Operative Dentistry

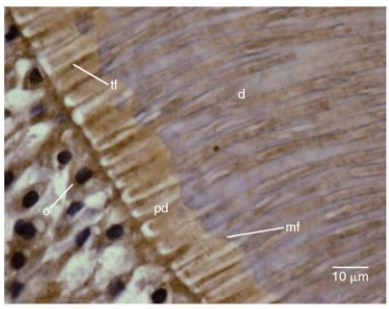
Lecture (4) **Dr. Raghad Noori**

Biologic Considerations of Dentin and their Clinical Significance in Operative Dentistry

(Part I-II)

❖ Dentin Formation

Dentin is a mineralized connective tissue that is covered by enamel in the crown and cementum in the root, as well as enclosing the innermost pulp. Dentin formation (dentinogenesis), is accomplished by cells called odontoblasts. Odontoblasts are considered part of pulp and dentin tissues because their cell bodies are in the pulp cavity, but their long, slender cytoplasmic cell processes (Tomes fibers) extend well (100–200 μm) into the tubules in the mineralized dentin, Because of these odontoblastic cell processes, dentin is considered a living tissue, with the capability of reacting to physiologic and pathologic stimuli (Fig). These processes extend beyond the DEJ into the enamel forming enamel spindles, which are responsible for sensitivity sensation during cavity preparation in enamel.



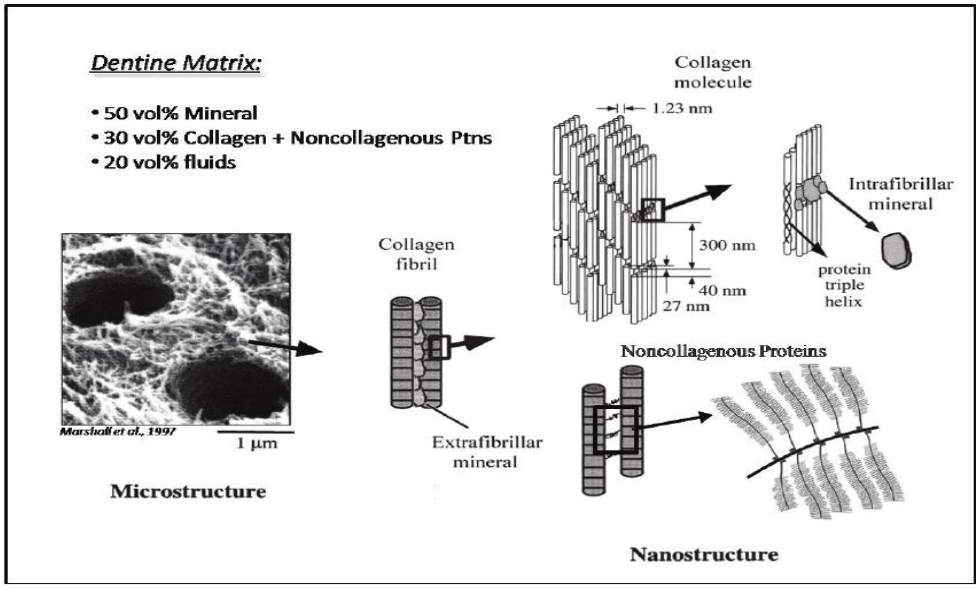
• **Fig. 1.14** Odontoblasts *(o)* have cell processes (Tomes fibers [tf]) that extend through the predentin *(pd)* into dentin *(d)*. *mf*, Mineralization front.



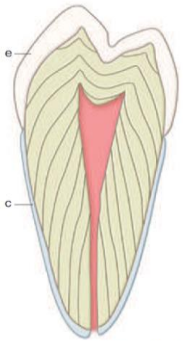
• **Fig. 1.15** Longitudinal section of enamel. Odontoblastic processes extend into enamel as enamel spindles *(A)*. (From Berkovitz BKB, Holland GR, Moxham BJ: *Oral anatomy, histology and embryology*, ed 4, Edinburgh, 2009, Mosby. Courtesy of Dr. R. Sprinz.)

Dentin formation begins immediately before enamel formation. The most recently formed layer of dentin is always on the pulpal surface. Odontoblasts generate an extracellular collagen matrix as they begin to move away from adjacent ameloblasts. Mineralization of the collagen matrix, facilitated by modification of the collagen matrix by various non-collagenous proteins,

gradually follows its secretion and deposition of intrafibrillar mineral crystals into the collagen fibrils (Fig)



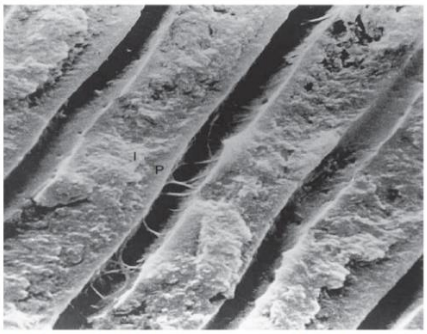
This unmineralized zone of dentin is immediately next to the cell bodies of odontoblasts and is called predentin (Fig 1.14). Dentin formation begins at areas subjacent to the cusp tip or incisal ridge and gradually spreads, at the rate of ~4 µm/day, to the apex of the root. In contrast to enamel formation, dentin formation continues after tooth eruption and throughout the life of the pulp. The dentin forming the initial shape of the tooth is called primary dentin and is usually completed 3 years after tooth eruption (in the case of permanent teeth).



❖ **Dentinal tubules:**

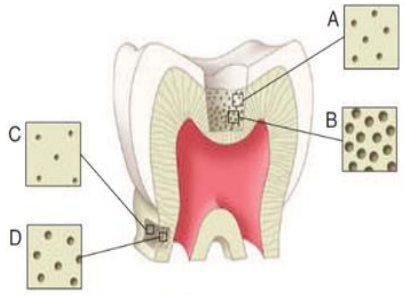
The dentinal tubules are small canal that remain from the process of dentinogenesis and extend through the entire width of dentin, from the pulp to the DEJ. Each tubule contains the cytoplasmic cell process (Tomes fiber) of an odontoblast and is lined with a layer of peritubular dentin, which is much more mineralized than the surrounding intertubular dentin (Fig). The tubules comprise about 10% of dentin volume. In mature dentin, the

odontoblastic process extends within the dentinal tubule to about one-third the dentinal thickness.

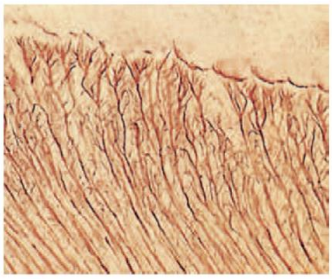


• **Fig. 1.18** Dentinal tubules in cross section, 1.2 mm from pulp. Peritubular dentin *(P)* is more mineralized than intertubular dentin *(I)*. (From Brännström M: *Dentin and pulp in restorative dentistry*, London, 1982,

The number of tubules increases from 15,000 to 20,000/mm2 at the DEJ to 45,000 to 65,000/mm2 at the pulp. The lumen of the tubules also varies from the DEJ to the pulp surface. In coronal dentin, the average diameter of tubules at the DEJ is 0.5 to 0.9 µm, but this increases to 2 to 3 µm near the pulp (Fig).
Tubules are generally oriented perpendicular to the DEJ. Along the tubule walls are small lateral openings called canaliculi or lateral canals. The lateral canals are formed as a result of the presence of secondary (lateral) branches of adjacent odontoblastic processes during **dentinogenesis.**



• **Fig. 1.19** Tubules in superficial dentin close to the dentoenamel junction (DEJ) *(A)* are smaller and more sparsely distributed compared with deep dentin *(B)*. The tubules in superficial root dentin *(C)* and deep root dentin *(D)* are smaller and less numerous than those in comparable depths of coronal dentin.

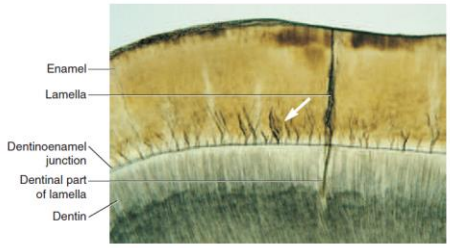


• **Fig. 1.21** Ground section showing dentinal tubules and their lateral branching close to the dentoenamel junction (DEJ). (From Berkovitz BKB, Holland GR, Moxham BJ: *Oral anatomy, histology, and embryology*, ed 4, Edinburgh, 2010, Mosby.)

❖ Dentinoenamel junction

It is the transition between the highly mineralized enamel and the collagen containing dentin. DEJ must resist fracture and separation under the extreme forces from occlusal loading. DEJ width falls within a range of 2 to 15 µm. Some collagen fibers from the dentinal material extend through this transitional area and are embedded in the more highly mineralized enamel. These embedded

collagen fibers may also add to the overall strength and resilience of the junction between the two tissues.



• **Fig. 1.10** Microscopic view through lamella that goes from enamel surface into dentin. Note the enamel tufts *(arrow)*. (From Fehrenbach MJ, Popowics T: *Illustrated dental embryology, histology, and anatomy*, ed 4, St. Louis, 2016, Saunders. Courtesy James McIntosh, PhD, Assistant Professor Emeritus, Department of Biomedical Sciences, Baylor College of Dentistry, Dallas, TX.)

❖ Dentin composition

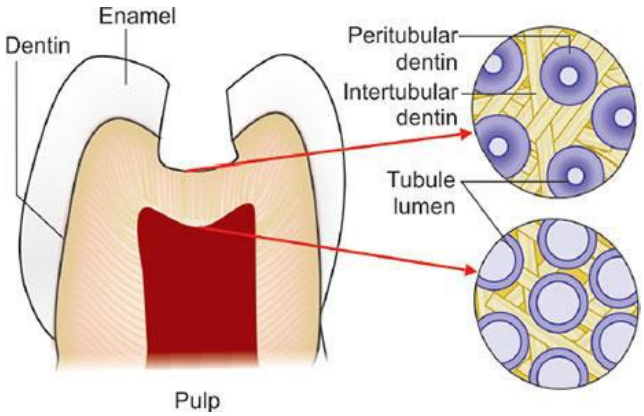
Mature dentin is a crystalline material that is less hard than enamel but slightly harder than bone. The odontoblast, with its cell body at the pulp periphery and its extended process within the dentinal tubule, secretes the organic dentin matrix and regulates mineralization. Mature dentin is 45% - 50% inorganic or mineralized material, 30% organic material, and 25% water. The organic material is approximately 90% type I collagen and 10% non-collagenous proteins. The crystalline formation of mature dentin mainly consists of calcium hydroxyapatite with the chemical formula of Ca10(PO4)6(OH)2. The calcium hydroxyapatite found in dentin is similar to that found in a higher percentage in enamel and in lower percentages in both cementum and bone tissue. In addition, the crystals in dentin are platelike in shape and 30% smaller in size than those in enamel. The thickness of the apatite crystals varies from 3.5 nm near the DEJ to about 2 nm close to the pulp. Small amounts of other minerals, such as carbonate and fluoride, are also present. The mineral content of dentin increases with age.

Structurally there are two main types of dentin which are:

1. Intertubular dentin: the primary structural component of it is the hydroxyapatite- embedded collagen matrix between tubules. Intertubular dentin forming the bulk of dentin structure.

2. Peritubular dentin: it is the hypermineralized dentin limited to the walls of dentinal tubules. The walls of the dentinal tubules (peritubular dentin) in the primary dentin gradually thicken, through ongoing mineral deposition, with age. The dentin therefore becomes harder, denser, and, because tubular fluid flow becomes more restricted as the lumen spaces become smaller, less sensitive.

The increased amount of mineral in the primary dentin is defined as dentin sclerosis. Dentin sclerosis resulting from aging is called physiologic dentin sclerosis.



The dentinal tubules in the Outer Dentin which is near the DEJ are relatively far apart and the Intertubular dentin makes up 96% of the surface area. In the Inner Dentin, the tubules diameters are larger and the distance between tubule centers is half that of tubules at DEJ. Thus, the intertubular matrix area is only 12% of the surface area, and the permeability of inner dentin is about eight times more permeable than the dentin near DEJ.

❖ Physiologic and Tertiary Dentin

Some of the variations in the dentinal tissue occur during its natural sequence of development or aging, while other variations result from the effects of external factors on the tooth, such as caries, injury, or wear. An understanding of these variations in the dentinal tissue is important because they are related to the long-term success of dental procedures and therapies.

❖ Physiologic dentin

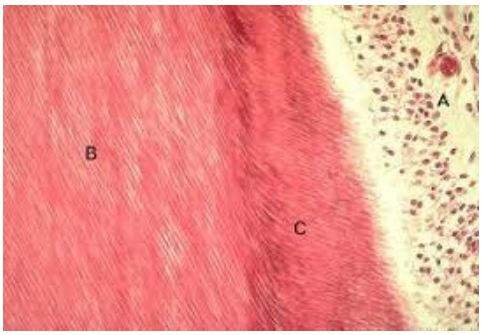
Odontoblasts synthesize and secrete extracellular organic matrix, which, following mineralization, forms the primary and secondary physiologic dentin (Fig).



1. Primary dentin: formed relatively quickly until root formation is completed by odontoblasts. The first-formed, 150-µm-thick layer of primary dentin subjacent to the enamel is termed mantle dentin. It differs from other primary dentin in that it is 4% less mineralized, and the collagen fiber orientation is

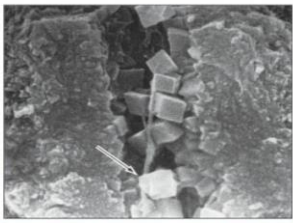
perpendicular rather than parallel to the DEJ. The odontoblasts produce primary dentin, mainly intertubular dentin, at a rate of 4 to 8 μm/day. Approximately 2 to 3 years following tooth eruption. The bulk of dentin surrounding the pulp chamber and canal systems, termed circumpulpal dentin. The synthesis of dentin then slows to 1 to 2 μm/day, decreasing in rate with age but continuing as long as the tooth is vital.

2. Secondary dentin: This is a slowly formed dentin of 1 to 2 μm/day, decreasing in rate with age but continuing as long as the tooth is vital. This continued deposition of secondary dentin led to constriction in the dimensions of the pulp chamber. In response to mild occlusal stimulus, secondary dentin is mainly deposited in the pulp horns and on the roof and floor of the pulp chamber so after many decades the chamber becomes quite narrow occluso-gingivally. This constriction in pulp chamber size reduced the possibility of pulp exposure in old patients during cavity or crown preparation. The dentist must pay attention for the size and location of the pulp chamber to decide the design of the preparation and placement of retentive features such as pins (secondary dentin is C in the figure).



❖ **Sclerotic dentin (transparent or peritubular dentin)**

This type of dentin results from aging or mild irritation (such as slow caries, attrition, abrasion, erosion and occlusal trauma) and causes a change in the composition of the primary dentin. The tubular content appears to be replaced by calcified material that obliterates the tubules, progressing from the DEJ pulpaly. The tubules become progressively hypermineralized and, with constriction of the lumen and less permeable. The sclerotic dentin is harder, denser, less sensitive and more protective of the pulp against subsequent irritation.



Sclerosis resulting from aging is **(physiological dentin sclerosis)** and that resulting from mild irritation called **(reactive dentine sclerosis).** The combination of peritubular wall thickness and intratubular crystals creates a zone of hypermineralized dentin beneath exposed or carious dentin, the zone of sclerosis or translucent zone (see Fig). Sclerotic dentin is frequently found beneath both active caries lesions and restorations and is an important defense reaction of the hard tissues because it limits the permeability of dentin.

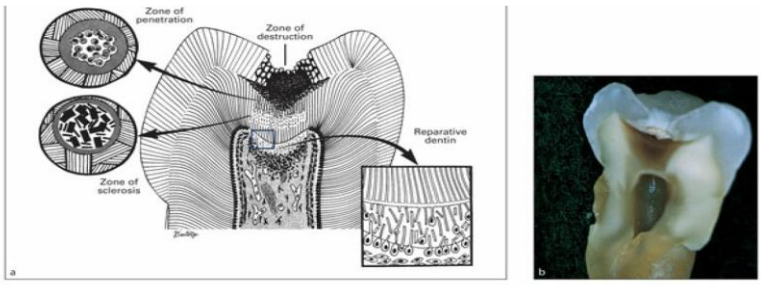
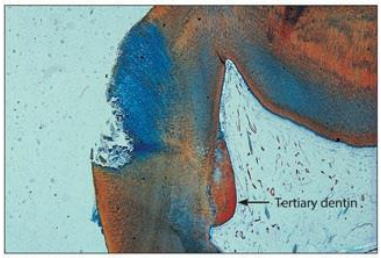


Fig 1-23 Tooth response to carious destruction of tooth surface. Acid demineralization and enzymatic destruction of the collagen matrix lead to cavitation, an irreversible change. *(a)* Bacteria fill and demineralize the lumens of the tubules peripherally, but dissolved minerals reprecipitate at a deeper level to augment sclerosis and hypermineralization of subcarious dentin. Reparative dentin with irregular and noncontinuous tubules forms a final barricade against bacterial metabolites. *(b)* Note the lateral spread of the caries lesion at the DEJ and a hypermineralized sclerotic zone around the pulp.

❖ **Reparative dentin (tertiary dentin)**

Intense traumatic insult (injury) to the tooth, whether caused by bacterial penetration associated with caries, or heat and trauma from a dental bur, may be severe enough to destroy the supporting odontoblasts in the affected location. Within 3 weeks, fibroblasts or mesenchymal cells of the pulp are converted or differentiated to simulate the activities of original odontoblast, and form irregularly organized a tubular dentin called reparative dentin



	Primary	*Secondary*	*Tertiary*
Definition	Dentin formed before root completion	Formed after root completion	Formed as a response to any external stimuli such as dental caries, attrition and trauma
Type of cells	Usually formed by primary odontoblasts	Formed by primary odontoblasts	Secondary odontoblasts or undifferentiated mesenchymal cells of pulps
Location	Found in all areas of dentin	It is not uniform, mainly present over roof and floor of pulp chamber	Localized to only area of external stimulus
Orientation of tubules	Regular	Irregular	Atubular
Rate of formation	Rapid	Slow	Rapid between 1.5 and 3.5 µm/day depending on the stimuli
Permeability	More	Less	Least

Dentin Function

There are different functions of dentin which can be summarized as follow:

1. **Structure:**
   Dentin forms the bulk of the tooth (both in the crown and root). It constitutes the largest percentage of tooth substrates.
2. **Color:**
   The coronal dentin (crown) provides color for the overlying enamel. Because of the translucency of overlying enamel, the dentin of the tooth gives the white enamel crown its underlying yellow hue, which is a deeper tone in permanent teeth. When the pulp undergoes infection or even dies, there is discoloration of the dentin, which causes darkening of the clinical crown.
3. **Elasticity:**
   Dentin also has great tensile strength, providing an elastic basis for the more brittle enamel. The tensile strength for enamel and dentin are 10 and 98 MPa, respectively. Although enamel is harder but enamel is very brittle and can be fracture without support of dentin which characterized by elasticity to absorb the heavy occlusal loads.
4. **Support:**
   Tooth strength and rigidity are provided by intact dentinal substrate. Resistance of tooth to fracture is significantly lowered with increasing depth and width of cavity preparation. For example, A posterior tooth with an endodontic access preparation retains only a third of the fracture resistance of an intact tooth. Therefore, a conservative initial approach that combines localized removal of carious tooth structure, placement of a bonded restoration, and placement of sealant is recommended. If large

preparations are required, the dentist should consider placement of inlays, onlay or crown. Also, higher concentrations of tooth-whitening (bleaching) agents, may have adverse effects on the fracture toughness of dentin.

5. **Protection:**
   Protective encasement for the pulp. As a vital tissue without vascular supply or innervation, it is nevertheless able to respond to thermal, chemical or tactile external stimuli because of the presence of dentinal tubules that permit stimuli transmission to the pulp.

❖ **Dentin can be distinguished from enamel (during tooth preparation), by:**

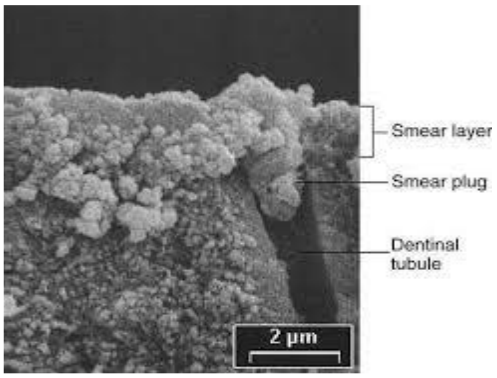
- **Color:** Enamel is translucent in color depending on its thickness, while, dentin is normally yellow-white and slightly darker than enamel. In older patient's dentin is darker and become brown or black in cases in which it has been exposed to oral fluids, old restorative materials or slowly advancing caries.
- **Reflectance:** dentin surfaces are opaquer and duller, being less reflective to light than enamel surfaces, which appear shiny.
- **Hardness:** dentin is softer than enamel, sharp explorer tends to catch and hold in dentin. The hardness of dentin averages one fifth that of enamel, and its hardness near the DEJ is about three times greater than near the pulp. Although dentin is a hard, mineralized tissue, it is flexible. This flexibility helps support the more brittle, less resilient enamel. Dentin is not as prone to fracture as is the enamel rod structure.
- **Sound:** when moving an explorer tip over the tooth, enamel surfaces provide a sharper, higher pitched sound than dentin surfaces

	Enamel	*Dentin*
Color	Whitish blue or white gray	Yellowish white or slightly darker than enamel
Sound	Sharp, high-pitched sound on moving fine explorer tip	Dull or low-pitched sound on moving fine explorer tip
Hardness	Hardest structure of the tooth	Softer than enamel
Reflectance	More shiny surface and reflective to light than dentin	Dull and reflects less light than enamel

❖ **Permeability of Dentin**

The permeability of dentin can compromise its protective function because of the presence of dentinal tubules, which are very important during formation of dentin, however, when the external layer of enamel and cementum is lost from the periphery of the dentinal tubules through caries, preparation with burs or abrasion and erosion, the exposed dentinal tubules become channels between

the pulp and the external oral environment. However, in case of dentin cutting with bur, these exposed dentinal tubules usually occluded by a layer of tenacious grinding debris called the smear layer, which adheres to the surface and partially plugs the tubular orifices. However, the removal of the smear layer with acid etching during dentin bonding procedures causes an increase in the local dentinal permeability along with an outward fluid movement from the tubules, which results in adverse conditions for adhesive bonding by creating wet environment which may reduce bond strength of bonding agent over time.



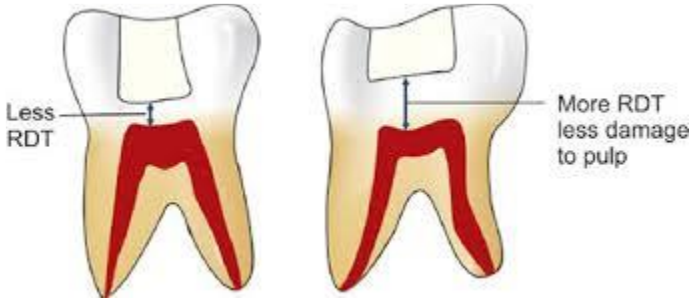
Also, the restored teeth are at risk of toxic leakage through the phenomenon of microleakage (which is defined as diffusion of the bacteria, oral fluids, ions and molecules into the tooth and the filling material interface) between the restorative material and the cavity wall, through capillary action, and diffusion of fluids containing various acidic and bacterial products can penetrate the gap between the tooth and restoration and initiate secondary caries of the internal cavity walls. Bacterial byproducts can continuously diffuse through permeable dentinal tubules to reach the pulp, putting the tooth at risk for pulpal inflammation and sensitivity.

No restorative material or technique can ensure a complete hermetic seal of the restoration-tooth interface, and leakage at the gingival (cementum or dentin) margins of resin-bonded restorations is commonly reported.



Dye experiment shows the microleakage through restoration tooth interface.

Restorative techniques with varnishes, liners or dentin bonding resin adhesives are effective to provide reliably sealed margins and sealed dentinal surface. However, the remaining dentin thickness is the key determinant of the rate of diffusion of gradient into the pulp, because thicker remaining dentin thickness with provide both smaller tubular diameters and greater tubular lengths, therefore thicker remaining dentin thickness can significantly reduce both the adverse toxic effect of restorative materials and bacterial irritation to the pulp.



There are also regional differences in dentinal permeability. Coronal dentin is much more permeable than root dentin. The coronal occlusal dentin (pulpal floor of a cavity preparation) is inherently less permeable than is the dentin around the pulp horns or axial surfaces. As a result, only about 30% of the subjacent dentinal tubules on occlusal surface are patent over their entire length. However, prepared proximal boxes or gingival margins, which are relatively more susceptible to microleakage and development of recurrent caries lesions, are located where the dentin is most permeable.

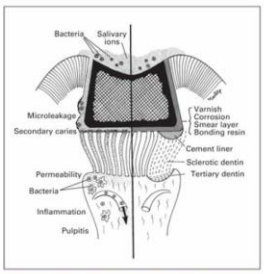


Fig 1-14 Leaking restoration interface *(left)*; sealed restoration interface *(right)*. Microleakage is exacerbated by polymerization shrinkage, condensation gaps around the restorative material, and/or differences in thermal expansion. When microleakage is present, the tubule openings in dentin form a potential pathway between the oral environment and the pulp. Various restorative materials, together with the tooth's defenses of tubule sclerosis and reparative dentin, restrict the noxious infiltration.

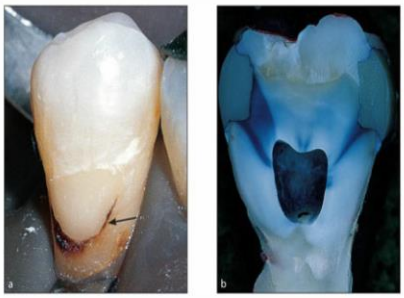


Fig 1-15 *(a)* Failed resin composite restoration. Polymerization shrinkage and cervical debonding created a restoration-wall gap defect (*arrow*, cervical margin), leading to microleakage and secondary caries. *(b)* In vitro dye penetration reveals microleakage and diffusion through dentinal tubules.

❖ Sensitivity of Dentin

Although dentin is sensitive to thermal, tactile and osmotic stimuli across its (3-3.5 mm) thickness. Dentin is neither vascularized nor innervated, except for about 20% of tubules that have nerve fibers penetrating inner dentin by few microns. Also, odontoblastic processes in the dentinal tubules reach only the inner third of dentin and their cell membrane is not conductive, however recent studies suggest that odontoblasts could possibly be directly involved in the

sensory transduction process of the tooth through some form of mechanosensory system.

❖ Theories of Dentin Sensitivity

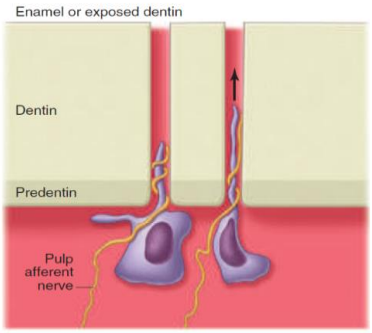
There are many theories proposed to explain dentin sensitivity:

1. Direct innervation theory: According to direct innervation theory, nerve endings penetrate dentine and extend to the dentino-enamel junction. Direct mechanical stimulation of these nerves will initiate an action potential. There are many shortcomings of this theory. There is lack of evidence that outer dentin, which is usually the most sensitive part, is innervated. Developmental studies have shown that the plexus of Rashkow and intratubular nerves do not establish themselves until the tooth has erupted; yet, newly erupted tooth is sensitive. Moreover, pain inducers such as bradykinin fail to induce pain when applied to dentine, and bathing dentine with local anesthetic solutions does not prevent pain, which does so when applied to skin.

2. Odontoblast receptor theory: Odontoblast receptor theory states that odontoblasts act as receptors by themselves and relay the signal to a nerve terminal. But majority of studies have shown that odontoblasts are matrix forming cells and hence they are not considered to be excitable cells, and no synapses have been demonstrated between odontoblasts and nerve terminals

3. Theory of thermal shock: This states that sensitivity is the result of direct thermal shock (temperature change) to the pulp via temperature changes transferred from the oral cavity through the tooth or restorative material, especially when the remaining dentin is thin. Protection from this insult would then be provided by an adequate thickness of an insulating material such as cements.

4. The hydrodynamic theory: This theory based on the capillary flow dynamics of the fluid-filled dentinal tubule. In a vital tooth with exposed dentin there is a constant slow movement of fluid outward through the dentinal tubules. The hydrodynamic theory proposed that when a stimulus such as air evaporation, cold or heat (i.e. generated from dental bur) or tactile pressure with probe cause the slow fluid movement to become more rapid causing displacement in the odontoblastic cell bodies and stretches the intertwined terminal branches of the nerve plexus to allow the entry of sodium to initiate depolarization and the perception of pain.



• **Fig. 1.23** Stimuli that induce rapid fluid movements in dentinal tubules distort odontoblasts and afferent nerves *(arrow)*, leading to a sensation of pain. Many operative procedures such as cutting or air-drying induce rapid fluid movement.

As dentin near the pulp, tubule density and diameter increase also the permeability increase, thus increasing both the volume and flow of fluid. This explains why deeper restorations are associated with more problems of sensitivity. In root dentin hypersensitivity the degree of tubule closure from intratubular crystals was directly correlated with decreased sensitivity to touch, air, or temperature stimuli. Tooth pastes that contain strontium chloride, oxalates can promote intratubular crystallization and desensitize dentin. Resin bonding agents, fluoride varnishes and potassium and silver nitrate can also reduce dentin hypersensitivity by occluding dentinal tubules or nerve desensitization. Calcium silicate cements can reduce dentin hypersensitivity by remineralization as well.

Comparison between thermal shock and hydrodynamic theories:

According to hydrodynamic theory, during temperature change, if the tubules can be occluded, rapid outward fluid flow is prevented and temperature do not induce pain. Therefore, reducing dentin sensitivity to thermal changes is by effective sealing of the dentinal tubules rather than placement of an insulating material (according to thermal shock theory). The hydrodynamic theory has gained general acceptance in recent years and has changed the direction of restorative procedures away from thermal insulation and toward dentinal sealing. Thus, there is increasing emphasis on the integrity of the interface between restorative material and cavity preparation in adhesive restorations. Also, it is worth noting that when the tooth exposed to thermal, mechanical and chemical stimuli for a long time, the rate of formation and the thickness and organization of reactionary dentin (as a response of pulp to stimuli) depend on the intensity and duration of the stimulus. The barrier of reactionary dentin is superior because there is no continuity between the affected permeable tubules of the regular primary dentin and those within the reactionary dentin. The tooth will be able to compensate for the traumatic or carious loss of peripheral dentin with deposition of new dentin substrate and reduction of pulpal irritation from

tubule permeability. Unless the lesion is either arrested or removed and a restoration placed, the diffusion of bacterial toxins reaching the pulp and initiate strong inflammatory response and result in pulpal necrosis