As the principal source of pain within the mouth and
as the major site of attention in endodontic treatment,
the pulp warrants direct inspection. By its very location
deep within the tooth, it defies visualization, other than
its appearance as radiolucent lines on radiographs.
Occasionally, when required to deal with an accidental-
ly fractured cusp, the dentist is afforded a glimpse of
the normal pulp. A pink, coherent soft tissue is noted,
obviously dependent on its normal hard dentin shell
for protection and hence, once exposed, extremely sen-
sitive to contact and to temperature changes.

When pulp tissue is removed en masse from a tooth
in the course of, say, vital pulpectomy, the dentist gains
a new perspective of the pulp. Here is connective tissue
obviously rich in fluid and highly vascular. After expo-
sure to air, the appearance and volume of the tissue
change as the fluid evaporates. Another dimension of
the physical characteristics of pulp tissue can be demon-
strated by grasping a freshly extirpated vital pulp
between thumb and forefinger in both hands and
attempting to pull the pulp apart. Surprisingly, this tiny
strand has much the feel of dental floss: it is tough,
fibrous, and inelastic. This is a reflection of an impor-
tant structural component of the pulp, namely collagen.

FUNCTION
The pulp lives for the dentin and the dentin lives by the
grace of the pulp. Few marriages in nature are marked
by a greater interrelationship. Thus it is with the pulp
and the four functions that it serves: namely, the for-
mation and the nutrition of dentin and the innervation
and defense of the tooth.1

Formation of the dentin is the primary task of the
pulp in both sequence and importance. From the meso-
dermal aggregation known as the dental papilla arises the
specialized cell layer of odontoblasts adjacent and inter-
nal to the inner layer of the ectodermal enamel organ.
Ectoderm interacts with mesoderm, and the odonto-
blasts initiate the process of dentin formation.2 Once
under way, dentin production continues rapidly until the
main form of the tooth crown and root is created. Then
the process slows, eventually to a complete halt.

Nutrition of the dentin is a function of the odonto-
blast cells and the underlying blood vessels. Nutrients
exchange across the capillaries into the pulp interstitial
fluid, which, in turn, travels into the dentin through the
network of tubules created by the odontoblasts to con-
tain their processes.

Innervation of the pulp and dentin is linked by the
fluid and by its movement between the dentinal tubules
and peripheral receptors, and thus to the sensory
nerves of the pulp proper.3

Defense of the tooth and the pulp itself has been
speculated to occur by the creation of new dentin in the
face of irritants. The pulp may provide this defense by
intent or by accident; the fact is that formation of lay-
ers of dentin may indeed decrease ingress of irritants or
may prevent or delay carious penetration. The pulp
galvanizes odontoblasts into action or produces new
odontoblasts to form needed hard tissue.

The defense of the pulp has several characteristics.
First, dentin formation is localized. Dentin is produced at
a rate faster than that seen at sites of nonstimulated pri-
mary or secondary dentin formation. Microscopically,
this dentin is often different from secondary dentin and
has earned several designations: irritation dentin, repara-
tive dentin, irregular secondary dentin, osteodentin, and
tertiary dentin.

The type and amount of dentin created during the
defensive response appear to depend on numerous fac-
tors. How damaging is the assault? Is it chemical, ther-
mal, or bacterial? How long has the irritant been applied? How deep was the lesion? How much surface area was involved? What is the status of the pulp at the time of response? A second defensive reaction, inflammation within the pulp at the site of injury, should not be ignored. This phenomenon will be explored in more detail in chapter 4.

**INDUCTION AND DEVELOPMENT OF DENTIN AND CEMENTUM**

Human tooth development spans an extremely long period of time, starting with the induction of the primary dentition during the second month of embryogenesis until completion of the permanent dentition toward the end of adolescence. The primary dentition is induced during the fifth week of gestation, and biomineralization begins during the fourteenth week of gestation. In tandem, the first permanent teeth have reached the bud stage, and they begin biomineralization just prior to birth. The first primary teeth begin to erupt in children at 6 months of age, and the first permanent teeth erupt at 5 to 6 years of age. The third molars are the last teeth to be formed, and their crown development is completed between 12 and 16 years of age. Therefore, the induction and development of the human dentition persists during embryonic, fetal, neonatal, and postnatal childhood stages of development. Detailed descriptions of the histology and timing of human tooth development can be readily found in a number of excellent textbooks.4

Inductive tissue interactions, specifically epithelium-mesenchyme interactions, have been extensively investigated and characterized throughout the various stages of tooth crown and root morphogenesis.5–8 The developing tooth system has become a well-characterized model for defining the molecular mechanisms required for reciprocal signaling during epithelium-mesenchyme interactions in crown and root morphogenesis and cytodifferentiation.9–13

It is now evident that the initial inductive signals for tooth formation are synthesized and secreted from specific sites defined as odontogenic placodes within the oral ectoderm that cover the maxillary and mandibular processes.13 The oral ectodermally derived inductive signals are received by multiple cognate receptors located on the cell surfaces of a specific subpopulation of cranial neural crest–derived ectomesenchymal cells during the initial induction process and the subsequent dental lamina, bud, and stages of tooth development8–13 (Figure 2-1).

During the transition from bud to cap stages, the ectomesenchyme provides several cell lineages, including one that becomes dental papilla mesenchyme and others that become progenitor cells for the subsequent development of the periodontium.8,14,15 At this time, multiple and reciprocal signals are secreted by the dental papilla mesenchyme, and these signals bind to the extracellular matrix and transmembrane cell receptors along the adjacent enamel organ epithelium. A discrete structure within the enamel organ, termed the “enamel knot,” synthesizes and secretes a number of additional signals that also participate in the determination for the patterns of morphogenesis within the maxillary and mandibular dentitions: incisiform, caniniform, and molariform.12,13

Figure 2-1 Early dental pulp, or dental papilla, exhibiting a cellular mass at the center of this tooth bud in the early bell stage. Nerve fiber bundles are evident in cross-section as dark bodies apical to dental papilla yet are absent in the papilla itself. Human fetus, 19 weeks. Palmgren nerve stain. Reproduced with permission from Arwil T, Häggströms I. Innervation of the teeth. Transactions Royal School of Dentistry (Stockholm), 3:1958.
The human genome was basically completed by the year 2000; essentially all of the 100,000 regulatory and structural genes within the human lexicon have been identified, sequenced, and mapped to specific chromosomal locations. Further, combinations of genes encoded within the human genomic deoxyribonucleic acid (DNA) are now known to be expressed during development that controls morphogenesis. A number of these genes have been identified and characterized as being expressed in the odontogenic placode, dental lamina, and the bud, cap, bell, and crown stages of tooth development in either the epithelial or mesenchymal cells or both. Significantly, these genes are expressed in various combinations during induction processes associated with many developing epidermal organ systems including the salivary gland, sebaceous glands, mammary glands, tooth, hair, and skin morphogenesis.

The specificity of induction reflects the particular combinations of signaling molecules, their cognate cell surface receptors, various intracellular signal pathways, and a large number of transcriptional factors that regulate gene expression. These combinations further are modified according to temporal and spatial information during development; the combination used to induce the dental lamina is different than that required for cap stage development and subsequent odontoblast cytodifferentiation. The hierarchy of these molecular mechanisms associated with the initiation and subsequent early stages of tooth development is shown in Figure 2-2.

Specific mutations or alternations in one or more of these molecules result in clinical phenotypes including cleft lip and/or palate and a range of dental abnormalities with enamel, dentin, cementum, periodontal ligament, or alveolar bone disorders; for example, hypodontia or missing teeth as seen in X-linked anhidrotic ectodermal dysplasia, which is caused by a mutation in the gene EDA; familial tooth agenesis, which is caused by a mutation in the gene MSX1; and X-linked amelogenesis imperfecta, which is caused by a mutation in the gene amelogenin. Recently, a mutation in the gene CBF-alpha, a transcription factor, was found to cause cleidocranial dysplasia with hyperdontia.

The development of dentin is intriguing for several reasons. First, signals within the inner enamel epithelia of the enamel organ induce adjacent cranial neural crest–derived ectomesenchymal cells to become progenitor odontoblasts. Mutations in either the signaling molecules, cognate receptors, intracellular signal pathways, transcription factors, or extracellular matrix molecules can result in severe dental anomalies including

![Figure 2-2](Image)

**Figure 2-2** Molecular controls for the initiation and early stages of tooth morphogenesis. The initiation or site selection for tooth development is controlled by oral ectoderm-derived epithelial fibroblast growth factor (FGF) and bone morphogenetic protein (BMP) through their signaling pathways. The actions of these signaling pathways or circuits are dependent on the stage and position of development and the combination of transcription factors that are either induced (→) or repressed (↓). This establishes the odontogenic placode. Thereafter, epithelium regulates the adjacent mesenchyme during the bud stage and the mesenchyme then promotes differentiation and influences the adjacent inner enamel epithelium through feedback loops in the cap, bell, and crown stages of tooth development. The reader is encouraged to see the following references for detailed analyses of these morphoregulatory molecules.
tooth agenesis, hypodontia, and oligodontia and defects in dentin deposition or biomineralization, termed dentinogenesis imperfecta. Several dentin extracellular matrix proteins—dentin sialoprotein, dentin matrix protein, and dentin phosphoprotein—are produced by alternative splicing from one single gene product. Mutations in any one of these encoded sequences, or in type I collagen, result in dentinogenesis imperfecta. Mutations that are limited to type I collagen produce osteogenesis imperfecta with a form of dentinogenesis imperfecta.

Following crown morphogenesis, cells from the cervical loop give rise to Hertwig’s epithelial root sheath (HERS) cells, which, in turn, induce adjacent dental papilla mesenchymal cells to engage in root formation.6–8 Progressive cell proliferation and migrations eventually outline the shape and size of the forming roots. In this developmental process, two interpretations are currently being considered. First, evidence is available to support the hypothesis that HERS cells transdifferentiate into cementoblasts and secrete acellular cementum matrix.8 Second, evidence is also available to support the hypothesis that peripheral ectomesenchymal cells penetrate through the HERS and become cementoblasts and secrete acellular cementum.6,7 Of course, both processes may take place at different times and positions of cementogenesis.

The understanding of the induction and development of cementum has also progressed in recent years. Hertwig’s epithelial root sheath cells provide signals that induce the differentiation of odontoblasts and the formation of the first peripheral layer of dentin. It has been known for almost 100 years that a small percentage of the human population expresses enamel pearls along the root surfaces of permanent teeth. These enamel-like aberrations in cementogenesis are intriguing and could offer new insights and strategies to regenerate acellular cementum.

Recent advances have indicated that molecules presumed to be uniquely restricted to inner enamel epithelium and ameloblasts associated with enamel formation are also expressed in HERS.6–8 Specifically, ameloblastin has recently been identified in both ameloblasts and HERS.20 This association between enamel and cementum has been previously discussed with respect to coronal cementum.21 These and other studies have suggested that enamel matrix contains molecular constituents, presumably with growth factor bioactivities, that can induce acellular regeneration when active on denuded human dentin root surfaces. Recently, an enamel matrix preparation has been demonstrated to induce acellular cementum regeneration on root surfaces associated with advanced periodontitis.22 The available evidence strongly supports the hypothesis that enamel organ epithelium-derived cell phenotypes control coronal enamel deposition and the initiation of acellular cementum formation.23,24

ANATOMY

The living pulp, as we have seen, creates and shapes its own locale in the center of the tooth. The pulp, under normal conditions, tends to form dentin evenly, faciolingually and mesiodistally.25 The pulp therefore tends to lie in the center of the tooth and shapes itself to a miniaturization of the tooth. This residence of the pulp is called the pulp cavity, and one speaks of its two main parts as the pulp chamber and the root canal. The clinical implications of pulp form and variation are extensively covered in chapter 10. Indeed, the key word in understanding the gross anatomy of the pulp is “variation.” Equally evident in any study of the pulp is the reduction in size of the chamber and canals with age. Such reduction in size thus becomes a new variation.

In addition to changes in pulp size and shape with aging, external stimuli also exert an effect. Caries, attrition, abrasion, erosion, impact trauma, and clinical procedures are some of the major irritants that may cause formation of irritation dentin. The clinician must appreciate the resultant alterations in internal anatomy that accompany disease and damage of the pulp and dentin.

Pulp Chamber

At the time of eruption, the pulp chamber of a tooth reflects the external form of the enamel.1 Anatomy is less sharply defined, but the cusp form is present. Often the pulp suggests its original perimeter (and threatens its future) by leaving a filament of itself, the pulp horn, within the coronal dentin. A specific stimulus such as caries leads to the formation of irritation dentin on the roof or wall of the chamber adjacent to the stimulus. Of course, with time, the chamber undergoes steady reduction in size as secondary, and irritation dentin is produced on all surfaces (Figure 2-3).

Root Canal

An unbroken train of connective tissue passes from the periodontal ligament through the apical root canal(s) to the pulp chamber. Each root is served by at least one such pulp corridor. Actually, the root canal is subject to the same pulp-induced changes as the chamber. Its diameter becomes narrowed, rapidly at first as the foramen takes shape in the posteruptive months but with increasing slowness once the apex is defined. The canal diameter
tends to decrease slightly with age; irritants such as periodontal disease may cause further constriction.

According to Orban, the shape of the canal, "to a large degree, conforms to the shape of the root. A few canals are round and tapering, but many are elliptical, broad and thin." A curve at the end of the root means almost invariably that the canal follows this curve. Meyer stated that "roots that are round and cone-shaped usually contain only one canal, but roots that are elliptical and have flat or concave surfaces more frequently have more canals than one." (Figure 2-4).

The foramen can change in shape and location because of functional influences on the tooth (eg, tongue pressure, occlusal pressure, mesial drift). The pattern that develops is the reverse of the changes in the alveolar bone around the tooth. Cementum resorption occurs on the wall of the foramen farthest from the force and apposition on the wall nearest. The net result is a deviation of the foramen away from the true apex (Figure 2-5).

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Figure 2-3  Schematic diagram of a mandibular molar showing hard tissue apposition with time and/or irritation. Black arrows indicate physiologic secondary cementum and dentin apposition; white arrows indicate formation of dentin in response to irritants. Pulp space undergoes continual reduction in size and volume. Note that the floor of the chamber is a region of maximum secondary dentin formation.

Figure 2-4  Canal size and shape are a reflection of the external root surface. The upper diagrams show the possible canal configurations within each shaped root. The maxillary molar root sections illustrate that all sizes and shapes of canals may be seen in a single tooth.

Figure 2-5  Features often noted in normal yet aging tooth: deviation of apical foramen as a result of mesial migration of the tooth (top arrow). Selective resorption and apposition of cementum have changed the position of the apex, causing narrowing of the apical third of the root canal by increments of secondary dentin and flaring of apical foramen from its smallest diameter at the dentino-cemental junction (bottom arrow) to its greatest diameter at the cemental surface. Note the abundance of collagen fibers in the radicular pulp. Reproduced with permission from Matsumiya S. Atlas of oral pathology. Tokyo Dental College Press; 1955.
Foramina

The anatomy of the root apex is partially determined by the number and location of apical blood vessels present at the time of formation of the apex. When the tooth is young and just erupting, the foramen is open. Islands of dentin may appear within the mainstream of connective tissue when the root sheath has induced them, but these islands are widely separated. Progressively, the main channel narrows. The primary vessels and nerves, though never directly threatened with strangulation, have a restricted passage. Increments of cementum apposition contribute to this continuous modeling. The possibilities of vascular branching are so varied at the apex that prediction of the number of foramina in a given tooth becomes impossible (Figure 2-6).

It is known that the incidence of multiple foramina is high. The majority of single-rooted teeth have a single canal that terminates in a single foramen. Less often, they possess an apical delta, which terminates in a major channel and one or more collateral exits. Occasionally, the delta has several channels of equal magnitude. Root canals of the multirooted teeth, on the other hand, tend to have a more complex apical anatomy. Multiple foramina are the rule rather than the exception. When accessory foramina are found in one root of a multirooted tooth, the other roots usually have a similar condition. Moreover, because the individual roots of such teeth often contain two or even three canals, a new factor is introduced. These canals can merge, but they need not and often do not merge before making their exit. Indeed, each may leave the root independently. Branching of the emergent canals within the apical area is a common finding because the preexisting vessels are linked.

It is also important to recall that cementum forms in abundance at the root apex. Because of the apposition of new layers of cementum, in response to eruption, the foramen anatomy is by no means constant. As stated above, the center of the foramen tends to deviate increasingly from the apical center. Also, many root canals have two apical diameters. The minor diameter at the level of the dentinocemental junction can be as small as one half that of the major diameter at the external surface of the root. Cementum deposition tends to produce an apical funnel of increasing divergence. Contributing to this is secondary dentin formation that narrows the dentinal orifices of the canal (see Figure 2-5).

Stereomicroscopic analysis of some 700 posterior root apices showed that at least half of the major foramina take eccentric positions that deviate as far as 2 mm from the apex. Accessory foramina, on average, were found to be located twice the distance of the major foramina from the vertex (Figure 2-7).

Accessory Canals

Communication of pulp and periodontal ligament is not limited to the apical region. Accessory canals are found at every level. Vascular perfusion studies have demonstrated vividly how numerous and persistent these tributaries are. Many, in time, become sealed off by cementum and/or dentin; however, many remain viable. The majority appear to be encountered in the apical half of the root. These generally pass directly from the root canal to the periodontal ligament (Figure 2-8).

A common area in which accessory canals appear is the furcation area of molar teeth (Figure 2-9). Burch and Hulen and Vertucci and Anthony found that molars frequently presented openings in the furcation areas. However, the studies did not determine how many of these represented patent (continuous) accessory canals all the way from the pulp to the periodontal ligment. Morphologic and scanning electron microscopic studies consistently show the presence of patent accessory canals or depressions that were assumed to be the openings to such canals. In other studies, dyes were injected or drawn by vacuum into the furcation of molars. Approximately one half of

Figure 2-6 Diverse ramifications of apical pulp space anatomy. These models were made by drawing pulps from serial histologic sections and "stacking" the drawings. Many regions are obviously inaccessible to conventional débridement methods. Adapted with permission from Meyer W. Dtsch Zahnaerztl Z 1970;25:1064.
Histology and Physiology of the Dental Pulp

the teeth studied demonstrate patent accessory canals from pulp space to furcation.38

HISTOLOGY

Regions

Classically, the pulp is described as having two defined regions, central and peripheral.39 Typically, however, studying sections of pulp under the microscope reveals that the classic description is not consistent; levels of activity dictate regional morphology. However, it is helpful to know the textbook description before studying the variations.

The pulp is in intimate contact with the dentin and survives only through the protection of its hard outer covering. As the price of this protection, the pulp contributes to a close symbiosis. The way in which the normal pulp relates to its immediate environment can be best explained by a review of its own morphology and that of the tissues with which it is confluent, namely, the dentin and the periodontal ligament. In general, the pulp demonstrates a homogeneity in its blend of cells, intercellular substance, fiber elements, vessels, and nerves.

**Peripheral Pulp Zone.** On the periphery of the pulp, adjacent to the calcified dentin, structural layers are apparent. These are usually evident in a medium-power photomicrograph (Figure 2-10). Next to the predentin lies the palisade of columnar odontoblast cells. Central to the odontoblasts is the subodontoblastic layer, termed the cell-free zone of Weil.40

![Figure 2-7](image1.png) Accessory foramen (arrow) in mandibular anterior tooth. Accessory foramina are usually located within the apical 2 to 3 mm of root. (Orban collection.) Reproduced with permission from Sicher H. Oral histology and embryology. 5th ed. St. Louis: CV Mosby; 1962.

![Figure 2-8](image2.png) A, Necrotic pulp in maxillary second premolar in which irritants egress through lateral canals to the periodontium, creating inflammatory lesions (arrows). Although often invisible on pretreatment radiographs, the presence of lateral canals may be confirmed following obturation. B, Bony lesions healed several months following root canal treatment. Accessory canals are important, not so much for the irritants they contain but because of their communication to the PDL. (Courtesy of Dr. Manuel I. Weisman.)
Plexuses of capillaries and small nerve fibers ramify in this subodontoblastic layer. Deep to the odontoblastic layer is the cell-rich zone, which blends in turn with the dominant stroma of the pulp. The cell-rich zone contains fibroblasts and undifferentiated cells, which sustain the population of odontoblasts by proliferation and differentiation. These zones vary in their prominence from tooth to tooth and from area to area in the pulp of the same tooth. The cell-free and cell-rich zones are usually indistinct or absent in the embryonic pulp and usually appear when dentin formation is active. The zones tend to become increasingly prominent as the pulp ages. Both of these zones are less constant and less prominent near the root apex.

Central Pulp Zone. The main body of the pulp occupies the area circumscribed by cell-rich zones. It contains the principal support system for the peripheral pulp, which includes the large vessels and nerves (Figure 2-11) from which branches extend to supply the critical outer pulp layers. The principal cells are fibroblasts; the principal extracellular components are ground substance and collagen. The environment of the pulp is unique in that it is surrounded by an unyielding tissue and fed and drained by vessels that pass in and out at a distant site. However, it is classified as an areolar, fibrous connective tissue, containing cellular and extracellular elements that are found in other similar tissues. These elements will be discussed in more detail.

Structural Elements, Cellular

Reserve Cells. The pulp contains a pool of reserve cells, descendants of undifferentiated cells in the primitive dental papilla. These multipotential cells are likely a fibroblast type that retains the capability of dedifferentiating and then redifferentiating on demand into many of the mature cell types. Beneath the odontoblasts in the cell-rich zone, are concentrations of such cells. However, Frank demonstrated by radioautography that these cells produce little collagen, which is circumstantial evidence that they are not mature fibroblasts. Bailey has reviewed ultrastructural studies that suggest cytoplasmic connections between the odontoblasts and these subjacent mesenchymal cells. Through such connections, on odontoblast injury or death, signals may be provided to these less differentiated cells that may cause them to divide and differentiate into odontoblasts or odontoblast-like cells, as required.

Also important are the reserve cells scattered throughout the pulp, usually in juxtaposition to blood vessels. These retain the capacity, on stimulation, to divide and differentiate into other mature cell types. For example, mast cells and odontoclasts (tooth resorbers) arise in the presence of inflammation.

Significant are the unique cells that differentiate to form the calcified tissue that develops under a pulp cap or pulpotomy when calcium hydroxide is placed in direct contact with the pulp. These unique cells are also frequently observed along the calcified tissue forming at the base of tubules involved with caries, restorations, attrition, or abrasion. This calcified tissue is not a true
Figure 2-10  A. Medium-power photomicrograph from human pulp specimen showing dentin (D), predentin (P), odontoblast layer (O), cell-free zone (CF), cell-rich zone (CR), and central pulp (CP). B. Region similar to area bracketed in A. Cell-free zone contains large numbers of small nerves and capillaries not visible at this magnification. Underlying CR does not have high concentration of cells but contains more cells than does central pulp. (A and B courtesy of Drs. Dennis Weber and Michael Gaynor.) C. Diagram of peripheral pulp and its principal elements. D, Scanning electron micrograph of dentin-pulp junction. Note corkscrew fibers between odontoblasts (arrow). Reproduced with permission from Jean A, Kerebel JB, Kerebel LM. Oral Surg 1986;61:592. E, Scanning electron micrograph of pulpal surface of odontoblast layer. Thread-like structures are probably terminal raveling of nerves. (Courtesy of Drs. R. White and M. Goldman.)
dentin, just as the cells that produce it are not true odontoblasts. However, like the odontoblast, these cells trace their origins to undifferentiated cells.

**Fibroblasts.** Most of the cells of the pulp are fibroblasts. These cells exhibit wide variation in their degree of differentiation. Baume refers to them as mesenchymal cells, pulpoblasts, or pulpocytes in their progressive levels of maturation. These distinctions are made, in part, because of the ability of these cells to form calcified tissues, something regular connective tissue fibroblasts apparently cannot accomplish.

Pulpal fibroblasts are spindle-shaped cells with ovoid nuclei (Figure 2-12). They synthesize and secrete the bulk of the extracellular components, that is, collagen and ground substance. The classic autoradiographic studies of Weinstock and Leblond, using $^3$H-proline, demonstrated the process of collagen synthesis and secretion by the fibroblast.

Not only are fibroblasts the principal producers of collagen, they also eliminate excess collagen or participate in collagen turnover in the pulp by resorption of collagen fibers. This has been demonstrated to occur intracellularly by the action of lysosomal enzymes, which literally digest the collagen components.

**Defense Cells.**

**Histiocytes and Macrophages.** Undifferentiated mesenchymal cells (see Figure 2-12) around blood vessels (pericytes) can differentiate into fixed or wandering histiocytes under appropriate stimulation. Wandering histiocytes (macrophages) may also arise from monocytes that have migrated from vessels. These cells are highly phagocytic and can remove bacteria, foreign bodies (endodontic paste, zinc oxide, etc), dead cells, or other debris. Pulpal macrophages and dendritic cells thought to function like Langerhans’ cells have been identified in normal rat pulp. These cells seem to be associated with pulpal immunosurveillance.

**Polymorphonuclear Leukocytes.** The most common form of leukocyte in pulp inflammation is the neutrophil, although eosinophils and basophils are occasionally detected. It is important to know that although neutrophils are not normally present in intact healthy pulps, with injury and cell death they rapidly migrate into the areas from nearby capillaries and venules. They are the major cell type in microabscess formation and are very effective at destroying and phagocytizing bacteria or dead cells. Unfortunately, their participation often injures adjacent cells and may contribute to the development of wider zones of inflammation.
Lymphocytes and Plasma Cells. These inflammatory cell types generally appear following invasion into the area of injury by neutrophils. These cells are not normally present in healthy pulp tissue but are associated with injury and resultant immune responses—attempts to destroy, damage, or neutralize foreign substance(s). Their presence would therefore indicate the presence of a persistent irritant.

Mast Cells. Interestingly, mast cells are seldom in large numbers in normal, healthy pulps but are commonly found in inflamed pulps. The granules of these cells contain histamine, a potent inflammatory mediator, and heparin. These cells release these granules or degranulate into the surrounding tissue fluid during inflammation.

Since these cells are generally found near blood vessels, degranulation of mast cells releases histamine close to vascular smooth muscle, causing vasodilation. This increases vessel permeability, allowing fluids and leukocytes to escape.

Odontoblasts. The principal cell of the dentin-forming layer, the odontoblast, is the first cell type encountered as the pulp is approached from the dentin (Figure 2-13). These cells arise from peripheral mesenchymal cells of the dental papilla during tooth development (see Structural Elements) and differentiate by acquiring the characteristic morphology of glycoprotein synthesis and secretion (Figure 2-14). Glycoprotein forms the predentin matrix, which is rendered mineralizable by the odontoblast, a unique cell producing a unique tissue, dentin. Synthesizing and secretory activities render the odontoblast highly polarized, with synthesis occurring in the cell body and secretion from the odontoblastic process.

The cell body contains organelles that represent different stages of secretion of collagen, glycoproteins, and calcium salts. Matrix secretion precedes mineralization with these two events separated in time and space by the predentin. As happens in bone, the initial mineral seeding of predentin at the dentinoenamel junction is by formation of “matrix vesicles.” Classic studies by Weinstock and colleagues, using an autoradiographic technique, have demonstrated the functional sequence of matrix production and secretion. This material has recently been reviewed by Holland.

In histologic sections viewed under a light microscope, odontoblasts appear to vary from tall, pseudostratified columnar cells in the coronal pulp (see Figure 2-10) to a single row of cuboidal cells in radicular pulp to a flattened, almost squamous shape near the apex. These squamous cells often form an irregular, atubular dentin.

Scanning electron microscopy has provided a better view of the external morphology of the odontoblasts (see Figure 2-10, C and D). The large nucleus is located in the base of the cell, giving it a pear-shaped appearance. From an exquisite scanning electron microscopic study, French dental scientists have demonstrated that odontoblast cell bodies “appear tightly packed in the pulp horn and successively pear shaped, spindle shaped, club shaped, or globular from the crown to the apex” (Figure 2-15).

During dentin formation in the crown, the odontoblasts are pushed inward to form the periphery of a pulp chamber, the circumference of which is increasingly smaller than the original circumference at the dentinoenamel junction. This explains why the cells are packed and palisaded into a pseudostratified appear-
ance of coronal odontoblasts. Conversely, because the space is not so compressed in the radicular pulp, the odontoblasts maintain a columnar, cuboidal, or (in the apical region) squamous shape. Also, the resulting cell and tubule density is much higher in the pulp chamber than in the root pulp. The increased tubule density in the chamber may explain the greater sensitivity and permeability of the dentin of the crown.

The cell body manufactures the matrix material; the material is transported to and secreted from the odontoblastic process. Classically, the odontoblastic process has been described as extending from the cell body to the dentinoenamel junction, a distance of 2 to 3 mm (ie, 2,000 to 3,000 μm). This concept was based on the observations of many light microscopists using a variety of special procedures and stains. When dentin was examined by electron microscopy, the odontoblastic process was determined to be limited to the inner third of dentin, with the outer two-thirds of the tubule devoid of processes or of nerves but filled with extracellular fluid. More recent investigations indicate that odontoblastic processes may indeed extend to the dentinoenamel junction. However, tubular structures in dentinal tubules are not necessarily odontoblastic processes. Unequivocal identification can be done only by identifying a trilaminar plasma membrane around the putative process using transmission electron microscopy. The extent of the odontoblastic process remains controversial.

Therefore, modern interpretations of pulpal injury following conservative cavity preparation may not be attributable to amputation of odontoblastic processes but to desiccation, heat, and osmotic effects. Further, dentin sensitivity may not be related to direct stimulation of either odontoblastic processes or nerves in peripheral dentin since the tubules may be devoid of such structures in the periphery of dentin.

After initial dentin formation, the odontoblast, via its process, can still modify dentin structure by producing peritubular dentin. This is a hypermineralized cuff with little organic matrix within the tubule, decreasing the diameter of the tubule. When irritated, the odontoblast can accelerate peritubular dentin forma-
tion to the point of complete occlusion of the tubule (Figure 2-16).\textsuperscript{81–83} When tubule occlusions extend over a large area, this is referred to as sclerotic dentin, commonly found in teeth with cervical erosion.\textsuperscript{44}

Alternatively, irritated odontoblasts can secrete collagen,\textsuperscript{84} amorphous material, or large crystals into the tubule lumen; these occlusions result in decreases in dentin permeability to irritating substances.\textsuperscript{81–83} Although these secretions have been described as a defensive reaction by the odontoblast to protect itself and the underlying pulp, this “protection” has never been proved.

**Extracellular.** The dental pulp has most of its volume primarily composed of fibers and ground substance. These form the body and integrity of the pulp organ.

**Fibers.** The morphology of collagen fibers, a principal constituent in the pulp, has been described at the level of both light and electron microscopy. At the ultrastructural level, typical 640 angstrom banding or electron-dense periodicity provides positive proof of collagen fiber identity.\textsuperscript{85} These fibers form a loose, reticular network to support other structural elements of the pulp. Collagen is synthesized and secreted by odontoblasts and fibroblasts. However, the type of collagen secreted by odontoblasts to subsequently mineralize differs from the collagen produced by pulpal fibroblasts, which normally does not calcify. They also differ not in basic structure but in the degree of cross-linking, and in slight variation in hydroxylysine content.\textsuperscript{86}

Tropocollagen is immature collagen fibers that remain thin and stain black with silver nitrate, described in light microscopy as argyrophilic or reticular fibrils. If tropocollagen molecules aggregate into larger fibers, they no longer stain with silver and are generally termed “collagen fibers.” If several collagen fibers aggregate (cross-link) and grow more dense, they are termed “collagen bundles.” Collagen generally becomes more coarse (ie, develops more bundles) as the patient ages. Age also seems to permit ectopic calcification of pulp connective tissue, ranging from the development of random calcifications to diffuse calcifications\textsuperscript{87} to denticle (pulp stone) formation. Elastic, the only other fibrous connective tissue protein, is found only in the walls of pulp arterioles.

Collagen has been described as having a unique arrangement in the peripheral pulp; these bundles of collagen are termed von Korff’s fibers. Most textbooks describe von Korff’s fibers as being corkscrew-like and originating between odontoblasts to pass into the dentin matrix (Figure 2-17). The tight packing of odontoblasts, predentin, capillaries, and nerves produces very narrow spaces between and around odontoblasts that can retain heavy metal stain (precipitates). Ten Cate’s electron microscopic studies of the distribution of these precipitates demonstrated their presence in narrow intracellular tissue spaces.\textsuperscript{88} He claimed that they represented artifactual “stains” that were not attached to collagen fibers.\textsuperscript{88} However, more recent scanning electron microscopic studies (see Figure 2-10,
D) of the predentin-pulpal border demonstrating screw-like fibrous material have stimulated new speculation that von Korff’s fibers are real structures.63

Ground Substance. This structureless mass, gel-like in consistency, makes up the bulk of the pulp organ. It occupies the space between formed elements. The ground substance resembles that of other areolar, fibrous connective tissues, consisting primarily of complexes of proteins and carbohydrates and water. More specifically, these complexes are composed of combinations of glycosaminoglycans, that is, hyaluronic acid, chondroitin sulfate, and other glycoproteins.89,90 The ground substance surrounds and supports structures and is the medium through which metabolites and waste products are transported to and from cells and vessels. Aging of the pulp alters the ground substance,91 although there is no substantive proof that these alterations significantly inhibit pulp functions.

Supportive Elements.

Pulpal Blood Supply. Numerous investigators have described the blood supply of the dental pulp.92–95 Because the pulp itself is small, pulp blood vessels do not reach a large size. At the apex and extending through the central pulp, one or more arterioles branch into smaller terminal arterioles or metarterioles that are directed peripherally (Figure 2-18, A). Before the arterioles break up into capillary beds, arteriovenous anastomoses often arise to connect the arteriole directly to a venule.96 These arteriole-venule shunts are identified by the presence of irregularly oriented myoepithelium-like cells surrounding them and by the cuboidal nature of the cells lining their lumen.97 The classic description of microcirculatory beds includes capillaries that branch off arterioles at right angles (Figure 2-18, B). Unfortunately, no such structures have been found in human pulps. Instead, arterioles branch into terminal arterioles, which, in turn, give rise to capillaries (Figure 2-19).

Capillary density is highest in the subodontoblastic region with loops passing between odontoblasts (Figure 2-20).98–100 In the subodontoblastic region, capillaries with fenestrations occur frequently and regularly in both primary and permanent teeth (Figure 2-21). The fenestrae (“windows”) are spanned by a thin diaphragm of plasma membrane. The frequency of fenestration falls off rapidly when examining central capillaries, being as low as 4% in the coronal pulp.98,101 Capillaries empty into small venules that connect with fewer and successively larger venules. At the apex, multiple venules exit the pulp. These venules connect with vessels that drain the periodontal ligament or adjacent alveolar bone. The vessels of the pulp have

Figure 2-18 A. Perfused pulp of dog premolar showing the size and location of vessels. Larger central vessels are arterioles and venules. Peripherally directed are smaller metarterioles that branch into the rich network of looping capillaries of peripheral pulp. B, Schematic diagram of classic microcirculatory vascular bed that would represent a region of central and peripheral vessels as seen in A. Arterioles are invested by a continuous layer of smooth muscle cells. Metarterioles have discontinuous clusters of smooth muscle with capillaries branching directly off metarterioles. Precapillary smooth muscle sphincters are strategically located to control capillary blood flow. True capillaries lack smooth muscle. Arteriovenous shunts represent direct connections between arterioles and venules. Their muscles are innervated by sympathetic nerve fibers. A reproduced with permission from Seltzer S, Bender IB. The dental pulp. 2nd ed. Philadelphia: JB Lippincott; 1975, p. 106.
thinner muscular walls (tunica media) than vessels of comparable diameter in other parts of the body. Undoubtedly, this is an adaptation to the surrounding protective and unyielding walls. Kim and his associates have obtained evidence that suggests that most vasodilating agents induce only a transient, brief increase in pulpal blood flow followed by a decrease in blood flow owing to collapse of local venules. Apparently, the vasodilation either directly impinges on venules or permits transudation of fluid across capillaries that indirectly compresses the thin-walled venules in the low-compliance system of the pulp chamber.

The above-described general vascular architecture is found in each tooth root. Alternate blood supply is available to multicanaled teeth, with the resulting rich anastomoses in the chamber. The occasional vessels that communicate via accessory canals have not been
demonstrated to contribute significantly as a source of collateral circulation.

Lymphatics. The presence of pulpal lymphatics is disputed. However, lymphatics have been identified in the pulp at the ultrastructural and histologic levels by the absence of red blood cells in their lumina, the lack of overlapping of endothelial margins, and the absence of a basal lamina. They arise as lymphatic capillaries in the peripheral pulp zone (Figure 2-22) and join other lymph capillaries to form collecting vessels. These vessels unite with progressively larger lymphatic channels that pass through the apex with the other vasculature. Numerous authors, using both histologic and functional methods, have described extensive anastomoses between lymph vessels of the pulp, periodontal ligament, and alveolar bone.

Functional Implications. The presence of arteriovenous shunts in the pulp provides the opportunity for blood to shunt past capillary beds since these arteriole-venule connections are “upstream” from the capillaries. Alternatively, the arteriole-venule shunts could remain nearly closed (in a constricted state), and most of the blood would pass peripherally in the pulp to perfuse capillaries and the cells that they support. It has been suggested that the distribution of blood flow might change during pulp inflammation. Increased dilation of arteriole-venule shunts may produce “hyperemia,” in which more blood vessels than normal are open and filled with blood cells; this may indicate more rapid blood flow or represent partial stasis. Further, this dilation of arteriole-venule shunts may “steal” blood from capillary beds, causing accumulation of waste products.

Capillary fenestration may indicate that these capillaries are more permeable to large molecules or that they allow more rapid fluid movement across the endothelium. However, studies on pulp capillaries
suggest a lower than normal permeability to large molecules. On the other hand, a higher rate of fluid movement has not been ruled out. Increased transudation of plasma and polymorphonuclear neutrophil leukocytes from the circulation occurs most often in venules rather than capillaries.

The structural identification of lymph capillaries complements the functional studies of Walton and Langeland, who demonstrated that substances placed in the pulp chamber can be found in regional lymph nodes. The open endothelial margins and incomplete basal lamina permit entry of large molecules and even bacteria. The fact that materials placed on pulps can migrate to lymph nodes indicates the possibility of immunologic reactions to substances that enter the pulp. Bernick described the appearance of lymphatics in the inflamed pulp and surmised that their function is to remove the excess fluid and debris that accompanies inflammation.

Unfortunately for the pulp, the lymphatics may collapse as pulp pressure rises, thus inhibiting removal of irritants and fluid. Clearly, more investigation is required before one can understand how lymphatic function is disturbed in pulp inflammatory conditions. The anastomoses of pulpal, periodontal, and alveolar lymphatics may be important routes for the spread of pulpal inflammation into adjacent tissues during the removal of irritants and fluid from the pulp. These structural interrelationships have not received the attention they deserve. Finally, the extent and degree of anastomoses of apical venules with those of the periodontal ligament and alveolar bone need investigation. Vessels may provide a route for local anesthetic movement during intraosseous or periodontal ligament injections rather than the fluid “dissecting” through perivascular tissue spaces. These same pathways have been implicated as routes of spread of inflammation from pulp to periodontal ligament and/or bone and vice versa.

Nerves. Several nerve bundles, each containing numerous unmyelinated and myelinated nerves, pass into each root via the apical foramen. The majority are unmyelinated nerves, most of which are part of the sympathetic division of the autonomic nervous system; these have been shown to cause reductions in pulp blood flow when stimulated. The remaining nerves are myelinated sensory nerves of the trigeminal system (Figure 2-23).

The myelinated nerve fibers branch extensively below the cell-rich zone to form the so-called plexus of Raschkow (Figure 2-24). From here, many fibers lose their myelin sheath and pass through the cell-free zone to terminate as receptors or as free nerve endings near odontoblasts (Figure 2-25); others pass between odontoblasts to travel a short distance up the dentinal tubules adjacent to odontoblastic processes. The nerve endings terminate far short of the dentinoenamel junction; rather, endings are found only in tubules of the inner dentin and predentin, on or between odontoblasts. Byers has found that intradental nerves pass approximately 100 µm into the tubules, regardless of the dentin thickness, in a wide variety of animal species. Perhaps nerve fibers cannot be nourished beyond a 100 µm diffusion distance. Some sensory axons exhibit terminal arborizations that innervate up to 100 dentinal tubules. Significantly, sensory nerves of the pulp respond to noxious stimuli with pain sensation only, regardless of the stimulus. This pain is produced whether the stimulus is applied to dentin or the pulp.

Cavity preparation in the unanesthetized tooth is painful at any depth of dentin. How can this occur if there are no sensory nerves in the outer two-thirds of dentin? The answer probably lies in the hydrodynamic
Figure 2-23  Schematic drawing showing sensory nerve location in pulp and dentin. Percentage of innervated tubules at regions A through D is indicated (left). Px = plexus of Raschkow; cfz = cell-free zone; O = odontoblasts; p = predentin. Reproduced with permission from Byers M.144

Figure 2-24  Branching of nerve bundles as they approach the subodontoblastic region (plexus of Raschkow). (Courtesy of Dr. James Avery.)

Figure 2-25  Nerve terminal (arrow) located between adjacent odontoblasts near calcifying dentin. Large number of vesicles is characteristic of nerve terminal. Terminal is closely applied to the adjacent odontoblastic process; this does not, however, represent a synapse. (Courtesy of Dr. James Avery.)
theory in which fluid movement within tubules stimulates distant sensory nerve endings (see Byers et al and Avery128–131 for reviews).

Highly organized junctions have been demonstrated between some nerve fibers and odontoblasts.132–136 Although they do not appear to be typical synaptic junctions, their existence must be functional. It is unclear whether the activity is sensory or motor.

An additional function of sympathetic nerves is the possible regulation of the rate of tooth eruption. Sympathetic nerve activity influences local blood flow and tissue pressure by opening or closing arteriovenous shunts as well as arteriolar blood flow; this may secondarily affect eruptive pressure.137 Activation of sympathetic fibers not only reduces pulpal blood flow138 but also decreases the excitability of intradental nerves.139 Thus, there is a very intimate relationship between pulpal nerves and their excitability and local blood flow.140

Numbers and concentrations of nerves vary with the stage of tooth development and also with location. Fearnhead and others have reported that very few nerves appear in the human pulp prior to tooth eruption.41,141,142 After eruption, the highest number of nerves is found in the pulp horns (about 40% of the tubules are “innervated”). The number of nerves per tubule drops off to about 4.8% in the more lateral parts of the coronal dentin to less than 1% in the cervical region (see Figure 2-23), with only an occasional nerve in radicular dentin.143 Patterns of branching nerves seen with the light microscope would confirm numbers of nerves at different levels. There is little branching off the main nerve bundles until the coronal pulp. Regions of sensitivity also correlate in that coronal pulp and dentin are more painful to stimuli than are radicular pulp and dentin. The same stimuli applied to dentin were described as “sharp” when applied to coronal dentin but “dull” when applied to radicular dentin.144 Restorative procedures in rat teeth cause sprouting of pulpal and intradental nerves that may modify both dentin sensitivity145 and local inflammatory reactions.146

Interestingly, removal of the pulp by extraction of the tooth or by pulpectomy and, presumably, pulpotomy results in the successive degeneration of the cell bodies located in the spinal nucleus of the trigeminal nerve, the main sensory ganglion, and the peripheral nerve leading to the tooth in the socket.147 Bernick observed the effects of caries and restorations on underlying nerves in the pulp.148 He found a degeneration of the subodontoblastic plexus of nerves associated with the production of irritation dentin. He concluded that “the terminal nerves in the injured pulp are sensitive to the noxious products of caries and the restorative procedures. An apparent decrease in sensitivity results in restored teeth.” The lack of sensitivity that accompanies the caries process may be attributable, at least partially, to degeneration of underlying nerves.

Calcifications. Basically, there are two distinct types of pulpal calcifications: formed structures commonly known as pulp stones (denticles) and tiny crystalline masses generally termed diffuse (linear) calcifications (Figure 2-26). Pulp stones seem to be found predominantly in the coronal pulp, whereas the calcifications found in radicular pulp seem to be of the diffuse variety.149

Calcifications are common in the dental pulp, with a tendency to increase with age and irritation. It has been speculated that these calcifications may aggravate or even incite inflammation of pulp or may elicit pain by pressing on structures; however, these speculations have not been proved and are improbable. Although these calcifications are not pathologic, their presence under certain conditions may be an aid in diagnosis of pulpal disease. Moreover, their bulk and position may interfere with endodontic treatment.

Pulp Stones. These discrete calcific masses appear with frequency in mature teeth.150 Although there is increased incidence with age, they are not uncommon

![Figure 2-26 Uninflamed pulp. Typical pattern of calcifications. Larger pulp stones in the chamber blend into linear diffuse calcifications in the canal. Reproduced with permission from Bernick S.155](image-url)
in young teeth. It has also been demonstrated that their occurrence and size often increase with external irritation. Pulp stones also may arise spontaneously; their presence has been identified on radiographs (Figure 2-27) and, on histologic examination, even in impacted teeth. Interestingly, there appears to be a predisposition for pulp stone formation in certain individuals, possibly a familial trait.

Pulp stones have been classified as two types, true or false. However, recent careful histologic examination has discounted the true pulp stone. Supposedly, true pulp stones are islands of dentin, demonstrating tubules and formative odontoblasts on their surface. However, serial sectioning has shown that these are not islands but peninsulas—extrusions from dentin walls. Therefore, the term “denticle,” which would imply dentin structure, is a misnomer. The term “pulp stone” is more correct, particularly because the “false” pulp stone so closely resembles gallstones and kidney or ureter stones.

Pulp stones, like other types of stones, are formed from clearly concentric or diffuse layers of calcified tissue on a matrix that seems to consist primarily of collagen. Their structure may help explain their origin; it has been shown that potential nidi of pulp stones may occur in the sheaths associated with blood vessels (Figure 2-28) and nerves. Other potential nidi are calcifications of thrombi in vessels or calcification of clumps of necrotic cells. Whatever the nidus, growth is by incremental layering of a matrix that quickly acquires mineral salts.

Pulp stones are also classified according to location. “Free” stones are those that are islands, “attached” stones are free pulp stones that have become fused with the continuously growing dentin, and “embedded” stones are formerly attached stones that have now become surrounded by dentin.

Pulp stones may be important to the clinician who attempts access preparation or to negotiate canals. Either free or attached denticles may attain large size and occupy considerable volume of the coronal pulp (Figure 2-29). Their presence may alter the internal anatomy and confuse the operator by obscuring, but not totally blocking, the orifice of the canal. Attached denticles may deflect or engage the tip of exploring instruments in the canals, thus preventing their easy passage down the canal.

Pulp stones of sufficient size are readily visible on radiographs, although the majority are too small to be seen except on histologic examination. The large, discrete masses, occasionally appearing to nearly fill the chamber (Figure 2-30), are likely to be those of natural occurrence. The chamber that appears to have a diffuse and obscure outline may represent a pulp that has been subjected to a persistent irritant and has responded by forming large numbers of irregular pulp stones. This finding is a diagnostic aid and indicates a pulp exposed to a persistent chronic irritant.

**Diffuse Calcifications.** Also known as linear calcifications because of their longitudinal orientation,
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these are common pulp findings. They may appear in any area of the pulp but predominate in the radicular region. Their form is that of tiny calcified spicules, usually aligned close to blood vessels and nerves or to collagen bundles (see Figure 2-26). Because of their size and dispersion, they are not visible in radiographs and are seen only on histologic specimens. Like pulp stones, diffuse calcifications also tend to increase with age and with irritation but otherwise have no known clinical significance.

Figure 2-29 Large pulp stone may have been formed by growth and fusion of smaller stones such as those below it. Examination of other serial sections may show that this apparently “free” pulp stone is actually attached to dentin walls. Reproduced with permission from Bernick S.155

PULP CHANGES WITH AGE

Teeth age, not only with the passage of time but also under the stimulus of function and irritation. Therefore, age is a chronologic occurrence, but even more importantly, an “aged” tooth may represent a premature response to the abuses of caries, extensive restorative procedures, and inflicted trauma. Since the pulp reacts to its environment and is in intimate contact with dentin, it responds to abuses by altering the anatomy of its internal structures and surrounding hard tissue.

Dimensional

With time and/or injury, the pulp volume decreases by forming additional calcified tissues on the walls (see Figure 2-30). Ordinarily, with time, formation of dentin continues, with the greatest increase on the floor of the chamber of posterior teeth (Figure 2-31) and on the incisal of anterior teeth. In such teeth, the location of the pulp chamber and/or root canals may be difficult. In anterior teeth, the clinician may have to search cervically to locate a remnant of the chamber. In molars, dentin formation may have rendered the chamber almost disk-like; while searching, it is easy to inadvertently pass a bur through the flattened chamber (Figure 2-32). If the preparation is continued, the next hemorrhage encountered will arise.

Figure 2-31 Pattern of dentin formation in “aged” posterior tooth from a 60-year-old patient. Typically irregular hard tissue apposition is greatest on the floor, decreasing chamber depth. (Courtesy of Dr. Sol Bernick.)
from the furcation, not from the chamber. Careful examination of radiographs to identify chamber size and location, followed by measurements of the occluso-chamber distance, will prevent this mishap. Irritation dentin formation will also alter internal anatomy. Therefore, when the dentin has been violated by caries or by attrition, one should expect increased amounts of hard tissue in the underlying pulp. Irritation dentin may occasionally be extensive enough to obscure or fill large areas of the chamber.

**Structural**

Although exacting quantitative studies have not been published, there is agreement that the number of cells decreases and the fibrous component increases with aging of the pulp (Figure 2-33). The increased fibrosis with time is not from continued formation of collagen but rather may be attributable to a persistence of connective tissue sheaths in an increasingly narrowed pulp space.

Bernick observed a decrease in the number of blood vessels supplying the aging pulp, noting that many of the arteries demonstrated arteriosclerotic changes similar to those seen in other tissues (Figure 2-34). These changes involve decreases in lumen size with intimal thickening and hyperplasia of elastic fibers in the media. Also common is calcification of arterioles and precapillaries. Although these structural changes are described, it is not clear whether the vascular and neural changes alter the function of the older pulp.

![Figure 2-32](image1.png) **Figure 2-32** Disk-like chamber (arrow) is a result of hard tissue apposition on the floor and roof. The center of the chamber is difficult to locate. An access preparation should be started by locating the large orifice of the distal canal first. (Courtesy of Dr. G. Norman Smith.)

![Figure 2-33](image2.png) **Figure 2-33** Age changes in cross-sections of human pulp. A, Young pulp with characteristic cellularity and relatively small scattered fibrous components of central pulp. B, Acellularity and large fiber bundles are common findings in mature pulps. (Courtesy of Dr. Dennis Weber.)

Not only do cells decrease in number, notably fibroblasts and odontoblasts, but the remaining cells are likely to appear relatively inactive. These ordinarily active cells demonstrate fewer organelles associated with synthesis and secretion.
“Regressive” Changes
The term “regressive” is defined as a condition of decreased functional capability or of returning to a more primitive state. Older pulps have been described as regressive and as having a decreased ability to combat and recover from injury. This has been surmised because older pulps have fewer cells, a less extensive vasculature, and increased fibrous elements. In fact, there have never been experiments proving that aged pulps are more susceptible to irritants or less able to recover. Until these have been conclusively demonstrated, the term “regression” is not appropriate, and the dentist should not assume that pulps in older individuals are less likely to respond favorably than are younger pulps.

Pulpal Response to Inflammation
Pulp structures and functions are altered, often radically, by injury and resulting inflammation. As a part of the inflammatory response, neutrophilic leukocytes are chemotactically attracted to the site. Bacteria or dying pulp cells are phagocytosed, causing release of potent lysosomal enzymes. These enzymes may attack surrounding normal tissue, resulting in additional damage.

For instance, by-products of the hydrolysis of collagen and fibrin may act as kinins, producing vasodilation and increased vascular permeability. Escaping fluid tends to accumulate in the pulp interstitial space, but because the space is confined, the pressure within the pulp chamber rises. This elevated tissue pressure produces profound, deleterious effects on the local microcirculation. When local tissue pressure exceeds local venous pressure, the local veins tend to collapse, increasing their resistance; hence blood will flow away from this area of high tissue pressure as it seeks areas of lower resistance. This process of blood diversion can be illustrated by applying slight pressure to the end of a fingernail. As the pressure increases, the nail bed blanches as blood is squeezed out of the local vessels, and new blood is prevented from flowing through this area of elevated tissue pressure. Persistent pressure continues to compromise circulation. The consequences of reduced local blood flow are minor in normal tissue but disastrous in inflamed tissue because the compromised circulation allows the accumulation of irritants such as injurious enzymes, chemotoxic factors, and bacterial toxins.

This event may lead to the development of the “compartment syndrome,” a condition in which elevated tissue pressure in a confined space alters structure and severely depresses function of tissues within that space. Depressed function often leads to cell death, which, in turn, produces inflammation resulting in fluid escape and increased pressure within the compartment. The increased tissue pressure collapses veins, thereby increasing the resistance to blood flow through capillaries. Blood is then shunted from areas of high tissue pressure to more “normal” areas. Thus, a vicious cycle is produced in which inflamed regions tend to become more inflamed because they tend to limit their own local nutrient blood flow (Figure 2-35).

This is not to say that the pulp “strangulation” theory is valid. As shown by Van Hassel, and more recently by Nahri and Tønder and Kvinsland, pressures are not readily transmitted throughout the pulp. Therefore, inflammation and increased pressure in the coronal pulp will not collapse veins in the apical region. Pulps physiologically have multiple compartments throughout. It is as if small volumes of pulp tissue are enclosed in separate connective tissue sheaths, each of which can contain local elevations in tissue pressure. Although no histologic evidence exists to support this notion, these functional compartments may break down individually to become necrotic and may coalesce to form microabscesses.

The recent micropuncture work by Tønder and Kvinsland demonstrated that there are highly localized elevations in interstitial tissue pressure in inflamed pulp. This is thought by some to be caused by the release of vasoactive neuropeptides such as substance P and calcitonin gene–related peptide, both found in pulp nerve fibers. During pulpal inflammation, there is an increase in the number of calcitonin gene–related peptide–containing nerves in areas previously devoid of nerves. The release of these peptides seems to promote and sustain inflammation, prompting some to call it neurogenic inflammation.

Pulpodontinal Physiology
As long as dentin is covered peripherally by enamel on coronal surfaces and cementum on radicular surfaces, the dental pulp will generally remain healthy for life, unless the apical blood supply is disrupted by excessive orthodontic forces or severe impact trauma. Most pathologic pulp conditions begin with the removal of one or both of these protective barriers via caries, fractures, or abrasion. The result is the communication of pulp soft tissue with the oral cavity via dentinal tubules, as has been demonstrated by dye penetration studies and radioactive tracer experiments.

It is apparent that substances easily permeate dentin, permitting thermal, osmotic, and chemical insults to act on the pulpal constituents. The initial stages involve stimulation or irritation of odontoblasts and may proceed to inflammation and often to tissue destruction.
To understand how these steps may lead to pulp damage, the pulpodental complex will be examined in its separate forms.

**Dentin Structure**

Dentin is a calcified connective tissue penetrated by millions of tubules; their density varies from 40,000 to 70,000 tubules per square mm. Tubules are from 1 µm in diameter at the dentinoenamel junction to 3 µm at their pulpal surface and contain fluid that has a composition similar to extracellular fluid. If the fluid becomes contaminated, for example, with caries bacterial endotoxins and exotoxins, then it develops a reservoir of injurious agents that can permeate through dentin to the pulp to initiate inflammation. It is useful to understand the important variables that control dentin permeability.

**Dentin Permeability.** Dentinal tubules in the coronal dentin converge from the dentinoenamel junction to the pulp chamber. This tends to concentrate or focus permeating substances into a smaller area at their terminus in the pulp. The surface area occupied by tubules at different levels indicates the effect of tubule density and diameter. One can calculate from Garberoglio and Brännström’s observations that the area of dentin occupied by tubules is only 1% at the dentinoenamel junction and increases to 45% at the pulp chamber. The clinical implications of this are enormous. As dentin becomes exposed to increasing depths by restorative procedures, attrition, or disease, the remaining dentin becomes increasingly permeable. Thus, dentin removal, although necessary, renders the pulp more susceptible to chemical or bacterial irritation. This functional consequence of tubule area is also responsible for the decrease in dentin microhardness closer to the pulp; as tubule density increases, the amount of calcified matrix between the tubules decreases. This relative softness of the dentin lining the pulp chamber somewhat facilitates canal enlargement during endodontic treatment.

Overall dentin permeability is directly proportional to the total surface area of exposed dentin. Obviously, a leaking restoration over a full crown preparation provides more diffusional surface for bacterial products than would a small occlusal restoration. Restorations requiring extensive and deep removal of dentin (ie, preparation for a full crown) would open more and larger tubules and increase the rate of injurious substances diffusing from the surface to the pulp—thus the importance of “remaining dentin thickness.”

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Figure 2-35 Vicious cycle of pulpal inflammation, which begins with irritation (top), leads to a localized response, and may progress to a lesion of increasing severity and eventual irreversible pulpitis.
permeability of the root is 10 to 20 times less than that of a similar thickness of coronal dentin. This may account for the lack of pulpal reactions to periodontal therapy that removes cementum and exposes root dentin to the oral cavity.

Recent evidence indicates that dentin permeability is not constant after cavity preparation. In dogs, dentin permeability fell over 75% in the first 6 hours following cavity preparation. Although there were no histologic correlates of the decreased permeability, dogs depleted of their plasma fibrinogen did not decrease their dentin permeability following cavity preparation. The authors speculated that the irritation to pulpal blood vessels caused by cavity preparation increased the leakage of plasma proteins from pulpal vessels out into the dentinal tubules, where they absorb to the dentin, decreasing permeability. Future study of this phenomenon is required to determine if it occurs in humans.

The character of the dentin surface can also modify dentin permeability. Two extremes are possible: tubules that are completely open, as seen in freshly fractured or acid-etched dentin, and tubules that are closed either anatomically or with microcrystalline debris. This debris creates the “smear layer” (Figure 2-36), which forms on dentin surfaces whenever they are cut with either hand or rotary instruments. The smear layer prevents bacterial penetration but permits a wide range of molecules to readily permeate dentin. Small molecules permeate much faster than large molecules. Smear layers are often slowly dissolved over months to years as oral fluids percolate around microleakage channels between restorative materials and the tooth. Removal of the “smear layer” by acid etching or chelation increases dentin permeability because the microcrystalline debris no longer restricts diffusion of irritants and also permits bacteria to penetrate into dentin. There is considerable debate as to whether smear layers created in the root canal during biomechanical preparation should be removed. Its removal may increase the quality of the seal between endodontic filling materials and root dentin. It may also increase the bond strength of resin posts.

**Pulp Metabolism.** The rate at which pulpal cells are metabolizing can be quantitated by measuring their rate of oxygen consumption, CO₂ liberation, or lactic acid production. Fisher and colleagues reported that zinc oxide–eugenol (ZOE) cement, eugenol, calcium hydroxide, silver amalgam, and procaine all depressed pulp oxygen consumption. Shalla and Fisher demonstrated that lowering the medium pH of pulp
cells below 6.8 caused a progressive decrease in oxygen consumption.\textsuperscript{203} This undoubtedly occurs during the development of pulp abscesses. Even though pulp respiration may decrease in an acid environment, Fisher and Walters have shown that bovine pulp has a very active ability to produce energy through anaerobic glycolysis.\textsuperscript{204} Oxygen consumption has also been measured in dental pulp tissue using an oxygen electrode.\textsuperscript{205} A more sensitive technique has recently been applied to studying pulp respiration.\textsuperscript{206–208} Pulp tissue was placed in $^{14}$C-labeled substrates such as succinate and measured the rate of appearance of $^{14}$CO$_2$ from the reaction vessel. Using this technique, reduced pulp metabolism was demonstrated when ZOE cement, Dycal, Cavitec, and Sargent’s formula/N$_2$ were used.\textsuperscript{206} It was also reported that the application of orthodontic force to human premolars for 3 days led to a 27% reduction in pulp respiration.\textsuperscript{207} The depressant effects of eugenol on pulp respiration were reported as well.\textsuperscript{208} Similar results were recently reported by Hume.\textsuperscript{209,210} Pulpal irritation generally causes elevated tissue levels of cyclooxygenase products. Eugenol in ZOE cement has been shown to block this reaction.\textsuperscript{211}

Pulp Reaction to Permeating Substances. What happens when permeating substances reach the pulp chamber? Although bacteria may not actually pass through dentin, their by-products\textsuperscript{212,213} have been shown to cause severe pulp reaction.\textsuperscript{177,212,214} The broad spectrum of pulp reaction, from no inflammation to abscess formation, may be related to the concentration of these injurious substances in the pulp. Although exposed dentin may permit substances to permeate, their concentrations may not reach levels high enough to trigger the cascade of events associated with inflammation. This would indicate that the interstitial fluid concentration of these substances can be maintained at vanishingly low concentrations. As long as the rate of pulp blood flow is normal, the microcirculation is very efficient at removing substances diffusing across dentin to the pulp chamber.\textsuperscript{184} There is enough blood flowing through the pulp each minute to completely replace between 40 and 100% of the blood volume of the pulp.\textsuperscript{215} Since blood is confined to the vasculature, which comprises only about 7% of the total pulpal volume,\textsuperscript{215,216} the blood volume of the pulp is replaced 5 to 14 times each minute.

If pulp blood flow is reduced,\textsuperscript{217–230} there will be a resultant rise in the interstitial fluid concentration of substances that permeated across dentin.\textsuperscript{184} The increased concentration of injurious agents may degranulate mast cells,\textsuperscript{49,50} release histamine\textsuperscript{231} or substance P,\textsuperscript{232–236} produce bradykinin,\textsuperscript{237} or activate plasma proteins.\textsuperscript{238,239} All of these effects would initiate inflammation. The endogenous mediators of inflammation produce arteriolar vasodilation, elevated capillary hydrostatic pressure, increased leakage of plasma proteins into the pulp interstitium,\textsuperscript{240} and increased pulp tissue pressure.\textsuperscript{167,169,241} These events, by causing collapse of local venules, lead to a further reduction in pulp blood flow,\textsuperscript{242} with an even higher interstitial concentration of irritants; thus, a vicious cycle\textsuperscript{243} is created that may terminate in pulp death (see Figure 2-35).

Techniques for accurately measuring pulp tissue pressures were developed in the 1960s.\textsuperscript{244–248} These methods all involve drilling carefully through the enamel and dentin to tap into the pulp chamber. Recently, several indirect methods of measuring pulp pressure through intact dentin have been devised. One group measured the pressure in a chamber cemented on cat dentin necessary to prevent outward movement of fluid as 15 cm H$_2$O.\textsuperscript{249} Ciucchi et al, using the same technique in humans, reported a normal pulp pressure of 14 cm H$_2$O (10.4 mm Hg), far below systemic blood pressure but close to pulp capillary pressure (Figure 2-38).\textsuperscript{250} Recent direct measurements of pulpal interstitial fluid pressures by micropuncture have given pressures of 6 to 10 mm Hg. However, they were done on pulps exposed to the atmosphere.\textsuperscript{169}

Pulp blood flow has been measured by numerous authors using many different techniques.\textsuperscript{251–254} Recently, Gazelius and his associates reported the use of
a laser Doppler blood flowmeter that was sensitive enough to measure changes in pulpal blood flow in intact human teeth.253 This method has begun to be used in pulp biology research. Blood flow in the pulp falls in direct proportion to any increase in pulp tissue pressure. Van Hassel,167 Stenvik and colleagues,241 and Tønder and Kvinnslæg169 reported that pulp tissue pressure is elevated in pulpitis but that the elevation is localized within specific regions of the pulp, being normal in noninflamed areas. The localized reduction in pulp blood flow, however, allows the accumulation of mediators of inflammation, which, in turn, causes a spread in the elevation of tissue pressure, reducing pulp blood flow to a larger volume of pulp, etc.167,243 The elevated pulp tissue pressure causes dull, aching, poorly localized pulp pain, a type of pain that differs from the brief, sharp, well-localized dentinal pain that is postulated to be caused by fluid movement within dentin.3 Accordingly, when teeth with elevated pulp pressures are opened to the pulp, the pain generally subsides rapidly as tissue pressures rapidly fall.

**Dentin Sensitivity.** Clinicians recognize that dentin is exquisitely sensitive to certain stimuli.255,256 It is unlikely that this sensitivity results from direct stimulation of nerves in dentin (Figure 2-39). As previously stated, nerves cannot be shown in peripheral dentin.143,144 Another speculation is that the odontoblastic process may serve as excitable “nerve endings” that would, in turn, excite nerve fibers shown to exist in deeper dentin, closer to the pulp.143,144,257 The experiments of Anderson and colleagues258 and Brännström259 suggest that neither odontoblastic processes nor excitable nerves within dentin are responsible for dentin’s sensitivity.

This led Brännström and colleagues to propose the “hydrodynamic theory” of dentin sensitivity, which sets forth that fluid movement through dentinal tubules, moving in either direction, stimulates sensory nerves in dentin or pulp.259,260 Further support for the hydrodynamic theory came from electron microscopic examination of animal67,261,262 and human dentin,64,66,67 demonstrating that odontoblastic processes seldom extend more than one-third the distance of the dentinal tubules. Work by LaFleche and colleagues suggested that the process may retract from the periphery during extraction or processing.77 Obviously, more investigation will be required before any definitive statement can be made regarding the distribution of the process. The tubules are filled with dentinal fluid that is similar in composition to interstitial fluid.176

The hydrodynamic theory satisfies numerous experimental observations. Although it cannot yet be regarded as fact, it has provided and will continue to provide a very useful perspective for the design of future experiments.263–265 (see Figure 2-39).

**Effect of Posture on Pulpal Pain.** Whenever an appendage is elevated above the heart, gravity acts on blood on the arterial side to reduce the effective pressure and, hence, appendage blood flow. This is why one’s arm rapidly tires when working overhead. The reduced pressure effect occurs in structures in the head that, in normal upright posture, are well above the heart. When the patient lies down, however, the gravitational effect disappears, and there is a significant increase in pulp blood pressure and corresponding rise in tissue pressure over and above that caused by endogenous mediators of inflammation. In this position, an irritated and inflamed pulp becomes more sensitive to many stimuli and may spontaneously begin to fire a message of pain. This is why patients with pulpitis frequently call their dentists after lying down at night. In the supine position, a higher perfusion pressure and, presumably, a higher tissue pressure develop in the patient, which cause more pulp pain. Patients often discover that they are more comfortable if they attempt to sleep sitting up, which again emphasizes the effects of gravity on pulp blood flow.
Another factor contributing to elevated pulp pressure on reclining is the effect of posture on the activity of the sympathetic nervous system. When a person is upright, the baroreceptors (the so-called “carotid” sinus), located in the arch of the aorta and the bifurcation of the carotid arteries, maintain a relatively high degree of sympathetic stimulation to organs richly innervated by the sympathetic nervous system. Tönder demonstrated that canine pulps showed large reductions in blood flow when the baroreceptor system was manipulated. If the human dental pulp is similar, it would result in slight pulpal vasoconstriction whenever a person is standing or sitting upright. Lying down would reverse the effect with an increase in blood flow and tissue pressure in the pulp. Lying down, then, increases pulp blood flow by removing both the effects of gravity and the effects of baroreceptor nerves, which decrease pulpal vasoconstriction. Thus, the increase in pain from inflamed pulps at night or the transformation of the pain from a dull to a throbbing ache has rational physiologic bases. The lack of documentation in the literature is owing to a lack of investigation.

Systemic Distribution of Substances from Dentin and Pulp. The rate of blood flow in the pulp is moderately high and falls between that of organs of low perfusion, such as skeletal muscle, and highly perfused organs, such as the brain or kidney (Figure 2–40). Since dentinal fluid (the fluid filling the tubules) is in communication with the vasculature of the pulp, in theory, substances placed directly on pulp or dentin diffuse to the interstitial fluid and are quickly absorbed into the bloodstream or into the lymphatics. In vivo evidence indicates that both may occur. Numerous authors have demonstrated that substances placed onto dentin or into pulp chambers are absorbed systemically. These substances include radioactive labeled cortisone, tetracycline, lead, formocresol, glutaraldehyde, and camphorated monochlorophenol.

![Figure 2-39](image)

Schematic diagram of essentials of three theories of dentin sensitivity. A, Classical theory proposed that stimuli applied to dentin caused direct simulation of nerves in dentin. B, Modified theory proposed that stimuli applied to the odontoblastic process would be transmitted along the odontoblast and passed to the sensory nerves via some sort of synapse. C, Hydrodynamic theory proposed that fluid movement within tubules transmits peripheral stimuli to highly sensitive pulpal nerves. C more accurately represents the actual length of the odontoblastic process relative to the tubules. Nerves are seldom found more than one-third the distance from pulp to surface. Modified with permission from Torneck CD.

![Figure 2-40](image)

Comparison of blood flows among various tissues and organs, adjusted according to weight. Pulpal blood flow is intermediate between muscle and heart blood flow. Reproduced with permission from Kim S. J Dent Res 1985;64:590.
This direct communication of dentin to systemic circulation was proved by Pashley, who demonstrated that radioactive iodide and albumin placed on dog dentin rapidly gave measurable blood levels of the substances. Systemic absorption of substances following pulp application was shown by Myers and colleagues, who measured the systemic appearance of 131I from pulpotomy sites in monkeys both before and after treatment of the pulp stumps with formocresol. Similar studies have recently been completed using 14C-formaldehyde and 14C-glutaraldehyde. Barnes and Langeland demonstrated that circulating antibodies were formed against bovine serum albumin and sheep erythrocytes placed on exposed pulps of monkeys. Thus, the pulp provides an access route not only to the systemic circulation but also to the lymphatic system.

Noyes and Ladd forced fluid into dog pulps and observed its collection in submaxillary lymph nodes. Kraintz and coworkers placed radioactive colloidal gold on dog dentin and found that it appeared in the lymphatic drainage. Walton and Langeland studied the distribution of zinc oxide and eugenol from pulpotomy sites in monkeys. Within days, the particles were distributed throughout the pulp and periapontium and appeared in the submandibular lymph nodes of the animals.

Feiglin and Reade deposited radioactive microspheres in rat pulps and found more microspheres in the submandibular lymph nodes in those rats whose pulps had been exposed for 5 days in comparison with those with acute pulp exposure. This suggests enhanced lymphatic function during inflammation.

The relationship of teeth to the cardiovascular and lymphatic systems is intimate and absolute. Clinicians should remember this when performing dental procedures since their placement of materials on dentin or pulp may result in widespread distribution of that material or medicament.

**HISTOLOGY OF THE PERIRADICULAR REGION**

At the periapex, the connective tissues of root canal, foramen, and periradicular zone form a tissue continuum that is inseparable. This intimate relationship is confirmed by the frequency of disease in the pulp, inciting disease beyond the tooth. When both the pulp and perirapex are jointly involved, immediate therapy must often focus on the periradicular region. More commonly, only pulp therapy is necessary. Healing of the periradicular tissue generally occurs spontaenously, demonstrating its capacity to repair. During preparation of the pulp space, the cardinal principles of instrumentation and obturation, aimed at confining everything to the canal space, indicate how necessary it is to respect the periradicular connective tissue.

The intimate communicative relationship of the structures at the periapex has been shown in experiments that traced substances placed in the coronal pulp to the periodontium. Markers migrated from the pulp and were observed in all areas of the periodontal ligament, the alveolar and medullary bone, and even in the marginal gingiva.

The periapex is the apical continuation of the periodontal ligament. Actually, the tissue at the immediate apex of the tooth is more akin to the content of the root canal than to the periodontal ligament. The concentration of nerves and vessels coursing into the pulp is such, in fact, that attachment fibers and bone normally associated with the ligament space are generally excluded. The radiographic appearance of the interruption in bone to permit passage of the neurovascular bundle must not be confused with the bone resorption that accompanies periradicular inflammation (Figure 2-42).

The connective tissue sheaths of vessel and nerve groups lie close together. It is small wonder that inflammatory change is found concentrated at this zone of vessel egress; the spread of inflammation occurs via the connective tissue sheaths of vessels as a pathway of spread.

Physiologically, as well as structurally, sharp contrasts set the periodontal ligament apparatus off from pulp tissue: (1) It is, for example, an organ of the finest tactile reception. The lightest contact on the tooth will stimulate its numerous pressor receptors. The pulp contains no such receptors. Proprioceptors of the periodontal ligament present the capability of spatial determination. It is for this reason that an inflamed periodontium can be more easily localized by the patient than can an inflamed pulp. (2) Collateral blood supply, so lacking within the pulp, is abundant in this area. This rich blood supply is undoubtedly a major factor in the periapex’s ability to resolve inflammatory disease. In contrast, the pulp often succumbs to inflammation because it lacks collateral vessels. (3) The apical periodontium communicates with extensive medullary spaces of alveolar bone. The fluids of inflammation and resultant pressures apparently diffuse through this region more readily than is possible in the confined pulp space.

Histologically, the periapex demonstrates the major features of the remaining periodontium. Collagenous fibers anchor cementum to alveolar bundle bone. The arrangement of bone and fibers is discontinuous where the neurovascular bundle passes through to the pulp. A significant component of the periodontal ligament at all levels is the cords of ectodermal cells derived from the
Figure 2-41  Experiment in monkeys showing time and spatial pattern of migration of material placed in contact with vital pulp. A, Human mandibular first and second molars after pulpotomy; application of a silver–zinc oxide–eugenol sealer and restoration with amalgam. Arrow indicates region shown histologically in B and C. B, Area of pulp canal indicated in A. Particles of silver and zinc oxide are visible in extracellular spaces (E) adjacent to vessels and intracellularly (I) within endothelial or perivascular cells. Material is also contained in venules (V) and in small vessels resembling capillaries, or in lymphatics (L). C, Same field as B; polarized light demonstrates particles seen in B, which are birefringent sealer particles. Reproduced with permission from Walton R and Langeland K.108
original root sheath, which form a tight network in this narrow zone between tooth and bone. These embryonic remnants, the epithelial rests of Malassez, may serve in a constructive capacity, and several such functions have been postulated. However, interest has focused on their potential to undergo rapid hyperplasia when stimulated by periradicular inflammation. As will be seen in chapter 5, it is these cells that provide the epithelial “seed” for the lining sheet of the apical cysts.

Beyond the ligament is the alveolar bone with its associated marrow. The transition from ligament space to marrow is made through the myriad perforations of the alveolar bone proper. This bone, at the periapex as much as on the lateral walls of the socket, is truly a cribriform plate. Interstitial connective tissue of the periodontal membrane passes through it, carrying vessels and nerves, to blend with the fatty marrow of the alveolar bone proper. This bone, at the periapex as much as on the lateral walls of the socket, is truly a cribriform plate. Interstitial connective tissue of the periodontal membrane passes through it, carrying vessels and nerves, to blend with the fatty marrow of the alveolar-supporting bone.

The potentials of this periradicular marrow are rich and significant. The reserve and other cells of the marrow contribute to nature’s débridement and repair in the diseased periradicular zone following adequate pulp therapy.

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