



## **Acute dental pain I pulpal and dentinal pain**

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*Published in:*  
Tandlaegebladet

*Publication date:*  
2016

*Document version*  
Publisher's PDF, also known as Version of record

*Document license:*  
[Unspecified](#)

*Citation for published version (APA):*  
Närhi, M., Bjørndal, L., Pigg, M., Fristad, I., & Rethnam Haug, S. (2016). Acute dental pain I: pulpal and dentinal pain. *Tandlaegebladet*, *120*(2), 110-119.

## ABSTRACT

## Acute dental pain I: pulpal and dentinal pain

The specialized anatomy of the pulp-dentin complex and the dense, predominantly nociceptive pulpal innervation from the trigeminal nerve explains the variety of pain sensations from this organ.

Brief, sharp pain is typical of A-fibre-mediated pain, while long-lasting, dull/aching pain indicates C-fibre involvement. A-fibres react to cold or mechanical stimuli, such as cold drinks or toothbrushing, whereas C-fibres are mainly activated by inflammatory mediators. Thus, lingering pain suggests presence of irreversible pulpal inflammation.

During pulpitis, structural changes of the pulpal nerves (sprouting) occur and neuropeptide release triggers an immune response; neurogenic inflammation. Pain sensations during pulpitis can range from hypersensitivity to thermal stimuli to severe throbbing. There might also be aching pain, possibly referred and often difficult to localize. Thus, diagnosis is challenging for the clinician.

Surface biofilm amplifies hypersensitivity of exposed dentin surfaces because irritants reach the pulp through open dentin tubules, producing inflammation. Removing the biofilm reduces dentin hypersensitivity, but supplemental treatment, aiming to reduce dentin permeability, is often necessary. Caries removal and filling therapy are adequate measures during reversible pulpitis if the pulp has maintained its ability to distance itself from the bacterial assault by producing reparative dentin. However, endodontic therapy is necessary when pulpitis has reached an irreversible stage.

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# Acute dental pain I: pulpal and dentinal pain

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Pain localized to teeth is among the most frequently experienced orofacial pain complaints, with a prevalence of 12% in the general population within a 6-month period (1). Tooth pain may be attributed to a variety of conditions, which may be acute or chronic in nature, local or systemic in origin, but is most frequently an indication of damage or disease in the tooth or its surrounding tissues. A good understanding of structures and mechanisms underlying the painful sensation is a prerequisite to pain management.

### Innervation of the dental pulp and dentin

The dental pulp resides in a rigid capsule consisting of dentin and enamel. This creates a low-compliant environment that makes the pulp tissue unique (2,3). The dental pulp is richly innervated mainly by axons from the trigeminal nerve, predominantly sensory in nature and mainly committed to pain perception (nociception). A smaller population of pulpal nerves are autonomic sympathetic fibres emanating from the superior cervical ganglion and associated with pulpal vasoconstriction (4).

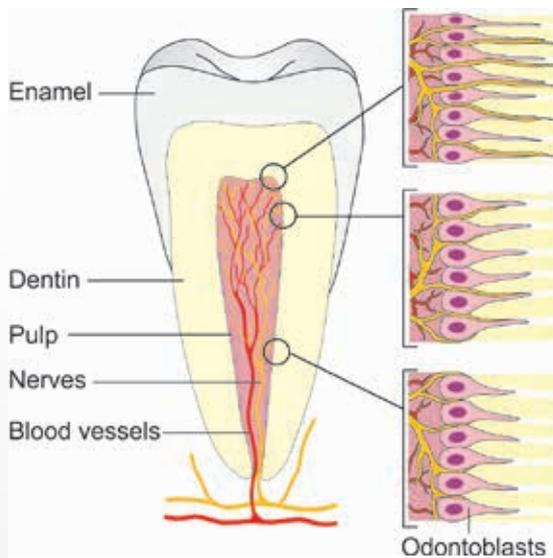
Extremely strong pain – reaching the maximum intensity at any pain score – can be induced by activation of intradental nerves (5-7). Such intense pain responses can be explained by the dense (Figs. 1 and 2) and predominantly nociceptive innervation of the pulp and dentin (6,8). The transmission of the pain-inducing stimuli through dentin from its exposed surface

#### EMNEORD

Dental innervation; pulpal and dentinal pain; dentin sensitivity/hypersensitivity; inflammation

is exceptionally effective and allows even very light stimuli, such as air blast and probing, to be intensified in a way that may induce tissue injury and subsequent nerve activation at the pulp-dentin border (5). Each tooth is innervated by about a thousand trigeminal axons (9-11), which may have branched before entering the apical foramen and may innervate more than one tooth. In the radicular pulp, the nerve fibres are bundled together, but once they reach the coronal pulp (8,12,13), they divide into smaller bundles. The axons then branch extensively and each may form 50-100 terminals in the peripheral pulp, forming a network under the odontoblast layer, known as the plexus of Raschkow. The density of nerve endings is especially high in the pulp horns, where as many as 50% of the dentinal tubules are innervated. Many of the tubules contain multiple nerve terminals (8). There are approximately 20 000-30 000 nociceptive nerve endings/mm<sup>2</sup> in the pulp-dentin border area in the most coronal pulp which accounts for the extremely high sensitivity of dentin.

### Intradental innervation



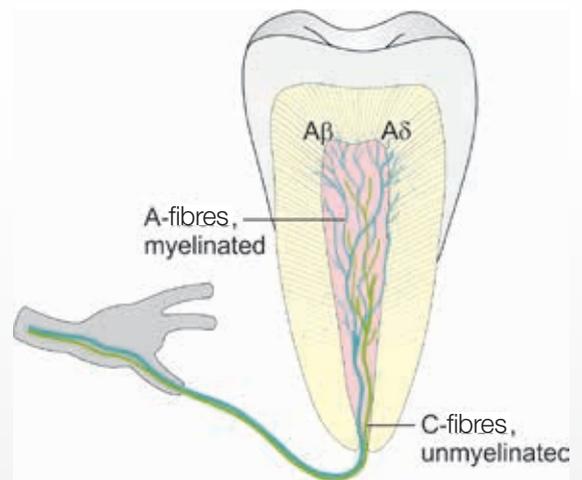
**Fig. 1.** Schematic presentation of the intradental innervation. The nerve bundles enter the pulp via the apical foramen/foramina and branch extensively especially in the coronal pulp. The pulp-dentin border zone in the peripheral pulp (pulp tips) is the most densely innervated area, where the nerve endings also extend the longest distance (100-150  $\mu$ m) into the dentinal tubules.

**Fig. 1.** Skematisk præsentation af den intradentale nerveinnervation. Den pulpale indgang for nervefibre sker gennem det apikale foramen/foraminae og forgrener sig ekstensivt særligt i den koronale del af pulpa. Pulpa-dentin-grænsen i den perifere del af pulpa (indbefattende pulpahorn) er det tætteste nerve-innerverede område, hvor de enkelte nerveender har deres længste udstrækning i dentintubuli (100-150  $\mu$ m).

### Nerve fibre types: A- and C-fibres, their functional differences

There are both myelinated (20-25%) and unmyelinated (75-80%) afferent nerve fibres in the pulp (8,12,13). These two fibre groups differ greatly in their functional properties (6,7,14,15). The myelinated fibres belong predominantly to the A $\delta$ -fibres but a part of them belong to A $\beta$ -group. These fibres are fast conducting (from 3 up to 50-60 m/s (6,7,14,15)). The A-fibre endings are located in the peripheral pulp and inner dentin (Figs. 1, 2 and Table 1). They are responsible for dentin sensitivity, and their activation in healthy teeth results in sharp and usually short-lasting pain, not outlasting the stimulus (5-7,16).

### Intradental A- and A-fibres



**Fig. 2.** Illustration showing the distribution of intradental A- and C-fibres. Unmyelinated C-fibres are located in the pulp proper, whereas myelinated A-fibres are extensively distributed in the peripheral pulp border, penetrating the inner part of dentin.

**Fig. 2.** Illustration af fordeling af intradentale A- og C-fibre. C-fiber har ikke en myelinskede og er lokaliseret i det centrale pulpavæv, imens A-fiber har en omsluttende myelinskede. Disse fibre er ekstensivt fordelt langs med pulpa-dentin-grænsen, hvor de penetrerer den inderste del af dentinen.

### Activation of intradental nerves

Activation of intradental nerves	
<b>Myelinated nerves</b> (Lower threshold)	<b>A<math>\beta</math>-fibres</b> (7%) – Vibration <b>A<math>\delta</math>-fibres</b> (93%) – Air, heat/cold, drilling/probing
<b>Unmyelinated nerves</b> (Higher threshold)	<b>C-fibres</b> – Inflammatory mediators, mechanical deformation, heat

**Tabel 1.** Stimuli capable of activating the intradental nerves.

**Table 1.** Stimuli, der aktiverer intradentale nerver.



There are also a number of larger Aδ-fibres (approximately 10% that enter the pulp at the apex). These are not active in the healthy pulp but become active when inflammation is present. This is an example of ‘peripheral sensitization’ when normally non-noxious nerve fibres are recruited to the pain system. All the sensory nerve fibres that enter the pulp branch and get narrower as they travel to the coronal part. Four times as many nerve fibres can be counted at the mid-crown level of the pulp than at the apical level. Myelinated nerve fibres commonly have non-myelinated terminals, making it difficult to differentiate the terminals of fast and slow fibres (13,17).

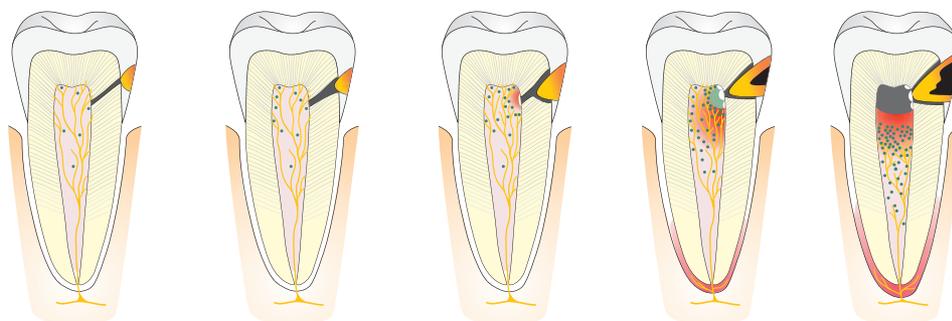
The non-myelinated nerves are C-fibres having slow conduction velocities (0.5-2.5 m/s) and their terminals are located in the pulp proper. They are predominately sensory with a small

population of sympathetics (10%). The majority (70%) of the axons entering the apex are C-fibres.

The C-fibres are polymodal and respond to several different noxious stimuli. In other sites they are activated by intense heat and cold and many inflammatory mediators such as histamine and bradykinin (7). In the pulp they are activated during inflammation, and increasingly so in its advanced stages (7). It seems that they may conduct the dull pain or ache in pulpal inflammation (5,7).

In addition, nerve fibres release biologically active peptides, known as neuropeptides, which influence neural activity and functioning (18). Neuropeptides in the dental pulp are released from the nerve terminals of mainly Aδ- and C fibres. There are numerous neuropeptides in the dental pulp which are com-

**Caries progression**



	STAGE 1	STAGE 2	STAGE 3	STAGE 4	STAGE 5
<b>Vascular effect</b>	No/few changes	Some vasodilation	Vasodilation	Vasodilation	Vasodilation in vital pulp
<b>Neural changes</b>	No/few changes	Little sprouting	Sprouting	Extensive sprouting	Extensive sprouting in vital pulp
<b>Immune cell infiltration</b>	No/few changes	Some	Some	More	Extensive with edema formation
<b>Histology</b>	No changes Some dentin sclerosis	Early signs of reparative dentin	Reparative dentin	Abscess formation Reparative dentin	Zone of necrosis Reparative dentin
<b>Symptoms</b>	Symptom free	Symptom free Sensitivity to hot/cold Hypersensitive	Symptom free Sensitivity to hot/cold Hypersensitive Tender to percussion	Symptom free Sensitivity to hot/cold Hypersensitive Acute pain Tender to percussion	Symptom free Sensitivity to hot/cold Hypersensitive Acute pain Tender to percussion Swelling
<b>Pulp diagnosis</b>	Normal	Reversible pulpitis	Reversible pulpitis	Irreversible pulpitis	Necrosis (partial to complete)
<b>Periapical diagnosis</b>	Normal	Normal	Normal Apical periodontitis	Normal Apical periodontitis	Normal Apical periodontitis (acute/chronic)
<b>Treatment</b>	Remineralization or caries removal and restoration	Remineralization or caries removal and restoration	Caries removal and restoration	Endodontic therapy	Endodontic therapy

**Fig. 3.** Schematic illustration of the 5 stages of caries progression from the enamel layer to pulp exposure with subsequent changes in the dental pulp (vascular, neural, immune, histological), possible symptoms and suggested treatment.

**Fig. 3.** Skematisk illustration af 5 stadier af caries. Progredierende fra en emaljeflesion til klinisk eksponering af pulpa med tilhørende reaktioner i pulpa (vaskulært, neuralt, immun-responsivt og histologisk), samt med mulige symptomer og forslag til behandling



monly classified as sensory, sympathetic or parasympathetic according to the origin of nerve fibres. Sensory and sympathetic neuropeptides are synthesized in the trigeminal and superior cervical ganglion, respectively (19).

Sensory neuropeptides are e.g. calcitonin gene-related peptide (CGRP), substance P (SP) and neurokinin A. Neuropeptide Y (NPY) is co-released with noradrenaline from the sympathetic nerve terminals. The most abundant neuropeptide in the dental pulp is CGRP, followed by SP. CGRP is a vasodilator while SP increases capillary permeability. NPY is a vasoconstrictor and modulates the immune function (20). When injected into the blood stream of experimental animals, CGRP, SP and NKA produce vasodilation (21), whereas activation of pulpal nerves by electrical stimulation produces long-lasting vasodilation in the pulp due to release of CGRP (22-24).

### Changes in the nerve function in inflammation, neurogenic inflammation, inflammatory mediators

Structural changes of nerve fibres occur in response to inflammation. Nerve fibres sprout or branch extensively (25,26), thereby increasing the release of neuropeptides resulting in "neurogenic inflammation". CGRP and SP are increased at initial stages of pulpal inflammation, whereas NPY increases in chronic stages (27). Neuropeptides released from sensory neurons not only act on the vasculature, but also directly attract and activate innate immune cells (dendritic cells) and adaptive immune cells (T lymphocytes) (28,29). Once immune cells are recruited to the site of inflammation, inflammatory mediators such as cytokines, histamine, bradykinin, prostaglandins, leukotrienes, and numerous other substances are released. Neural sprouting increases neuropeptide content and release, resulting in neurogenic inflammation (30,31). Fig. 3 schematically illustrates the 5 stages of changes to the dentin-pulp complex according to caries progression, possible symptoms and suggested treatment. Caries, even limited to the enamel layer may already have some minor effect on the dental pulp (5), e.g. in terms of neurogenic inflammation and onset of dentin sclerosis can occur. This corresponds with alterations along the odontoblast layer (32) (Stage 1). Sprouting of sensory neuropeptide containing nerve fibres occurs with deeper carious lesions (Stage 2 and 3) coinciding with hyper- and thermal sensitivity of the tooth (26,31,33). This sprouting is reversible and subsides to normal after caries arrestment or restoration. Irritation of the dental pulp due to caries leads to reparative dentin formation by odontoblasts. With the progression of caries (stage 4), localized microabscesses may form in the dental pulp with sprouting of nerve fibres. There is also increased release of neuropeptides (34).

As the contaminated demineralized carious dentin reaches the dental pulp (stage 5), pulpal inflammation becomes extensive with partial necrosis combined with reparative dentin formation and vital inflamed pulp apically. Due to the loss of a functional barrier against infection and limited capacity for

## CLINICAL RELEVANCE

From the clinical point of view it is important to note that the sharp, short lasting, non-lingering, pain due to stimulation of exposed dentin can be evoked when the pulp is healthy or has some minor reversible injury and, thus, can be successfully managed without root canal treatment. Considering the response characteristics of the C-fibres it can be concluded that their activation, inducing dull aching pain, which is often long-lasting or

lingering in nature, may suggest that the pulp is irreversibly damaged and might need root canal treatment. Increased release of sensory neuropeptides in the dental pulp causes vasodilation, leading to increased local tissue pressure and increased capillary permeability, causing plasma extravasation and edema formation. Due to the non-compliant nature of the dental pulp, clinically this can be felt as throbbing pain.

healing in the coronal portion of pulp at this stage, necrosis progresses apically. Symptoms can be numerous and variable at this stage and when left untreated, infection and inflammation progresses, eventually leading to complete pulpal necrosis and apical periodontitis.

### Mechanism of nerve activation in response to dentinal stimulation, dentin sensitivity

How stimuli are relayed from the peripheral dentin to the sensory terminals located in the region of the dentin-pulp border zone has been a subject of interest for many years. Evidence indicates that movement of fluid in the dentinal tubules is a crucial factor in dentinal pain. Pain-producing stimuli, such as heat, cold, air blasts, and probing with the tip of an explorer, have the ability to displace fluid in the tubules (35,36). This is referred to as the *hydrodynamic mechanism of dentin sensitivity*.

#### Hydrodynamic theory

The hydrodynamic theory suggests that dentinal pain associated with stimulation of a sensitive tooth ultimately involves mechanotransduction. Recently, classical mechanotransducers have been recognized on pulpal afferents, providing a mechanistic support to this theory (37). Thus, fluid movement in the dentinal tubules is translated into electrical signals by activation of mechanosensitive ion channels located in the axon terminals. Using single-fibre recording techniques, a positive correlation was found between the degree of pressure change and the number of nerve impulses leaving the pulp (38-40). The outward fluid movement (negative pressure) produces a much stronger nerve response than inward movements (36,40). →

A short application of cold or heat to the outer surface of dentin can evoke pain that is not dependent on temperature changes in the pulp (38,41). The response to thermal stimulation is rapid, although the thermal conductivity of dentin is relatively low. Heat expands the fluid within the tubules, causing the fluid to flow towards the pulp, whereas cold causes the fluid to contract, producing an outward flow.

It is principally the A-fibres that are activated by a rapid displacement of the tubular contents (Table 1 and Fig. 1) (42). C-fibres, however, may be activated by heat (above 43°C). The polymodal C-fibre nociceptors contain numerous receptors which respond to different types of stimuli (43,44). Particularly, a receptor termed the “transient receptor potential, subtype vanilloid 1” or TRPV1 is expressed, and responds to heat above 43°C, certain inflammatory mediators, and acid (pH < 6) (45). Eugenol activates and ultimately desensitizes TRPV1, and this may explain the anodyne action of zinc oxide eugenol temporary restorations (46).

It has also been shown that pain-producing stimuli are more readily transmitted from the dentin surface when the exposed

tubule apertures are open and the fluid within the tubules is free to flow outward. For example, acid treatment of exposed dentin to remove the smear layer opens the tubule orifices and makes the dentin much more responsive to stimuli such as air blasts and probing.

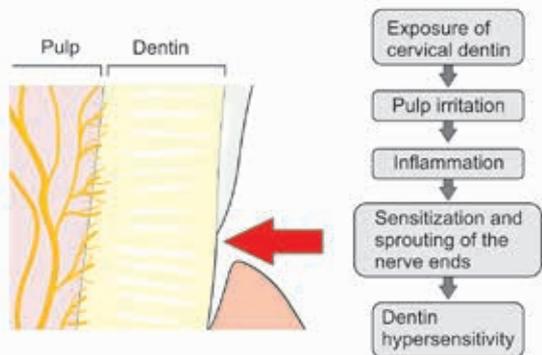
### Wear facet



**Fig. 5.** A wear facet is present at the buccal cervical surface of a lower canine. Due to pain the hygiene procedures were avoided. Eventually a carious lesion has started to progress at the gingival border (black arrow).

**Fig. 5.** En slidfacet er til stede svarende til facialfladen på en -3. På grund af smerter har tandbørstning været undgået igennem en længere periode. En carieslæsion er efterfølgende tilkommet ved grænsen til gingiva (sort pil).

### Mechanisms in the development of cervical dentin sensitivity



**Fig. 4.** Possible neurogenic mechanisms playing a role in the development of cervical dentin sensitivity: After gingival recession external irritants may induce local inflammation in the pulp-dentin border and result in sprouting of the nerve endings and, consequently, more extensive innervation of the tissue compared to healthy teeth, which may increase dentin sensitivity due to the increased release of the neuropeptides together with many other inflammatory mediators, which may sensitize the nociceptive nerve endings.

**Fig. 4.** Mulige neurogene mekanismer, der kan spille en rolle for udvikling af cervikal dentinsensitivitet: Efter gingival retraktion kan eksterne irritanter måske inducere lokal inflammation i pulpa-dentin-grænsen og dermed resultere i en vækst (spiring) af de pulpale nerveender. Herved opstår en øget innervation af vævet sammenlignet med sundt væv. Dette kan føre til dentinsensitivitet pga. forøget frigivelse af neuropeptid, og sammen med mange andre inflammatoriske mediatorer kan dette føre til sensibilisering af de nociceptive nerveender.

### Excavation of caries



**Fig. 6.** During excavation of caries an extensive peripheral excavation procedure may lead to a severe dentine hypersensitivity following restoration.

**Fig. 6.** Under ekskavering af caries kan en ekstensiv perifer ekskaveringsprocedure føre til øget dentinsensitivitet, efter at en fyldning er lagt.

The hydrodynamic theory is also applicable to explain hypersensitive dentin. It has been questioned whether exposed dentin is simply sensitive or becomes truly hypersensitive. However, evidence indicates that new sodium channels, capable of activating nerves, are expressed in nerve tissue exposed to inflammation. An increase in the density of sodium channels or their sensitivity may therefore contribute to dentinal hypersensitivity. Hypersensitivity typically occurs in the cervical area where the dentin is exposed because the protective enamel/cement was not formed or is worn out or etched away (Figs. 4 and 5). The odontoblasts and/or pulp cells respond by forming intratubular deposits or eventually tertiary dentin is laid down. This results in narrowing or closing of the dentinal tubuli. Deposition of tertiary dentin leads to decreased conductivity compared to the primary and secondary dentin. In addition, deposition of tertiary dentin without involvement of primary odontoblast cells over the pulpal ends of the exposed tubules may also reduce the sensitivity, as reparative dentin is less innervated by sensory nerve fibres. Some hypersensitive dentin, however, does not spontaneously desensitize, indicating either an on-going inflammatory change or mechanical changes in the patency of dentinal tubules.

#### Dentin hypersensitivity, development, prevention and treatment

The prevalence of individuals claiming to have dentin hypersensitivity has been reported to between 3–57% and most frequently in patients between 20 and 40 years (47).

#### Bacteria and dentin hypersensitivity

A wear facet or non-carious cervical lesion may be very painful, and consequently the patient may avoid daily use of this particular tooth and oral hygiene procedures. This may in turn develop into even more severe pain. In cases where a biofilm develops, the bacteria and their metabolites penetrate the dentin, resulting in local inflammatory changes in the pulp, including neurogenic inflammation as described above. Due to the pain, the person may tend to leave the site undisturbed. This may have two clinical effects; firstly, an altered sensitivity of the nerves, which become more reactive, including the sequence of sprouting and secondly, there may be onset of caries progression (Fig. 5). Taken together, the bacteria may play a role in severe dentin hypersensitivity, where only improved professional cleaning of the cervical area may lead to significant and permanent pain relief (48).

#### Iatrogenic development of hypersensitivity

During excavation the clinician may overextend the cavity preparation thereby exposing sound dentin (Fig. 6), where the permeability of the dentinal tubules is higher than in subjacent carious dentin. This scenario may be accompanied with suboptimal cooling and dehydration of the dentin. Consequently, the patient may experience severe dentin hypersensitivity following excavation and restoration.

#### The role of pulpal inflammation in dentin sensitivity

Pulpal A- and C-fibres can be sensitized by many external irritants, which can induce an inflammatory response in the pulp tissue. Sprouting of the nociceptive nerve terminals takes place in response to inflammation and may widen the receptive fields of the nerve fibres (43,49), which may result in increased overlap of the receptive fields (= the area where a single neuron can be activated, when stimulated). Thus, stimulation of a small spot, e.g. in dentin, may result in activation of a much greater number of pulpal nociceptors and, consequently, increased sensitivity compared to a non-inflamed tooth (Fig. 7). Moreover, inflammation and the consequent sprouting of the axons may result in more extensive innervation in pulp and pulp-dentin areas which are normally sparsely innervated in healthy teeth. This may be one mechanism playing a significant role in increased cervical sensitivity (Fig. 4). Also, fillings with open margins can induce pulpal inflammation, affecting the sensitivity of dentin in other parts of the pulp. Open dentinal tubules next to such a filling may allow the diffusion and penetration of external irritants into the pulp, resulting in inflammation, activation and also sprouting of the nerve endings in the pulp-dentin complex (Fig. 8). In fact, it may well be that inflammation of some degree could in general play a role in dentin hypersensitivity.

In addition, dental pulp seems to contain a considerable number of so called “silent” or “sleeping” nociceptors that cannot be activated in healthy, but only in inflamed teeth (43). Electrophysiological experiments indicate that approximately 40% of the nociceptive afferents can be activated in healthy teeth, whereas the proportion will increase to 60% when the pulp is inflamed. Considering the total number of the intradental afferents (approximately 1000) in each tooth, such an increase in number of nociceptors is significant regarding the dental pain sensitivity.

#### Silent and “hot tooth”

It seems that the activation of pulpal nociceptors can vary to a great extent (5,43,50). In many cases acute pulpitis can be extremely painful. However, most often pulpal inflammation may proceed to total pulp necrosis with minor symptoms or with no symptoms at all (23,50,51) (Fig. 3). This is puzzling considering the rich nociceptive innervation of the pulp. Such a variation in the symptoms can also be a serious diagnostic problem from a clinical point of view. A number of local mediators may be involved in the prevention of the nerve activation (43,51). Those include e.g. local opioids, somatostatin, noradrenalin and nitric oxide (43,52-54). These mediators are also important for regulation of the intensity of pulpal inflammation. The inhibition of nociceptor activity results in reduced release of the neuropeptides and other inflammatory mediators and also attenuation or even complete prevention of pain symptoms (43,51). In addition to the local or peripheral sensitization and inhibition described above, mechanisms on brainstem level or

higher in the complex nociceptive pathways may play an important role to regulate the pain (central sensitization/inhibition), like in all pain development and modulation (55).

### Clinical cases of pulpal and dentinal pain and their treatment

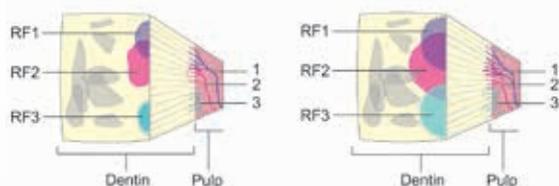
#### Treatment of dentin hypersensitivity

With reference to classical literature, the clinical impression and interpretation of dentin pain is something that will be triggered and provoked by well-defined external stimuli. In the following clinical scenarios, the accumulation of biofilm may play a huge role in pain development.

#### Scenario 1

The patient has not visited the dentist for years. The patient is completely unable to drink or have cold or hot fluids in the oral cavity due to pain. Clinically, the patient has a poor hygiene status and a high number of defective restorations. Accumulation of biomass is noted during the clinical examination. Not

### Receptive fields of single intradental nerve fibres

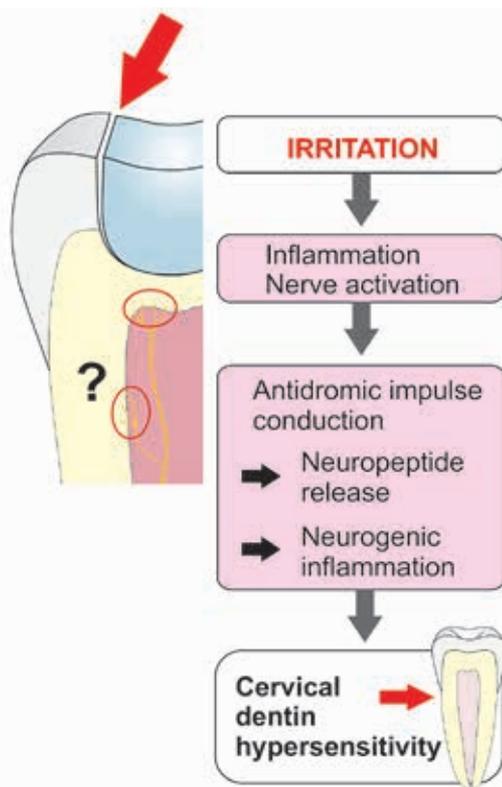


**Fig. 7.** Schematic presentation of the receptive fields of single intradental nerve fibres in a block of dentin of a healthy (A) and an inflamed (B) tooth. Three nerve fibres with their nerve endings in the pulp-dentin border are indicated by numbers (1-3). They are colored (blue, red and brown) and the same colors are used to show their receptive fields on the exposed dentin surface. The receptive fields (RF) of the fibres are considerably wider and more extensively overlapping in the inflamed (B) compared to the healthy tooth (A). Consequently, irritation of a standard area in dentin results in activation of considerably higher number of nerve fibres and higher sensitivity in the inflamed tooth compared to the healthy one.

**Fig. 7.** Skematisk repræsentation af de receptive felter fra en enkelt intradental nervefiber i en dentinblok fra en sund (A) og en inflammeret (B) tand. Nervefibrene svarende til pulpa-dentin-grænsen er angivet med tal (1-3). De er farvet (blå, rød og brun), og de samme farver er anvendt for at vise sammenhæng med deres respektive felter på den eksponerede dentinoverflade. De enkelte receptive felter (RF) af fibrene er betydelig større og mere overlappende i den inflammerede (B) sammenlignet med den sunde tand (A). Heraf følger, at ved en given irritation af dentin vil det medføre en aktivering af et betydeligt større antal af nervefibre samt øget dentinsensitivitet i den inflammerede tand sammenlignet med den sunde.

only are the restorations suboptimal, but also larger parts of the teeth are broken down with exposed dentin. This has created an ecosystem with biofilm formation on exposed dentin (Fig. 9a,b). Following several visits with only professional biofilm removal (Fig. 9c), the patient arrives with a marked decrease in the pain level. Of course, in the real life scenario the clinician

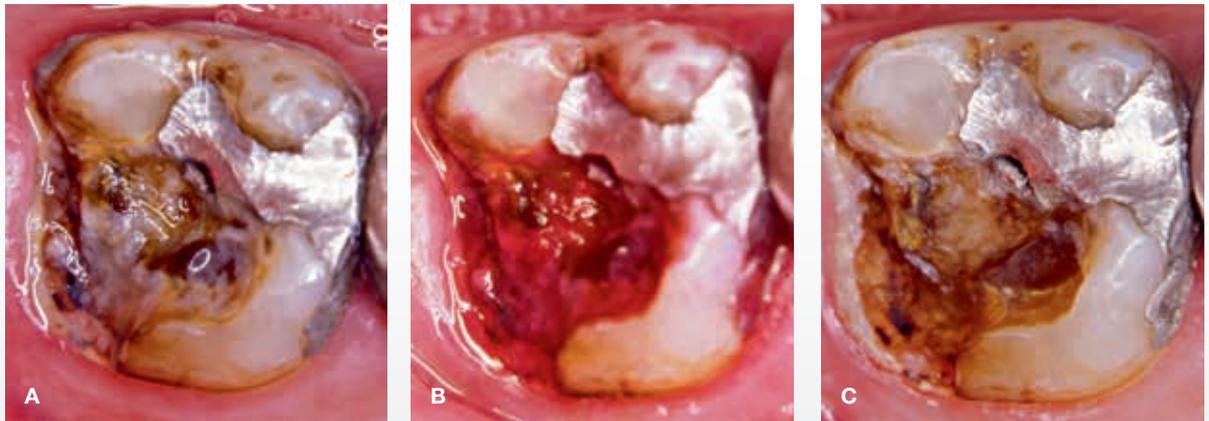
### Leaky fillings



**Fig. 8.** Even considerably remote leaky fillings may increase dentin sensitivity due to neurogenic inflammatory effects with branching of the intradental axons in other areas of the pulp-dentin complex. The neurogenic inflammation and related release of the neuropeptides induce activation of other inflammatory mediators as well. The induced inflammation increases the sensitivity of the pulpal nociceptors and, consequently, the sensitivity of the pulp and dentin.

**Fig. 8.** Utætte fyldningsdefekter, der ligger relativt langt fra pulpa, kan bidrage til en øget dentin-sensitivitet pga. de neurogene inflammatoriske virkninger herunder deres forgrening af intradentale axoner i store områder af pulpa og pulpa-dentin-organet. Den neurogene inflammation og dens tilhørende frigivelse af neuropeptider inducerer tillige aktivering af andre inflammatoriske mediatorer. Den samlede inducerede inflammation øger følsomheden af de pulpal nociceptorer og dermed følsomheden af pulpa og dentin. Derfor kan det være nødvendigt at omlave selv en gammel pulpaferm fyldning ved behandling af dentin hypersensitivitet.

## Large cavities



**Fig. 9.** Patient presenting with severe dentin hypersensitivity and large cavities covered with biofilm. (a) before biofilm staining, (b) after biofilm staining and (c) after professional biofilm removal.

**Fig. 9.** Patient med svær dentin hypersensitivitet med stor kavitet som følge af både caries samt fraktur af undermineret emalje. Hele den eksponerede overflade er belagt med en biofilm; a) før biofilmindfarvning, b) efter biofilmindfarvning og c) efter professionel biofilmfjernelse.

would initiate both hygiene procedures and restorations, but the present case reflects the important impact of biomasses on exposed dentin and dentin hypersensitivity.

### Scenario 2

If improved hygiene procedures have been introduced without pain relief, the treatment plan should be aimed at reducing dentin permeability (Table 2). This can be achieved by either physically blocking the dentinal tubules or by depolarizing the nerves (56). A wide range of materials for desensitization have been sold, but without universal success. Dentin hypersensitivity can be very difficult to control, indicating that the materials either have no permanent effect and/or that the inflammatory changes are so profound that a natural healing process is prevented. Several treatment modalities have shown an occluding or desensitizing effect in animals, but it has been difficult to demonstrate in the clinic (56). A problem with materials that are intended to block fluid flow in dentinal tubules, for example by precipitation of salt crystals, is that the precipitation may be washed out, is dissolved in an acid medium or is worn away so that the potential occluding effect is temporary.

There are many published studies on toothpastes with strontium and potassium salts (57). Several of these report that these toothpastes have some desensitizing effect, whereas others have not been able to demonstrate an effect. The design of these studies have been questioned and the effect is uncertain (58). Before conducting a costly and invasive treatment, a practical test could be to evaluate whether the individual patient

### Treatment of hypersensitive dentin

Biofilm removal using improved professional hygiene procedures

Formation of a smear layer by burnishing the exposed root surface

Application of agents, such as oxalate compounds, forming insoluble precipitates within the tubules

Application of agents such as hydroxyethyl methacrylate (HEMA) with or without glutaraldehyde that are thought to occlude tubules with precipitated plasma proteins in dentinal fluid

Application of dentin bonding agents to seal off the tubules

**Table 2.** Treatment modalities for treatment of hypersensitive dentin.

**Table 2.** Behandlingsregimer af hypersensitiv dentin.

feels improvement in symptoms using these toothpastes. A paste consisting of arginine and calcium carbonate, claimed to close the dentinal tubuli, has been introduced as a gentle treatment of a sensitive area. So far, only preliminary results are available from the manufacturer (59,60).

If the above methods are ineffective the next step would be to seal the dentinal tubules with fluoride varnish, dentin primer and resin or with a resin restoration. In some cases it may end with endodontic treatment. As in other contexts, primary prevention is of course the optimal solution, by using a gentle

brushing technique as well as a low intake of soft drinks with low pH as opposed to the attempt to treat the established hypervulnerable area/lesion.

### Conclusion

Diagnosis and management of dentinal pain is often a challenge to the clinician. The dental pulp is exceptionally richly innervated by nociceptive afferents, and pulpal and dentinal

pain can cause patients considerable discomfort. Many factors are involved in the development and persistence of pain. Caries, iatrogenic damage, changes in dentin structure and permeability caused by erosion or tooth wear, aggregation of biofilm on unprotected dentin; all can lead to activation of nociceptive nerves, which initiates local inflammatory changes in the pulp, and also triggers central changes in pain processing – both of which are complex and may be difficult to reverse.

### ABSTRACT (DANSK)

#### Akutte tandsmerter I: smerter fra pulpa og dentin

Den specialiserede anatomi i pulpa-dentin-organet samt den rige pulpale innervation fra trigeminusnerven forklarer de forskellige typer af smertefølelser i en tand. En kort skarp smerte er typisk for en A-(nerve) fibermedieret smerte, imens en langvarig, bankende smerte indikerer C-(nerve) fiberaktivitet. A-fibre reagerer på termiske eller mekaniske stimuli, såsom kolde drikke eller tandbørstning, imens C-fibre hovedsagelig aktiveres ved inflammatoriske mediatorer.

Således vil en dvælende smerte indikere en irreversibel pulpal inflammation. Ved pulpitis vil der opstå strukturelle ændringer i de pulpale nerver, der samtidig frigiver neuropeptider, som udløser et immunrespons: neurogen inflammation. Smertefornemmelser

under pulpitis kan variere fra hypersensibilitet overfor termiske stimuli til svære dunkende og uudholdelige smerter. Smerterne kan være meddelte og ofte vanskelige at lokalisere, hvorfor diagnostik af inflammation i pulpa er en klinisk udfordring.

En biofilm forstærker hypersensitivitet af eksponerede dentinoverflader, fordi de mikrobielle irritanter kan nå pulpa gennem åbne dentintubuli, hvorved der fremkaldes inflammation. Fjernelse af biofilm reducerer isninger i tænderne, men supplerende behandling er ofte nødvendigt med det formål at opnå en reduktion af dentinens permeabilitet. Cariesekskavering samt fyldningsterapi er en tilstrækkelig behandling ved en klinisk bedømt reversibel pulpitis, hvorimod endodontisk behandling er nødvendigt, når pulpitis har nået et irreversibelt stadium.

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