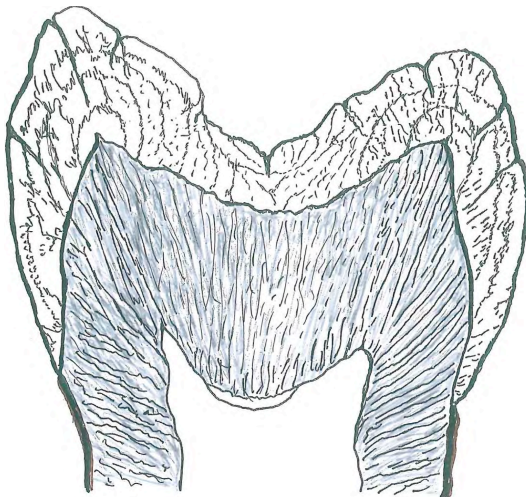


DENTINE FACTOID

This FACTOID focuses upon the morphology & physiology of vital dentine. Until histologists in the 1800's were able to suitably demineralize enamel & dentine for proper thin sectioning—histologists were unable to understand the microstructure of tooth tissues. Knowledge of dentine had been limited to thick ground sections by such US clinicians as Chapin Harris, Greene V. Black & Marshall H. Webb.

Dentine is the hardest vital tissue of the human body, a **variable substrate** composed of unique **morphological**, biochemical & physiological differences—the clinician should realize dentine is **NOT** a uniform substrate. It is covered by non-vital enamel that is dense & rigid (99% mineral - 1% organic) that ends at the cervical-gum margin, whereas vital dentine (65% mineral - 32% organic & approximately 3% fluid) constitutes the bulk of the tooth. Dentine is a flexible substrate that provides elasticity during the chewing of food—preventing severe catastrophic fracture.



The enamel cap is seen—above left—with incremental lines & thin lamella spaces that project from outer surface grooves & run to the EDJ that separates the 2-mineral substrates. The blue-gray is dentine showing tubules running from the EDJ to the pulp. A small zone of reparative dentine is seen directly below the central bulk of dentine. Cementum—covers the entire root surface—extending apically from the enamel-cervical edge that covers the external root-dentine interface as a thin vital tissue, which is biologically similar to bone—except it does **not** have any innervation or blood vascular supply.

STRUCTURAL FEATURES OF DENTINE

Dentine has **two** unique components made of **calcium hydroxy apatite (CHA)**:

Intertubular dentine (ITD) is the greater morphological fraction that lies between the tubules. **ITD** is composed of CHA that is distributed amongst the fibers of collagen, proteoglycans, glycoprotein & other organic components. The area of **ITDentine** is largest at the EDJ where small tubules emerge from the tissue interface—the area of dentine at the EDJ interface composes about 94%—as small tubules project towards the dentine-pulp interface. Whereas the area of **ITDentine** occupies about 94% of the dentine-pulp interface region. Each tubule is shaped like a funnel (ferrule effect) being smallest at the EDJ & largest at the dentine-pulp interface—like an old-fashioned ice-cream cone placed directly over another cone.

Peritubular dentine (PTD) is the most dense (99.5%) portion of the dentine substrate being composed almost exclusively of CHA with less than 0.5% collagen or other organic protein components. The **PTDentine** completely lines each tubule wall as a uniformly thin circumferential ring of dense CHA mineral, being deposited by the odontoblast process & cells that line the pulp-dentine matrix along the predentine interface of the pulp border.

The dentine morphology of each tooth is composed of **dentinal tubules** that project from the EDJ to the pulp interface, where the odontoblast cells are arranged as a specialized sheet of cells. As mentioned above, each tubule is smallest at the EDJ & largest at the pulp interface. Each tubule contains an **odontoblast process**, which actually represents the greatest volume—approximately 70%—of each cell. Other tubule contents are collagen fibers, fine non-myelinated nerves found in about 1 of 10 tubules (Gunji 1982) & small calcium granules, which are involved in the ongoing deposition of peritubular dentine (Matthews (1978). As first reported by John Neill in 1839 again by Alfred Gysi in 1900 & later Martin Brännström in 1967, a fluid that flows from the pulp, through the tubules into the enamel is known to be directly responsible for the initial stimuli of pain in dentine. Each dentine-tubule follows a rather long-lazy S-shaped course from the EDJ to the pulp interface. Odontoblast cell differentiation is

due to an inductive message from the ameloblast, which crosses the interface membrane (membrane preformata). Occasional odontoblasts leave remnants of their process within the enamel matrix that are called an **enamel spindles**, only seen in the crown. The first zone of dentine that is deposited before eruption of the tooth is called **mantle dentine**—an almost perfect substrate free of imperfections. Once eruption of the tooth commences along its eruption pathway (gubernaculum dentis), the dentine becomes subjected to various metabolic & physiological factors, which leaves a hypomineralized area called the **interglobular dentine**. After eruption is complete, the underlying dentine is called secondary-dentine that often presents small irregularities & defects. Below the enamel-cervical margin, the root dentine continues as epithelial cell nest remnants of ameloblasts deposit a thin epithelial **acellular cementum** against the root dentine (Randi Furseth 1968). In the root, dentine, odontoblasts continue their rhythmic deposit of mineralized dentine in which most of the odontoblasts—for some unknown reason—literally spin around upon themselves & leave a hypomineralized zone called the **granular layer of Tomes**—Dr. Tomes described this unique granular layer in 1842 & Professor A. R. Ten Cate explained its formation in 1972.

As the clinical crown becomes subjected to various oral factors such as poor-to-none oral hygiene, carious biofilm may penetrate through the enamel lamella or other defects to the EDJ where it spreads laterally & then invades the dentine tubule complex. During the initial carious bacterial insult, lactic acid is produced, which demineralizes the CHA of the PTDentine & the **ITDentine**, leaving the collagen & other dentine proteins unaffected. If at that time, the caries is halted—due to fluoride or oral hygiene intervention—a zone of **affected dentine** is left. At this point the remaining vital odontoblasts processes process calcium-matrix granules into the tubule space where they become incorporated into the dense hard mass of mineral that fills the tubule lumen as **sclerotic dentine**. This process is a normal physiological response that occurs without any clinical intervention.

If a secondary invasion of **anaerobic** bacteria follows the initial invasion of *Strep mutans*, the new anaerobes secrete proteolytic enzymes, which completely destroy

the remaining collagen & other proteins—this dead zone of dentine is called **infected caries**. This outer infected caries zone is easily removed with gentle excavation with hand instruments following its identification by a biological dye agent (**DiscovRED**, or **C-BLUE**) that stains the bacteria in the dead tissue.

When a minor carious insult proceeds the length of the dentinal tubule complex to a localized area of the vital pulp interface, some of the **original odontoblasts** become stimulated from certain physiological means & they may secrete a thin layer of **reactionary dentine**. However, if a number of original primary odontoblasts die from the carious lesion, a new population of odontoblastoid cells are recruited, which proliferate to the dentine interface below the zone of caries & commence to deposit a new zone of **reparative dentine** that attempts to replace the volume of original dentine that was removed by operative procedures.

CLINICAL CONSIDERATIONS

Dentine is **NOT** a uniform substrate---it is **extremely variable**.

The greatest concentration of CHA mineral in dentine is at the EDJ.

The dentine tubules are smallest at the EDJ.

The organic component of dentine is greatest at the dentine-pulp interface.

Adhesives that rely on CHA for bonding will have higher bond values at the EDJ.

Larger tubules present higher fluid concentrations with lower expected bond strength.

Moisture control is an extremely variable consideration in a multidimensional cavity.

Fluid flow is greatest towards the pulp & least evident towards the EDJ.

As a cavity becomes larger with removal of more dentine, the amount of subjacent reparative dentine below the cut tubules becomes larger.

The bulk of dentine continues to be deposited throughout life making the pulp chamber smaller & less subject to sensory stimulation.

As dentine thickness increases throughout life, the color of the tooth becomes **darker** & more subject to environmental factors—aged dentine is more difficult to whiten.

You must think of using an adhesive system that is less technique sensitive in a moist environment when the cavity is placed in deeper dentine.

Above all, attention to detail & material technique sensitivity remain most important.