Fig. 9.17   **Collagen bundles in an older pulp organ.** Trauma may also have contributed to collagen in this pulp.

the pulp proper but are in small amounts and not well characterized.

Vascularity

The pulp organ is highly vascularized, with vessels arising from the external carotid arteries to the superior and inferior alveolar arteries. It drains by the same veins. Although the periodontal and pulpal vessels both originate from these vessels, their walls are different. The walls of the periodontal and pulpal vessels become quite thin as they enter the pulp, because the pulp is protected within a hard, unyielding container of dentin. These thin-walled arteries and arterioles enter the apical canal and pursue a direct route up the root pulp to the coronal area **(Fig. 9-18)**. Along the way, vessels produce branches that pass peripherally to a plexus that lies in and adjacent to the odontogenic zone of the root **(Fig. 9-19)**. Blood flow is more rapid in the pulp than in most areas of the body, and the blood pressure is quite high. The diameter of the arteries varies from 50 to 100 μm, which equals the size of arterioles in other areas of the body. These vessels have three layers: the inner lining, or **intima,** which consists of oval or squamous-shaped endothelial cells surrounded by a closely associated fibrillar basal lamina; a middle layer or **media,** which consists of muscle cells from one to three cell layers thick **(Fig. 9-20)**; and an outer layer, or **adventitia,** which consists of a sparse layer of collagen fibers forming a loose network around the larger arteries. Smaller arterioles with a single layer of muscle cells range from 20 to 30 μm, and **terminal arterioles** of 10 to 15 μm are also present. **Precapillaries** measuring 8 to 12 μm and **capillaries** measuring 8 to 10 μm in diameter are present in the peripheral pulp. Capillaries are endothelial cell–lined tubes that form a network among the odontoblasts (see Fig. 9-19). Numerous investigators have shown that lymphatic vessels
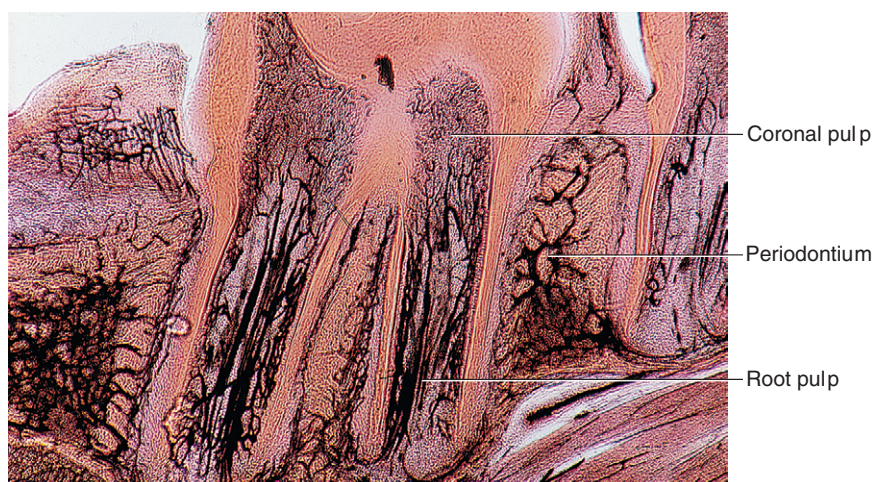

Fig. 9.18   **Vascular injection to illustrate blood vessel organization in pulp and periodontium.** Larger vessels conduct blood in the central pulp, and smaller capillaries are in the peripheral pulp.

Fig. 9.19 Vascular injection into blood vessels to illustrate the network of capillaries among odontoblasts in the odontogenic zone. Dentin, which protects pulp, is seen at the top of the picture. The central pulp is in the lower part of the micrograph. *(From Avery JK: Oral development and histology, ed 3, Stuttgart, 2002, Thieme Medical.)*
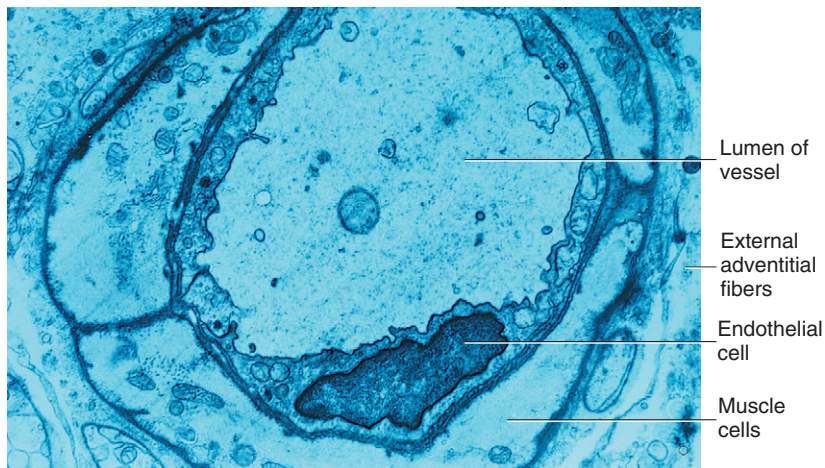
Fig. 9.20 Ultrastructure of a pulp arteriole in the central pulp area. The lumen is surrounded by endothelial cells; a nucleus is seen below. These cells compose the intima layer. Surrounding the intima is a layer of muscle cells that form the media. External adventitial fibers are also present. *(From Avery JK: Oral development and histology, ed 3, Stuttgart, 2002, Thieme Medical.)*

are present in pulp. These vessels are thin walled, irregularly shaped, and larger than capillaries and have an incomplete lamina supporting the intima and media.

Nerves
Several large nerves enter the apical canal of each molar and premolar, and single nerves enter the anterior teeth. These nerve trunks traverse the radicular pulp, proceed to the coronal area, and branch as they extend peripherally **(Fig. 9-21)**. Nonmyelinated axons also enter with the myelinated axons, but they are smaller. A young molar may have as many as 350 to 700 myelinated axons and 1000 to 2000 nonmyelinated axons entering the apex.

The large nerve trunks are invested with Schwann cells (see Figs. 9-14 and 9-15). Later, as the pulp organ matures, the subodontoblastic plexus is apparent in the roof and lateral walls of the coronal pulp and, to a lesser extent, the root canals. This network, comprising both myelinated and nonmyelinated axons, is known as the *parietal layer of nerves* or nerve **plexus of Raschkow** (see Fig. 9-21 and **Fig. 9-22**). From the parietal layer, the nerves lose their myelin sheath, pass into the odontogenic zone, and then terminate among the odontoblasts or extend into the dentinal tubules with the odontoblastic process.

> ### ◎ CLINICAL COMMENT
>
> The vitality of pulp results in part from the apical canal's ability to remain open. This opening can become blocked, however, as the tooth ages and cementum becomes deposited around the apical canal. Thin walls of veins are the first structure affected by cemental constriction of the apices; vascular congestion can occur, leading to pulpal necrosis.
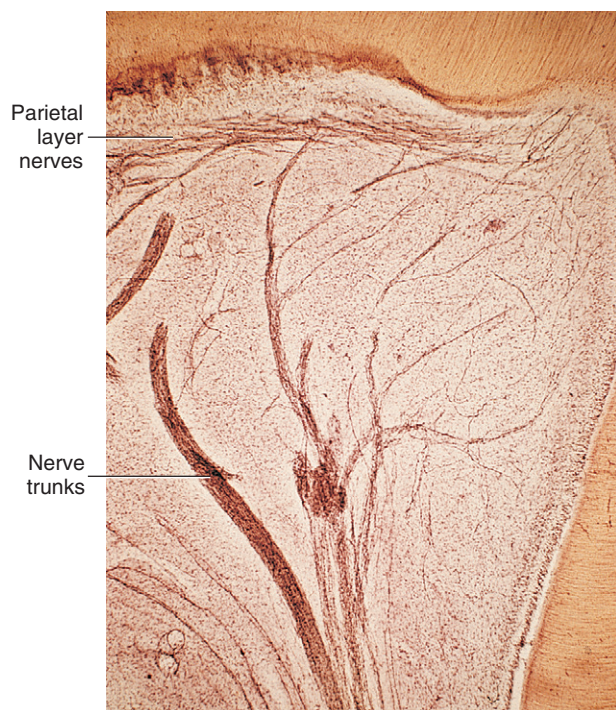
Fig. 9.21 Nerve trunks pass from the radicular pulp into the coronal area. These nerves extend to the periphery, where they form a plexus of nerves adjacent to the odontogenic zone above. *(From Bhaskar SN, editor: Orban's oral histology and embryology, ed 11, St. Louis, 1991, Mosby.)*

Nerve Endings

Most pulpal nerve endings are in the odontogenic region of the pulp horns. Some terminate on or in association with the odontoblasts (**Figs. 9-23** and **9-24**). Others are found in the predentinal tubules, usually in the region of pulp horns or roof of the coronal area (see Fig. 9-24). These nerve endings are presumed to function in pain reception. Few nerve endings are located along the larger muscular blood vessels in the central pulp. All these nerve endings have a similar appearance and are highly vascular. They are believed to function in regulation of blood flow, constriction, or dilation of large blood vessels of the pulp. These nerve endings are postganglionic sympathetic nerves with cell bodies located in the superior cervical ganglion.

PAIN AND THE PULP-DENTIN COMPLEX

Pain is a function of the high concentration of nerve endings within the tooth. Pulp is highly sensitive to temperature changes, electrical and chemical stimuli, and pressure as applied to the inner enamel, dentin, or pulp. Teeth are one of the few body structures that perceive only the modality of pain. The close relationship between nerve endings and the odontoblasts and their processes is significant. Moreover, the nerve endings in the dentinal tubules and the pulp may be some distance from where the pain is perceived, at the dentinoenamel junction and the inner enamel. Several theories attempt to explain this phenomenon.

The first theory is called the **direct innervation theory,** which is based on the belief that the nerves extend to the dentinoenamel junction. However, studies have not shown nerves present at this junction. In a second theory, other scientists believe the odontoblastic process is the receptor and that it conducts the pain to nerve endings in the peripheral pulp and in the dentinal tubules. This theory has been termed the **transduction theory** (**Fig. 9-25**).

A third theory, the **hydrodynamic theory,** was developed to explain the transmission of pain through the thickness of dentin (see Fig. 9-25). This theory is based on the premise that when dentin is stimulated, fluid and the odontoblastic process move within the tubules, making contact with the nerve endings in the inner dentin and adjacent pulp. When these nerve endings are contacted, they deform and act as mechanoreceptors to produce an impulse. Several factors support this theory. For example, when a stimulus such as cold is applied to
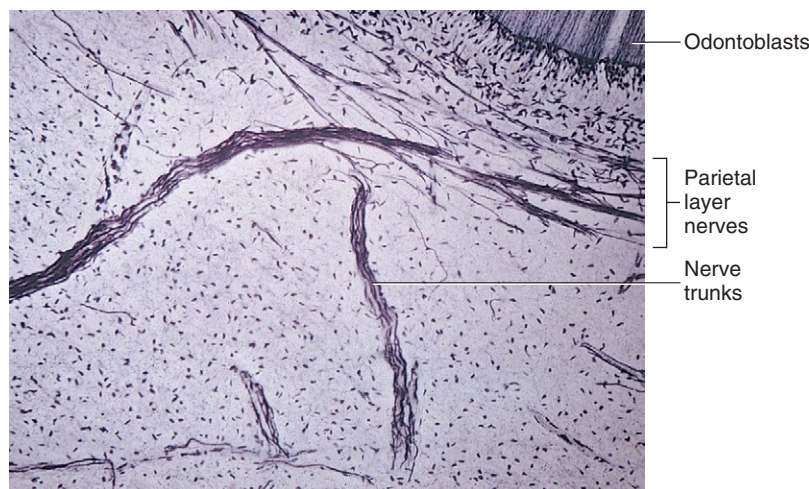

Fig. 9.22 Myelinated nerves extending into the parietal nerve plexus in the peripheral pulp. From this area, they extend between odontoblasts to terminate among them or in dentinal tubules. *(From Avery JK: Oral development and histology, ed 3, Stuttgart, 2002, Thieme Medical.)*
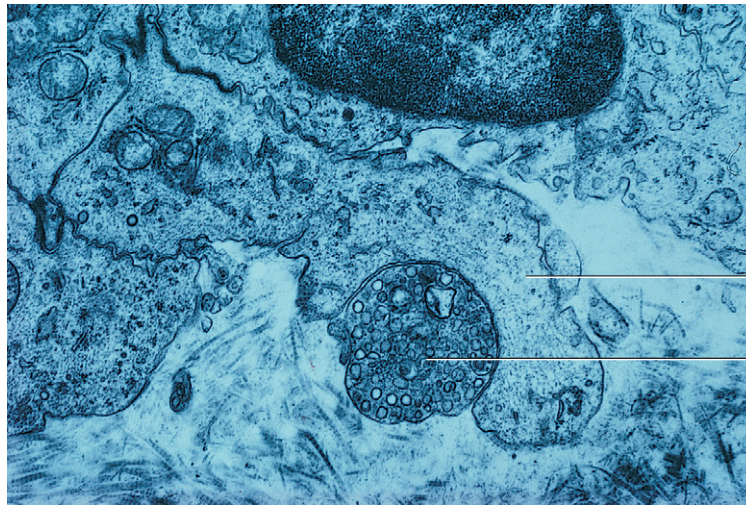
Fig. 9.23 **Ultrastructure of a nerve ending in close contact with the odontoblastic process in predentin.** The nerve contains small vesicles believed to contain a neurotransmitter substance. The nerve terminal interdigitates with the odontoblastic process. *(From Bhaskar SN, editor:* Orban's oral histology and embryology, *ed 11, St. Louis, 1991, Mosby.)*
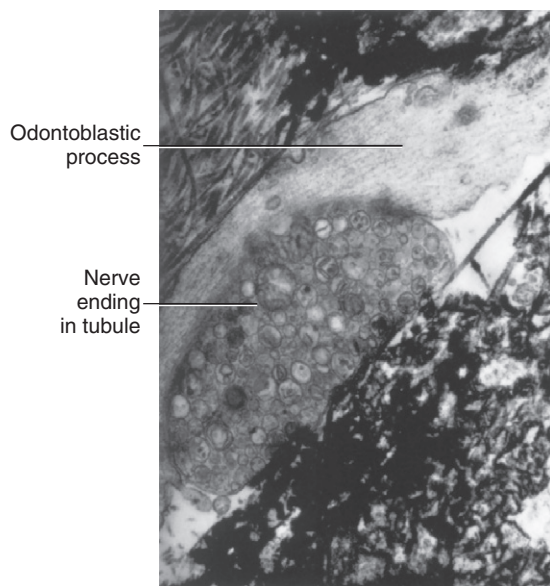
Fig. 9.24 **Vesiculated nerve terminal in a dentinal tubule making contact with odontoblastic process.** The nerve and process are close to each other. Dark-stained mineral of dentin is shown above and below the tubule.

the dentin, the odontoblastic process moves outward, but when heat is applied, the odontoblastic process moves inward. Other evidence is seen in the close relationship of the nerve endings and the odontoblastic process.

The odontoblast is a unique cell that forms dentin throughout life. It forms reactionary or response dentin, for example, in response to various stimuli. In addition, it plays a role in conducting stimuli through dentin and in affecting nerve endings in the peripheral pulp.

FUNCTIONS OF THE PULP

Pulp has several functions, none of which is more important than providing vitality to the teeth with its cells, blood vessels, and nerves. The loss of pulp after a root canal does not mean the tooth will be lost; on the contrary, the tooth will function without pain. The tooth, however, has lost the protective mechanism its pulp nerves provided.

Pulp has several other functions. It is **inductive,** because in early development the pulp (papilla) interacts with the oral epithelium and initiates tooth formation. Pulp organs are **formative,** because odontoblasts of the pulp form the dentin that surrounds and protects pulp. Pulp is **protective** in its response to stimuli, such as heat, cold, pressure, and operative cutting procedures. The formation of sclerotic dentin, the process of mineral deposition in the tubules, originates in pulp and protects pulp from invasion of bacteria and bacterial products. Pulp is **nutritive,** because it carries oxygen and nutrition to the developing and functioning tooth. Finally, pulp has the ability to be **reparative** (**Fig. 9-26**) through its response to operative cutting or dental caries by the formation of reactionary and reparative dentin.

REGRESSIVE CHANGES

Numerous regressive changes in the pulp and surrounding dentin are related to environmental stimuli and to aging. It is

> ◎ **CLINICAL COMMENT**
>
> A cracked tooth may result from masticatory impact on a hard object. It can cause a fracture of a restoration margin. As a result, bacterial organisms or their toxins may penetrate the tooth and cause inflammation of the pulp, pain, and eventually pulpal pathosis.
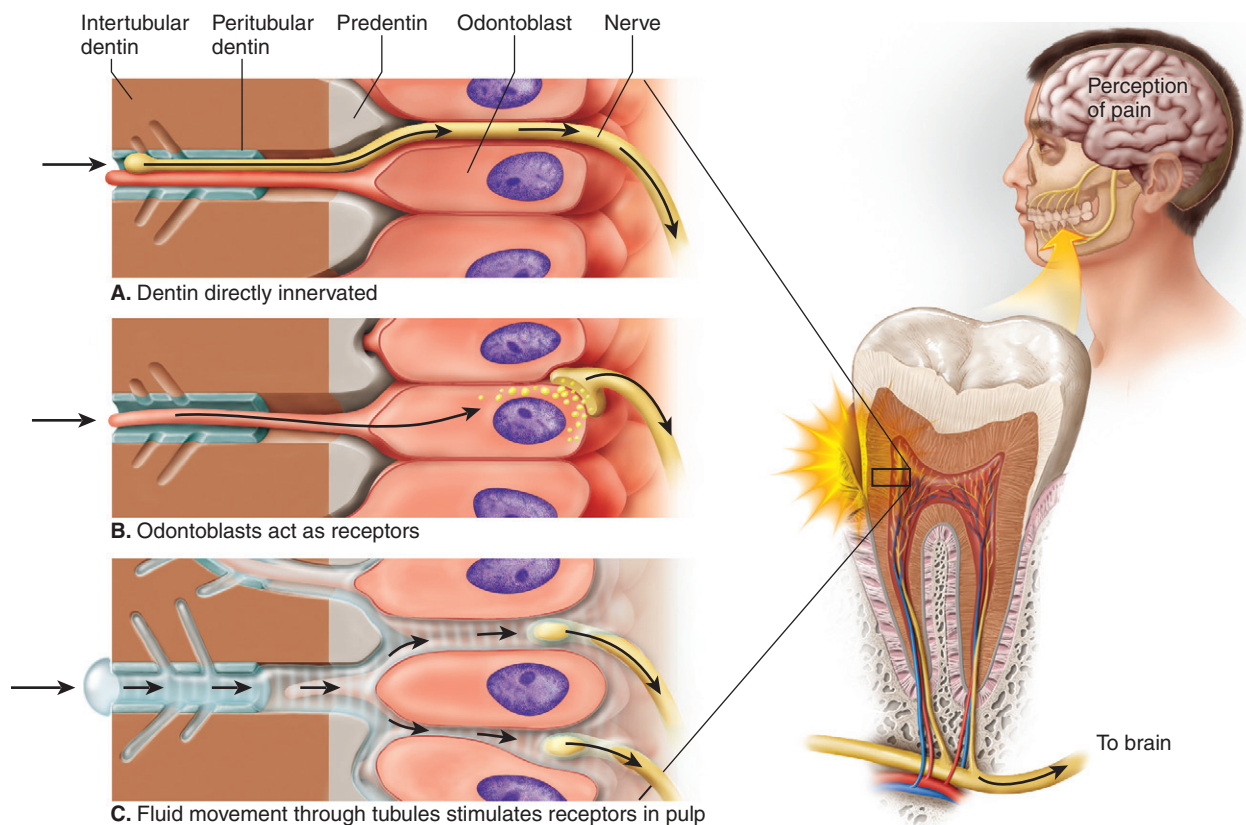
Fig. 9.25 **Summary of theories on the passage of nerve impulses through dentin.** At the top *(A),* impulses are shown stimulating nerves in dentin; this is termed the *direct stimulation theory.* In the center *(B),* an odontoblast is depicted as receptor passing impulses to nerves in the peripheral pulp and hence on to the brain, which is the transduction theory. At the bottom *(C),* the diagram displays concept of fluid and odontoblast movement. This movement causes pressure on the nerve endings, which stimulates them. The odontoblast thus acts as a mechanoreceptor to nerve endings, which, in turn, conduct impulses to the brain. This is termed the *hydrodynamic theory.* *(From Nanci A: Ten Cate's oral histology, ed 8, St. Louis, 2013, Mosby.)*

often difficult to determine which factor has caused the specific change seen. As the tooth ages, pulp decreases in size because of the continued deposition of dentin. This decrease in size usually occurs because of uniform deposition around the entire perimeter of the pulpal border **(Fig. 9-27)**. In addition, changes occur in the dentin with both aging and injury. Areas of dentinal changes, such as dead tracts and mineral deposits, appear in zones of trauma. Reparative dentin usually forms under traumatized areas (see Fig. 9-27). In addition, as a result of both aging and trauma, pulpal cells decrease in general, as do cellular perinuclear cytoplasm and organelles in the cytoplasm, such as mitochondria and endoplasmic reticulum. This indicates that cell activity has decreased. Therefore, aging decreases the ability of the pulp to respond to injury and to repair itself. With injury, however, deposition of dentin appears in a specific location (see Fig. 9-27).

Fibrous Changes

Fibrosis, which is seen in some pulps more than others, is believed to be caused more by injury than by aging. In some cases, diffuse fibrosis with collagen fibers appears throughout the pulp. Occasionally, the fibers nearly obliterate the pulp. What mechanism causes this condition is not certain, although it is believed to result from pulpal injury, at least in part. Scarring caused by injury is an important factor. One characteristic of aging is an increase in collagen fibers, which become more evident with the decreasing size of the pulp (see Fig. 9-17). Some pulps contain diffuse areas of collagen, and others have bundles of them probably because of injury, as well as unknown systemic factors.
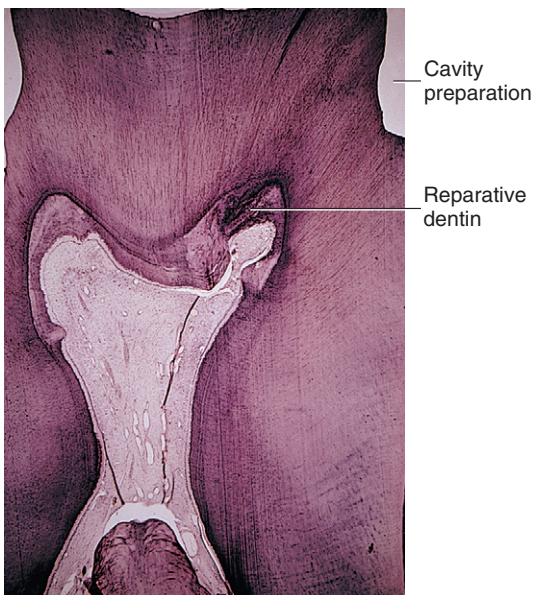
Pulp Stones

Pulp stones or **denticles** are round to oval calcified masses appearing in either the canal or coronal portions of the pulp organ **(Fig. 9-28)**. They appear in teeth that have suffered injury such as microtrauma, as well as in otherwise normal pulps. Pulp stones also occur in unerupted, as well as erupted, teeth. These denticles are noted in most pulps of permanent teeth, especially in individuals more than 50 years of age. They are classified according to their structure as true or false. **True denticles** have dental tubules like dentin. Odontoblasts may be on the surface of these denticles, and their processes are evident in their tubules. **False denticles** are concentric layers of calcified tissue **(Fig. 9-29)**. In the center of these false stones may be a group of cells that appear necrotic. These cells are believed to serve as the nidus of denticle formation.
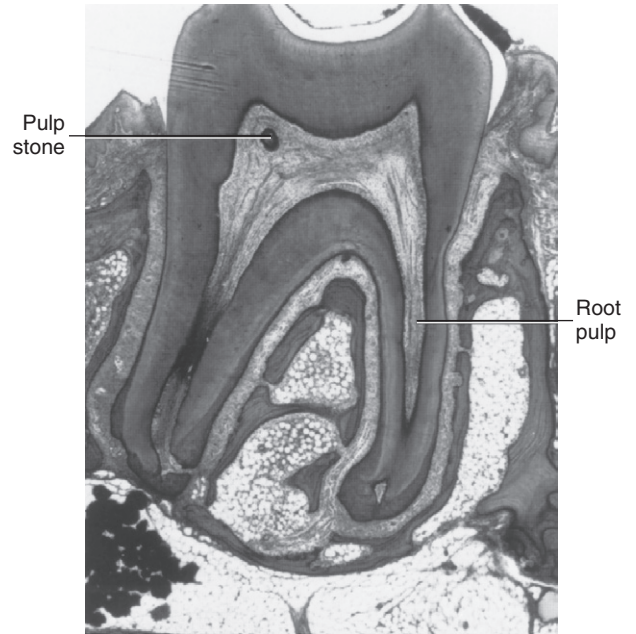
All denticles begin small and grow, sometimes nearly obliterating the pulp. Denticles may appear free in pulp, attached
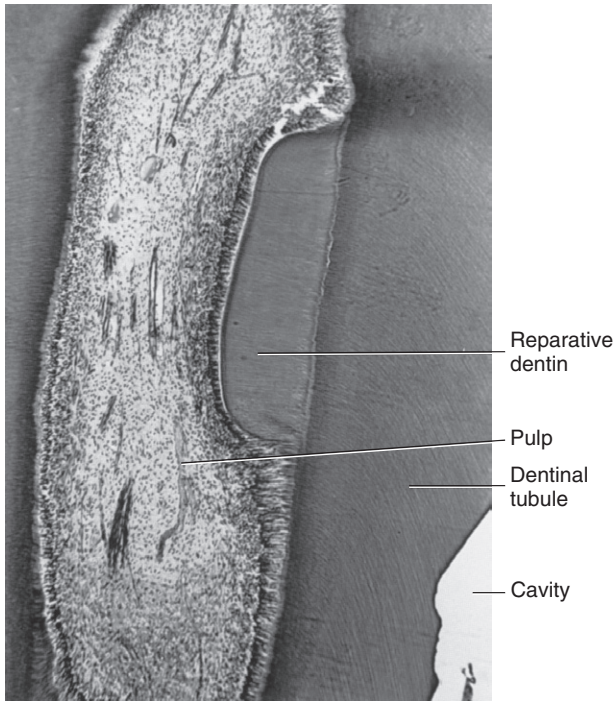
Fig. 9.26   Reparative dentin is deposited underlying areas of stimulation by caries, abrasion, cavity preparation, and restorations. It is limited to area underlying dental tubules leading from the cavity floor. *(From Avery JK: Oral development and histology, ed 3, Stuttgart, 2002, Thieme Medical.)*

Fig. 9.28   Fibrous changes and pulp stone in coronal pulp. A pulp stone appears in the coronal area of a molar tooth.

Fig. 9.27   Reparative dentin underlying cavity preparation on the mesial-occlusal-distal aspects of a crown. Reparative dentin on roof and sides of coronal pulp chamber underlie cut dentinal tubules that lead from cavity preparation. *(From Avery JK: Oral development and histology, ed 3, Stuttgart, 2002, Thieme Medical.)*
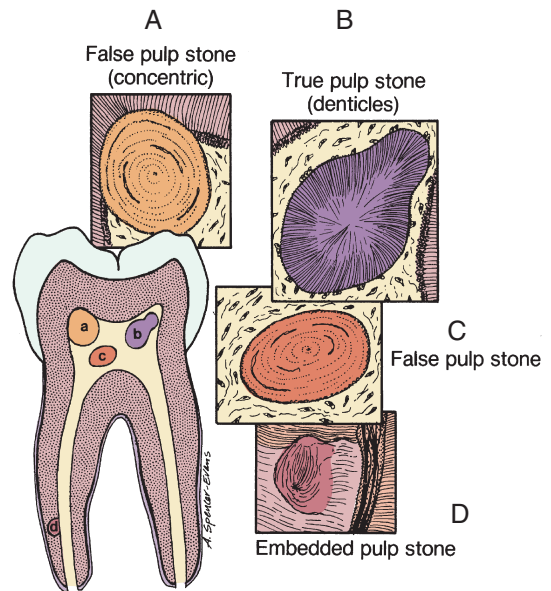
Fig. 9.29   Diagram of types of pulp stones. A, False attached denticle. **B,** True denticle with tubules. **C,** False free denticle. **D,** Embedded denticle.

to dentin, or embedded in dentin. Therefore, they are classified as **free, attached,** or **embedded denticles.** One pulp may have all three types (see Fig. 9-29). Investigators believe that a free denticle may become attached and later embedded as dentin is deposited around the denticle. Most denticles are false stones that are free in the pulp.

Diffuse Calcifications

Diffuse calcifications appear as irregular calcified deposits along collagen fiber bundles or blood vessels in the pulp. This is considered a pathologic condition and usually appears as a sprinkling of small or occasionally large masses of mineral. These calcifications appear more often in the root canal than in the coronal area of the pulp.

Self-Evaluation Questions

1. Describe the characteristics of the odontogenic zone.
2. Compare the odontoblast in coronal pulp with the odontoblast in root pulp.
3. What are the most prominent cells of pulp, and what are their functions?
4. What are five other cell types found in normal pulp?
5. Describe the various blood vessels of pulp and how they differ from blood vessels of the periodontium.
6. Give descriptions and locations of nerve endings in pulp.
7. Name five functions of pulp.
8. Name and describe various types of denticles.
9. What are the types of junctional complexes found between odontoblasts?
10. Name and describe the types of reparative dentin.

◎ CLINICAL COMMENT

Pulp stones begin to develop as early as functional occlusion. They normally are asymptomatic unless they impinge on blood vessels or nerves and usually do not present a problem to the dentist. Pulp stones are thought to be a result of microtrauma to the pulp resulting in ectopic calcifications.

✠ CONSIDER THE PATIENT

Discussion: This condition can be caused by internal resorption of the root and crown dentin. The crown appears pink because the transparent enamel reveals the blood vessels in the pulp.

★ QUANDARIES IN SCIENCE

The dental pulp contains a heterogeneous population of cells that maintain the pulp proper and the dentin protecting this sensitive organ. The dense innervation of the pulp belies the importance of this tissue in maintaining the overall health of the body, as does the number of reserve cells (various types of stem cells) contained within the stroma of the matrix. Because scientists do not fully understand the biology of the pulp, it is interesting to speculate on why it is so densely innervated with sensory nerves that contain many different types of neurotransmitters that are usually co-localized with other neurotransmitters within discrete nerve terminals located in the dentinal tubules, around the odontoblasts, and within the pulp proper. Do the different conformations of nerve terminals and different neurotransmitters function only during the transmission or modulation of nociception, or do they have other roles such as modifying the responsiveness of the odontoblasts to iatrogenic or environmental insult or in the recruitment of replacement odontoblasts? The varieties of nerves, nerve terminals, and putative neurotransmitters that have been reported in the mature dental pulp have raised many questions relative to their functional significance. Evidence suggests that pulpal neurons have a significant role in modulating wound healing by releasing substances that can upregulate protein synthesis and modulate inflammation. Could changes in odontoblastic activity be mediated by neuropeptides, neuromodulators, and/or neurotransmitters released by sensory nerves that maintain the homeostatic balance of the pulp-dentin complex and affect how this balance is restored after injury?

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