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Pulpal Pain Diagnosis—A Review

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Evidence gathered from our studies and the work of others appears to support the presence of two distinct nerve pain pathways in the dental pulp, represented by fast conducting A-delta and slow conducting C-fibers. Each of these types of fibers has different pain characteristics: A-delta fibers evoke a rapid, sharp, lancinating pain reaction, and C-fibers cause a slow, dull, crawling pain. Pain response thresholds vary in different regions of the tooth, and thermal, osmotic, ionic, and electric stimuli involve different mechanisms to provoke nerve excitation of the dental pulp. Evidence also points to the fact that the incidence of pain increases as the histopathosis worsens. On interrogation, patients who manifest severe or referred pain almost always give a previous history of pain in the tooth with the ache. Eighty percent of patients who give a previous history of pain manifest histopathologic evidence of chronic partial pulpitis with partial necrosis, the untreatable category, for which endodontics or extraction is indicated. The other 20% exhibit histopathosis of the pulp with slight inflammation to chronic partial pulpitis without necrosis, a treatable category. Clinically, one can determine the degree of pulp histopathosis by asking the patient about a previous history of pain in the involved tooth. This history of previous pain adds another dimension in diagnosis for the clinician as to whether the painful pulpitis is reversible. This information also aids in referred pain localization.

Pulpal pain symptoms occur with different intensities, e.g. mild, moderate, or severe. These differences often aid the clinician in establishing the proper diagnosis or treatment plan and determine whether a pulpectomy is indicated or whether the pulp can be salvaged. In other words, is the inflammatory condition of the pulp treatable or nontreatable? The purpose of this article is to present data on how to distinguish clinically between reversible and irreversible painful pulpitis and how to localize referred pain.

Pain symptoms often challenge the clinician's diagnostic acumen, for it is well known that a correct diagnosis implies correct treatment. Generally, pain is conceptualized as a psychobiologic phenomenon which has two components: perception of pain, which is influenced by anesthesia, and reaction to pain, e.g. fear, anxiety, anguish, depression, or crying, which is influenced by drugs and emotions. These emotional states vary from patient to patient and the disturbances at times can exaggerate the perception of pain.

One's ability to diagnose different pain disorders depends on understanding that pain can be of either systemic or odontogenic origin; it must be recognized that different pain intensities are associated with numerous different diseases which manifest pain and that pain can be referred from one part of the body to another. Evaluation of the dental history, particularly the history of pain in the same tooth before the present pain experience, is an important consideration in rendering proper treatment.

The pain process occurs mainly while tissue damage is going on, not after tissue injury. When tissue damage is occurring, the intensity and duration of pain differ from when a stimulus is applied to a normal tooth. For example, when a thermal stimulus is applied to a tooth with a normal pulp, pain perception is immediate, and upon stimulus removal, the pain disappears immediately. When the same thermal stimulus is applied to a tooth with pulpal damage, such as in the case of deep caries, there is a lower pain response threshold and the pain lingers.

Besides the different perceptions and reactions to pain, patients attach different significances to different pain locations. A greater concern is expressed when pain is present in the eye or belly than when pain is due to a cut finger. Also, there is greater anxiety when an anterior rather than a posterior tooth is involved.

All nerves are alike in that they transmit electrical impulses. However, they differ as to modalities of sensation and conduction velocity. Their respective tracts terminate in specific areas of the CNS. Regardless of the stimulus, the sensation is specific. It can be pain, touch, taste, smell, sound, or sight. The dental pulp is an excellent example of a specific response following any stimuli, be it touch, thermal, electric, or chemical: the response is the same—pain. Sensory receptors that detect different stimuli are connected or wired to specific areas in the CNS which discriminate the sensation in the brain. Table 1 classifies the respective sensory functions of the different sensory receptors.

Intensity of pain, i.e. mild, moderate, or severe, is governed by frequency of firing, number of nerves, and the type of nerve fibers. There are the fast-conducting myelinated A-delta nerve fibers and slow-conducting unmyelinated C-fibers. The A-delta fibers have a fast conduction velocity of 12 to 30 m/s. The slow C-fibers have a conduction velocity of 0.5 to 2 m/s. Conduction velocity is also governed by the diameter of the nerve fiber. Differences in the

TABLE 1. Sensory receptors

Mechanoreceptors
Tactile, hearing
Thermoreceptors
Hot, cold
Nociceptors
Pain, A-delta and C-fibers
Electromagnetic receptors—vision
Rods and cones of eye
Chemoreceptors
Taste, smell, blood O_2-CO_2
Drugs
Glucose, amino and fatty acids

Modified from Guyton AC. In: Basic neuroscience. 2nd ed. Philadelphia: W. B. Saunders, 1991:103.

TABLE 2. Differences in excitation of A-delta and C-fibers

Symptoms and Reactions	A-Fibers Myelinated	C-Fibers Unmyelinated
Conduction	Fast	Slow
Pain	Sharp	Dull
Location	PDJ	Core of pulp
Cold	Yes	No
Heat	Yes	Yes
Ion effect	Yes	No
EPT	Yes	No
Localization	Good	Poor
Referred pain	No	Yes
Excitability threshold	Low 9.9 μA	High 37.4 μA
Hypoxia	No response	Responds
Increase in pulp pressure	No	Yes
Inflammatory mediators	No	Yes
Hyperosmotic solutions	Yes	No

PDJ, pulp dentin junction (border).

character of pain, such as pricking (i.e. pin sticking, sharp electric-like) produced by the electric pulp tester (EPT), are due to stimulation of the A-delta type myelinated nerve fibers, whereas the burning, crushing, crawling, aching-type pain results from stimulation of the unmyelinated C-fibers. The latter nerve fiber requires a stronger electric current than the conventional EPT that is used on the A-delta fibers (1). C-fibers have a higher excitability threshold than the A-delta fibers. Differences in excitability between myelinated and unmyelinated nerve fibers are illustrated in Table 2.

SYSTEMIC CONSIDERATIONS

At times it needs to be determined whether intradental pain is of systemic, nonodontogenic, or odontogenic origin before a diagnosis can be made. Patients with pain of systemic origin present themselves infrequently in the dental office. In the author's sixty-eight years of experience in active practice and service in a large general hospital, pain of systemic origin mimicking dental pain (e.g. trigeminal neuralgia, acoustic neuroma, herpes zoster, diabetic odontalgia, myocardial infarction) was encountered infrequently; only once or twice was each of the foregoing diseases encountered. However, pain of atypical facial neuralgia is most frequently referred by physicians to dentists for consultation. The pain in atypical facial neuralgia does not follow the path of the fifth nerve distribution and is not spasmodic. Pain usually occurs bilaterally, in contrast to trigeminal neuralgia, which occurs unilaterally. The pain appears to localize itself mainly in the upper molars and creates a diagnostic problem when deep fillings are present. This disorder is present in a 4:1 ratio in females and occurs mainly during or after menopause. The pain symptoms seem to involve the C-fibers, representative of the dull, crawling, boring, continuous pain associated with a burning sensation in the tongue, lips, and roof of the mouth. The pain comes on spontaneously and, after a long, lingering, painful period, leaves spontaneously. Patients often insist on endodontic therapy or extraction, neither of which gives relief. The reaction component to pain is the main feature in this disease.

TABLE 3. Dentinal pain evoked by electrical stimulus varies in different regions of human teeth

Region	Average Calibrations of Response Threshold*
Cervical ⅓	35.91
Middle ⅓	31.44
Incisal ⅓	28.51
Incisal edge	21.81

* Calibrations on Analytic Technology Digital Pulp Tester, based on 2387 pulp test recordings (3).

The nonodontogenic group represented by temporomandibular joint syndrome, pericoronitis, mouth ulcers, and sialolithiasis offers no difficulty in differential diagnosis. However, in sialolithiasis, a stone in the salivary duct causes pain and swelling in the angle of the mandible and may create a diagnostic problem. This diagnosis is often missed because of the location of the swelling.

REGIONAL PAIN DIFFERENCES

Pain of odontogenic origin involves dentinal and pulpal pain derived from two distinctly different types of pain nerve fibers located in two different regions of the dental pulp. The terminals of the fast-conducting A-delta fibers are located principally at the pulp dentin border zone at the pulp periphery. Their free nerve endings penetrate into the dentinal tubules for a distance of 150 to 200 μm. Almost all of the A-delta fibers are located in the coronal portion of the pulp, with the greatest nerve density in the pulp horns (2). In addition, electric pulp testing studies indicate that there are regional differences in dentinal pain. The lowest response threshold is present at the incisal edge or the incisal one-third facial region, followed by the mid-third region. The highest response threshold is at the cervical third (Table 3). The results appear to indicate that the differences in response threshold are due to the difference in nerve densities located in different regions of the pulp (3), most likely to meet the protective functional demands of the pulp tissue. In contrast, the slow-conducting C-fibers are located in the pulp proper, extending most likely into the cell-rich zone.

THERMAL STIMULI

Dentinal pain consists of a sharp, brief, localized pain of short duration. This response follows any stimulus that alters the hydrodynamics, osmotics, or ionics within the dentinal tubules. The stimuli can be thermal, mechanical, osmotic, or electric. Thermal stimuli, e.g. heat or cold, give superior pain reactions when more extreme differences in temperature are applied to the crown of the tooth (Table 4). The fluid within the dentinal tubules moves with a stronger or faster wave movement to deform the cell membrane

TABLE 4. Temperatures of different thermal agents

CO_2 snow	−78.5°C
DDM (Frigen)	−50.0°
Ethyl chloride	−18.0°
Ice	−0.0°
Liquid nitrogen	−150.0°
Hot gutta-percha	+75.0°

of the free nerve endings, acting as a mechanoreceptor. Gradual temperature change causes no immediate pain response; there is no rapid fluid movement, which is essential to excite the A-delta nerves. Ultimately, a response occurs as the gradual temperature change causes a thermoreceptor activity with a C-fiber response (4).

Clinically, carbon dioxide snow or hot gutta-percha application give better test results than ice or hot water (5). Exposure of teeth to these extreme temperatures causes no ill effects, according to histologic findings (6).

The application of a cold stimulus to the clinical crown evokes a rapid A-delta pain response followed by an immediate pain cessation. Continuous cold application compromises the blood flow due to vasoconstriction of the blood vessels and can cause anoxia, a condition under which the A-delta fibers cease to function. When the application of cold is prolonged, there is a drop in mitochondrial activity, with a fall in impulse frequency and decreased excitability, which is considered to be a cryogenic effect.

Heat application on the other hand, has a biphasic effect. First, the initial heat application evokes a rapid, brief pain response due to the rapid fluid movement caused by the sudden temperature change. As the heat application is continued, there is a period of pain cessation followed by a more intense pain with a greater frequency and a rise in mitochondrial activity. This is presumably due to the dilation of blood vessels caused by heat with a transient increase in intrapulpal pressure (7). This action now involves the C-fibers, which are effected by heat. The A-delta fibers seldom cause intense or referred pain. Dentinal pain, caused by A-delta fibers, is more transitory and is used more often than C-fibers for diagnostic vitality tests. C-fibers are usually associated with pathophysiology.

A-delta and C-fibers transmit pain signals through different pain pathways in the CNS to different regions of the thalamus. The C-fibers terminate in the caudal nucleus of the trigeminal brain stem complex and in the intralaminar nuclei of the thalamus, both of which are parts of the reticular activating system. From the thalamus, which is considered a relay station, the C-fiber impulses are relayed to the cerebral cortex and the hypothalamus.

The A-delta fibers also terminate in the nucleus caudalis and then activate a central pain pathway that terminates in the most caudal portion of the ventrobasal thalamus complex. From here signals are transmitted to other areas of the thalamus and to the somatic sensory cortex, the area of discrimination and localization from both A-delta and C-fiber systems (8). It is in the cortex that critical comparisons with other previous sensations are made. The cerebral cortex is the information-processing center that recalls data stored from previous pain experiences (9).

ELECTRIC STIMULUS

While thermal stimuli evokes pain due to fluid movement in the dentinal tubules, electric stimuli, using the EPT, evokes a pain response due to ionic movement. Electric conductivity causes an ionic imbalance across the neural membrane, inducing an action potential with a rapid saltatory or hopping action which occurs at the nodes of Ranvier in myelinated nerves. In addition, nerve transmission, like any electric current, cannot be conducted through an ion-free solution (e.g. distilled water).

OSMOTIC AND IONIC CHANGES

Hypersensitivity, a common clinical pain entity is caused by stimulating exposed dentinal tubules with sugars or $CaCl_2$. These cause an hyperosmotic pressure stimulation that induces an immediate transient excitation of the A-delta fibers. The nerve excitation effect becomes greater when the dentin is acid-etched or when the smear layer is removed (10).

ION ACTIVITY

A relationship of intradental nerve activity and ionic composition of intracellular fluid exists. Thus an increase in calcium ions effects the cell-membrane permeability (11). Calcium ions have a stabilizing effect on all cell membranes. An increase in calcium ions intracellularly reduces sodium ion entry through the neural membrane by reducing the membrane permeability to sodium, resulting in a lower excitability with less pain and muscle activity. This action is observed in patients who suffer from hyperparathyroidism in which there is a hypercalcemia. They require twice the amount of electric current to cause an excitable reaction (12). On the other hand, when there is a decrease in intracellular calcium ions, there is an increase in sodium ion entry. This increase in membrane permeability to sodium causes a rapid action potential. Clinically, this condition is known as hypoparathyroidism (clinical tetany). In this disease, there is a marked increase in muscular activity.

It is indeed enticing to speculate, as suggested by Olgart et al. (13), that the pain in acute painful pulpitis may become intermittently reduced by the calcium ions elaborated from dentin demineralization during the caries process. This concept is somewhat analogous to the effect of the endorphins or encephalins, the body opiates, that are secreted for systemic pain relief (14). The former may be considered as the local opiate.

Potassium is another ion which controls pain such as that in dentin hypersensitivity. The addition of potassium ions to dentifrices (14) and applications of potassium nitrate solution can induce a hyperpolarization, which decreases the excitability of the nerve fiber (15). Trowbridge et al. (16) found that eugenol increased potassium permeability of the nerve fiber membrane and depressed the rate of sodium influx during membrane excitation, thus creating a state of hyperpolarization. The increase in potassium ions produces the anodyne effect that follows the application of a zinc oxide-eugenol dressing. Potassium in the form of potassium oxalate is also used to control pain, but the mechanism differs. The application causes an insoluble precipitate of calcium oxalate, blocking the fluid movement and ionic exchange within the dentinal tubules.

PULPALGIA

Previous studies (Table 5) have shown that 90% of patients who suffer from pulpalgia give a history of pain prior to the present pain

TABLE 5. Correlation of clinical signs and symptoms with histologic diagnosis

Histologic Diagnosis	Pain Intensity			Previous History of Pain	Pulp Exposure (%)	Pain on Percussion (%)
	Incidence of Pain (%)	Mild to Moderate (%)	Severe (%)			
Reversible category						
Normal pulp	13	13	—	No	—	4
Early inflammation	11	11	—	No	11	5
Atrophic pulp	25	25	—	No	—	8
Acute pulpitis	25	25	—	No	—	—
Chronic partial pulpitis without necrosis	42	37	5	Yes	21	17
Irreversible category						
Chronic partial pulpitis with necrosis	64	21	43	Yes	79	43
Chronic total pulpitis	78	60	18	Yes	78	36
Total pulp necrosis	54	29	25	Yes	71	38

Modified from Seltzer S and Bender IB. The dental pulp. 3rd ed., Chap. 19, 1983.

episodes, and histologic findings manifest moderate to severe pulp inflammation with some areas of necrosis. Marked inflammation and some necrosis of the pulp is frequently present in patients with moderate to severe pain. Severity and duration of pain appears to be related to the status of pulp pathosis. Severe pulpal pain usually indicates the presence of liquefaction necrosis with an increase in intrapulpal pressure of 34.5 mm/Hg. In the normal pulp, the average intrapulpal pressure is 10 mm/Hg (9). Severe pain happens most often when the histologic diagnosis is chronic partial pulpitis with partial necrosis (Table 5). The severity of the pain lessens in pulps with histologic diagnosis of total pulp necrosis. This may be due to drainage and a drop in intrapulpal pressure, as in an isobaric chamber, in the more advanced stages of pulp disease. Presence of severe pulpal pain almost always reveals a history of previous pain in the involved tooth, with an incidence of carious pulp exposure of 79% (Table 5). Thus, when moderate to severe pain is present, the pulp is in the state of irreversible painful pulpitis, and endodontics or extraction is indicated (Table 5). However, when mild to moderate pain is present with no previous history of pain in the tooth under evaluation, the histologic picture usually manifests as a mild inflammation or a chronic partial pulpitis without histologic areas of necrosis. Furthermore, the standard clinical thermal pulp test exhibits normal pulp vitality with no lingering pain or positive percussion test. Thus the pulp can be considered in a reversible state and amenable to pulp conservation (Table 5). Thus, for patients who manifest severe pain or give a previous history of pain in teeth with symptoms of mild or moderate pain, endodontics or extraction is indicated. In teeth with no previous pain history, the aching pulpitis is reversible. Also, pulps in the untreatable category show an increased number of teeth with a lingering pain symptom and a positive percussion sign (Table 5).

In addition, comparative immunohistologic studies of reversible and irreversible pulpitides have shown distinct ratio differences between T-helper and T-suppressor lymphocytes (17). The predominant T-suppressor lymphocytes are able to suppress the inflammatory process and reverse the condition in the pulp, as suggested by Baumgartner (Table 6).

Clinical observations indicate that 60% of patients with pulpal inflammation are asymptomatic, whereas 40% experience mild, moderate, or severe pain (Table 7). These results confirm that pain

TABLE 6. Ratio of T-helper (T-H) to T-suppressor (T-S) lymphocytes in reversible and irreversible pulpitides

Pulp Status	Ratio T-H/T-S Lymphocytes
Normal pulp	0.5
Reversible pulpitis	0.56
Irreversible pulpitis	1.14

From Baumgartner JC. Curr Op Dent 1991;737–43.

TABLE 7. Incidence of various degrees of pain

Degrees of Pain	No. of Cases	% of Cases
No pain	388	60
Slight (no medication)	132	20
Moderate (aspirin)	71	10
Severe (codeine)	42	7
Intolerable (drainage, Demerol)	20	3

occurs while inflammation is going on and not after damage is done. After damage, the pulp may be in a necrotic state with limited impulse conduction. At times, some sensitivity is felt upon entry with an instrument into a necrotic pulp with a periapical radiolucency. This is due to the action of C-fibers, which can function at times under hypoxic conditions. The unmyelinated C-fiber does not need as much oxygen as the A-delta nerve fibers to function (18).

REFERRED PAIN

Referred pain is usually provoked by an intense stimulus acting on the C-fiber. A slight stimulus provokes no referred pain irrespective of any type nerve fiber, be it A-delta or C-fiber. Stimulation studies with the conventional EPT excites only the A-delta nerves and shows an overall localization accuracy of 82%, based on 1035 stimulation tests, with no evidence of pain crossover or decussation (19). Only strong stimulation of C-fibers provokes referred pain.

Accurate localization of referred pain ensues when tactile fibers are activated. In teeth with referred pain, the inflammation remains confined within the pulp. The inflammatory process has not yet extended into the periapical region to engage the tactile fibers, the Ruffini mesencephalic fibers of the periodontal ligament, to give the clinical percussion sign (20). Clinicians often adopt a "wait and see policy" in teeth with referred pain for correct localization. Referred pain, characterized by an intense, boring, continuous pain, always occurs with a previous history of mild to moderate pain in the tooth causing the present ache. Upon questioning, patients often recall the location of previous mild or moderate tolerable pain. Usually, they are fairly certain as to its localization. However, with the increase in pulpal inflammation over time and an increase in pain intensity, there is confusion as to localization and an inability to identify the involved tooth.

Sites of referred pain are always the posterior teeth and always unilateral, involving only one tooth in either maxilla or mandible. Anterior teeth seldom if ever refer pain to teeth in opposite jaws or to each other. Anterior teeth do not refer pain to posteriors and posteriors do not refer pain to anterior teeth. There is no pain decussation. When the pain is on the left side, it is emanating from a posterior tooth on the left side and vice versa if the pain is on the right side. Besides, referred pain is usually absent in the following: teeth undergoing endodontic treatment, cases of periodontitis, teeth with sinus tracts or fistuli, and endodontically treated teeth. These suggestions are based on the author's clinical observations.

Since the EPT excites only the A-delta fibers, no valid conclusion can be drawn from toothache studies involving referred pain because the pulpitides are associated mainly with C-fibers. All frank toothaches primarily involve C-fibers with their characteristic slow pain reaction.

An interpretive account is offered here as to the development of the severe pain category or the mild to moderate pain category with a previous history of pain. In pulp inflammation, the nerve fibers are in a hyperalgesic state, i.e. the nerve fibers have a low threshold of response. Under this circumstance, a low stimulus, which under normal conditions could not evoke a response, can provoke and exacerbate a pain response in this hyperalgesic state. This quick pain response is due to an action potential from a much lower nerve membrane negativity (i.e. the nerve fibers are in a lower state of excitability). As an example, under normal conditions, the negativity within the neural membrane may be about -70 to -80 mV, whereas under inflammatory conditions it can be -35 to -40 mV, requiring a lower stimulus for excitation. Thus, when a normal stimulus is applied to a tissue in an hyperalgesic state, the stimulus causes a more intense reaction. A classic example of hyperalgesia is the extreme sensitivity of sunburned skin—even wearing a shirt causes severe pain.

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