiltrated with acute inflammatory cells (Figs. 2.7a, b).

We now know that the ground dentin was covered by a smear layer that became covered with bacterial plaque within days. This biofilm created sufficient lactic acid to dissolve the smear layer and smear plugs (Kerns et al. 1991) within 1 week so that the dentin tubules would become hyperconductive. Thus, their increased dentin sensitivity was due, in part, to hyperconductive dentin (Brännström 1962).

2.6 Dynamics of Smear Layer Loss and Tubule Occlusion

Many dentists ask "how long do smear layers last on planed root surfaces?" Others ask how long it takes acid-etched dentinal tubules to close by remineralization. The answer to both of these questions is "about 1–2 weeks." In a now classic paper (Kerns et al. 1991), we used teeth extracted for periodontal reasons. Their cervical regions were root planed (Fig. 2.8a) and then divided into multiple slabs (2×3×1 mm): one served as a smear layer-covered control; another served as an EDTA-etched control with open tubules; another EDTA-etched specimen was treated with 3 % monopotassium-monohydrogen oxalate (pH 2.4) for 2 min to occlude the open tubules with crystals of calcium oxalate. Then the various dentin slabs were placed in denture flanges of denture patients for 1–4 weeks. At 1-week intervals, the dentin slabs were removed and processed for SEM.

Figure 2.8b shows dentin covered with a smear layer before it was placed in a denture flange. The image on the right shows a similar

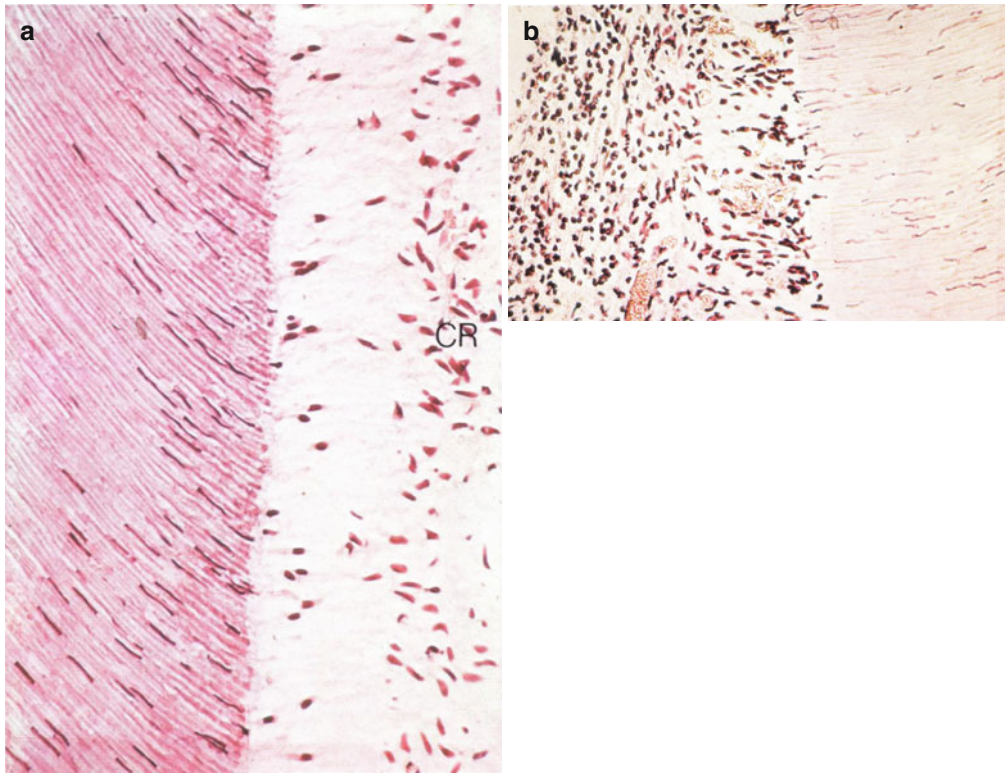

Fig. 2.7 (**a**) This tooth was allowed to air dry for 4 min and became painful after 20 s. Note the odontoblast layer has disappeared from beneath the dentin. Odontoblast nuclei are seen up inside the tubules, but no inflammation has occurred yet. One can easily identify the cell-free zone and the cell-rich (CR) zone (From Figs. 14 and 15, Brännström 1981, with permission). (**b**) This tooth's class V cavity was exposed to oral fluids for 1 week. The dentin was very painful. The pulp was heavily infiltrated with acute inflammatory cells. Pulpal capillaries were disrupted and venules were dilated (From Figs. 14 and 15, Brännström (1981), with permission)

sample that had been worn in the mouth for 1 week. Note that more than half the smear layer was lost and many open tubules were exposed. Figure 2.8c shows dentin treated with 18 % EDTA (pH 7) to remove the smear layer and expose open dentinal tubules. In the right panel is shown the appearance of similar dentin after 1 week where some of the tubules begin to be occluded by salivary salts. Fig. 2.8d show dentin that has remineralized for 2 weeks (left panel), while the right panel shows dentin that remineralized for 4 weeks. In Fig. 2.8e (left panel), dentin surfaces that were treated with 2.7 % acidic potassium oxalate for 1 min were covered with calcium oxalate crystals. In the right panel, similar dentin that had been in the mouth for 1 week showed loss of most of the calcium oxalate crystals and reappearance of some open tubules. However, some tubules remained occluded by calcium oxalate crystals below the surface.

Clinically, many patients do not complain of dentin hypersensitivity immediately after scaling and root planning (Fischer et al. 1991) but do so after 7–10 days. We believe that the lack of initial sensitivity is due to the smear layer created on root dentin by scaling; that then slowly dissolves over the next 7–10 days, making exposed dentinal tubules hyperconductive. If the patient remains untreated, these open sensitive dentinal tubules slowly begin to close by remineralization over the next 1–2 weeks. Thus, some patients may spontaneously heal but may be angry with their clinician. If patients who become hypersensitive in 7–10 days are then treated with 3 % potassium oxalate (BisBlock, Bisco Dental, Chicago, IL; SuperSeal, Phoenix, Fenton, Michigan; RemeSense), then

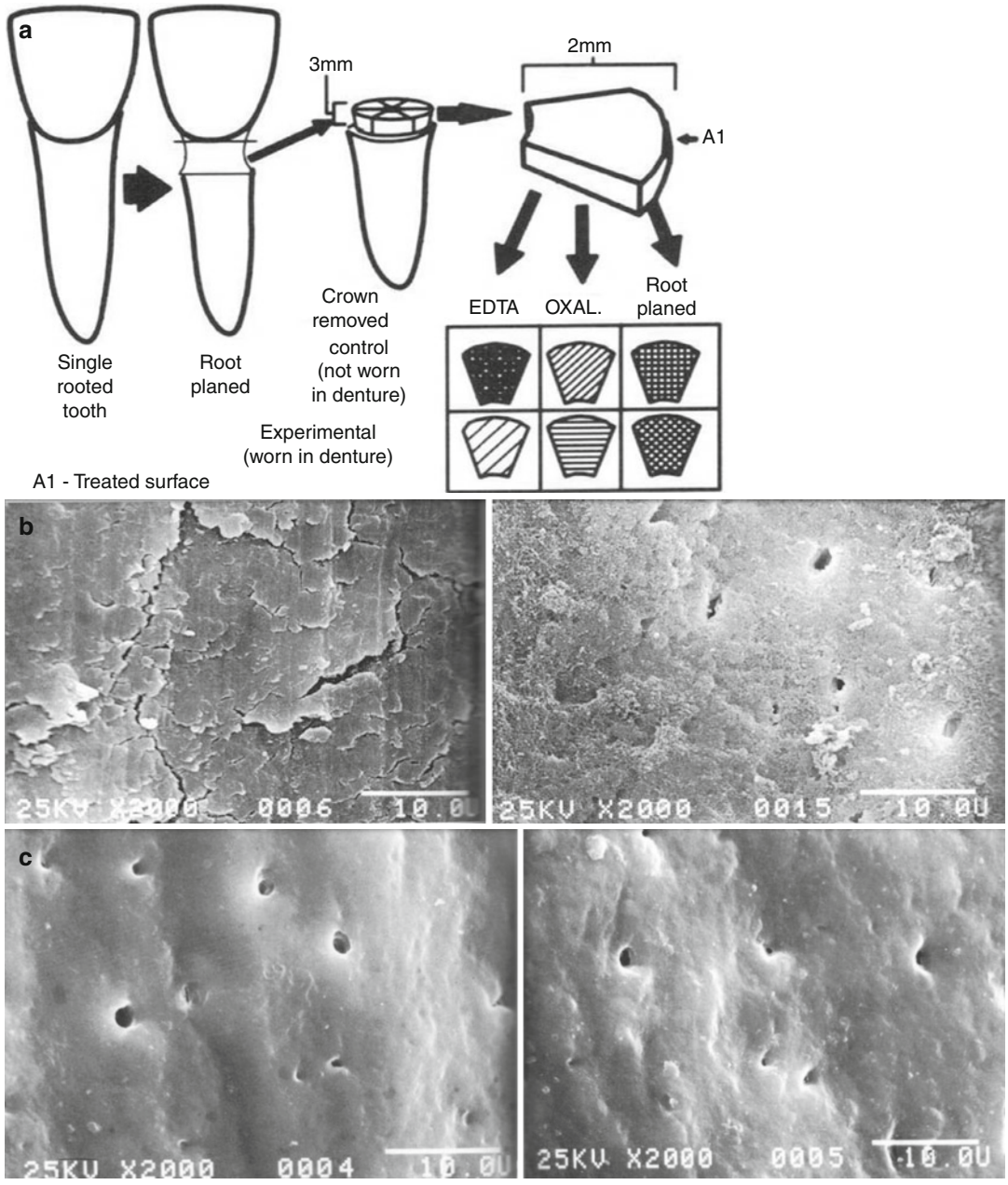

Fig. 2.8 (**a**–**e**) Kerns et al. (1991) placed dentin chips on denture flanges that were followed for up to several weeks. (**b**) Shows smear layer-covered dentin on the left. After 1 week, more than half the smear layer dissolved (*right*) opening many tubules. (**c**) If tubules were first opened with 18 % EDTA (*left*) and then exposed to saliva for a week, the tubules began to close by deposition of salivary mineral (*right*), which became more mineralized by 2 weeks more so (**d**, *left*) and more so after 4 weeks (*right*). (**e**) Dentin surface is covered with calcium oxalate crystals (*left*). After 1 week in the mouth, most of the calcium oxalate crystals on the dentin surface dissolved (*right*) (Kerns et al. (1991) with permission from the American Academy of Periodontology)

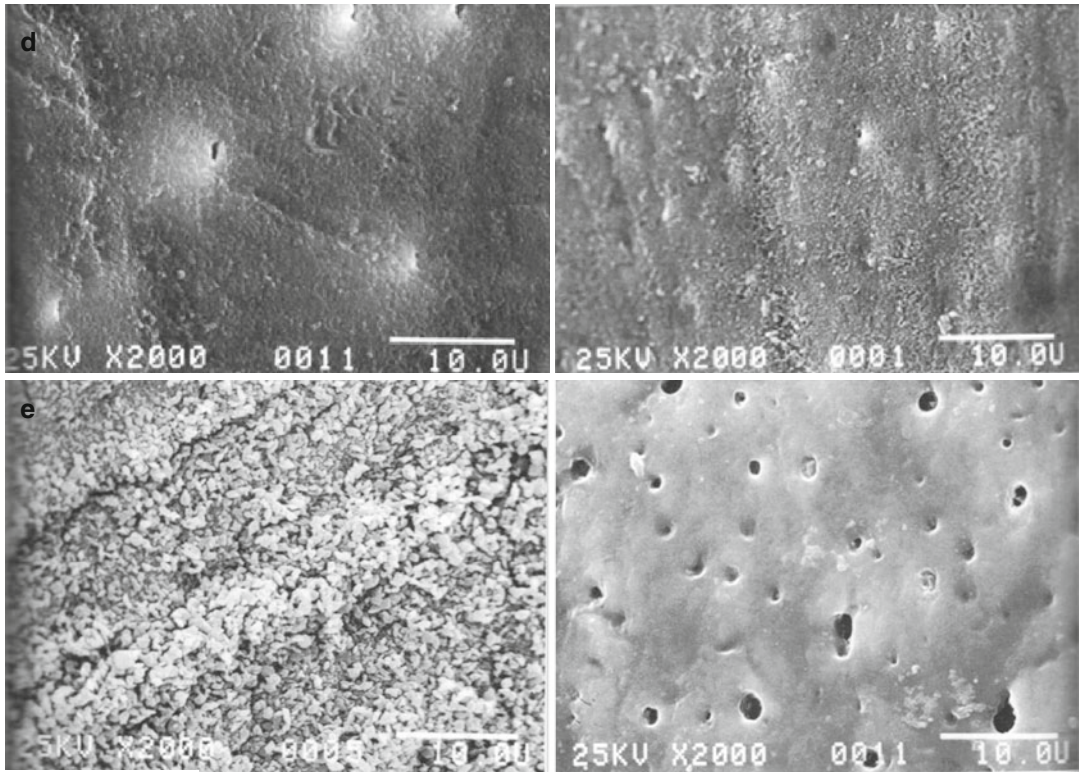

Fig. 2.8 (continued)

those open tubules can be closed temporarily with calcium oxalate crystals. An alternative to these professionally applied topical oxalates is the new potassium oxalate-containing desensitizing Listerine mouthwash called Listerine Advanced Defence Sensitive (LADS) (Fig. 2.9). This is only available in England but should be available in the USA this summer.

Remineralization of open dentinal tubules assumes that the patient has effective plaque control. If excessive plaque is frequently fed with fermentable carbohydrates, the plaque-derived lactic acid will etch away any attempts to remineralize. Patients who suffer from xerostomia do not have sufficient saliva to remineralize dentin.

2.7 Spontaneous Healing Mechanisms

The success of Brännström's (1962) use of oral fluids to induce pulpal inflammation under exposed, intact dentin encouraged Lundy and Stanley (1969) to repeat this experiment on a much wider range of patient ages (28–54 years), and to correlate their pain responses with pulpal histopathology over much longer times. Those authors recruited adult patients with asymptomatic teeth that were scheduled for extraction. Lundy and Stanley cut deep class V cavity preparations in those teeth under local anesthesia. The empty cavities were left exposed to saliva for 1–120 days. Just before extracting the teeth, the authors exposed these teeth to air blasts, probing, hot and cold stimuli, electric pulp testing, etc. and recorded the results. The teeth were then extracted and processed for light microscopy. When the authors correlated their clinical pain tests with the corresponding histopathologic results, they found that dentin sensitivity to air blasts and probing increased profoundly during the first week. The authors said that "it was quite evident that the degree of dentin sensitivity became very profound in the first week, sometimes becoming intolerable" (Table 2.1). The teeth with hypersensitive dentin were associated

Fig. 2.9 A dentin disk with open tubules was treated with listerine advanced defence sensitive (LADS) desensitizing mouthrinse containing 1.4 wt % potassium oxalate (pH 4.2) twice a day for 60 s, for 1 week. The disk was then fractured to permit examination of intratubular contents below the surface. This scanning electron micrograph shows a funneled tubule orifice of a dentinal tubule. Acid-etching, used to open the tubule of smear layer debris removed the hypermineralized peritubular dentin matrix from the top 3–4 µm of the tubule. When treated with soluble potassium oxalate (KOx) in Listerine, the KOx diffused down the tubule until it could interact with intact peritubular dentin. The acidic (pH 4.2) mouthrinse liberated ionized calcium from the peritubular matrix, that reacted with the soluble KOx to form insoluble crystals of calcium oxalate (*double white arrows*). *T* indicates the continuation of the tubule lumen. When dentin fractures, the fracture plane is not always straight. The tubule lumen disappeared between the opposing white arrows and the region below the *T*. Note that the calcium oxalate crystals become larger the deeper they penetrate down the lumen. (Sharma et al. 2013, with permission)

Table 2.1 Histopathologic pulpal reactions in teeth with open cavities

	Less than 12 days	Over 12 days
Number of specimens[a]	26	22
Average postoperative time interval	5.0	84.0
(days)	(1–11 days)	(25–240 days)
Average age of patients	41.1	42.0
Average tooth size (code)[8]	3.4	3.0
Average remaining dentine thickness (mm)	0.9	1.2
Average degree of displacement	1.9	0.6
Average degree of superficial response	2.2	0.5
Average degree of deep response	2.5	0.8

[a]For these computations, those specimens that presented preoperatively formed irregular dentine were not included. From Lundy and Stanley (1969)

with acute inflammatory cells on their pulpal terminations of only the cut tubules. However, the subjective symptoms and histologic reactions were completely different at longer time periods (e.g., >12 days). The patients no longer complained of sensitivity to hot and cold food and drink even though the cavities remained open.

Bergenholz and Lindhe (1975) were intrigued by the possibility that bacterial plaque on dentin could cause pulpal inflammation. They prepared

Table 2.2 Histological response of monkey pulps to place extracts for 8 h (monkey 1) or 30 days (monkeys 2 and 3)

		Monkey 1		Monkey 2		Monkey 3	
		Test	Control	Test	Control	Test	Control
Mean remaining dentin thickness ± s.d. (mm)		1.1 ± 0.5	0.8 ± 0.4	1.0 ± 0.5	0.9 ± 0.5	1.1 ± 0.3	1.0 ± 0.2
Degree vascular labeling	0	0	4	8	7	2	5
	1	0	1	0	1	4	0
	2	6	1	0	0	0	0
	P-value	$P<0.01$		$P>0.1$		$P>0.01$	
Degree cell infiltration	0	5	5	2	6	0	4
	1	0	0	3	2	5	1
	2	1	1	3	0	1	0
	P-value	$P>0.1$		$0.05<P>0.1$		$P<0.05$	

Bergenholtz and Lindhe (1975) tested effects of water-soluble plaque extracts applied to exposed dentin for 8 h (monkey #1) or 30 days (monkeys 2 and 3). Each monkey received intravascular injection of colloidal carbon to label pulpal blood vessels just before sacrifice. Note high vascular labeling in monkey 1 and high leukocyte cell infiltration in pulps of monkeys 2 and 3

class V cavities in the cervical root dentin of monkeys. Then an extract of human dental plaque were sealed in the test cavity for 32 h causing the accumulation of bacterial plaque on those surfaces. After 32 h, they sacrificed the first monkey and examined the pulps by light microscopy. Those teeth treated with plaque extract exhibited pulpal inflammation, while control teeth treated with sterile buffer had no signs of pulpal inflammation (Bergenholz and Lindhe 1978). They also cut deep class V cavities in monkeys and then applied pooled human supragingival plaque extract to dentin every 5 min for 8 h (Table 2.2). Even water extracts free of bacteria caused pulpal inflammation under intact root dentin (Bergenholz 1981).

We believe that the works of Brännström (1962), Lundy and Stanley (1969), and Bergenholz (1977, 1981) provide the rationale for why patients with exposed cervical dentin show less dentin hypersensitivity if they maintain good plaque removal habits (Drisko 2007).

They showed that freshly exposed dentin is sensitive, but not hypersensitive. It becomes hypersensitive when bacterial products from septic saliva or bacterial plaque diffuse down exposed dentinal tubules and induce localized pulpal inflammation beneath the exposed tubules. Inflammation induces nerve sprouting, upregulation of a new class of sodium channels, and elevations in pulpal tissue pressure. These changes, combined with the loss of smear layers over the initial 5–7 days, cause an elevation in baseline outward fluid flow that is sufficient to bring intradental nerves closer to their activation thresholds, allowing previously innocuous hydrodynamic stimuli to cause severe pain.

Although Lundy and Stanley (1969) did not say it in their classic paper, Stanley later concluded that because there was no irritation dentin formation in the severely inflamed pulps, the disappearance of symptoms in the unfilled cavities over 12–90 days must have been due to decreases in dentin permeability that had no histologic correlates.

2.8 Spontaneously Healing of Hypersensitive Dentin

To investigate this possibility, Pashley et al. (1984a) cut class V cavities in the molar teeth of anesthetized dogs. They measured the permeability of the cavity dentin every hour for 6–8 h. They were surprised to find that in dogs with vital pulps, the permeability of the dentin fell about 15 % per hour for 6–8 h (Fig. 2.10). This was done under isolation so that neither saliva nor bacteria could touch the dentin. When pulpotomies were done in teeth before preparing the cavities, the permeability of the teeth did not decrease at all but increased over time. This confirmed that vital pulps somehow could decrease the permeability of dentin over time.

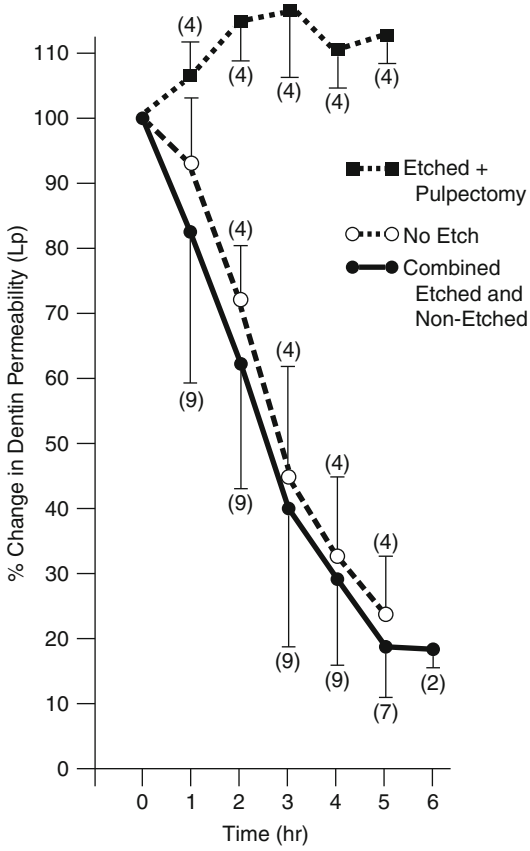

Fig. 2.10 Dentin permeability was measured in dog molars every hr for 6 h after preparing and acid-etching class V cavities. The *dotted line* shows results obtained in nonvital (pulpotomized) teeth. In those teeth, permeability increased over time. The *solid line* shows a 15 % decrease in dentin permeability each hr for 6 h. Each value is mean of number of teeth shown in parentheses ± 1 SD (From Pashley et al. (1984b), with permission)

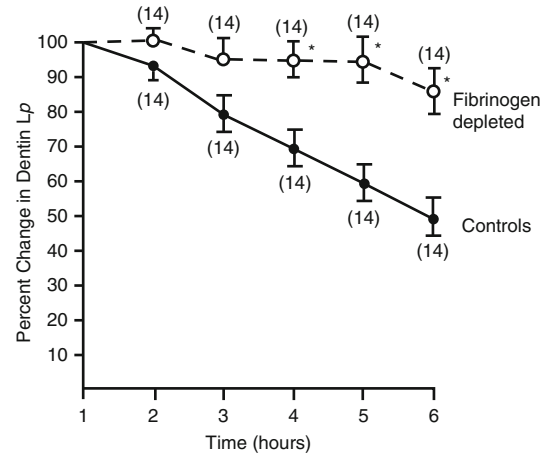

Fig. 2.11 Changes in dentin permeability of dog dentin in vivo over time. The *solid lines* are data obtained from control dogs over 6 h. The *dotted lines* indicate results obtained in dogs that were depleted of all of their plasma fibrinogen before the experiment began. Apparently, fibrinogen and other large plasma proteins leak out of irritated pulpal capillaries and partially occlude dentinal tubules over time (From Pashley et al. (1984a), with permission)

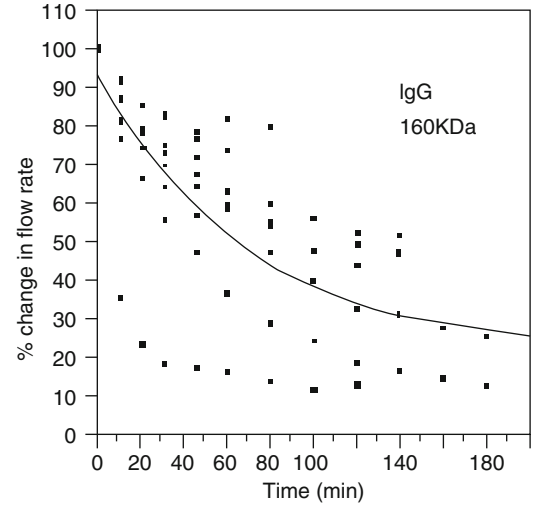

Fig. 2.12 Decrease in dentin permeability in vitro when dilute IgG (100 μg/ml) was filtered from pulp side of dentin to occlusal side for 3 h (From Hahn and Overton 1997 with permission)

Apparently pulpal irritation by cavity preparation and fluid filtration induces pulpal inflammation that increases the permeability of pulpal capillaries and venules to plasma proteins. In the patients of Lundy and Stanley (1969), this outward leakage of plasma proteins may have stopped the inward diffusion of salivary solutions of bacterial products, allowing the pulps to undergo healing even though the dentin remained exposed. When Pashley et al. (1984a) depleted dogs of fibrinogen in vivo prior to cutting cavities into dentin, the decrease in dentin permeability over time was greatly reduced (Fig. 2.11). Large plasma proteins like fibrinogen and immunoglobulins (Fig. 2.12) leak out of these vessels and adsorb to the walls of the tubules where they partially occlude them.

Thus, the pulpodentin complex is not a passive set of structures but is a vital, dynamic

complex that reacts to external trauma by a series of events designed to protect the pulp from external threats.

The inflammatory mediators that are released during pulpal inflammation include histamine, bradykinin, prostaglandins, and complement. The inflammatory mediators sensitize pulpal nerves and increase local pulpal blood flow and local tissue pressure, causing an increase in outward fluid flow, much like the well-documented increase in outward gingival fluid flow that is seen in inflamed gingival tissues (Pashley 1976). If this increase in outward fluid flow approaches the critical outward fluid velocity reported by Charoehlaro et al. (2007) of 5.8 nL/s mm^2, the A-δ nociceptors beneath that exposed dentin might become "hypersensitive" to hydrodynamic stimuli relative to their preinflammed sensitivity.

2.9 Clinical Considerations: Methods of Dentin Sensitization

We speculate that during periodontal surgery, roots are planed free of calculus, inadvertently removing cementum and exposing cervical dentinal tubules. The newly exposed dentin is covered by a smear layer generated by curettes. Over a period of 5–10 days, this surface becomes colorized by multiple species of oral bacteria to form a biofilm. As this biofilm metabolizes ingested sucrose, it produces sufficient organic acids to remove the smear layer over 5–7 days and make that dentin both hyperconductive and more sensitive. The loss of the smear layer increases the permeability (Kerns et al. 1991) of dentin to bacterial products (Ferraz et al., 2011), causing pulpal inflammation that makes pulpal nerves hypersensitive. This localized pulpal inflammation increases capillary permeability to plasma proteins including immunoglobulins, fibrinogen, and albumin; these molecules diffuse into dentinal tubules and adsorb to the tubule walls causing decreases in dentin permeability (Pashley et al. 1981, 1984a, 1985) over the next 7–8 days. This allows the transient dentin hypersensitivity to spontaneously resolve without any treatment. However, in a minority of patients, this spontaneous resolution does not occur and these patients remain hypersensitive. The mechanism of this prolonged hypersensitivity is unknown. Perhaps these patients have experienced multiple episodes of pulpal inflammation that have healed by scar formation causing a loss of pulpal capillaries near dentin. The lack of local capillaries might prevent leakage of plasma proteins into exposed dentin. Alternatively, patients may generate too much outward dentinal fluid flow that flushes these plasma proteins from the tubules preventing tubule occlusion. Such patients need to be treated with a topical tubule occlusion agent (Gluma or potassium oxalate-containing products like SuperSeal or Listerine Advanced Defence Sensitive) to reverse their hypersensitivity. Tubule occlusion lowers the inward diffusion of bacterial products, allowing resolution of local pulpal inflammation, reversing nerve sprouting, allowing fewer more sensitive sodium channels (Na_v 1.8 and 1.9) to be expressed in pulpal nerves, and decreasing dentin sensitivity. In this scheme, bacterial products and pulpal inflammation must be controlled if we are to prevent the development of dentin hypersensitivity.

Future occluding treatments for dentin sensitivity should include antimicrobial agents (Sharma et al. 2004; Charles et al. 2013) and anti-inflammatory agents that can all act in concert to prevent the development of localized pulpal inflammation beneath open, sensitive dentinal tubules.

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