Dental Pulp Neurophysiology: Part 1. Clinical and Diagnostic Implications

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The purpose of diagnosis in endodontics is to assess the condition of a tooth — the object of the patient’s complaint — and to identify the cause of the pain or discomfort. Diagnosis is the art of identifying the problem and using scientific knowledge to determine the cause of the problem. Knowledge of the physiology of pain and methods of interpreting it with available clinical diagnostic devices is essential to reach a proper diagnosis.

The patient’s history, or more specifically, the history of the patient’s pain, is the first clinical data that the dentist must collect and consider. The dentist should pay careful attention to the patient’s answers about the pain, such as the type, duration, frequency, aggravating factors, effect of analgesics and tenderness when biting.

Once a preliminary or differential diagnosis is reached, further clinical examination is needed to confirm the diagnosis, such as inspection of the extraoral soft tissues, examination of regional lymph nodes and an intraoral examination that includes looking for signs of a sinus tract, swelling of the soft tissues, a mobile tooth or teeth, the condition of the gingiva, and the number of decayed and restored teeth. A transillumination test may reveal hidden decay or a fractured tooth and may result in the diagnosis of a necrotic tooth if enamel translucency is lost. In this simple test, a strong light is placed behind
fibres regulate the blood flow; no consensus about the role of parasympathetic fibres exists.7

The cell bodies of the sensory neurons of the pulp are located in the trigeminal ganglion. Hundreds, perhaps thousands, of axons found in the canines and premolars5,8 enter the pulp through the apical foramen where they branch following the distribution of the blood supply all over the pulp. The majority of the nerve bundles reach the coronal dentin where they fan out to form the nerve plexus of Raschkow. There, they anastomose and terminate as free nerve endings that synapse onto and into the odontoblast cell layer (approximately 100–200 μm deep in the dentinal tubules) and the odontoblastic cell processes.7,9

The 2 types of sensory nerve fibres in the pulp are myelinated A fibres (A-delta and A-beta fibres) and unmyelinated C fibres. Ninety percent of the A fibres are A-delta fibres, which are mainly located at the pulp–dentine border in the coronal portion of the pulp and concentrated in the pulp horns. The C fibres are located in the core of the pulp, or the pulp proper, and extend into the cell-free zone underneath the odontoblastic layer.10,11

The ratio of myelinated to unmyelinated fibres is difficult to ascertain because the nerve fibres in recently erupted teeth with open apices may not yet have acquired the myelin sheath.8,12

**Clinical Implications for Intrapulpal Sensory Nerve Fibres**

The A-delta fibres have a small diameter and therefore a slower conduction velocity than other types of A fibres, but are faster than C fibres. The A fibres transmit pain directly to the thalamus, generating a fast, sharp pain that is easily localized. The C fibres are influenced by many modulating interneurons before reaching the thalamus, resulting in a slow pain, which is characterized as dull and aching.10

The A fibres respond to various stimuli such as probing, drilling and hypertonic solutions through the hydrodynamic effect.13–16 This effect depends on the movement of the dentinal fluid in the dentinal tubules in response to a stimulus. Although the normally slow capillary outward movement does not stimulate the nerve endings and cause pain,17–19 rapid fluid flow, as in the case of desiccating or drying dentin, is more intense and is likely to activate the pulpal nociceptors.11 Heat or cold stimuli cause fluid movement through the dentinal tubules, resulting in a painful sensation in a tooth with a viable sensory pulp20,21 (Fig. 1). This response is due to the rapid temperature change that causes a sudden fluid flow within the tubules and deforms the cell membranes of the free nerve endings. A gradual change in temperature, however, does not cause an immediate pain response because rapid fluid movement exerts the A-delta fibres. The C fibres elicit a response to a gradual temperature change.10,22,23

A recent study24 attributed the pain caused by thermal changes to the mechanical deformation of the enamel and

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**Figure 1:** Illustration of the movement of dentinal fluid inside dentinal tubules in response to a hot stimulus (red arrow) and a cold stimulus (blue arrow).
The ionic concentration of the material also affects the diffusion of the acid’s chemical composition in the induction of pain because this osmotic pressure causes the outward fluid flow in the tubules, together with aspiration of the odontoblastic nucleus.26–28 The ionic concentration of the material also affects the reduction of pain in sensitive dentin. A normally irritant substance such as potassium chloride temporarily relieves pain because the high concentration of potassium temporarily blocks the conduction of nerve impulses, causing a hyperpolarization that decreases the excitability of the nerve fibres. This hyperpolarization is the basis for the addition of potassium ions to dentifrices.

In addition to pain from sensitive or exposed dentinal tubules, persistent pain may decrease the threshold of the nociceptors, usually during pulpal inflammation in which some of these mediators are manifested clinically by diffuse pain, called referred pain, from a specific tooth because nerve fibres innervate multiple teeth with multiple pulps.35 These fibres have less excitability than the A fibres and a higher threshold, so they need more intense stimuli to be activated. The C fibres may survive in the presence of hypoxia,33,36 which may explain pain sensed during preparation for the root canal of a necrotic pulp.37 The dentist should tell the patient that the pain will not be completely resolved after the dental visit, and that this pain may be caused by deafferentation, or the interruption of the afferent input into the central nervous system.38

All functional changes to the nociceptors are reversible on removal of the cause. For example, in the case of dentin hypersensitivity, the tubules are treated by blocking, which directly affects the A fibres (hydrodynamic cessation) and resolves the neural changes in the pulp, causing the pain to subside.39

In contrast to these morphologic and functional changes in pulpal nerve endings, the pulp, through its defensive mechanism, responds by secreting endogenous opioids, noradrenalin, somatostatin and specific chemical mediators in response to the toxins secreted by carious lesions to regulate the activity of nociceptors.40–42 Some of these mediators are excitatory; others, such as morphine, have an inhibitory effect. The neuroinflammatory and the neuropulpal interactions (nerve–odontoblast interactions) still need to be clarified.35

Based on this discussion of fibres and their responses, we can relate the type of fibres to clinical pulp testing methods:

- **Thermal pulp testing** depends on the outward and inward movement of the dentinal fluid, whereas electric pulp testing depends on ionic movement.43
- **Because of their distribution** larger diameter than that of C fibres, their conduction speed and their myelin sheath, A-delta fibres are those stimulated in electric pulp testing.44–46
- **C fibres** do not respond to electric pulp testing. Because of their high threshold, a stronger electric current is needed to stimulate them.46
- **Based on the hydrodynamic effect** outward movement of dentinal fluid caused by the application of cold (contraction of fluid) produces a stronger response in A-delta fibres than inward movement of the fluid caused by the application of heat.16,42,43
- **Repeated application** of cold will reduce the displacement rate of the fluids inside the dentinal tubules, causing a less painful response from the pulp for a short time, which is why the cold test is sometimes refractory.10
- **The A-delta fibres** are more affected by the reduction of pulpal blood flow than the C fibres because the A-delta fibres cannot function in case of anoxia.33,34
- **An uncontrolled heat test** can injure the pulp and release mediators that affect the C fibres.44,45
- **A positive percussion test** indicates that the inflammation has moved from the pulp to the periodontium, which is rich in proprioceptors, causing this type of localized response.
Conclusions
The dental clinician should not rely solely on the type of pain to determine a diagnosis. Other nonodontogenic types of pain, as well as psychological pain, may obfuscate the correct diagnosis. Type of pain has not been correlated with the histopathologic condition of the pulp. The A and C fibres are activated by different stimuli and different inflammatory mediators, producing changes in the quality of pain that range from a sharp shooting pain to a dull and prolonged pain. Mechanical stimulation such as probing causes a sharp pain by stimulating the A-delta fibres, whereas prolonged pain manifests after the removal of a thermal stimulus (mainly heat) activates the C fibres. The stimulus itself may indicate the type of pain, but does not indicate the changes occurring in the pulp tissue or the stage of inflammation occurring.

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