Acute dental pain I
pulpal and dentinal pain
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The specialized anatomy of the pulp-dentin complex and the dense, predominantly noci-
ceptive 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 indi-
cates 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, lin-
gering pain suggests presence of irreversible pulpal inflammation.

During pulpitis, structural changes of the pul-
pal nerves (sprouting) occur and neuropeptide
release triggers an immune response; neuro-
genic inflammation. Pain sensations during pul-
pitis 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 ex-
posed dentin surfaces because irritants reach
the pulp through open dentin tubules, produc-
ing inflammation. Removing the biofilm reduces
dentin hypersensitivity, but supplemental treat-
ment, aiming to reduce dentin permeability,
is often necessary. Caries removal and filling
therapy are adequate measures during revers-
able pulpitis if the pulp has maintained its ability
todistance itself from the bacterial assault by
producing reparative dentin. However, endo-
dontic therapy is necessary when pulpitis has
reached an irreversible stage.

**Pain localized to teeth is among the most frequently ex-
perienced 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 in-
ervated mainly by axons from the trigeminal nerve, predomi-
nantly sensory in nature and mainly committed to pain percep-
tion (nociception). A smaller population of pulpal nerves are
autonomic sympathetic fibres emanating from the superior cer-
vical ganglion and associated with pulpal vasoconstriction (4).

Extremely strong pain – reaching the maximum intensity
at any pain score – can be induced by activa-
tion of intradental nerves (5-7). Such intense
pain responses can be explained by the dense
(Figs. 1 and 2) and predominantly nocicep-
tive innervation of the pulp and dentin (6,8).
The transmission of the pain-inducing stim-
uli through dentin from its exposed surface
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/mm² in the pulp-dentin border area in the most coronal pulp which accounts for the extremely high sensitivity of dentin.

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 Aβ-fibres but a part of them belong to Aδ-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).

Table 1. Stimuli capable of activating the intradental nerves.

<table>
<thead>
<tr>
<th>Nerve Fibre Type</th>
<th>Activating Stimuli</th>
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<tbody>
<tr>
<td>Myelinated Nerves</td>
<td>Aβ-fibres (7%) – Vibration</td>
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<td></td>
<td>Aδ-fibres (93%) – Air, heat/cold, drilling/probing</td>
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<tr>
<td>Unmyelinated Nerves</td>
<td>C-fibres – Inflammatory mediators, mechanical deformation, heat</td>
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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 µm) into the dentinal tubules.

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 pulp-dentin border, penetrating the inner part of dentin.

Fig. 1. Skematisk præsentation af den intradentale nervenervation. Den pulpale indgang for nervfibre 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 pulpaområder) er det tætteste nerve-innerverede område, hvor de enkelte nerveender har deres længste udstrækning i dentintubuli (100-150 µm).
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

<table>
<thead>
<tr>
<th>STAGE 1</th>
<th>STAGE 2</th>
<th>STAGE 3</th>
<th>STAGE 4</th>
<th>STAGE 5</th>
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</thead>
<tbody>
<tr>
<td>Vascular effect</td>
<td>No/few changes</td>
<td>Some vasodilation</td>
<td>Vasodilation</td>
<td>Vasodilation</td>
</tr>
<tr>
<td>Neuronal changes</td>
<td>No/few changes</td>
<td>Little sprouting</td>
<td>Sprouting</td>
<td>Extensive sprouting</td>
</tr>
<tr>
<td>Immune cell infiltration</td>
<td>No/few changes</td>
<td>Some</td>
<td>Some</td>
<td>More</td>
</tr>
<tr>
<td>Histology</td>
<td>No changes</td>
<td>Early signs of reparative dentin</td>
<td>Reparative dentin</td>
<td>Abscess formation</td>
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<tr>
<td>Symptoms</td>
<td>Symptom free</td>
<td>Sensitivity to hot/cold</td>
<td>Hypersensitive/Tender to percussion</td>
<td>Symptom free</td>
</tr>
<tr>
<td>Pulp diagnosis</td>
<td>Normal</td>
<td>Reversible pulpitis</td>
<td>Reversible pulpitis</td>
<td>Irreversible pulpitis</td>
</tr>
<tr>
<td>Periapical diagnosis</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Treatment</td>
<td>Remineralization or caries removal and restoration</td>
<td>Remineralization or caries removal and restoration</td>
<td>Caries removal and restoration</td>
<td>Endodontic therapy</td>
</tr>
</tbody>
</table>

**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.
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 pulp inflammation, whereas NPY increases in chronic stages (27). Neuropeptides released from sensory neurones 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), pulp 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 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 pulp 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).

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.
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.

**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.](image)

![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).](image)

![Fig. 6. During excavation of caries an extensive peripheral excavation procedure may lead to a severe dentine hypersensitivity following restoration.](image)
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 pulp 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 ongoing 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-curious 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 pulp 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 pulp 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 pulp 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, noradrenaline 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 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

**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. 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. To achieve the full effect of a given irritation of dentin the treated area, it is necessary to remove the biofilm and the debris from the dentinal walls. This can be achieved by using a combination of mechanical instrumentation, ultrasonic scaler and prophylaxis paste to remove the biofilm and debris from the dentinal walls.

**Leaky fillings**

**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.
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.

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

Tabel 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 hypersensitive area/lesion.

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

Aktele tandsmerter I: smerten fra pulpa og dentin

Den specialiserede anatomii i pulpa-dentin-organet samt den nje pulpale innervation fra trigeminsensnerven forklarer de forskellige typer af smertefølelser i en tand. En kort skarp smerte er typisk for en A- (nerve) fibermediert smerte, imens en langvarig, bankende smerte indebærer C- (nerve) fiberaktivitet. A-fibre reagerer på terminiske eller mechaniske stimuli, såsom kolde drikke eller tandbørstering, imens C-fibre hovedsaglig aktiveres ved inflammatoriske mediatorer.


En biofilm forstærker hypersensitivitet af eksponerede dentinoverflader, fordi de mikrobielle irritamenter kan nå pulpagen 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. Cariesoksekaverende samt fyldningstherapi er en tilstrækkelig behandling ved en klinisk bedømt reversibel pulpitis, hvormod endodontisk behandling er nødvendigt, når pulpitis har nået et irreversiblet stadium.

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