Table 1.1	Evolution of Endodontics (*Contd...*)
1990	Carlsen and Alexandersen described Radix entomolaris
1993	Torabinejad developed mineral trioxide aggregate (MTA)
1993	Lussi, Nussbacher Grosrey described non-instrumentation technique
1993	APIT/Endex (third gen Electronic apex locator) developed by Frank and Torabinejad
1994	Ben Johnson introduced Profile instruments
1995	McSpadden introduced Quantec instruments
1996	Buchanan introduced Hand Greater taper files
1996	Hoshino and Colleagues introduced Triple antibiotic paste
1999	Endox system was introduced by Haffner and colleagues
1999	Bingo 1020 (fourth generation apex locator)
2000	Edward Lynch and Aylin Bayson developed Healozone
2000	Arias introduced electrochemically activated water as irrigation solution
2001	Clifford Ruddle and team developed protaper system in co-operation with Dentsply
2001	Micro-mega developed Hero Shapers
2002	John McSpadden introduced K3 Endo
2003	Jai and Albert introduced Resilon as an obturating material
2003	Torabinejad introduced irrigant MTAD (4.25% citric acid, 3% doxycycline, 0.5% Tween 80)
2004	Dr Goodis introduced V-taper
2005	Zehnder introduced irrigant HEBP (7% 1-Hydroxyethylidene-1,1-bisphosphonate)
2005	Liberator was introduced by Miltex
2006	Protaper Universal system was introduced
2006	Fukumoto introduced intracanal aspiration technique
2006	Koch and Brave introduced Activ GP
2007	Franklin introduced Monobloc concept
2007	Endovac was introduced by Hoafs and D Edson
2007	Tulsa dental specialities introduced M wire technology
2007	Greater taper series X was introduced by Dr S Buchnan
2008	Dr Richard Mounce introduced Twisted file
2008	Ghassan Yared introduced a concept of canal preparation using only one NiTi rotary instruments
2009	Pierre Machtou, Bob Sharp and Cliff Ruddle introduced endoactivator irrigation system
2009	George Eliades introduced vibringe irrigation system
2009	Endosequence post preparation technique was developed by Dr Ali Nasseh
2010	Redent Nova introduced self adjusting file
2010	Ghasson Yared introduced Reciproc
2011	Dentsply introduced Wave One
2012	FKG Dentaire introduced RaCe
2012	Coltene-Whaledent introduced Hyflex-CM
2012	Micro-Mega introduced Revo-S file
2013	Micro-Mega introduced One shape
2013	Dentsply introduced ProTaper Next
2014	Neolix France introduced NeoNiTi file system
2015	Coltene-Whaledent introduced Canal Pro Cr-2 Endomotor with reverse motion
2015	File retrieval kit by Dr Yoshi Teruachi
2016	Autosyringe introduced by vista dental
2017	Carl Zeiss introduced Civil Zeiss extara 300

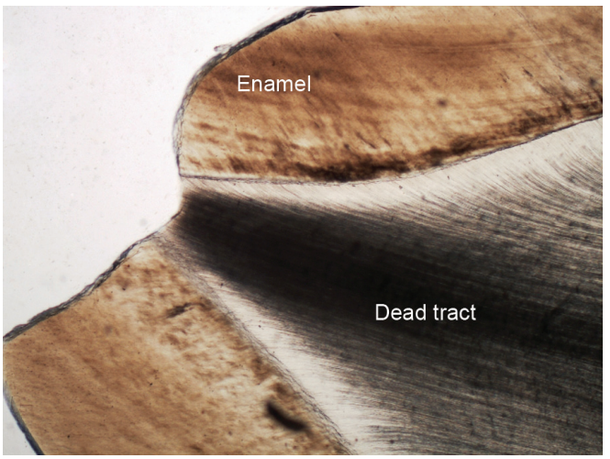

Fig. 2.5 Dead tracts in a ground section of a tooth. Under transmitted illumination the tracts appear dark because air in them refracts the light

from 45000–55000 cells/mm^2. The cell body of odontoblasts is located on the pulp side of dentin and cytoplasmic processes are inserted into tubules of mineralized dentin. A large nucleus is located in the basal portion of the cell, close to the pulp along with Golgi apparatus, mitochondria and endoplasmic reticulum. The cell body is 20–40 µ tall and 3.0–5.0 µ wide, depending upon the dentinogenic activity. They produce the organic matrix of pre-dentin and dentin including Type I collagen and proteoglycans. Odontoblasts intracellularly transport calcium ions to the mineralization front, and have the capacity to degrade organic matrix.

The cytoplasmic feature of the odontoblasts varies according to the functional activity of the cell. Odontoblasts have all the characteristic organelles, which are associated with protein and proteoglycans production. The activity of odontoblasts is reflected in the number and types of organelles present in the cytoplasm.

The adjacent odontoblasts are attached together with extension junctional complexes and with gap junction. The junctional complex consists of tight junction, known as zonula occludens. These tight junctions are thought to contribute in the final polarization of odontoblasts. Unlike other polarized cells, e.g. epithelial cells, the tight junctions in odontoblasts may be related to cell differentiation than to permeability of cell layers. The interodontoblastic collagen fibers (von Korff's fibers) may pass through the odontoblast-predentin layer from pulp into dentin.

iv. *Odontoblastic processes*: The odontoblastic processes are the cytoplasmic extension, gradually narrowing, that penetrates into mineralized dentin, falling the lumen of dentinal tubules. The odontoblastic process lacks cell organelle, but contains secretory vacuoles and microtubules. Its diameter is 3–4 µ at the pulp predentin border and gradually narrows as it passes within the dentinal tubules.

The depth of penetration of cytoplasmic processes is controversial. Various studies have observed different depth of penetration. It is hypothesized that in developing young teeth, the processes might be up to dentin-enamel junction, while in other teeth, the processes may only penetrate up to 0.3 mm into coronal dentin (may be slightly more in root dentin).

It is hypothesized that the cytoplasmic process extends about one-third of the distance from predentin to enamel, indicating that vital tissue changes occur only in the inner-third coronal dentin towards pulp. Changes occurring in the outer two-thirds of the dentin may be of physiochemical nature by precipitation of mineral salts within the tubules or growth of the peritubular dentin via components secreted into the periodontoblastic space. These components may diffuse peripherally to form a matrix that will mineralize.

v. *Dentinal fluid*: It has been hypothesized that a transport mechanism exists between blood circulation and dentin. It is suggested that odontoblasts regulate the transport of substances into dentin. Dentin contains several serum proteins, mainly in dentinal tubules. The origin of the fluid in dentinal tubules (either from pulp tissue extracellular space or from blood vessel or both) may not be clear, but the fluid present, containing inorganic and organic constituents, is a significant component of pulp-dentin complex. The space between odontoblast process and the tubule wall is filled with fluid, commonly referred to as '*dentinal fluid*'. The dentinal fluid is under control of odontoblasts, until tissue is damaged because of caries, cavity preparation, etc. It is established that the odontoblastic cell layer forms a functional barrier that restrains the passage of fluid, ions and other molecules along the extracellular pathway.

It has been accepted that dentin sensitivity to extrinsic irritants is mediated through alteration in hydraulic conductance of the dentinal fluid. The accepted hydrodynamic theory implicate fluid movement through dentinal tubules as a transducing mechanism, explaining dentin sensitivity (dentin is more sensitive to outward than inward flow).

Adhesive restorations are also dependent on the dentinal fluid. Increased dentinal wetness and fluid

i. *Zone of odontoblasts*: A layer of odontoblasts, circumscribes the outermost part of the pulp. They form a single layer lining in the most peripheral portion of the pulp. The cytoplasmic extension (odontoblastic process) extends into dentinal tubules. The shape of the cell body of odontoblasts is not uniform, rather these cells are tall and columnar in the coronal pulp, short and columnar in the mid portion of the tooth and cuboidal to flat in root portion. The *primary function* of odontoblasts is to produce and deposit dentin.

ii. *Zone of Weil (cell-free zone)*: Subjacent to odontoblast layer, an area relatively free of cells, known as 'cell-free zone' or 'zone of Weil' is seen. Major constituents of this zone are the rich network of mostly unmyelinated nerve fibers, blood capillaries and few fibroblasts. The function of capillaries is to provide nutrition to the odontoblasts, especially during dentinogenesis; the nerve plexus are involved in the neural sensation of pulp.

iii. *Cell-rich zone*: Next is the cell-rich zone. The constituents of this zone are ground substance, fibroblasts, undifferentiated mesenchymal cells, defense cells (macrophages and lymphocytes), blood capillaries and nerves. The higher density of fibroblasts in this zone is much more prominent in the coronal pulp than in root pulp. The *functions* of cell-rich zone are:

- Fibroblasts form as well as degenerate collagen fibers
- By depositing calcified tissues, they help in reparative dentin formation
- Collagen fibers secreted by odontoblasts form the dentinal matrix; whereas; collagen fibers secreted by fibroblasts support the pulp.
- Ground substance acts as barrier against the spread of bacteria. It acts as transport medium for metabolites and other waste products.
- Collagen fibers collectively protect the neurovascular bundle, especially in the apical third area.
- Undifferentiated mesenchymal cells may differentiate into other cells during repair and regeneration, as per need.

iv. *Pulp proper*: From the cell-rich zone inward is the central connective tissue mass known as pulp proper. This zone contains fibroblasts, larger blood vessels and nerves. Undifferentiated mesenchymal cells and defense cells such as macrophages are frequently located in perivascular area. Collagen fiber bundles are more numerous in root pulp than coronal pulp.

The clinical implication of this higher density of collagen fiber bundles in apical region is the use of barbed broach during removal of pulp. The removal of pulp tissue is facilitated when the broach is passively placed apically engaging these collagen bundles.

The ground substance in pulp, the extracellular matrix, is consisted of polysaccharides, glycoproteins and proteoglycans. The proteins bind to cell surfaces and other matrix molecules. The water content is approximately 90%, which may decrease in aged pulp.

The blood vessels and nerves are embedded in the pulp matrix in the central portion of pulp, emanating branching to the periphery of the pulp. The nerve fibers enter through the apical foramen; whereas, lymphatics and venules exit the pulp mainly through apical foramen.

Components of Pulp

a. Cells

The main cells of pulp are:

i. *Odontoblasts*: Odontoblasts are also part of pulp; the highly differentiated cells of the pulp. The cell body of odontoblasts is located on the pulp side of dentin and cytoplasmic processes are inserted into tubules of mineralized dentin. The cell structure of odontoblasts is described under 'components of dentin'.

ii. *Fibroblasts*: These are the most numerous connective tissue cells with the capacity to synthesize and maintain connective tissue matrix. Synthesis of collagen is a main function of fibroblasts in the pulp. Fibroblasts are also responsible for synthesis and secretion of a wide range of non-collagenous extracellular matrix components such as proteoglycans and glycoproteins (fibronectin). In addition to synthetic activity, fibroblasts are also involved in the degradation of extracellular matrix. Fibroblasts are able to phagocytose collagen fibrils and digest them intracellularly by lysosomal enzymes.

iii. *Undifferentiated mesenchymal cells*: These are distributed throughout the cell-rich zone occupying the perivascular area. These cells appear as stellate shaped cells with a relatively high nucleus to cytoplasmic ratio. When stimulated, they may give rise to fibroblasts or odontoblasts as per need (also known as reserve cells). In older pulps, the number of undifferentiated mesenchymal cells may diminish, which reduces the regenerative potential of pulp.

iv. *Immunocompetent cells*: The ability of connective tissue to generate local inflammatory and immune reactions makes it an active participant in host defense. This capacity depends on immune-competent cells, which include lymphocyte, macrophages and dendritic cells. They increase in number during inflammation and may play role in repair process in the pulp.

permeability. The increased permeability allows odontoblasts to get affected by toxic bacterial products. The early inflammatory reaction is containment of bacterial growth within the pulp due to phagocytosis by neutrophils but at the expense of pulp tissue. At this stage, since the carious lesion has not penetrated into the pulp, removal and restoration of the lesion will allow the pulp to heal. Certain chemical mediators (bradykinin, serotonin and substance P) sensitize the A-delta fibers and lower their threshold, thereby increasing their response to stimulants.

It is established that the inflammatory mediators, capable of causing pain, are released in the pulp in direct proportion to the insult. Serotonin (5 HT), able to sensitize intra-dental A fibers and Bradykinin have shown to be in significant higher concentration in irreversibly inflamed human pulps. It has also been demonstrated that neuropeptides from the nociceptive nerve fibers present in the pulp [calcitonin gene-related peptide (CGRP), Neurokinin A and substance P] are found in significantly higher concentrations in symptomatic pulp compared with healthy pulps. Sharp pain due to external stimulus like cold which gets relieved after the stimulus is removed, is associated with the pulpodentinal myelinated A-delta neurofibres.

b. Irreversible Pulpitis

The classic symptom of irreversible pulpitis is lingering pain induced by thermal stimuli. Even mild temperature changes (tap water, breathing cold air) may induce pain. The initial reaction is a very sharp pain to hot or cold stimuli, which lingers for a couple of minutes to hours even after the stimulus is removed. The lingering pain is usually a dull ache or a throbbing pain. Spontaneous (unprovoked) pain, which may wake the patient at night and may become worse when lying down, is another hallmark feature of irreversible pulpitis. The more C-fiber-mediated pain (dull, throbbing, poorly localized), the more severe the inflammation and more likely to be irreversible in nature. Patients with irreversible pulpitis may have difficulty locating the precise tooth (source of pain). They may even confuse the maxillary and mandibular arches (but not the left and right sides of the mouth) because of the extensive branching of dental nerve axons and perhaps fewer proprioceptive fibres in the pulp.

Patients may complain of a dull continuous pain, exacerbated by heat but relieved by cold. Exact mechanism is not clear; however, it is speculated that micro-organisms in the infected pulp produce gases, which contract with cold water, relieving pressure on the nerve endings, particularly in the apical portion of the canal. The relief with cold water is temporary and the pain returns as soon as the tooth warms up and the gases expand. It is hypothesized that the apical part of the pulp nerves are the last to necrose.

When the carious lesion penetrates through the dentin and contacts the pulp, the nature of inflammatory response changes from a collection of mostly mononuclear leukocytes to a localized collection of polymorphonuclear leukocytes forming micro-abscesses within the chronic inflammatory lesion. It is at this stage that reversible pulpitis becomes irreversible. Eventually, initial microabscess leads to numerous other microabscesses. When they are large enough to coalesce, the pulp undergoes liquefaction necrosis.

A few authors suggested that inflammation alone would not cause pulp death. The injured pulp often becomes necrotic even when the injury is minor and intrapulpal pressure rises very high (up to 80 mm Hg). Others opined that the pulp pressure rises initially after injury but declines to its normal level and the pulp may recover.

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 reduced pressure effect occurs as the head in normal upright posture, is above the heart (gravity keeps the pressure low). When the patient lies down, the gravitational effect disappears, significantly increasing the pulp pressure, over and above that caused by endogenous mediators of inflammation. In the supine position, a higher tissue pressure develops in the pulp, which causes more pain. 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.

Irreversible pulpitis is difficult to diagnose until periradicular tissues are involved. As the periradicular tissues get involved, the tooth becomes sensitive to percussion and the affected tooth can be identified.

c. Pulp Necrosis

The pulp inflammation leads to necrosis followed by infection and eventually loss of tissue because of its ingestion by bacteria. It has been reported that in 50–60% cases, pulps can progress from vitality to necrosis without pain. This phenomenon has been termed as 'painless pulpitis'. It is not known how