牙體復形學 Operative dentistry

Biologic Considerations

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學習目標

- 期許同學在瞭解病患的需求及材料的特性後，能依據所學善加利用，做到不僅是幫病患解決病痛的牙醫師，同時也是個讓病患永遠也忘不了的藝術家。

1. 牙齒的生理，解剖形態
2. 齲齒的生理，診斷及治療計劃
3. 窩洞的修形及材料的選擇
4. 窩洞的充填方式及其修飾
5. 美觀性材料的選擇及其運用
6. 變色牙的修飾

參考資料

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2. Fundamental of operative dentistry. A contemporary approach 3rd edition, James B. Summitt.

Summary

Operative dentistry is the basic science in clinical dental practices.It included dental physiology,morphology,cariology,tooth preparation for restoration.The purpose of Operative dentistry is to complete the function and create the aethestic outlook.

Enamel

Enamel provides a hard, durable shape for the functions of teeth and a protective cap for the vital tissuesof dentin and pulp. Both color and form contribute tothe esthetic appearance of enamel. Much of the art of restorative dentistry comes from efforts to simulate the color, texture, translucency, and contours of enamel with synthetic dental materials such as resin composite or porcelain. Nevertheless, the lifelong preservation of the patient's own enamel is one of the defining goals of the dentist.6 Although enamel is capable of lifelong service, its crystallized mineral makeup and rigidity, as well as stress from occlusion, make it vulnerable to acid demineralization (caries), attrition (wear), and fracture (Fig 1-2). Compared to other tissues, mature enamel is unique in that, except for alterations in the dynamics of mineralization, repair or replacement is only possible through dental therapy.

Permeability

At maturity, enamel is about 90% inorganic hydroxyapatite mineral by volume. Enamel also contains a small amount of organic matrix, and 4% to 12% water, which is contained in the intercrystalline spaces and in a network of micropores opening to the external surface. The micropores form a dynamic connection between the oral cavity and the systemic, pulpal, and dentinal tubule fluids. When teeth become dehydrated, as from nocturnal mouth breathing or rubber dam isolation for dental treatment, the empty micropores make the enamel appear chalky and lighter in color (Fig 1-3). The condition is reversible with return to the "wet" oral environment. Various fluids, ions, and low-molecular weight substances, whether deleterious, physiologic, or therapeutic, can diffuse through the semipermeable enamel. Therefore, the dynamics of acid demineralization, caries, reprecipitation or remineralization, fluoride uptake, and vital bleaching therapy are not limited to the surface but are active in three dimensions.

Permeability

Gradual coloration and improved caries resistance are two results of lifelong exposure of semipermeable enamel to the ingress of elements from the oral environment into the mineral structure of the tooth. The yellowing of older teeth may be attributed partly to accumulation of trace elements in the enamel structure and perhaps to the sclerosis of mature dentin. Surface enamel benefits from incorporation of salivary or toothpaste fluoride to increase the ratio or conversion of hydroxyapatite to larger, more stable crystals of fluorohydroxyapatite or fluoroapatite. Therefore, with aging, color is intensified and acid solubility, pore volume, water content, and permeability of enamel are reduced.

Clinical Appearance and Diagnosis

The dentist must pay close attention to the surface characteristics of enamel for evidence of pathologic or traumatic conditions. Key diagnostic signs include color changes associated with demineralization, cavitation, excessive wear, morphologic faults or fissures, and cracks (Fig 1-2).

Color

Enamel is relatively translucent; its color is primarily a function of its thickness and the color of the underlying dentin. From approximately 2.5 mm at the cusp tips and 2.0 mm at the incisal edges, enamel thickness decreases significantly below deep occlusal fissures and tapers to a negligible thickness cervically at the junction with the cementum or dentin of the root. Therefore, the young anterior tooth has a translucent gray or slightly bluish enamel tint at the thick incisal edge. A more chromatic yellow-orange shade predominates cervically, where dentin shows through thinner enamel. Coincidentally, in about 10% of teeth, a gap between enamel and cementum at thisjuncture leaves vital, potentially sensitive dentin completely exposed.

Color

Anomalies of development and mineralization, extrinsic stains, antibiotic therapy, and excessive fluoride can alter the natural color of the teeth. However, because caries is the primary disease threat to the dentition, color changes related to enamel demineralization and caries are critical diagnostic observations. The translucency of enamel is directly related to the degree of mineralization. Subsurface enamel porosity from carious demineralization is manifested clinically by a milky white opacity termed a white spot lesion when located on smooth surfaces (Figs 1-2, 1-4a, and 1-4b). In the later stages of caries, internal demineralization of enamel at the dentinoenamel junction (DEJ) imparts a whiteness or opacity seen through the more translucent overlying enamel.

Color

Subsurface cavitation imparts a blue or gray tint to the overlying enamel. With the advent of remineralization and sealant techniques, several authorities have suggested that invasive restorative procedures or replacement restorations should be initiated only if caries extension to dentin can be confirmed visually or radiographically, Smooth surface enamel that is chalky white and roughened from prolonged contact with acidic plaque (Fig 1-4a) generally indicates that the patient has inadequate oral hygiene, has a cariogenic diet, and is at a higher risk for caries.

Cavitation

Unless prevention or remineralization can abort or reverse the carious demineralization, dentin is affected until the undermined enamel breaks away to create a "cavity"; a restoration must then be placed. Untreated, the cavitation expands to compromise the structural strength of the crown, and microorganisms infiltrate into deep dentin to jeopardize the vitality of the tooth. When the carious lesion extends gingival to the cementoenamel junction (CEJ), as in root caries (Fig 1-2), isolation, access, and gingival tissue response complicate the restorative procedure.

Wear

Enamel is as hard as steel, with a Knoop Hardness Number of 343 (compared with a Knoop Hardness Number of 68 for dentin). However, enamel will wear because of attrition or frictional contact against opposing enamel or harder restorative materials, such as porcelain. Normal physiologic contact wear for enamel is as much as 29 micrometer per year. Restorative materials that replace or function against enamel should have compatible wear, smoothness, and strength. Heavy occlusal wear is demonstrated when rounded cuspal contacts are ground to flat facets. Depending on factors such as bruxism, other parafunctional habits, malocclusion, age, and diet, cusps may be completely lost and enamel abraded away so that dentin is exposed and occlusal function compromised. However, the effects on vertical dimension from tooth wear may be offset by apical cementogenesis and tooth eruption. Cavity outline form should be designed so that the margins of restorative materials avoid critical, high-stress areas of occlusal contact.

Faults and Fissures

Various defects of the enamel surface may contribute to the accumulation and retention of acidic plaque. Perikymata (parallel ridges formed by cyclic deposition of enamel) (Fig 1-5), pitting defects formed by termination of enamel rods, and other hypoplastic flaws are common, especially in the cervical area. Limited linear defects or craze lines result from a combination of occlusal loading and age-related loss of resiliency but are not clinically significant. Organic films of surface pellicle and dendritic cuticles extending 1 to 3 micrometer into the enamel may play key roles in ion exchange and in adhesion and colonization of bacterial plaque on the enamel surface.

Faults and Fissures

Of greater concern are the fissure systems on the occlusal surfaces, and often on other surfaces, of posterior teeth. A deep fissure is formed by incomplete fusion of lobes of cuspal enamel in the developing tooth. The resulting narrow clefts provide a protected niche for acidogenic bacteria and the organic nutrients they require (Figs 1-4b, 1-4c, 1-6a, and 1-6b). Because of these fissure faults, 57.7% of total decayed, missing, and filled surfaces (DMFS) of school children in the United States occur on the occlusal surfaces; only 12.0% are found on the mesial and distal surfaces. Altogether, pit and fissure defects are eight times more vulnerable to caries than are smooth surfaces. Careful observation of enamel surrounding fissures for evidence of demineralization or cavitation is necessary to determine the need for restorative intervention.

Cracks

Although craze lines in the surface enamel are of little consequence, pronounced cracks that extend from developmental grooves across marginal ridges to axial surfaces, or from the margins of large restorations, may portend a coronal or cuspal fracture. This defect is especially critical when the crack, viewed with cavity preparation, extends through dentin or when the patient has pain when chewing. A cracked tooth that is symptomatic or involves dentin requires a restoration that provides complete cuspal coverage.

Crystal Structure

Enamel is a mineralized epidermal tissue. The organic matrix gel is first formed and then later partly digested by ameloblastic ceils of the developing tooth organ. Calcium and phosphorus in the form of hydroxyapatite are seeded throughout the developing matrix and immediately begin to crystallize, enlarge, and supplant the organic matrix.

Crystal Structure

The majority of hydroxyaparite crystals, $Ca_{10}(PO_4)_6(OH)_2$, exist in an impure form in which ions or molecules are missing, or extrinsic substitutions occur to destabilize the crystal and make it more soluble. An important therapeutic exception is the incorporation of fluoride ion through systemic intake or topical application via remineralization dynamics. In mature enamel, the closely packed, hexagonal crystals are 25 to 39 nm in thickness and 45 to 90 nm in width (Fig 1-7). Crystal length, whether columns of full enamel thickness or segmented units, is yet to be determined. The matrix proteins, enamelins, and water of hydration form a shell, or envelope, around each crystal. Because the crystals are oriented perpendicular to the concave contours of the secreting ameloblastic cells, the crystal orientation gradually varies by as much as 70 degrees from the center of the cell (corresponding to the core center of the enamel rod) to the periphery (Fig 1-8). The closely packed crystal deposition, repeated in a symmetric pattern, forms the basic structural units of enamel, the rods.

Enamel Rods

The enamel rods are described as keyhole- or mushroom-shaped, with a circular core, or head, 4 to 5 micrometers in diameter, in which the long axis of crystals runs approximately parallel to the rod. Cervically, the progressive disinclination of the crystals produced from the boundaries of adjacent ameloblasts forms a fanshaped tail known as the interrod area (Fig 1-9). Except for a narrow, highly mineralized zone without rod structure both at the surface and at the DEJ, each rod runs the full thickness of the enamel. Because each row of rods is offset, the core of each rod is surrounded by the interrod substance of adjacent rods. As a result, the occlusal three-fourths of each core boundary is characterized by a junction of crystals meeting at acute angles. This interface, termed the rod sheath, is only increased intercrystalline space in which micropores and greater amounts of organic matrix are located.

Enamel Rods

The spacing and orientation of the crystals and amount of organic matrix make the enamel rod boundary and central core differentially soluble when exposed for a brief time to weak acids. Acid etchanrs remove about 10 pm of surface enamel and then preferentially dissolve either the rod core or periphery to form a three-dimensional, pitted surface with microporosities greater than 20 μm in depth (Fig 1-10). The acid-treated enamel surface has a high surface energy, so that resin monomer flows into and intimately adheres to the etched depressions to polymerize and form retentive resin tags. Because there are 30,000 to 40,000 enamel rods/mm2 and the etch penetration increases the bondable surface area 10-to 20-fold, micromechanical bonding of resin restorative materials to enamel is significant.

Enamel Rods

Transverse sections of enamel rods promote optimum tensile bond strengths to etched enamel of 9 to 20 MPa, compared to 11 MPa for longitudinal or parallel sections. Acid-etch modification of enamel for restoration retention provides a conservative, reliable alternative to traditional surgical methods of tooth preparation and restoration.

Enamel Rods

The enamel rod boundaries form natural cleavage lines through which longitudinal fracture may occur. The fracture resistance between enamel rods is especially imperiled if the underlying denrinal support is pathologically destroyed or mechanically removed by a dental instrument (Figs 1-1la and 1-11b). Loss of enamel rods that form the cavity wall or cavomargin of a dental restoration creates a gap defect similar to an occlusal fissure. Leakage or ingress of bacteria and their products may lead to secondary caries. Therefore, a basic tenet of cavity wall preparation is to bevel or parallel the direction of the enamel rods and to avoid undercutting them.

Enamel Rods

However, a common precept, that cavity preparations should always be cut perpendicular to the external coronal surface, is not supported histologically. Each successive row of enamel rods runs a slightly different course in a wave pattern, both horizontally and vertically, through the inner half of the enamel thickness, and then continues in a relatively straight parallel course to the surface. However, on axial surfaces and cuspal slopes, the path of each row terminates at an oblique angle to the surface rather than at a perpendicular tangent of 90 degrees (Fig 1-12). Starting at 1.0 mm from the CEJ, the rods on the vertical surfaces run occlusally or incisally at approximately a 60-degree inclination and progressively incline approaching the marginal ridges and cusp tips, where the rods are essentially parallel to the long axis of the crown.

Enamel Rods

The rods beneath the occlusal fissures are also parallel to the long axis, but rods on each side of the fissure vary up to 20 degrees from the long axis. Therefore, if cut perpendicular to the external surface, occlusal walls of preparations on axial surfaces might incorporate compromised enamel (Fig 1-12). An obtuse enamel-cavosurface angle would more closely parallel the rod direction and preserve the integrity of the enamel margin.

Enamel Rods

Considering the wide variation in direction of enamel rods and the structural damage caused by high-speed eccentric bur rotation, a finishing step of planing the cavosurface margin with hand or low-speed rotary instruments to remove any friable or fragile enamel structure is recommended.

Resilience

Although enamel is vulnerable and incapable of self-repair, its protective and functional adaptation is noteworthy. Carious mineralization, to the point of cavitation, generally takes 3 to 4 years. Demineralization of enamel is impeded because the apatite crystals are 10 times larger than those ill dentin (see Fig 1-7). Enamel apatite crystals offer less surface-to-volume exposure and little space for acid penetration between the crystals. With preventive measures and exogenous or salivary renewal of calcium, phosphate, and especially fluoride, the dynamics of demineralization can be stopped or therapeutically reversed.

Resilience

Enamel thickness and degree of mineralization are greatest at the occlusal and incisal surfaces where masticatory contact occurs. If enamel were uniformly crystalline, it would shatter with occlusal function. A substructure, organized into discrete, parallel rods with a scalloped DEJ, minimizes the transfer of occlusal stress laterally and directs it anisotropically or unidirectionally to the resilient dentinal foundation. The interwoven paths and interlocked keyhole morphology of the enamel rods help control lateral cleavage. As a functional adaptation to occlusal stress, the spiraling weave of rod direction is so pronounced at the cusp tips of posterior teeth that it is referred to as gnarled enamel. Finally, the further subdivision of enamel rods into distinct crystals separated by a thin organic matrix provides additional strain relief to help prevent fracture.

Dentin

-Function

The coronal (crown) dentin provides both color and an elastic foundation for enamel. Together with the radicular (root) dentin, which is covered with cementum, dentin forms the bulk of the tooth and a 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.

Support

Tooth strength and rigidity are provided by an intact dentinal substrate. Several investigators have reported that resistance to tooth fracture is significantly lower with increasing depth and/or width of cavity preparation. A tooth with the deepest possible Class 1 amalgam preparation, that of an endodontic access preparation, retains only a third of the fracture resistance of an intact tooth. To appreciate the magnitude of occlusal loading, mean maximum bite force of 738 N (166 lbs) applied to an average contact area of 4 mm^2 distributed over 20 centric contacts yields more than 26,744 psi. In vitro studies report that large mesio-occluso-distal preparations increase the strain or deflection of facial cusps by three times that of an intact tooth and decrease coronal stiffness by more than 60%.

Support

Elastic deformation and excessive cuspal flexure are etiologic factors contributing to noncarious cervical lesions, cervical debonding of restorations. marginal breakdown, fatigue failure, crack propagation, and fracture. Removal and replacement of dental restorations over a patient's life-time generally result in successively larger/deeper preparations. Therefore, to preserve coronal integrity, 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 an onlay or a crown.

Morphology

Dentin is composed of small apatite crystals embedded in a cross-linked organic matrix of collagen fibrils. The extended cytoplasmic processes of the formative cells, the odontoblasts, form channels or tubules traversing the full thickness of the tissue. Unlike enamel, which is acellular and predominantly mineralized, dentin is, by volume, 45% to 50% inorganic apatite crystals, about 30% organic matrix, and about 25% water. Dentin is pale yellow and slightly harder than bone. Two main types of dentin are present: (I) intertubular dentin, the primary structural component of the hydroxyapatite-embedded collagen matrix between tubules; and (2) peritubular dentin, the hypermineralized tubular wall. The relative and changing proportions of mineralized crystals, organic collagen matrix, and cellular and fluid-filled tubular volume determine the clinical and biologic response of dentin. These component ratios vary according to depth of dentin, age, and traumatic history of the tooth.

Depth

Outer dentin (Figs 1-13 to 1-15). During formation of dentin, the odontoblastic cells converge from the dentinoenamel junction pulpally, creating a tapered channel surrounding their extended cytoplasmic processes. By secreting precursor collagen, these cells produce and nourish the developing dentinal matrix. In the first-formed dentin near the DEJ, the tubules of the outer dentin are relatively far apart and the intertubular dentin makes up 96% of the surface area. Although the tubules are 0.8 μm in diameter and constitute about 4% of the surface area of outer dentin, there are as many as 20,000 tubules/mm^2. In addition, there is extensive terminal branching of the tubules in the outer dentin along with regularly spaced connections, or canaliculi, between tubules, so that the cellular processes make up a highly interconnected system. This interconnected structure may account for the paradox that superficial dentin, though furthest from the pulpal nerve receptors, is sensitive to a stimulus as localized as an explorer tip.

Inner dentin

The dentinal substrate near the pulp is quite different from that near the DEJ; these differences affect the permeability and bonding characteristics of the inner dentin. The formative odontoblast cells converge concentrically to terminate in a single, tightly packed layer at the wall of the pulp chamber. At the pulp-dentin interface, the number of odontoblasts (and therefore tubules) ranges up to 76,000/mm^2. The tubule diameters are larger, 2.5 to 3.0 μm, and the distance between tubule centers within the inner dentin is half that of tubules at the DEJ. Thus, the intertubular matrix area is only 12% of the surface area, and the volume occupied by fluid-filled tubule lumens at the inner or predentin level is 22% of the surface, 20 times that at the DEJ. Even though the tubule space is partially occluded by the cellular process, collagen, and mineral deposits, the dentin close to the pulp is still about eight times more permeable than the dentin near the DEJ.

Permeability

The permeability of dentin is directly related to its protective function (Fig 1-16). When the external "cap" of enamel or cementum is lost from the periphery of the dentinal tubules through caries, preparation with burs, or abrasion and erosion, the exposed tubules become conduits between the pulp and the external oral environment. Restored teeth are also at risk of toxic seepage through the phenomenon of microleakage between the restorative material and the cavity wall. No restorative material can provide a completely hermetic seal of the cavity wall. Gaps of 10 μm or more may exist between newly placed amalgam and cavity walls, and increased leakage at the cemental margins of resin-bonded restorations is commonly reported (Figs 1-17a and 1-17b).

Permeability

Through capillary action, differential thermal expansion, and diffusion, fluids containing various acidic and bacterial products can penetrate the gap between tooth and restoration and initiate demineralization (secondary caries) of the internal cavity walls. From this base, bacterial substances can continue by diffusion through permeable dentinal tubules to reach the pulp. Open tubule conduits to the external oral environment create a micropulpal exposure, putting the tooth at risk for pulpal inflammation and sensitivity. The remaining dentinal thickness is the key determinant of the diffusion gradient. Restorative techniques that incorporate varnishes, liners, or dentin bonding resin adhesives are effective to the extent that they provide reliably sealed margins and a sealed dentinal surface.

Sensitivity

Although sensitive to thermal, tactile, chemical, and osmotic stimuli across its 3.0- to 3.5-mm thickness, dentin is neither vascularized nor innervated, except for about 20% of tubules that have nerve fibers penetrating inner dentin by no more than a few microns. Therefore, attention has been focused on the odontoblast and its process as a possible stimulus receptor. This role is doubtful, however. Neither electron microscopy nor new research technologies have proven that the odontoblastic cellular process extends to the peripheral DEJ in mature dentin. In addition, the cell membrane of odontoblasts is nonconductive, and there is no synaptic connection between the odontoblastic cell and the adjacent terminal branches of the pulpal nerve plexus. Finally, pain sensation remains even when the odontoblastic layer is disrupted.

Sensitivity

Brainnstrom et al proposed a theory based on the capillary flow dynamics of the fluid-filled dentinal tubules (Fig 1-18). Tubular fluid flow of 4 to 6 mm/s is produced by application of a stimulus, such as air evaporation, cold, or heat (ie, generated from a dental bur), or tactile pressure. The "current," or hydrostatic pressure, displaces the odontoblastic cell bodies and stretches the intertwined terminal branches of the nerve plexus to allow entry of sodium to initiate depolarization. Evidence supporting the hydrodynamic theory includes in vivo correlation of tubule patency with hypersensitive roots) Also, Ahlquist et al correlated intensity of pain with rapid hydrostatic pressure changes applied to the sealed, smear-free dentinal axial walls of cavity preparations. Therefore, the knowledge that permeable dentin is sensitive dentin may help the dentist avert postoperative discomfort associated with tooth restoration.

Substrate for Bonding

Early attempts to bond resins to etched dentin were relatively unsuccessful because of the variability of the dentinal substrate, the hydrolytic deterioration of the bonding agents, or interference by the smear layer, a tenacious, semipermeable film of organic and inorganic debris on the prepared dentinal surface. Many of the newer dentin bonding systems remove the smear layer, acid-etch the intertubular dentinal surface and peritubular walls to provide the porosity required for microretention, and penetrate and surround the exposed collagen fibrils. Hydrophilic bonding resin then forms a limited-depth interdiffusion or hybrid zone of resin and collagen/microetched dentin between the restorative resin composite and normal dentin.

Substrate for Bonding

Deeper levels of dentin offer an altogether different substrate for bonding that is wetter and less solid (see Figs 1-13 to 1-15). A positive pulpal pressure of 14 cm H_20 creates an outward flow of dentinal tubular fluid. The odontoblastic process, mineral deposits, and intertubular collagen partially occlude the dentin tubule and provide some resistance to flow. When dentin is cut at deeper levels, flow is unimpeded, and the dentinal surface becomes wet, which can interfere with the adhesion of hydrophobic resin polymers. Not all bonding systems are equally effective in both deep and peripheral dentin.

Substrate for Bonding

Another substrate variable that reduces bond strength is excessive sclerosis or hypermineralization of exposed root surface dentin characterized by a deeper yellow color and glossy surface. Aging and response to external stimuli are associated with tubule.

Physiologic and Tertiary Dentin

Primary dentin is formed relatively quickly until root formation is completed; the odontoblasts then become relatively quiescent. After this, the slowly formed dentin that continues to constrict the dimensions of the pulp chamber is termed secondary dentin. The morphology of the pulp chamber approximates the external tooth contours, but pronounced extensions, or pulp horns, into buccal cusps of premolars and mesiobuccal cusps of molars must be carefully avoided during cavity preparation in young teeth to prevent exposure. Perhaps in response to a mild occlusal stimulus, secondary dentin is preferentially deposited in the pulp horns and on the roof and floor of the pulp chamber so that, after many decades, the chamber becomes quite narrow occlusogingivally. The dentist must allow for the size and location of the pulp chamber, for they may be deciding factors in the design of the preparation and placement of retentive features, such as pins.

Physiologic and Tertiary Dentin

Another physiologic or age-related process, perhaps mediated by the odontoblastic process, is the continual mineralization of tubule walls (see Fig 1-15). As a result, the peritubular dentinal wall progressively thickens and occludes the tubule lumen. Deposition of both peritubular and secondary dentin is considered physiologic, because reduction of the pulp chamber is found in unerupted teeth, and sclerosis is reported in radicular dentin in the premolars of 18 year olds. However, with sufficient external stimulus or irritation, such as caries, attrition, or restorative procedures, the rate of dentin deposition can be accelerated. Unlike primary and secondary dentin, which are deposited uniformly, this added or tertiary dentin is localized to the affected area of the pulp-dentin complex. The tertiary dentin caused by an external stimulus that reactivates the dentinogenic activity of the pre-existing odontoblasts is termed reactionary dentin. If the odontoblasts are killed by a pernicious stimulus, the tertiary dentin generated by osteoblastlike replacement cells is termed response dentin.

Sclerotic Dentin

With age, tubule lumens are gradually constricted by the continuing physiologic mineralization of the peritubular dentin. As with tertiary dentin, external stimuli can accelerate and augment the mineralization of the peritubular dentin or the tubular contents to make the dentin less permeable. Open tubules on exposed root surfaces predispose as many as one in seven adults to hypersensitivity. Treatment modalities occlude the tubules, either by sealing the tubule orifice with bonded resin or by precipitating intratubular protein or crystals with application of fluoride or potassium nitrate compounds to the root surface. When dentin is suddenly exposed by fracture or a lost restoration, plasma proteins are transported to the exposed tubules. Within 6 to 24 hours, a fibrinlike intratubular clot forms to constrain the hydrodynamic flow and diminish sensitivity.

Sclerotic Dentin

With initial enamel caries or microleakage, the outward flow of the dentinal fluid under positive pulpal pressure counteracts pulpal diffusion of endotoxins and acids. As caries progresses, immunoglobulins are transported and concentrated in the tubular fluid and cellular processes within the dentin pulpal to the caries lesion. Withdrawal, injury, or necrosis of the cytoplasmic contents leaves the tubule open to bacterial toxins and deeper invasion. However, calcium and phosphorus released by carious acid demineralization of the peripheral hydroxyapatite crystals reprecipitate deeper within the tubules, pulpal to the infected lesion. Platelike or rhomboid mineral crystals fill and barricade the open lumen (Figs 1-19a and 1-19b). Also, accelerated mineralization, with constriction of the peritubular walls pulpal to the level of bacterial penetration, forms a protective confinement barrier, the sclerotic or translucent zone.

Sclerotic Dentin

With the protective response of stimulus-generated new or tertiary dentin, the in vitro permeability or hydraulic conductance of chronically carious dentin is reduced to only 7.6% of that recorded for normal dentin. Thus, even a minimal width of remaining dentin below a deeply excavated carious lesion and restoration may transform into a protective impermeable barrier. One unfortunate consequence, however, is that sclerosed, insensitive dentin blocks painful warning symptoms that might alert the patient to the presence of caries. Occasionally, with virulent bacteria and in young, permeable teeth, there is insufficient time for sclerosis. The tubules, empty and vulnerable, are described as “dead tracts”. Fortunately, sclerosis is a predictable, protective dentinal response observed in more than 95% of carious teeth and often in conjunction with the production of tertiary or reparative dentin, another protective response that occurs in more than 63% of affected teeth.

Reparative Dentin

Intense traumatic insult 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 (Figs 1-19a and 1-20). Within 3 weeks, fibroblasts or mesenchymal cells of the pulp are converted or differentiated to simulate the organization, matrix secretion, and mineralizing activities of the original odontoblasts. The matrix includes cellular and vascular components of the pulp and sparse, irregularly organized tubules. The rate of formation, the thickness, and the organization of the reparative dentin are commensurate with the intensity and duration of the stimulus. Reparative dentin usually forms at a rate of about 1.5 μm/day, but the rate may be as high as 3.5 μm/day. At 50 days after trauma, a 70-μm thickness of reparative dentin has been reported.

Reparative Dentin

The barrier protection of reparative dentin is superior because there is no continuity between the affected permeable tubules of the regular primary dentin and those within the reparative dentin. Nevertheless, some porosity defects have been reported in reparative dentin so that, with vital pulpal exposures, sterile technique and durable cavity liners/sealers are needed to preserve pulpal vitality. But with reparative dentin, the tooth is able to compensate for the traumatic or carious loss of peripheral dentin with deposition of new dentin substrate and reduction of pulpal vulnerability from tubule permeability.

Reparative Dentin

Unless the lesion is either arrested or removed and a restoration placed before it is about 0.5 mm from the pulp, the diffusion gradient of bacterial metabolites reaching the pulp can initiate a strong inflammatory response. If the reparative dentin is breached to allow sufficient bacteria to overwhelm the vascular, inflammatory, and phagocytic defenses of the pulp, the result is pulpal necrosis.

Pulp

The dental pulp, 75% water and 25% organic, is a viscous connective tissue of collagen fibers and ground substance supporting the vital cellular, vascular, and nerve structures of the tooth. It is a unique connective tissue in that its vascularization is essentially channeled through one opening, the apical foramen at the root apex, and it is completely encased within relatively rigid dentinal walls. Therefore, it is without the advantage of an unlimited collateral blood supply or an expansion space for the swelling that accompanies the typical inflammatory response of tissue to injurious conditions. However, the protected and isolated position of the pulp belies the fact that it is a sensitive and resilient tissue with a great potential for healing.

Pulp

The dental pulp fulfills several functions for the pulpodentin complex: (1) formative, creating the primary and secondary dentin as well as the protective response of reactionary or reparative dentin; (2) nutritive, providing the vascular supply and ground substance transfer medium for metabolic functions and maintenance of cells and organic matrix; (3) sensory, transmitting afferent pain response (nociception) and proprioceptive response; and (4) protective, responding to inflammatory and antigenic stimuli and removing detrimental substances through its blood circulation and lymphatic systems.

Morphology

The pulpal tissue is traditionally described in histologically distinct, concentric zones: the innermost peripheral pulp core, the cell-rich zone, the cell-free zone, and the peripheral odontoblastic layer (Figs 1-21a to 1-21c). The radicular and coronal pulp core is largely ground substance, an amorphous protein matrix gel surrounding cells, discrete collagen fibers, and the channels of vascular and sensory supply. The gel serves as a transfer medium, between widely spaced pulp cells and vasculature, for transport of nutrients and by-products. Terminal neural and vascular components, which divide and multiply extensively in the subodontoblastic zones, converge into larger vessels and trunks and together form a main trunk passing through the pulp core to or from the apical foramina. Both matrix and collagen components are formed and maintained by a dispersed network of interconnected fibroblastic cells.

Morphology

Fibrocytes and undifferentiated mesenchymal cells are particularly concentrated in the outer coronal pulp to form the cell-rich zone subjacent to the peripheral layer of odontoblastic cells. Functioning like troops in reserve, the mesenchymal cells and/or fibrocytes are capable of accelerated mitotic differentiation and collagen matrix production to serve as functional replacements for destroyed odontoblastic cells. They produce reparative dentin when bacteria or their by-products breach the permeable dentinal wall or a pulpal exposure occurs. A dense and extensive capillary bed and nerve plexus form the cell-free zone, infiltrate the cell-rich zone, and separate it from the cellular bodies of the peripheral odontoblastic layer.

Vascular System

The circulatory system supplies the oxygen and nutrients that dissolve in and diffuse through the viscous ground substance to reach the cells. In turn, the circulation removes waste products, such as carbon dioxide, by-products of inflammation, or diffusion products that may have permeated through the dentin before they accumulate to toxic levels: (see Fig 1-16). The equilibrium between diffusion and clearance may be threatened by use of long-acting anesthetics that contain vasoconstrictors such as epinephrine. An intraligamental injection of a canine tooth with 2% lidocaine with 1:100,000 epinephrine will cause pulpal blood flow to cease for 20 minutes or more. Fortunately, the respiratory requirements of mature pulp cells are low so that no permanent cellular damage ensues.

Vascular System

Inflammation, the normal tissue response to injury and the first stage of repair, is somewhat modified by the unique location within the noncompliant walls of the pulp chamber. A stimulus producing cellular damage initiates neural and chemical reactions that increase capillary permeability so that proteins, plasma fluids, and leukocytes spill into the confined extracellular space, producing elevated tissue interstitial fluid pressure. Theoretically, elevated extravascular tissue pressure could collapse the thin venule walls and start a destructive cycle of restricted circulation and expanding ischemia. However, the pulpal circulation is unique because it contains numerous arteriole "U-turns," or reverse flow loops, and arteriole-venule anastomoses, or shunts, to bypass the affected capillary bed. Also, at the periphery of the affected area, where high tissue pressure is attenuated, capillary recapture and lymphatic adsorption of edematous fluids are expedited. These processes confine the area of edema and elevated tissue pressure to the immediate inflamed area. Although tissue pressure at an area of pulpal inflammation is two to three times higher than normal, it quickly falls to nearly normal levels approximately 1.0 mm from the affected area.

Vascular System

Another protective effect of elevated but localized pulpal tissue pressure is a vigorous outward flow of tubular fluid to counteract the pulpal diffusion of noxious solutes through permeable dentin. However, an inflammatory condition and higher tissue pressure may also induce hyperalgesia, a lowered threshold of sensitivity of pulpal nerves. Thus, an afflicted tooth exposed to the added stress of cavity preparation and restoration may become symptomatic or hypersensitive to cold or other stimuli.

Innervation

Dental nerves are either efferent autonomic C fibers to regulate blood flow or afferent sensory nerves derived from the second and third divisions of the fifth intracranial (trigeminal) nerve. Nerves are classified according to purpose, myelin sheathing, diameter, and conduction velocity. Although a few large and very high-conduction velocity A-β (beta) nerves with a proprioceptive function have been identified, most sensory interdental nerves are either myelinated A-δ(delta) nerves or smaller, unmyelinated C fibers. The innervation of a premolar, for example, consists of about 500 individual A-δ(delta) nerves that gradually lose their myelin coating and Schwann cell sheathing as they branch and form a sensory plexus of free nerve endings around and below the odontoblastic layer. The A-δ nerves have conduction velocities of 13.0 m/s and low sensitization thresholds to react to hydrodynamic pressure phenomena. Activation of the A-δ system results in a sharp, intense "jolt."

Innervation

There are three to four times more of the smaller, unmyelinated C fibers, which are more uniformly distributed through the pulp. The conduction velocities of C fibers are slower, 0.5 to 1.0 m/s, and C fibers are only activated by a level of stimuli capable of creating tissue destruction, such as prolonged high temperatures or pulpitis. The C fibers are also resistant to tissue hypoxia and are not affected by reduction of blood flow or high tissue pressure. Therefore, pain may persist in anesthetized, infected, or even nonvital teeth. The sensation resulting from activation of the C fibers is a diffuse burning or throbbing pain, and the patient may have difficulty locating the affected tooth. The afferent transmission of painful sensations, commonly experienced although unreliable as a warning signal, may not be the primary protective function of pulpodentin innervation. Experimentally denervated teeth exposed to trauma suffer greater pulpal damage than innervated controls. The initiation and coordination of the inflammatory cascade; the vascular, tissue, and tubular fluid dynamics; and the immunocompetent response are important protective functions of the neural components.

Odontoblastic Layer

The peripheral cellular layer of the pulp, the odontoblasts, produce primary, secondary, and reactionary dentin. This layer may also regulate or influence tubular mineralization and sclerosis as a defense mechanism (see Fig 1-15). Postmitotic and irreplaceable, the columnar cell bodies line the predentin wall of the pulp chamber in a single layer. From each cell, a single process extends into at least one third of the tubule and adjacent dental substrate that it formed. Each cell has an indefinite life span, but crowding from continued deposition of secondary dentin constricts the pulpal chamber to reduce the initial number of cells by half. The odontoblastic cells are packed closely together, with both permanent and temporary junctions between the cellular membranes. Just as the peripheral processes of the odontoblasts are physically interconnected, a third type of intercellular interface, a communicating junction, mediates transfer of chemical and electronic signals that permit coordinated response and reaction of the odontoblastic layer,

Odontoblastic Layer

Thus, as an additional protective response, the integrity and spacing of the odontoblastic layer mediates the passage of tissue fluids and molecules between the pulp and the dentin. Routine operative procedures, such as cavity preparation and air drying of the cut dentinal surface, can temporarily disrupt the odontoblastic layer and may sometimes inflict permanent cellular damage.

Restorative Dentistry and Palpal Health

Surgical and restorative treatments generate considerable physical, chemical, and thermal irritation of the pulp. However, if the dentist uses an acceptable technique and achieves bacterial control, even a mechanical pulp exposure or use of acidic restorative materials poses few problems for pulpal health. Although microleakage round restorations is ubiquitous, the fact that almost all pulps remain healthy is related to diminished virulence of the bacteria, relative impermeability of the dentin, and healing potential of the pulp. But the capacity for pulpal healing is restricted by the effects of aging and by extensive and/or repeated restorative procedures. Two clinical reviews of patients who had received either a fixed prosthesis or single complete crowns reported that 6% and 13% of crowned teeth revealed some sign of pulpal necrosis requiring endodontic treatment.

Restorative Dentistry and Palpal Health

Although pressure, desiccation, and surgical amputation of cellular processes accompany dental intervention, excessive heat generated by the friction of rotary instrumentation is considered the most damaging insult to the pulp. Heat may cause coagulation, extensive burn lesions, and temporary stasis of the pulpal circulation. Studies of heat applied externally to enamel surfaces or cavity floors have produced equivocal results. Zach and Cohen reported 15% irreversible pulpal necrosis after a 5.5°C increase of intrapulpal temperature and up to 60% necrosis after an 11°C rise. However, against prepared cavity walls of orthodontically condemned teeth with about 0.5 mm of remaining dentinal thickness, a 30-second heat application of 150°C (200°F) produced few symptoms, relatively minor pulpal changes, and no necrosis, Interpretation of the studies indicates that the presence of a sufficient remaining dentinal thickness (0.5 mm or more) is a critical factor in limiting thermal conduction (providing insulation), just as it is in limiting dentinal permeability.

Restorative Dentistry and Palpal Health

Other in vitro studies have shown damaging temperature thresholds are possible if improper amalgam finishing and polishing techniques, such as continuous contact and excessive speed or pressure, are used. Another study reported that 25 seconds of continuous contact of a rotating dental bur against a tooth without water coolant could produce a critical 6°C rise in intrapulpal temperature. Therefore, an important safeguard to prevent the buildup of pulpal heat is use of a water coolant combined with intermittent instrument-tooth contact, especially during high-speed enamel or dentin reduction. Another potential thermal threat to the pulp is from high-energy light-curing devices for polymerization of resin composites. These devices are capable of raising the pulpal chamber temperature as much as 8°C. Other steps recommended to minimize pulpal damage associated with restorative procedures include the following: use of sharp burs or single-use diamonds, use of concentrically rotating instruments, avoidance of overdrying and prolonged desiccation of cut dentin, and accurate fitting of provisional restorations.

Restorative Dentistry and Palpal Health

Although the aged tooth is less permeable, some biologists suggest that it has less reparative potential. Age-related changes include reduced blood supply, a smaller pulp chamber, lower ratio of cells to collagen fiber, loss and degeneration of myelinated and unmyelinated nerves, loss of water from the ground substance, and increased intrapulpal mineralizations (denticles). Restorative procedures easily tolerated in the younger patient may pose problems for the older patient. Nevertheless, the newer concepts of treatment, including preventive measures; improved restorative materials; reliable bonding and sealing of enamel fissures, margins, and dentinal tubules; and conservative tooth preparations should extend durability and biocompatibility of dental services.

Gingiva

The gingiva is that part of the oral mucosa that covers the alveolar bone, defines the cervical contours of the clinical crown, and seals the tooth root and periodontal structures from the external environment. A normal, healthy gingiva presents a scalloped marginal outline, firm texture, coral pink or normally pig-mented coloration, and, in about 40% of the population, a stippled surface. A healthy, stable gingiva without hyperplastic, swollen, bleeding, or receding tissue is essential to both esthetic and restorative success.

Gingiva

Gingivitis, an inflammatory soft tissue response to bacterial plaque, affects up to 44% of the adult population in the United States. Gingival bacteria, associated with poor oral hygiene or defective restorations, can cause periodontal disease. Because periodontitis is a major cause of adult tooth loss, the status of the alveolar bone and soft tissues of the periodontium must be evaluated along with that of the teeth.

External Appearance

The two primary components of the gingival tissues are keratinized gingiva and alveolar mucosa (Figs 1-22a and 1-22b). The keratinized gingiva includes both the attached gingiva and the marginal gingiva. Attached gingiva is firmly affixed to the periosteum of the alveolar bone and hard palate and to the supraalveolar cementum of the root. It extends coronally around the teeth to form the free gingiva, a scalloped, unattached cuff that also fills the gingival embrasure between adjacent teeth with a facial and lingual papilla. Attached and marginal gingiva are separated by an external free gingival groove in about 30% to 40% of the healthy adult population. The vertical width (height) of the keratinized gingiva (attached and marginal gingiva) is clearly measured from the mucogingival junction separating it from the alveolar mucosa, which is mobile, darker red, and nonkeratinized. The usual width of keratinized gingiva varies by location, from less than 2.0 mm on the lingual aspect of the mandibular incisors to 9.0 mm on the lingual aspect of mandibular molars.

External Appearance

Histologically, the keratinized gingiva is composed of an underlying connective tissue, the lamina propria with an irregular boundary of projecting ridges supporting the oral epithelium. With attached gingiva, these layers are affixed directly to the periosteum. Oral epithelium in flexible areas, such as the cheek, overlays a vascular submucosal layer (Figs 1-23a and 1-23b). The epithelial layer is characterized by progressive strata in which the active mitotic cells of the basement membrane completely differentiate into scales of synthesized keratin protein as they migrate to the surface for desquamation, The significance of keratinized tissue in restorative dentistry is somewhat controversial. Lang and Loe concluded that a minimum width of 2.0 mm of keratinized gingiva is required to prevent chronic gingival inflammation. However, several laboratory and clinical studies report that, with good oral hygiene, a healthy and stable gingival margin is possible even when the attached gingiva is minimal, missing, or remodeled.

Dentogingival Junction

The complex of epithelial cell types and connective tissue forming the gingival attachment to the tooth and alveolar bone is called the dentogingival junction (Figs 1-23a to 1-23c). Coronally, the keratinized marginal gingiva invaginates against the cervical enamel to form a partial or nonkeratinized epithelium-lined gingival sulcus with an average depth of 1.0 to 2.0 mm. A depth of more than 3.0 mm is generally considered pathologic and termed a periodontal pocket.

Dentogingival Junction

From the base of the sulcus, which corresponds to the level of the CEJ in the young adult tooth, a layer of junctional epithelial cells forms an adhesive basement membrane seal against the cementum of the root. The thickness of the junctional epithelium narrows from 15 to 30 cells at the base of the sulcus to one to three cells apically. Over time, cumulative bacterial and mechanical irritation often result in a lower gingival level (longer clinical crown) and a corresponding increase in the width of the junctional epithelium. Like other oral epithelial cells, the junctional epithelial cells exhibit high mitotic activity, and the cells migrate coronally to the base of the sulcus to be desquamated. An extensive vascular plexus underlies the junctional epithelial cells, which are widely spaced to facilitate the passage of vascular and inflammatory cells into the gingival fluid of the sulcus. Bacterial colonization within the sulcus is discouraged by the combination of a rapidly disrupted cellular base and the lavage and antibacterial action of the vascular transudate of gingival fluid.

Dentogingival Junction

The supra-alveolar connective tissue and lamina propria of the gingiva are made up of dense interlaced bundles of collagen fibers supporting the gingiva and affixing it to the periosteum and cementum of the hard tissues (Fig 1-24). The fibers are classified by attachment and function into the following groups: (1) dentogingival, attaching the gingiva to the cementum; (2) alveologingival, affixing gingiva to alveolar bone; (3) transseptal, connecting interproximal cemental surfaces; (4) dentoperiosteal, from alveolar crest to cementum, an extension of the periodontal ligament; and (5) circular, around the tooth.

Restorative Dentistry and Gingival Health

Although dental restorations with supragingival margins and physiologic contours would best sustain gingival health, apical extension of coronal caries lesions, root caries, tooth fracture, and esthetic considerations often dictate a subgingival placement of restorations. The relationship of plaque retention and local irritants to gingival inflammation is well documented. Iatrogenic factors and restoration defects such as gingival overhangs, excessive axial contours, marginal defects, and surface roughness of the restorative materials may exacerbate or even cause localized gingival inflammation. Elimination or control of restorative defects is essential. Nevertheless, a clinically acceptable periodontium is often maintained in the presence of less-than-perfect restorative dentistry, thereby implicating bacterial virulence and patient susceptibility as key etiologic cofactors, Margins placed near the base of the sulcus are increasingly associated with problems of inflammation, bleeding, hyperplasia, gingival recession, and pathologic bacteria. Assuming dental restorations and technique are of good quality, the most critical factors in preserving the health of the restorative-periodontal interface are the appropriate placement of the restorative margin, within the sulcus or supragingivally, and the preservation of the dentogingival junction, known clinically as the biologic width.

Biologic Width

A histologic autopsy study of individuals of various ages and gingival levels revealed variations in depths of the sulcus and junctional epithelium, but the supra-alveolar connective tissue attachment consistently measured approximately 1.07 mm (see Fig 1-23a).In health, the connective tissue and juntional epithelium occupy the space between the base of the sulcus and the alveolar crest and measure approximately 2.0 mm; this is termed the biologic width. This dimension is assumed to be a physiologic minimum required to preserve the attachment and health of the supporting periodontium. Caries, tooth fracture, or operator error may create conditions in which the 2.0-mm biologic width is surgically traumatized or excised. Chronic and unsightly gingival inflammation and bone loss may occur as the body attempts to restore the biologic width to a more apical level.

Biologic Width

In a review of clinical application, Kois reported variability in the dentogingival complex and suggested that the location of the base of the sulcus is problematic because the junctional epithelium is readily penetrated with a probe. Therefore, under anesthesia, a periodontal probe should be used to measure, or "sound," the true dentogingival complex (biologic width + sulcus depth). The majority of midfacial depths from the free gingival margin to the alveolar bone level have a dimension of approximately 3.0 mm, but deviations are confirmed using a periodontal probe for sounding. With a 3.0-mm reading, predictable periodontal healing and health are assured, with the gingival margin of the restoration 0.5 to 1.0 mm apical to the free gingival margin. This preserves a 2.0-to 2.5-mm biologic width. If the biologic width cannot be maintained, prerestorative osseous surgery (crown lengthening) should be performed to reduce bone level and regain the 2.0 to 2.5 mm needed for the biologic width.