Orofacial pain is a major public health problem. This fact was recently emphasized by a report from the US National Center for Health Statistics (NCHS). Although their figures and estimates apply only to the United States, they may generally be extrapolated worldwide.

The NCHS interviewed “45,711 households in the US civilian population.” This was the 1989 National Health Interview Survey (NHIS). Using the statistics developed from this survey, the National Institute of Dental Research reported in 1993 that “about 39 million people or about 22% of the U.S. population 18 years of age or older are estimated to have experienced at least one of five types of orofacial pain more than once during the past six months.”

This alarming cohort of 39 million can be further broken down into 22 million toothaches (12.2%), 15 million oral sores (8.4%), 9.5 million “jaw joint” pains (1.4%), and 1.3 million burning mouth pains (0.7%). The total adds up to more than 39 million because some respondents suffered from more than one type of orofacial pain. Of the 45,711 households interviewed, 9,072 people reported orofacial pain. These figures, extrapolated nationwide, may actually be too low because the Armed Forces, institutions (prisons, etc), and children under age 18 were not included in the statistics.

The shocking revelation that 22 million people in the United States suffer from a toothache within a 6-month period is an overall prevalence rate of 12,261 persons per 100,000 population. The prevalence for African Americans (14,584) and Hispanics (14,226) was even higher.

By all odds, the most frequently seen “pain” patient will be experiencing acute, true intraoral pain, toothache and its sequelae being the most common. Pain accompanying intraoral lesions and infections is the next most commonly seen. After that, the field thins somewhat, with top priority going to the acute pains of everyday general, endodontic, and oral surgery practice.

Less commonly seen, but a good deal more baffling, are the chronic pains found in and around the mouth, a number of them referred there from faraway sites. These are the craniofacial pains to be discussed in detail in Chapter 8. Because these two types of oral and perioral pain are so different in diagnosis and management, they will be dealt with separately, even though there is often a confusing overlap.

The shocking revelation that 22 million people in the United States suffer from a toothache within a 6-month period is an overall prevalence rate of 12,261 persons per 100,000 population. The prevalence for African Americans (14,584) and Hispanics (14,226) was even higher.

Solving these problems is rarely rewarding financially, but they are the few moments we as a profession enjoy that set us apart from the lay public. These problems also try one’s patience and ingenuity; only the skilled are successful in diagnosing and managing the really difficult cases.
Diagnosis is a personal and cognitive experience; therefore, many of the qualities of a good diagnostician are of an interpersonal nature and based on knowledge, experience, and diagnostic tools. Diagnosing orofacial disease is similar to medical diagnosis. The pulp test, radiographs, percussion, palpation, and other tests and procedures can facilitate the diagnosing of facial disease, just as the electrocardiograph, electroencephalograph, liver and kidney function tests, echocardiograph, computed axial tomographic scanners, and a host of other radiographs can facilitate medical diagnosis.

REQUIREMENTS OF A DIAGNOSTICIAN
A dentist can develop a number of assets to become a successful diagnostician. Again, the most important of these are knowledge, interest, intuition, curiosity, and patience. The successful diagnostician must also have acute senses and the necessary tools for diagnosis. For a detailed discussion of these assets, see Chapter 6.

HISTORY, EXAMINATION, DIAGNOSTIC TESTS, AND CONSULTATIONS
The important steps leading to a diagnosis and establishing a plan of treatment have been dealt with in depth in the previous chapter. Additional information, necessary to establish a diagnosis in chronic extracranial and intracranial pain complexes, will be detailed in the next chapter dealing with craniofacial pain.

PULP PAIN
Pulp pain, or pulpalgia, is by far the most commonly experienced pain in and near the oral cavity and may be classified according to the degree of severity and the pathologic process present:

1. Hyperreactive pulpalgia
   a. Dentinal hypersensitivity
   b. Hyperemia
2. Acute pulpalgia
   a. Incipient
   b. Moderate
   c. Advanced
3. Chronic pulpalgia
   a. Barodontalgia
4. Hyperplastic pulpitis
5. Necrotic pulp
6. Internal resorption
7. Traumatic occlusion
8. Incomplete fracture

The mildest pulp discomfort, experienced when no inflammation is present, is hyperreactive pulpalgia.

Hyperreactive Pulpalgia
Hyperreactive pulpalgia is characterized by a short, sharp, shock—that is, “pain” best described as a sensation of sudden shock. The sensation is as sharp as it is sudden and must be elicited by some exciting factor. It is never spontaneous. The pain is of short duration, lasting only slightly longer than the time during which the irritating element is in contact with the tooth. In some manner, the odontoblastic cellular body within the dentin must be excited by a noxious stimulus, either hot or cold, sweet or sour, or touch. Excitation of the odontoblast conducts the excitation to the pulp nerves. These “dentinal receptors have the characteristics of slow adaptation.”

It is difficult to explain to a patient that the severe pain experienced when eating ice cream—a blinding pain that extends upward through the eye and into the forehead—is really normal and not pathologic. In “lay terms,” one can only tell the patient that the cold “excites” the nerve in the tooth, and the pain is so severe it is referred upward through the eye.

The dentist, however, requires a more sophisticated explanation. Although this is hard to come by, the best explanation revolves around thinking of the fluid in the dentinal tubules, along with the odontoblast cells, as a “pump”—a hydrodynamic theory, if you will, that proposes that the fluid moves back and forth to stretch, compress, and excite the pulp nerves. Bränström pointed out that “the displacement of tubule contents, if the movement occurs rapidly enough, may produce deformation of nerve fibers in the pulp or predentin or damage to the cells; both of these effects may be capable of producing pain.” Such a mechanical transmission of the stimulus would account for the hitherto inexplicably great sensitivity of the dentin to pain, in spite of the apparent absence of nerve fibers in this tissue.

Bränström further confirmed the damage and pain generated by blowing air over exposed dentin. A short air blast evaporates from 0.1 to 0.3 mm of fluid from the dentinal tubule. This results in immediate capillary fluid replacement from the pulp’s blood supply, sucking the odontoblasts and nerve fibers up into the tubule. The nerves are stretched or even torn off, eliciting pain (Figure 7-1).

On continued exposure to an air blast, however, a plug of fluid protein builds up in the tubule, preventing fluid outflow. This plug “closes the pump” and leads to dentin insensitivity. When water is applied to the dentin surface, however, the plug “melts” and sensitivity returns (see Figure 7-1).
The same phenomenon is produced by the dental drill, the frictional heat and surface pressure displacing the tubule fluid and causing pain. Scraping or chiseling the dentin produces similar pressure and pain. Pulp pain produced by cold is a similar phenomenon. When cold is applied, the fluid in the tubules contracts, thus redirecting the fluid volume. Fluid contraction within the tubules again produces fluid outflow owing to the normal pulp pressure, and the nerves are once again stretched into the tubules along with the odontoblasts (Figure 7-2). Beveridge measured a fall in intrapulp pressure when cold was applied to a tooth (Figure 7-3). Researchers in Israel found this to be an “interstitial” pressure fall, whereas the “arterial transmural” pressure rose.

Hyperreactive pulpalgia owing to the application of heat is more easily explained. Again, Beveridge easily demonstrated a true increase in intrapulp pressure when heat was applied to the tooth (Figure 7-4). An increase of pressure within the pulp serves to excite the sensory pulp nerves.

Three different types of response to heat have also been recorded from pulp nerve fibers: (1) a transient type of response when pulp nerve fiber was excited by heat over 43°C (the response ceased as soon as the temperature fell below the firing points), (2) a long-lasting type of response that started at over 45°C and continued even after the temperature had returned to the initial level for a few minutes, and (3) a pulsating type of response in which the discharge of the fiber was synchronized with the heartbeat.

Brännström added to the understanding of the “pumping” excitation by dentinal fluid when he pointed out, “Fluids have a considerably greater coefficient of expansion than solids—a sudden rise in temperature of 20°C at the outer one-third of the closed tubule

Figure 7-1 Pain produced by air blast. A, Air evaporates dentinal fluid, causing rapid outflow (arrows) owing to capillary pressure from the pulp’s vessels. B, Odontoblast and accompanying nerve fiber aspirated into tubule, stretching nerve and causing pain. C, Prolonged air blast caused a protein plug to form in the tubule, preventing outward flow. Redrawn with permission from Brännström M. Dentin and pulp in restorative dentistry. Nacka (Sweden): Dental Therapeutics AB; 1981. p. 15.

Figure 7-2 The effect of cold stimulus on the pulp. A, Cold is applied to the tooth, causing a contraction of fluid in tubule. B, Pulp capillary pressure forces replacement fluid into the tubule along with the odontoblast and afferent nerve. Stretching of nerve (arrow) produces intense pain. Redrawn with permission from Brännström M. Dentin and pulp in restorative dentistry. Nacka (Sweden): Dental Therapeutics AB; 1981. p. 17.

Figure 7-3 Effect of intrapulp pressure by application of cold ethyl chloride spray to anesthetized mandibular premolar in a 13-year-old boy. Within 8 seconds, intrapulp pressure had dropped from 30 mm Hg to nearly zero. After the irritant was removed, pressure returned to initial baseline within 1 minute. Reproduced with permission from Beveridge EE, and Brown AC.
might result in an immediate pulpward movement of about 5 micra of the content of the tubules, once again stretching the afferent nerves.

Hyperreactive pulpalgia is common following the placement of a new restoration. Patients also complain after root planing and curettage or following periodontal surgery, which exposes the root surface. Hyperreactive pulpalgia also may be present in the tooth with a carious lesion. Teeth traumatized by bruxism or incompletely fractured teeth are generally more hyperreactive, as are the maxillary teeth involved in maxillary sinusitis.

It is possible, with our present level of knowledge, to divide the sensations of hyperreactive pulpalgia into hypersensitivity and hyperemia.

Dentinal Hypersensitivity

The exciting factors of a hypersensitive pulp are usually cold food or drink or cold air, contact of two dissimilar metals that will yield a galvanic shock, or stimulation of the exposed dentin on the root surface by cold, sweet or sour substances, vegetable or fruit acid, salt, or glycine, or often just touching the surface with a fingernail, a toothbrush, an interdental stimulator, or an explorer. One should not be surprised at this latter reaction when the microanatomy of the dentin and pulp is reviewed. The cementum covering the gingival root dentin frequently is missing or has been removed by curettage or brushing, exposing the dental tubules. It has also been reported that the use of the new “calculus-removing” toothpastes leads to an increased dentinal hypersensitivity. Evidently, these agents remove the surface smear layer and open the dentinal tubuli orifices (Palm Springs Seminars, Palm Springs, California, 1990, Lecture, Ingle JI.).

When one considers that “one square millimeter of dentin contains approximately 30,000 tubules” and that “approximately 25% of the volume of the dentin is occupied by fluid, most of which is in the dentinal tubules,” one must be struck by the capacity for fluid dynamic hypersensitivity emanating from exposed dentin.

This is a frequent complaint following periodontal surgery when whole areas of root are exposed by the apically repositioned gingiva. Add to this the careful root preparation that removes most of the cementum covering the dentin. The problem is then compounded by two other avenues leading to irritation: the use of citric acid on the root surface to remove the smear layer that may “plug” the tubuli and the formation of dental plaque on the root surfaces. The acid-releasing bacteria in the plaque set up a steady barrage of irritation into the dentinal tubules.

This is a “catch-22” equation: the hypersensitive dentin is “painful” to brush and floss and therefore is avoided by the patient. The bacterial plaque that then forms causes greater sensitivity, so the area is avoided all the more during home care, which, in turn, leads to more plaque and greater sensitivity. Relieving the sensitivity is the only solution to the problem.

One is led to conjecture about the pulp sensation stimulated by sour substances, fruit juices, sugar, salt, and dissimilar metals, which may be described as an electric current flowing between the oral cavity and the pulp. Sicher postulated that the oral cavity is positively charged and the pulp is negatively charged (personal communication, 1958). Any electrolyte, such as salt or fruit acid, upsets this ionic balance, and the resultant current stimulates the nerve endings to the odontoblasts. The sensation disappears as soon as the electrolyte is diluted away or metal (such as aluminum foil) is removed. In addition to the current flow theory, Anderson believes that “pain can be evoked from dentin by applying to it solutions which exert high osmotic pressure.” Brännström pursued this idea further, although he believes it is not simply a question of osmosis but relates again to the hydrodynamic “pump”—concentrated solutions of sugar, salt, etc dehydrate the tubule contents, causing their rapid outflow and deformation of the nerves within the tubule. Brännström also pointed out that a similar mechanism is operative when cracks develop in the dentin—that is, as a cusp flexes with biting pressure, the fluid in the tubules is pumped back and forth (especially on release), which stimulates the pain response.

*This figure varies between 10,000 and 30,000 depending on the location in the tooth—crown or root.
Hyperemia

All minor pulp sensations were once thought to be associated with hyperemia, an increased blood flow in the pulp. The investigations of Beveridge and Brown demonstrated, however, that an increase in intrapulp tissue pressure is produced only when heat is applied to the tooth, not when cold is applied.

The increased pressure against the sensory nerve endings in the pulp might well produce the sensation associated with hyperemia. Quite possibly, this will explain why the pain appears to be of a different intensity and character with applications of cold or heat, the cold producing a sharp hypersensitivity response and the heat producing true transient hyperemia and a dull pain.

This difference in the character of the painful response between cold and hot might well be explained by the difference in the nerve fibers supplying the pulp:

The pulp contains both myelinated A nerve fibers and unmyelinated C nerve fibers. The former [A] are fast-conducting and have a low response threshold, whereas the latter [C] are slow-conducting with a higher activation threshold. Activation of the A fibers...will cause a sharp localized response, whereas activation of C fibers will cause a dull, poorly localized response.

Cold stimulates the fast-conducting A fibers, producing the sharp, localized pain. Continued heat application, on the other hand, will more likely stimulate the slower-conducting C fibers, deeper in the pulp, with a resulting dull pain of longer duration, the pain also experienced with early pulpitis.

Trowbridge concurred in reviewing the action of the A and C fibers and pointed out that approximately 25% of the dentinal tubules contain nerve fibers.

The converse of “pain from pressure” also appears to be true. Beveridge and Brown have shown the effect of pain on intrapulp pressure. Paradoxically, pulp pain causes first a fall and then, when removed, a rise in intrapulp tissue pressure (Figure 7-5): “This again raises the question of the role of neural control in the regulation of intrapulp pressure.” It was also discovered that intrapulp pressure decreased when the patient fell asleep and increased when she awakened.

Examination

Determining which tooth is hyperreactive by examination is not always as simple a step as it might seem. A patient may complain of the symptoms of hypersensitivity on taking cold water into the mouth. On the other hand, ice on the suspected tooth during examination may not elicit an unusual reaction. In this case, the entire tooth must be surrounded by cold for the pulp to react. This particular condition is best checked by isolating the teeth adjacent to the suspected tooth behind a heavy rubber dam and then playing a stream of ice water onto the tooth being examined.

If the tooth has had a recent restoration, it usually responds to applications of ice, carbon dioxide “ice,” or Fluori-Methane (Gebauer Chemical Co., Cleveland, Ohio) or ethyl chloride sprayed on a large cotton pellet. Cervical dentin exposed by scratching with an explorer may also elicit a pain response.

Hyperreactive teeth are also said to be more sensitive to the pulp tester; that is, “they require lower levels of electrical stimulation to produce a response.”

“Electrical stimulation is different from other types of stimuli in that it does not cause movement of the fluid within the dentinal tubules.” The sensation derived from electrical stimulation has been described as a “prepain” sensation—“tingling, hot, sharp or warm; rarely is it described as painful.”

Some of the fast-conducting A fibers are initially stimulated by electricity and are described by Nahri as A beta fibers with conduction velocities well beyond the A delta fibers stimulated by tubule fluid movement. At higher levels of electrical stimulation, the slower C fibers “kick in” so that the summation of A and C fibers produces the painful response “associated with higher electrical stimulation.”

Treatment

Grossman has stated, “The best treatment for hyperemia lies in its prevention.” This is sound advice. Application of the new resin adhesives or placement of...
an insulating base under metallic restorations will materially reduce most hypersensitivity. Moreover, this sensation usually diminishes gradually as irritation dentin builds to protect the dental pulp.

There is, however, another source of continuing irritation often overlooked—microleakage.17 Virtually every restoration placed—amalgam, resin, cemented restorations—will share some degree of microleakage around and under the filling. The bacteria collected here will again produce acidic irritants that could affect the pulp through the dentinal fluid. The resulting degree of sensitivity will, in great measure, depend on the presence or absence of a smear layer that obstructs the tubuli.17 Removal of the smear layer (which is very fragile to acids such as those found in soda drinks and fruit juices) and its replacement with one of the new resin bonding agents will materially overcome the problem of microleakage. These adhesives have been shown to be a substitute for an insulating cement base.

Since a true hyperreactive pulp is not a pathologic condition, it may continue to be sensitive for years, acting as a distress signal, warning against insult to a particular tooth. The patient learns to avoid the involved tooth and often becomes a unilateral masticator in the process. The pulp seems well able to accept constant insult, and the statement that long-standing “hyperemia” eventually terminates in pulp inflammation and death is patently false. Apparently, something more than hypersensitivity or hyperemia must be present to lead to necrosis. One would suspect inflammation and/or infection.

Recent interest in eliminating dentinal hypersensitivity has stimulated the revival or development of a number of modalities—physiologic, chemical, or mechanical in nature. Physiologic methods are remineralization of the dentinal tubuli from the “calcium phosphate-carbohydrate-protein complex” in the saliva and/or from the formation of irritation dentin from the pulp. Both of these techniques can take place naturally over long periods of time, but artificially stimulating salivary flow and/or pulp activity are too time consuming and painful to be practical.18

For chemical/mechanical obstruction, “the ideal desensitizing agent should be non-irritating to the pulp; be relatively painless on application; be easily applied; be rapid in action; have long-term or permanent effectiveness; and produce no staining.”19 Krauser pointed out the obverse, “that an agent may be effective (1) in one individual but not in another, (2) on one tooth but not on others, and (3) against one stimuli but not others.”20

“Various agents have been used in attempts to seal the peripheral ends of tubules in sensitive dentin.”18 Agents that have been tried and found wanting are calcium hydroxide, formalin, and silver nitrate. Tubule-sealing agents that have proved successful are potassium oxalate, strontium chloride, sodium and stannous fluoride, and the resins, including the new adhesives. Another approach, using potassium nitrate, blocks sensory nerve activity at the pulpal end of the tubules by altering the excitability of the nerves.

**Potassium oxalate** as a desensitizing agent was developed by Greenhill and Pashley.21 It is sold commercially as PROTECT (John O. Butler Co., Chicago, Ill.). Applying potassium oxalate to the dentin surface, which, in turn, produces “calcium oxalate crystals of different particle sizes within the dentinal tubules, is a means of obstructing the tubules’ apertures (Figure 7-6).” “Calcium oxalate is poorly soluble and is formed when the potassium oxalate contacts the calcium ions in the dentinal fluid.”218 A single-dose applicator permits pinpoint delivery, to the sensitive area, of monopotassium-monohydrogen oxalate. Although the degree and duration of relief will vary from patient to patient, the effectiveness of a single application by the dentist can last up to 6 months.

One rather crude study was less than enthusiastic about oxalate dentin desensitization after 3 months using a monopotassium-monohydrogen oxalate agent,22 whereas a more sophisticated American and two Japanese reports conveyed a good impression of the oxalate solution for desensitization.23–25

**Strontium chloride** is contained in two toothpastes on the market, Sensodyne (Block Drug Co., Jersey City, N.J.) and Thermadent (Mentholatum Co., Buffalo, N.Y.). Strontium combines “with phosphate in the dentinal fluid and exchanging for calcium in the hydroxyapatite of the dentinal tube walls may produce strontium phosphate crystals and dentinal tubules closure.”218 Goodman believes that the strontium ion alters neural transmission, which may account for the immediate improvement in relieving sensitivity.26 Strontium may also stimulate the formation of irritation dentin, and it has been reported “as well to bind to the matrix of the tubule, thus reducing its radius.”27

The fluorides, sodium and stannous, have been used as desensitizing agents longer than any of the other mineral salts. Initially, sodium fluoride was used in paste form (33%) and burnished into the sensitive areas. Repeated applications were necessary. Neither stannous nor sodium fluoride anticaries rinses or toothpastes, however, are particularly effective desensitizers.

Goodman has stated that, “Fluoride is thought to work by reaction between the fluoride ion and ionized calcium in the tubular fluid to form an insoluble calci-
um fluoride precipitate.\textsuperscript{26} It may also stimulate the formation of irritation dentin.

Stannous fluoride with carboxymethylcellulose in a glycerine gel was found to be significantly more effective than a placebo gel in reducing hypersensitivity,\textsuperscript{18} and an acidulated sodium fluoride solution decreased conduction in the tubuli by 24.5%. If sodium fluoride was applied by iontophoresis, hydraulic conduction in the dentinal tubules was decreased by 33%.\textsuperscript{21}

Fluoride iontophoresis has been recognized for years as a consistently successful treatment for dentinal hypersensitivity. Gangerosa is credited with popularizing this treatment when he introduced the Electro Applicator. The Desensitron (Parkell Products, Farmingdale, N.Y.) has also proved effective.

To use these battery-powered devices, the patient holds the positive electrode in his hand and the dentist, using the negative electrode, applies a 2% solution of sodium fluoride to the sensitive areas of the teeth. Using this technique, Simmons reported 94 to 99% reduction in hypersensitivity.\textsuperscript{28} According to a report from India,\textsuperscript{29} a comparative evaluation of the desensitizing effects of the topical application of 33.3% sodium fluoride paste, of iontophoresis with a 1% solution of sodium fluoride, and of iontophoresis with the patient’s own saliva was made. Iontophoresis with sodium fluoride produced immediate relief after one application, whereas topical application required two to three applications. The authors concluded that “iontophoresis with 1% sodium fluoride is the method of choice for the treatment of hypersensitive dentin, as it meets all the requirements of an ideal desensitizing agent except permanency of effect, which requires further investigation” (Table 7-1).\textsuperscript{29}

Gangerosa reported very similar results as well as recommending the iontophoretic application of the fluoride in a tray to desensitize a number of teeth.\textsuperscript{30–33} Carlo and colleagues reported 100% desensitization after two iontophoretic fluoride treatments 73.9% of the time.\textsuperscript{34} Brough et al., on the other hand, found one

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**Figure 7-6** A, Smear layer–covered dentin treated with 3% monopotassium-monohydrogen oxalate for 30 seconds (original ×1,000 magnification). Enlarged inset (×10,000 original magnification) reveals a crack over the tubule. Much of the surface is angular crystals of calcium oxalate. (Courtesy of David H. Pashley.) B, Smear layer treated with neutral 30% dipotassium oxalate. Note large crystals growing out of the smear layer. C, Surface of B treated with 3% monopotassium-monohydrogen oxalate, pH 2.0, acid etches the smear layer away but reacts with calcium from tubular fluid to release a host of finer crystals effectively plugging the tubules. B and C reproduced with permission from Pashley DH, Galloway SE. Arch Oral Biol 1985;30:731.
application of 2% sodium fluoride by iontophoresis to be no more effective to cold response than a similar application with distilled water or 2% sodium fluoride without iontophoresis.\textsuperscript{35}

Potassium nitrate as a desensitizing agent was developed by Hodash, who reported the use of saturated solutions and pastes to be used for home care that contain up to 5% potassium nitrate.\textsuperscript{36} These pastes are sold over the counter as Promise and Sensodyne Fresh Mint (Block Drug Co., Jersey City, N.J.) and Denquel (Vicks Oral Health Group, Wilton, Conn.).

Hodash reported that “Relief of hypersensitivity was notable and rapid in most instances,” and that “Potassium nitrate is also an extremely safe chemical.”\textsuperscript{36} Goodman has shown some impressive clinical results using dentifrices containing potassium nitrate.\textsuperscript{26} He suggested that desensitization may occur either by the oxidizing nature of potassium nitrate or by crystallization, which blocks the tubules, or both.\textsuperscript{26} Pashley, on the other hand, believes that potassium nitrate does not block the tubules but instead reduces the sensitivity of the mechanoreceptor nerves to the movement of dentinal fluid in the tubuli, which normally would produce painful stimuli. Although the fluid still shifts, according to Pashley, “the nerves would not fire because they would be rendered unexcitable.”\textsuperscript{37} Goodman also believes that the “potassium ion depolarizes the nerve fiber membrane…in which few or no action potentials can be evoked.”\textsuperscript{26} Patients should be encouraged to use the desensitizing dentifrices frequently.

Composite resins and bonding adhesives have also been used very successfully to reduce or eliminate dentinal hypersensitivity. Early on, isobutyl cyanoacrylate was found to be effective by blocking the dentinal tubules. Bahram stated that “Cyanoacrylate should be repeated after 6 weeks.”\textsuperscript{38}

<p>| Table 7-1 Comparison of Desensitizing Method\textsuperscript{29} |
|---------------------------------|---------|--------|--------|</p>
<table>
<thead>
<tr>
<th>Degree of Relief</th>
<th>Group I\textsuperscript{+}</th>
<th>Group II\textsuperscript{†}</th>
<th>Group II\textsuperscript{‡}</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good</td>
<td>33.33</td>
<td>55.55</td>
<td>—</td>
</tr>
<tr>
<td>Moderate</td>
<td>52.94</td>
<td>44.45</td>
<td>35.13</td>
</tr>
<tr>
<td>None</td>
<td>13.73</td>
<td>—</td>
<td>64.87</td>
</tr>
</tbody>
</table>

\textsuperscript{+}Treatment by topical application using a 33.3% solution of sodium fluoride.

\textsuperscript{†}Treatment by iontophoresis using a 1% solution of sodium fluoride.

\textsuperscript{‡}Treatment by topical application using a 33.3% solution of sodium fluoride.

In another study, a light-curing dentin bonding agent, Scotchbond (3-M Co., St. Paul, Minn.), was painted onto sensitive areas of exposed dentin and light-cured for 20 seconds.\textsuperscript{27} After one treatment, all sensitivity was eliminated in 89% of the extremely sensitive surfaces and in 97% of the moderately sensitive surfaces. “After 6 months, 85 percent of these teeth were [still] without sensitivity and only 15 percent exhibited any sensitivity at all.”\textsuperscript{27} In contrast, a “control” group treated with a sodium fluoride/strontium chloride solution received virtually no relief.\textsuperscript{27} More recently, Amalgambond (Parkell Products, Farmingdale, N.Y.), a 4-META bonding agent, was tested: “Initially all teeth treated had an immediate response of no sensitivity.” At 6 months, 18 of 19 treated teeth showed decreased sensitivity, 15 of those showing no sensitivity. All control teeth remained sensitive.\textsuperscript{39} A similar study, in which “several coats of a dentin primer (NPG-GMA, BPDM) were applied to root sensitive teeth, achieved similar six months results—all patients symptomless except one who was slightly sensitive to ice.”\textsuperscript{40}

The authors of this study are very positive that the success of resin adhesive therapy depends on careful preparation of the root surface and application of the resin before curing. If or when the resin wears away and sensitivity returns, additional application should eliminate the discomfort once again. Many of the manufacturers of dental adhesives are making extended claims for the effectiveness of their product to reduce hypersensitivity, and this appears justified.

In conclusion, there are a number of alternative modalities that will desensitize hypersensitive dentin. It “boils down” to what works best in the dentist’s and/or the patient’s hands. One must remember that the placebo effect is always present and that at least 30% of the time, anything that is done will achieve a result, for example, the relief achieved over 3 months using only water.\textsuperscript{35}

It should also be remembered that “molars are usually less sensitive than cuspsids or premolars, which are, in turn, usually less sensitive than incisors…older teeth are less sensitive than younger teeth.”\textsuperscript{18} Also, dental plaque elimination should be the first priority before any treatment is undertaken.

Acute Pulpalgia

True pulpalgia begins with the development of pulp inflammation or pulpitis. Beveridge and Brown have shown that an increased intrapulp tissue pressure is possible.\textsuperscript{14} It may be postulated that this pressure might well be the stimulus that is applied to the sensory nerves of the pulp and leads to severe toothache.
**Incipient Acute Pulpalgia.** The mild discomfort experienced as the anesthetic wears off following cavity preparation is a good example of *incipient pulpalgia*. The patient may be vaguely aware that the tooth feels different, “as though it has been worked on,” but the sensation disappears by the next morning.

Stanley and Swerdlow have shown extravascular migration of inflammatory cells following even modest irritation by a controlled and cooled cavity preparation.\(^4\) It is most fortunate that, from the incipient stage, pulpitis is *reversible*, and the discomfort vanishes. One would suspect that incipient pulpalgia is so mild that the pulpitis it predicts is often ignored by the patient until it is too late. This could well be true of the initial sensation developing with a new carious lesion, the slight ache in response to cold or sweets (see Figures 6–2 and 6–3).

**Excitation.** Incipient acute pulpalgia must be stimulated by an irritant such as cavity preparation, cold, sugar, or traumatic occlusion.

**Examination.** If the pulpalgia follows cavity preparation, the involved tooth is obvious. If dental caries is the nosuous stimulus, the cavity is found by an explorer and radiographs. The lesion may be quite small, just into the dentin. The patient can usually tell which quadrant is involved and may even point out the involved tooth. Cold is the best stimulus to initiate incipient acute pulpalgia. The pulp tester is of questionable value in these cases.

When traumatic occlusion is causing the pain, the diagnosis becomes more difficult (see “Traumatic Occlusion,” below).

**Treatment.** Removal of the carious lesion followed by calcium hydroxide application and a sedative cement for a few days may be all that is required to arrest *incipient acute pulpalgia*. Watchful waiting following cavity preparation should not extend to procrastination, leading to moderate or advanced acute pulpalgia. Corticosteroids placed in the cavity following preparation or used on the dentin surface prior to cementation of extensive restorations have proved effective for reducing postoperative pain (HR Stanley, personal communication, 1984).

**Moderate Acute Pulpalgia.** The pain of *moderate acute pulpalgia* is a true toothache, but one the patient can usually tolerate. Many patients report for dental attention after hours, or sometimes days, of discomfort from the developing pulpitis. The pain is frequently described as a “nagging” or a “boring” pain, which may at first be localized but finally becomes diffuse or referred to another area. The pain differs from that of a hyperreactive pulp in that it is not just a short, uncomfortable sensation but an extended pain. Moreover, the pain does not necessarily resolve when the irritant is removed, but the tooth may go on aching for minutes or hours, or days for that matter.

**Excitation.** *Moderate pulpalgia* may start spontaneously from such a simple act as lying down. This alone accounts for the seeming prevalence of toothache at night. Some patients report that the pulp aches each evening, when they are tired. Others say that leaning over to tie a shoe or going up or down stairs—any act that raises the cephalic blood pressure—will start the pain. The list of inciting irritants would not be complete without mentioning hot food or drink, sucking on the cavity, and biting food into the cavity. Most pain of *moderate pulpalgia*, however, is started by eating, usually something cold.

Hahn and his associates have reported a correlation between thermal sensitivity in irreversible pulpitis cases and the microorganisms present in deep carious lesions. Using anaerobic testing methods, they found that *Fusobacterium nucleatum* and *Actinomyces viscosus* were associated with sensitivity and prolonged pain induced by cold. Other bacteria produced heat-sensitive responses.\(^4\)

A warm water rinse does not usually relieve the pain, and cold water makes it worse. The patient may find, however, that two or three aspirin or acetaminophen tablets bring relief. He may continue to take analgesics for days, while wishfully thinking that the pulp will recover. Too many dentists also practice this same game of self-deception.

**Examination.** Attempting to determine which tooth is involved with *moderate acute pulpalgia* is often a difficult experience. The patient may report after days of discomfort, and by this time the pain, though still present, may be widespread and vague. The patient believes he can pinpoint the exact tooth, but then he becomes confused. The typical statement, “the tooth stopped aching as soon as I entered the office,” is commonly heard. No amount of irritation will start it again. If the patient is on heavy analgesics or mild narcotics, it is best to postpone examination until responses will not be clouded by drugs.

If the pain has been constant for some time, all of the pulps on the affected side seem to ache, and, frequently, two or three give approximately the same response to the pulp tester or thermal testing. This is where intuition comes into play. The examiner gets a “feeling” about a particular tooth. It might respond a bit sooner to the pulp tester, or it may ache just a bit longer after cold is applied. The restoration seen in the radiograph may be just a little deeper. All too frequent-
ly, in this day of “full coverage,” a number of teeth may be restored with full crowns, a situation that manifestly compounds the problem.

If the pain is only vague when the patient is first seen, the dentist should attempt, by careful questioning, to obtain a general idea of the area of the pain. Usually, the patient can tell which side is involved and frequently whether pain is in the maxilla or the mandible. This may not be absolute, however, for the pain may be referred from one arch to the other. The patient may remember where the pain started initially, hours or days before. Examination of the suspected area may immediately reveal the involved tooth, made obvious by a large carious lesion or huge restoration. Then again, nothing unusual may be present.

Radiographs may give an immediate clue in the form of a huge interproximal cavity or a restoration impinging on the pulp chamber. If nothing is learned from radiographic examination, the electric pulp tester is then employed, but generally without great success.

A tooth involved in moderate acute pulpalgia is hypersensitive and will respond sooner, or lower, on the scale of the pulp tester. Then again, all of the teeth in the area may be hypersensitive and respond in the same way to pulp testing so that no definite conclusions may be drawn from this test. This leaves the thermal test as the final arbiter since percussion and palpation rarely reveal any response, although the tooth may be slightly sensitive to percussion.

The first thermal evaluation to use is the cold test because the pulp is more likely to respond to this stimulus. The tooth under the greatest suspicion should be tested first. The examiner should block the adjacent teeth with his fingers, being careful that melting ice does not run onto these teeth. The immediate response to cold may be quite sudden, violent, and lasting. On the other hand, the initial pain may go away immediately when the cold is removed. This is the time to stop! Do not test any more teeth for about 5 minutes. The reason for this is quite obvious: The pain in the tested tooth that stops aching may rebound within a few minutes, and if the dentist has proceeded to test other teeth, neither he nor the patient will be able to differentiate the aching tooth. If the pulp starts to ache, however, reapplying the cold should increase and prolong the pain.

Infrequently, heat is the stimulus that starts the symptoms. Sometimes, however, nothing will start the ache, and the patient must be dismissed and asked to return when the tooth is again painful.

Occasionally, the search is narrowed down to a maxillary and a mandibular tooth, both prime suspects because both are aching. One molar is the problem tooth and the other the “referred” tooth. If anesthetic is injected into the suspected arch, and the hunch was correct, the pain should stop in both teeth. If the pain does not stop, the offending tooth is in the opposite arch. Again, by means of the anesthetic test, aching mandibular premolars may be differentiated from molars by the use of a mental injection that will anesthetize from the second premolar toward the midline. A zygomatic injection in the maxillary arch for the maxillary molars, or a careful slow infiltration for the maxillary premolars injected well forward toward the canine, may differentiate between these confusingly similar pains. Nor should one forget the interligamentary injection (see chapter 9). Injecting down through the periodontal ligament allows each individual tooth, even each individual root, to be anesthetized. Although this analgesia may not be profound enough for pulpectomy, it may prove adequate to stop pulpalgia from referring. The anesthetic test is a last resort and should be used after all other means have been exhausted.

In diagnosing moderate acute pulpalgia, above all, the examiner must think, must be shrewd, and must not panic. If in doubt, hesitate! Often one more day may make a difference. The patient should be warned to return to the office without having taken any analgesics.

Treatment. The treatment for moderate pulpalgia is quite simple: pulpectomy and endodontic therapy if the tooth can and should be saved or extraction if the tooth should be sacrificed. If endodontic therapy is indicated, it may be completed in one appointment.

Hodosh and colleagues, who reported favorably on the use of potassium nitrate as a desensitizing agent, also used the chemical mixed with carboxylate cement as a pulp-capping medium in teeth with pulpitis. In a preliminary report, they noted that all of the teeth became asymptomatic immediately but that 2 of 86 failed.

Glick used Formocresol to treat pulps that continue to ache after root canal therapy has been completed. His supposition is that vital, inflamed tissue still exists in a canal that is impossible to locate. The tooth may even respond to thermal and electric testing. The Formocresol “embalms” the microscopic remainder of the pulp, and the pain is alleviated. In the same vein, a US Army dentist reported two endodontically treated teeth that still ached when heat was applied. After re-entry, a careful search revealed additional untreated canals. Total pulpectomy and root canal filling completely eliminated the postoperative pain.

In the Orient, toothache has long been alleviated with acupuncture. A favorite site to place the acupuncture needle is the Hoku point—midway in the web of
tissue between the thumb and index finger on either hand. Temporary relief of pain is achieved after a few minutes of “needing” this point. The respected pain center group at McGill University has reported similar results by massaging the Hoku point for about 5 minutes with an ice cube wrapped in a handkerchief. “Ice, an analgesic, helps overload the circuits, quickly closing the pain gate,” according to the researchers.46 This simple method of pain control might well be recommended to a patient unable to appear immediately at the dental office.

**Advanced Acute Pulpalgia.** There is never any question about the patient suffering the pain of advanced acute pulpalgia. He is experiencing one of the most excruciating acute pains known to humanity, comparable to otic abscess, renal colic, and childbirth. If every dentist personally experienced the pain of advanced acute pulpalgia, he would be a more sympathetic practitioner for the experience.

This patient is in exquisite agony and sometimes becomes hysterical from the pain. The patient often is crying and virtually unmanageable. One patient, who had to drive 40 miles to a dentist, reported that he could stand the pain no longer, so he stopped the car, took out a pair of pliers, and pulled his own tooth. Patients have confessed contemplating suicide to escape the pain.

The relief for this pain is embarrassingly simple: cold water, preferably iced. Cold water rinsed over the tooth is all that is usually needed to arrest the pain temporarily. The patient might discover this fact while taking an analgesic and, in so doing, receive immediate relief. He then reports to the dentist with a thermos or jar of ice water in hand, only stopping to sip as the pain gradually returns. Frequently, he times the periods of relief much as the expectant mother times her labor pains. The relief often lasts 30 to 45 seconds.

When a patient telephones reporting a toothache, especially late at night, the dentist should always inquire, “Have you tried rinsing cold water on the tooth?” If the answer is negative, request that the patient do so and return to the telephone to report results. If the cold gives relief, the compassionate professional meets the patient as soon as possible to provide permanent relief. If cold aggravates the pain, the patient has moderate pulpalgia, which might well become advanced pulpalgia by morning. The patient with advanced pulpalgia would have to continue rinsing with cold water throughout the night, and, even then, the cold may no longer give relief. Thus, a tired and “frazzled” patient becomes a hysterical one.

**Examination.** The examination for advanced acute pulpalgia, in comparison to that for moderate pulpalgia, is relatively simple, even if the tooth is not aching when the patient presents himself. The involved tooth always has a closed pulp chamber, as revealed by the radiograph. Otherwise, the tremendous intrapulp pressure could not develop. In addition, the radiograph may reveal a thickened periodontal membrane space at the apex as the inflammation spreads out of the pulp.

The history is self-incriminating. The symptoms are violent! The involved tooth usually can be pointed out by the patient and is sometimes tender to percussion as well. These teeth are said to be less sensitive to the pulp tester (requiring a higher reading), but the performance of this test is merely “gilding the lily.” Heat is the merciless offender. Hahn reported that cavities filled with “black pigmented Bacteroides, Streptococcus mutans, and total anaerobic colony counts were positively related to the heat sensitivity” in irreversible pulpitis cases.42

Because the inflamed pulp reacts so violently to heat, the most decisive test is the heat test, although one must have a cold water syringe in the other hand, ready to give immediate relief. As soon as the hot gutta-percha touches the involved tooth, the patient develops what Sicher has called the subgluteal vacuum; he suddenly rises up in the chair as if stabbed. Cold water is instantly applied, and the pain subsides.

The thermal test is conclusive! When the patient is again comfortable, however, the adjacent teeth should also be tested to ascertain that no more than one tooth is involved or that the suspected tooth gives the most violent reaction. The patient should be assured that the involved tooth will not again be warmed.

Local anesthesia gives blessed relief, and the dentist has, from that moment, made a friend for life. The friendship will be more lasting if the tooth is saved by endodontic therapy rather than extracted.

**Treatment.** The treatment for advanced pulpalgia is the same as for moderate pulpalgia: pulpectomy and endodontic therapy for the salvageable tooth and extraction for the hopeless one.

Complete anesthesia of an inflamed pulp may be difficult even though all outward signs would indicate the conduction or infiltration injection to be successful. In this case, an intrapulp injection of lidocaine or pressure anesthesia with lidocaine or an interligamentary injection may be necessary.

Following pulpectomy, the pulpless tooth should be relieved of occlusal contact by grinding. Endodontic therapy should be completed at a later appointment.
Chronic Pulpalgia

The discomfort from chronic pulpalgia is best described as a “grumble,” a term commonly used by patients who withstand the mild pain for weeks, months, or years. Often the pain can easily be kept under control with one or two analgesic tablets, two or three times daily. Frequently, the patient seeks relief only when the pulp begins to ache every night.

The pain from chronic pulpalgia is quite diffuse, and the patient may have difficulty locating the source of annoyance. Patients frequently say that they have a “vague pain in my lower jaw.” Chronic pulpalgia is likely to cause referred pain, which is also mild. Other patients may appear with beginning acute apical abscess and confess to knowing that something was “wrong” with the tooth for months. Other patients comment on the bad taste or odor constantly noted.

Excitation. The pulp involved in chronic pulpalgia is not affected by cold but may ache slightly on contact with hot liquids. The most common report is that the tooth is sore to bite on.” If meat or a bread crust, for example, is crushed into the cavity, the pain lasts until the irritant is dislodged. The patient may report that the tooth begins to hurt late in the day, “when I’m tired,” or, more frequently, “when I lie down.”

Barodontalgia. One patient confessed to discomfort each Monday morning and Friday evening. These were the times each week when he crossed a 4,000-foot mountain pass in his travel across Washington state. Here the slight difference in barometric pressure was enough to excite the pain response. The same may be true during an airplane flight. (Planes are actually pressurized at 5,000 feet, not sea level.)

Kollman tested 11,617 personnel of the German Luftwaffe who participated in simulated high-altitude flights up to 43,000 feet: “Only 30 (0.26%) complained of toothache (barodontalgia).” Chronic pulpitis was the principal culprit, followed by maxillary sinusitis.47

Rauch classified barodontalgia (formerly called aerodontalgia) according to the chief complaint.48 If the patient has pulpitis, he will have pain on ascent—sharp momentary pain (Class I) in the case of acute pulpitis, and dull throbbing pain (Class II) in the case of chronic pulpitis. These pains are caused by the extraoral decompression of the ambient pressure in the plane, which, in turn, allows for a compensating increase of pressure within the pulp chamber and root canal. Descent (compression of the ambient pressure) brings relief in the pulpitic tooth. If the pulp is necrotic, the reverse is true, a dull throbbing pain (Class III) on descent (compression) and asymptomatic on ascent (decompression). In a case of periradicular abscess or cyst, severe persistent pain (Class IV) occurs with both ascent and descent.

Rauch pointed out that “even though the highest incidence factor is less than 2 percent, because of the vast number of people” who fly, barodontalgia must be considered in the differential diagnosis of oral pain.48

Examination. Determining which tooth is involved with chronic pulpalgia is often ridiculously simple and, on other occasions, most difficult. Frequently, a large carious lesion is present, or an amalgam restoration is fractured at the isthmus. Another common offender is recurrent caries under a restoration, usually an inlay. These are the lesions that are painful when compressed by food packed into the cavity.

The leathery dentin covering these lesions may be removed with a spoon excavator, often without anesthesia and with no great discomfort. The pulp lies revealed, covered with a gray scum of surface necrosis. Biopsy would show degeneration of the remainder of the pulp, accounting for the lack of severe pain.

The chronic pulpalgia that is the most difficult to diagnose lies under a full crown because it is impossible to pulp test electrically, or under a three-quarter crown, where recurrent caries is not revealed by radiographs. In these cases, carbon dioxide “ice” should be used as the stimulant.

The pulp tester and the radiograph are the best tools for locating the tooth involved with chronic pulpalgia, which will sometimes respond as “necrotic” to electric testing—that is to say, it will take the maximum discharge from the tester. In any case, a high reading on the rheostat should be expected. England and colleagues demonstrated intact nerve fibers, with some variations from normal, in pulp specimens with “irreversible pulpitis.”49 In the necrotic pulp, dissolution of the fibers was apparent.

The radiograph often reveals interproximal or root caries, or recurrent caries under a restoration. In chronic pulpalgia, the so-called “thickened” periodontal membrane also may be present, indicating that the inflammatory process is not confined completely to the pulp. These cases may also demonstrate condensing osteitis of the cancellous bone at the apices. Interestingly, this osteosclerosis disappears after successful endodontic therapy (see Figure 5-9).

The apices of the involved roots also show external resorption, although this condition is more prevalent following pulp necrosis and complete periradicular involvement (see Figure 6–49).

Thermal tests are of little value in a positive sense in diagnosing chronic pulpalgia, although, in some cases, slight pain may be experienced in response to extreme
heat. This is in accord with the patient’s history of no response to iced drinks but an occasional response to hot coffee.

Percussion has a good deal to offer in many of these cases. Often the patient is vaguely aware that something feels “different” about the involved tooth when it is percussed. Palpation is virtually useless. However, having the patient bite on an applicator stick sometimes reveals soreness of a particular tooth.

Chronic pulpalgia has the aggravating habit of referring its vague pains throughout the region. The patient may insist that a mandibular molar is aching, whereas examination reveals that a maxillary molar is the offender. Often anesthetizing the involved tooth is the only convincing proof to the patient that he is wrong. Patients have reported with aching of a maxillary molar when the maxillary lateral incisor has been found to be the offender. If the tooth suspected by the patient appears normal to all examination and testing, the examiner should be suspicious of chronic pulpalgia in another tooth on the same side. The mandibular molar involved in chronic pulpalgia is not as apt to refer pain to the ear as it is in acute pulpalgia.

Treatment. The treatment for chronic pulpalgia is quite basic: pulp extirpation and endodontic therapy if the tooth is to be saved and extraction otherwise. Anesthesia is no problem.

Hyperplastic Pulpitis. The exposed tissue of a hyperplastic pulp is practically free of symptoms unless stimulated directly.

Excitation. The discomfort of a hyperplastic pulp is quite simple. It “erupts” out of its open bed of caries for all to see. Differential diagnosis is concerned with only one problem, namely that of discerning whether the polyp is pulp or gingival in origin because both are covered by epithelium (see Figures 4-72 and 4-73).

The pulp polyp may be lifted away from the walls with a spoon excavator and the pedicle of its origin thus revealed. It is remarkably painless to handle and may even be excised with a sharp spoon excavator with no great discomfort.

Treatment. Frequently, the teeth involved in hyperplastic pulpitis are so badly decayed that restoration is virtually impossible. Hence, extraction is usually indicated. On the other hand, if the tooth can be restored, pulpectomy and endodontic therapy are recommended prior to restoration. Glick reported limited success with pulpotomy in these cases, done originally as an experiment on three cases with good bleeding (personal communication, 1964). He was surprised to see periradicular repair take place.

Necrotic Pulp. There are no true symptoms of complete pulp necrosis for the simple reason that the pulp, with its sensory nerves, is totally destroyed. Often, however, only partial necrosis has occurred, and the patient has the same vague, comparatively mild discomfort described for chronic pulpalgia.

The examiner also must bear in mind that the pulp in one or two canals in multirooted teeth may be necrotic, and the pulp in a second or third canal may be vital and quite probably involved in acute or chronic pulpitis. The results of examination in these cases are most bizarre because each level of pulp vitality is represented by a confused response.

Examination. A routine radiographic survey or coronal discoloration may present the first indication that something is amiss in the case of the tooth with a necrotic pulp. On questioning, the patient may recall an accident of years ago or a bout of pulpalgia long since forgotten.

Many cases of pulp necrosis are discovered because of the discoloration of the crown. This applies primarily to the anterior teeth and ranges from a vague discoloration, visible only to the trained eye, to frank discoloration of the darkened tooth. A discernible difference may sometimes be demonstrated by transillumination with a fiber optic.

The radiograph may be helpful if a periradicular lesion exists because its presence usually indicates associated pulp death. Radiographically, the tooth with the necrotic pulp may exhibit only slight periradicular change; in other words, a radiolucency is usually found by hindsight rather than foresight. Then again, a sizable periradicular bony lesion may accompany the necrotic pulp. No changes in the canal are noted radiographically to indicate necrosis.

One of the first lessons to be learned, however, is never to trust a radiograph alone in diagnosing pulp necrosis. A snap judgment of the periradicular radiolucency that exists with periradicular osteofibrosis associated with perfectly normal, vital pulps will lead to error if the examiner depends on radiograph evidence alone. It is imperative always to pulp-test the tooth.

The electric pulp tester, therefore, is the instrument of choice for determining pulp necrosis. With complete necrosis, no response will be given at any level on the tester. With partial necrosis, a vague response that can easily be tolerated may be elicited at the top of the scale. The tooth with a necrotic pulp may also be slightly painful to percussion.

Treatment. There is no treatment for pulp necrosis per se because the necrotic pulp has long since been
Internal Resorption. Internal resorption is an insidious process when the afflicted pulp is completely free of symptoms. On the other hand, this condition has been known to mimic moderate acute pulpalgia in pain intensity. The usual case, however, closely resembles the chronic pulpalgia syndrome, that is, mild pain at the tolerable level. When confined to the crown, enough tooth structure may be destroyed for the pulp to show through the enamel—hence the synonym for internal resorption, “pink tooth.”

Excitation. Symptoms of internal resorption depend primarily on whether the process has broken through the external tooth surface. If the pulp destroys enough tooth structure to finally erupt into the oral cavity, it responds much as the hyperplastic pulp, painful only to pressures of mastication.

Because the pulp is undergoing dystrophy localized to a single area, it is not as likely to be excited by the drinking of hot or cold fluids. The pulp that erodes through the root surface may give vague symptoms, primarily with mastication, but the patient usually remembers these symptoms in retrospect after the condition is pointed out on the radiograph.

Examination. Two methods of examination reveal the case of internal resorption: visual examination if the crown is involved and radiographic examination for the crown and root. Thermal tests and the electric pulp tester may provide confirming, yet only partially reliable, evidence.

The case of internal resorption that is truly difficult to diagnose is the one of coronal involvement often hiding behind the full or three-quarter crown and thus not revealed in the radiograph. The patient complains of vague symptoms and referred pain, but the response of the involved pulp to the testing procedures may be similar to that of the other teeth. Percussion may be of slight value. In these cases, an intuitive hunch is needed. If one is fortunate, the correct tooth, when tested, may exhibit slight variances from the other teeth in the area. On the basis of these minor variances, the suspected tooth is chosen; however, the presence of internal resorption is not confirmed until the coronal pulp is entered.

Treatment. Pulpectomy is the only treatment for internal resorption. As long as the pulp remains, it is most likely to continue its destructive process. If the tooth can be saved by endodontic restoration, the defect can best be obturated by thermoplasticized and compacted gutta-percha.

Traumatic Occlusion. A tooth traumatized by bruxism or traumatized because a restoration is in hyperocclusion often responds much like the tooth with mild pulpalgia. First, the pulp is usually hypersensitive, reacting primarily to cold. In addition, the pain may be vague, reminiscent of chronic pulpalgia.

The patient may complain of being bothered by pulp discomfort on awakening in the morning or possibly of being awakened by the discomfort. The story of pain at the end of a rather trying day is also characteristic. Pathognomonically, the patient reports relief after only one aspirin. Moreover, he usually says that the tooth is not painful on mastication; at least this is not his chief complaint.

Paradoxically, even a well-treated pulpless tooth being traumatized by bruxism presents the vague symptoms of pseudopulpalgia. It, of course, does not respond to thermal stimuli but still feels like a mild toothache.

Examination. From the patient’s history usually comes the clue to diagnosing the pain from trauma. History of “toothache” on awakening is an unusual symptom and should direct one’s thinking toward bruxism at night. The discussion of a tense daily situation is another clue. The vagueness of the pain is most important because one expects to be dealing with chronic pulpalgia, and yet the thermal and pulp tester response is often like that of a normal or hyperreactive pulp. The fact that a low dosage of a mild analgesic can control the pain is pathognomonic.

If one suspects pain from trauma, one should look for facets of wear on the tooth. Articulating paper may be helpful; however, the point of contact may not be readily apparent. One young patient shifted her mandible forward during sleep and ground the distal of the mandibular second molar against the mesial of the maxillary first molar, a protrusive shift of a full centimeter. It was difficult to believe that the two well-worn facets would match, and yet, when the youngster was teased into protruding her mandible to this extent, causing contact between the two surfaces, her eyes lighted in delight, and she began compulsive bruxism.†

It should be remembered that the mandible also may be retruded in sleep, causing facets distal to masticatory facets to be involved. Examination for these annoying contacts should be carried out with the patient supine in the dental chair.

Too many dentists examine the median occlusion position (centric) and the lateral excursion of function (working bite) and completely neglect to examine for nonfunctional (balancing bite) traumatic contacts. So

†Contraction or stretch of muscles (as in yawning) is often pleasurable!
often the nonfunctional contact is the patient’s compulsive position. Some patients even bring diagrams to the office to describe the point of interference, demonstrating an abnormal and exaggerated oral awareness.

Peculiarly enough, the involved tooth or teeth are frequently not sensitive to percussion but are sensitive to mastication. Biting or chewing on a narrow cotton roll or Burlew disk will sometimes elicit discomfort.

The radiograph may show no periradicular changes or may exhibit a widened periodontal space and apical external root resorption (see Figure 6–46).

Treatment. Treatment for these cases obviously involves relieving the point of occlusal trauma by judicious grinding to reshape the involved tooth and its opponent. Actually, the tooth should be completely disoccluded to give the inflamed tissue a chance to recover.

Many times, the dentist is unsure of his diagnosis, especially in cases that closely resemble or actually are pulpitis. The pulp should be given the benefit of the doubt, particularly if a fully restored crown is involved and complete testing is difficult. If symptoms and signs are vague, the case should first be handled as a problem of traumatic occlusion, especially if there is some evidence that this might be true. If, after careful adjustments, the pulp does not respond with almost immediate relief, the possibility of pulpitis should be reconsidered, but only after all of the excursions of the mandible and the patient’s history are rechecked.

Sometimes the patient reports relief as soon as the occlusion corrections are completed, even before leaving the chair.

Incomplete Fracture or Split Tooth. The tooth that is split or cracked but not yet fractured presents some of the most bizarre symptoms encountered in practice. These symptoms range from those of a constant, unexplained hypersensitive pulp to constant, unexplained toothache.

The tooth may be uncomfortably only occasionally during mastication, and at that time the pain may be one quick, unbearable stab. This is when the crack in the dentin suddenly spreads as the cusp separates from the remainder of the tooth. The pulp may only be hypersensitive, possibly for years. In one case, follow-up continued for 8 years, and the pulp hypersensitivity immediately ceased when the buccal cusp fractured away.

Many of the cases involve noncarious, unrestored teeth; hence it is hard to believe that anything could be wrong with the tooth. If the split has extended through the pulp, bacterial invasion occurs, and true pulpitis results. These cases are comparatively easy to diagnose because of the obvious symptoms.

The most frequent complaint is that of a tooth painful to bite on, with an occasional mild ache. One case was diagnosed over the telephone on the basis of a report of these classic symptoms by the harassed referring dentist. The patient confirmed the diagnosis by reporting the same day with the buccal cusp of a maxillary premolar in hand.

Excitation. The discomfort of the split tooth is elicited by biting on the tooth or contacting cold fluids. If the pulp is involved in fracture, any exciting agent for pulpalgia will bring on discomfort.

Examination. First, one thinks of carefully examining the tooth, dried and under good light, to find the crack in the enamel. Usually, the search goes unrewarded because the examiner sees no cracks at all or finds similar enamel crazing in every tooth.

The pulp tester customarily gives a normal reading unless the pulp is involved. Thermal tests may be valuable if a cold or hot stream is played on the tooth or hot or cold fluids are rinsed against the possible culprit. Hot gutta-percha or a stick of ice, on the other hand, is usually valueless.

Percussion alone, surprisingly enough, is usually not helpful, yet biting on an applicator stick or cotton roll may give the spreading action needed to elicit pain.

The crown may also be painted with tincture of iodine, which is washed off after 2 minutes. The crack often appears as a dark line (see Figure 4–12).

A piece of Burlew rubber disk can be used to stress a possibly fractured tooth. Held in a locking pliers, it can be shifted around to different positions on the occlusal surface while the patient is asked to bite on it. An even more definitive device is the Tooth Slooth (Laguna Niguel, California, USA), a triangular plastic tip on a handle (Figure 7–7). With this device, it can be determined quite accurately which cusp is splitting away.

The radiograph records an obvious split only if it is in correct alignment to the central rays (Figure 7–8). It will completely fail to reveal the almost microscopic split, which elicits the really bizarre syndrome.

Treatment. If an incomplete fracture is suspected but the pulp is not involved, the crown should be pre-
pared for a full crown, which should then be cemented temporarily with zinc oxide–eugenol cement. The full crown binds the remaining tooth structure much as the hoops contain the staves of a barrel.

If the incomplete fracture has entered the pulp and a true pulpalgia indicates that pulpitis is present, then root canal therapy should be completed first, followed by full coverage to prevent a total fracture. If the fracture has extended completely through, into the periodontal ligament and pulp, the chances of saving the entire tooth are remote indeed. The possibility of saving a portion of the tooth is discussed in chapter 12.

POSTOPERATIVE PAIN: INCIDENCE, PREVENTION, AND TREATMENT

There is no question that a good deal of postoperative pain is associated with endodontic therapy. This is one of the factors that continues to affect the public adversely concerning root canal therapy. Some of these problems are unavoidable, but many are iatral. Apical overextension of necrotic debris (infected or otherwise), instruments, paper points, medicaments, and filling materials lead to postoperative pain. Apical perforation is a common occurrence that can mainly be avoided by careful attention to establishing and maintaining correct working length.

In surveying 1,204 teeth treated endodontically, Dutch investigators reported an incidence of 30% postoperative pain: 7% with severe symptoms and 23% with moderate symptoms. By far the greatest number of cases of postoperative pain (65%) were related to patients who reported at the first appointment with preoperative pain. In contrast, only 23% of those who developed postoperative pain were free of pain initially. Most postoperative pain occurred on the first day after initiating endodontic treatment. Emergency treatment was necessary in 57% of the patients to relieve the pain. Analgesics satisfied the rest.

In a subsequent study, the Dutch team further analyzed a subsection of the cohort of 803 patients with 1,204 teeth, mostly completed in one appointment—1 hour for single canal cases and 2 hours for molars. A positive correlation was found “in the case of a nonvital pulp in conjunction with preoperative pain on the day of treatment [and] when a radiolucency larger than 5 mm in diameter is present.” The chance of postoperative pain also increases with the number of root canals in the tooth. The probability of postoperative pain was reduced in any case with a vital pulp.

A US Navy group has also reported that patients who were asymptomatic at the start of treatment experienced a low incidence of postoperative pain. As one would expect, they also found that pulpectomy signifi-
cantly reduced postoperative pain in patients who had reported initially with preoperative pain. More recently, it was reported that complete pulpectomy was the most effective method of preventing postoperative pain in those patients presenting with preoperative toothache in teeth with vital pulptis. The next most effective method was pulpotomy and the least effective was partial pulpectomy, which was twice as likely to allow pain to continue.53

Chronic postoperative pain following endodontic surgery is quite unusual, even though immediate post-surgical acute pain is expected. One survey, however, classified 5% (6 cases) of a cohort of 118 cases as endodontic failures because of continuing pain for an average of 21 months.54 It was determined that three of the six patients may have suffered from post-traumatic dysesthesia, “pain associated with the manipulation of the root or apical bone...If nerve injury occurs, an abnormal repair process is possible.” The remaining three “failures” were thought to be phantom tooth pain. The duration of pain in this group prior to treatment was 1, 4, and 36 months, respectively.54

PREOPERATIVE THERAPY
A number of studies have been done evaluating the efficacy of preoperative medication of cases suspected as potential “troublemakers.”55,56–58 The Navy group (above) found that the preoperative administration of flurbiprofen (a nonsteroidal anti-inflammatory drug [NSAID]) significantly reduced postoperative pain compared to placebos.52 A group in Pittsburgh also recommended the use of an NSAID, the shorter-acting ibuprofen, as a preoperative prophylactic against the possibility of postoperative pain.59

Morse and his group at Temple University achieved similar results with the prophylactic administration of diflunisal (a long-acting NSAID).55 They also found that the intracanal use of a corticosteroid solution following pulpectomy was efficacious.56 These results were similar to those of their previous studies with antibiotics.57

The value of oral dexamethasone (corticosteroid) following initial endodontic treatment has also been reported.58

Questioning the time-honored procedure of prophylactically relieving the occlusion of posterior teeth being treated endodontically, a group at Iowa University found that there was no statistically significant difference between routine occlusal relief and placebo relief.60 Although they could state that their results “cast doubt on the practice of routinely relieving the occlusion of posterior teeth receiving endodontic treatment,” they did not imply that occlusal relief should be abandoned in cases of acute apical abscess (AAA) or acute apical periodontitis (AAP).60

PERIRADICULAR PAIN
Periradicular pain may be almost as excruciating as pulp pain and may often continue for a longer period of time. Periradicular lesions that may produce discomfort are (1) SAP, (2) SAA, (3) asymptomatic apical abscess (AAA), and (4) apical cyst. The adjective “acute,” as used here, refers to the severity and the rapidity of the course of the lesion. Acute apical periodontitis is by far the most distressing periradicular lesion.

Symptomatic Apical Periodontitis
Symptoms. This acute form of periradicular pain can be most excruciating and sometimes lasts for days. The tooth is exquisitely painful to touch, and even contacting the tooth in closure may bring a flood of tears. The pain is most persistent, lasting 24 hours a day.

The pain has been described as constant, gnawing, throbbing, and pounding. Eventually, the patient may gain blessed relief, only to bite on the tooth while eating or during sleep, which starts the pain cycle once more. Many patients beg to have the tooth extracted. Yielding to their wishes, this has been done only to have the pain continue for another 48 hours owing to osteitis.

There is no overt swelling involved, just a grossly painful tooth elevated slightly in its socket. One week of discomfort is to be expected if nothing is done!

Etiology. The degree of discomfort described in the preceding paragraphs may be iatral. That is, the clinician perforates the root apex during endodontic therapy, forces caustic medicaments or irritating solutions through the apical foramen, or forcibly deposits necrotic, infected, and toxic canal contents into the periradicular tissue. These irritants produce a violent inflammatory reaction. If bacteria are present in the canal and are extruded apically, an acute abscess also develops to complicate the picture.

Typically, SAP follows initial endodontic treatment. The mandibular premolars and molars are the teeth most frequently and violently involved, the premolars especially. This fact could be attributed to their invitingly straight, tapered canals, which encourage abuse of the periapex with a reamer or file. Furthermore, the thick bony cortex and the small amount of cancellous bone found in this area limit the space allowable for swelling. This limitation greatly increases the pressure in the area and hence the pain.

Examination. Diagnosis of SAP is relatively easy; the patient is in severe pain, and the involved tooth is
The advent of corticosteroids as anti-inflammatory agents has improved the treatment of SAP. Hydrocortisone, combined with neomycin (Neo-Cortef 1.5% eye/ear drops, sterile suspension, Upjohn Co., Peapack, N.J.), is recommended as an anti-inflammatory/antibacterial medicament. The canal is flooded with this liquid suspension and then, very gently, the fluid is “teased” out of the trephined apex with a fresh sterile instrument. A loose cotton pellet is then placed in the chamber and a thin temporary filling placed without undue pressure. The canal should not be filled to overflowing with the corticosteroid solution to allow space for inflammatory swelling. There will be some “lag time” before the anti-inflammatory effect of the hydrocortisone takes place.

If the tooth continues to be painful after the analgesia wears off, the patient is instructed to return to the office to have the procedure repeated. Removing the temporary (with a dam in place) once again allows for drainage, and the canal is again medicated with neomycin 1.5%. The temporary is replaced to prevent secondary contamination. Each time the rubber dam is removed, the occlusion should again be checked. Frequently, further adjustment is necessary because the tooth has again been elevated in the alveolus. The patient is warned not to eat on this side; however, the warning is usually superfluous.

When the patient is leaving the office, he should be instructed that if the pain becomes unbearable at night, he can remove the temporary at home. He should be shown in a mirror how to pick out the thin temporary filling by using a safety pin that has been straightened into a right angle. Exposed to the saliva, the canal, of course, becomes contaminated. This is a modest problem, however, compared to a sleepless night of pain that might be suffered by the patient.

In a further attempt to reduce or eliminate post-treatment pain, Liesinger and colleagues reported the successful use of dexamethasone (corticosteroid), injected intraorally or intramuscularly, to suppress pain.61,62 In a more objective laboratory study, a group at San Antonio, Texas, quantitated the effect of dexamethasone as an anti-inflammatory drug.63 After producing acute periapical lesions in rat molars by overinstrumentation, they injected either dexamethasone or a saline control in the buccal vestibule opposite the no-insulted teeth. They found that “dexamethasone produced a significant anti-inflammatory effect” as measured by the number of polymorphonuclear neutrophil leukocytes that were counted in the area.63

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The patient should also be given a narcotic for analgesia: 30 to 60 mg (0.5 to 1 grain) of codeine is the initial prescription, taken every 3 hours with 10 g aspirin. If the patient does not obtain relief from codeine or cannot tolerate the drug, 50 to 100 mg meperidine (Demerol) is prescribed every 4 hours, depending on the patient’s age and weight. A few patients have had such intense, prolonged pain that they required morphine. Other patients have found it necessary to return every 5 or 6 hours for injections of local anesthetic into the affected area.

Alveolar trephination is another means of relieving the severe pain of SAP. Trephination, or surgical fistulation, is thoroughly discussed in Chapter 12.

In any event, the patient should be seen daily until the symptoms have resolved. Endodontic therapy should not be undertaken until the tooth is comfortable.

**Acute Apical Abscess.** The pain of AAA is similar to that described for AAS but somewhat lower in intensity. After all, necrosis is an extension of the inflammatory cycle, which begins with acute apical periodontitis and continues to the abscess state if not checked.

Necrosis of the acute abscess usually destroys enough tissue to permit fluid dispersion. The extravasated fluid breaks out into the soft tissue and narrow spaces where swelling is not as confined as it was at the periapex. This is not to say that the AAA is not painful. On the contrary, it is quite painful, but in comparison with SAP, the unbearable pain has gone and in its place is a full systolic throbbing pain, particularly on palpation. The involved tooth is also painful to movement or mastication.

**Etiology.** The discomfort of AAA develops gradually as the abscess grows in size. The condition is invariably related to bacterial invasion of the periradicular region from a necrotic, infected pulp canal. The abscess may develop spontaneously from an infected pulpless tooth or may follow initial endodontic treatment if bacteria are forced into the periradicular tissue.

In any event, the initial discomfort may be mild but gradually builds in intensity as the abscess becomes indurated or hardened. When the alveolar plate is “eroded” by the process and the abscess gathers into frank pus, the entire area softens and feels fluctuant to palpation, and the pain is greatly reduced.

**Examination.** Diagnosis of AAA is a relatively simple matter. The patient has pain and, invariably, swelling. Although the swelling may not always be observable to the examiner, the patient feels the tense-ness of the swollen area. The degree of swelling varies from the initial, undetected swelling to gross cellulitis and massive asymmetry (see Figure 5–11). The involved tooth also is extremely painful to percussion or palpation. Radiographically, the picture may vary from a widened periodontal space to a large alveolar radiolucency. Actually, the radiograph is not the best means of diagnosis because it frequently reveals nothing of true diagnostic value.

Outside of percussion, electric pulp testing is the best method of diagnosis because the pulp of the tooth involved in AAA is invariably necrotic. The vitality test, moreover, is the best criterion to differentiate an AAA from an acute periodontal abscess. In the case of the periodontal abscess, the pulp of the involved tooth is not likely to be necrotic, although, by chance, it could be. Percussion proves that the periodontal abscess is not as painful as the apical abscess. The reason is quite clear. The periodontal abscess is a “lateral” abscess, found on the side of the root, so that percussion causes little increase in pressure. On the other hand, percussion against the inflamed periapex of the tooth with AAA induces a great increase in pressure owing to the wedging effect of the tapering root.

The adjacent teeth involved in the swollen area may also be painful to percussion, and they register an increased reading on the pulp tester owing to the collateral edema. The adjacent teeth, however, are not nearly as painful to percussion as the involved tooth and usually register within normal limits in pulp testing. Multiple loss of vitality may follow an accident so that a number of adjacent teeth could test nonvital, but usually only one is abscessed.

In contrast to percussion, thermal tests have little value. Extremes of heat may increase gas expansion in the area and thereby increase the pain momentarily. Cold may give slight relief but usually does nothing at all. Palpation of the area reveals the swelling, and the pressure increases the discomfort.

**Treatment.** Under a special section devoted to these problems in Chapter 12, treatment of the AAA is discussed in detail. Suffice it to say here that drainage is established through the root canal if the abscess is in its initial stage, or by incision if the abscess is fluctuant. Trephination may also be performed to establish drainage and relieve pressure. The occlusion is relieved and a regimen of systemic antibiotics and either hot rinses or cold applications is prescribed for the patient depending on the stage of development of the abscess.

The pain often can be controlled with mild analgesics such as acetaminophen. However, hydrocodone or meperidine (Demerol) must be prescribed for severe cases.

Endodontic therapy or extraction, whichever is indicated, is completed after the acute symptoms have sub-
sided and while the patient is still receiving antibiotics. Periradicular surgery is rarely necessary in treating these cases.

**Chronic Apical Periodontitis.** Chronic apical periodontitis is seldom painful and is thoroughly discussed in chapter 5 under "Periradicular Pathology."

**Treatment.** Endodontic therapy is usually indicated for the tooth involved with CAP. This is sometimes followed by periradicular surgery, but only where indicated.

**Chronic Apical Abscess.** Also called suppurative apical periodontitis, CAA is generally free of symptoms. There may be stages in the long history of such a lesion when a draining fistula closes, and mild swelling and discomfort ensue. The patient reports that the abscess drains daily or that opening the abscess with a needle relieves the discomfort.

Many cases of suppurative apical periodontitis are so painless that they go undetected for years until revealed by radiography.

**Etiology.** Chronic apical abscess is the inflammatory response to an infection by bacteria of low virulence from the root canal. As stated previously, the only discomfort associated with a CAA is that related to the occasional closing of the draining fistula with attendant pressure. This chronic lesion, however, may develop an acute exacerbation, the phoenix abscess, and when this happens, the patient has all of the problems of an AAA. In this event, the pain and swelling are magnified owing to the large preexisting lesion.

**Examination.** On questioning, the patient with a previously undetected CAA may remember a particularly stormy session in the involved area or perhaps a traumatic incident in which the pulp was devitalized by a blow. There has been no discomfort since, however.

**Chronic apical abscess** is frequently associated with long-standing dental restorations such as full gold or jacket crowns, large composites or amalgams, and extensive bridgework. Occasionally, a routine radiograph reveals a CAA, associated with a discolored anterior tooth. This may appear as an area of diffuse radiolucency around the apex of the tooth in question and may vary from a minor lesion to a massive loss of bone. External resorption of the root end is also a common finding.

The lesion of CAA, easiest to detect, has an associated draining fistula, usually intraoral, seldom cutaneous. This sinus tract, lined with inflammatory tissue, drains the abscess through a stoma into the oral cavity. It is the closing of this tract that causes the patient discomfort.

**Treatment.** If the tooth involved with CAA can be saved, it may be retained by endodontic therapy. Periradicular surgery is sometimes indicated for these pathologic lesions. The chronic lesion that becomes acutely infected must be treated as an AAA until the symptoms have subsided. The tooth may then be handled as an endodontic case or extracted, as conditions indicate.

**Apical Cyst.** The apical cyst, per se, is painless unless it becomes infected. In that event, the case should be handled as an AAA. The apical cyst is discussed in chapter 5 under "Periradicular Pathology."

**Treatment.** When treated endodontically, the apical cyst may be enucleated during periradicular surgery.

**PERIODONTAL LESION PAIN**

Few periodontal lesions are severely painful. The causes of these lesions are divided into diseases that attack just the gingiva and those that involve the deeper periodontal complex. Two uncomfortable lesions that involve the gingiva and mucosa are acute necrotizing ulcerative gingivitis and herpes simplex. These diseases offer no severe problems in the differential diagnosis of pain because both lesions are diagnosed from their appearance and/or odor.

Two painful conditions that involve the pericemen tal structures and must be differentiated are the acute gingival or periodontal abscess and pericoronitis.

**Acute Gingival or Periodontal Abscess**

The patient with an acute periodontal abscess seeks treatment for a tooth that is painful to move or to bite on. The pain, however, is not as deep-seated or throbbing as that of an AAA. Although some localized swelling is present, it is not as extensive as with the AAA.

**Etiology.** The acute periodontal or gingival abscess develops from a virulent infection of an existing periodontal pocket or as an apical extension of infection from a gingival pocket. Most gingival abscesses are associated with traumatic injury to the gingiva or periodontium by a mechanical force. Both types of abscess are frequently seen in patients who have compulsive clenching or bruxism.

**Examination.** Although the involved tooth may be painful to movement, it is not as painful as the tooth involved in an AAA. Furthermore, the location of the abscess is usually different; the periodontal abscess "points” opposite the coronal third of the root, whereas the apical abscess generally “points” opposite the apex.

The electric pulp tester is the surest method of differentiation. The necrotic, infected pulp causing an apical abscess always gives an essentially negative response to testing, whereas the tooth involved with the
**periodontal abscess** is generally vital. Use of the periodontal probe often reveals a tract from the gingival sulcus to the abscess.

**Treatment.** The reader is referred to a periodontics text for information on treatment.

**Pericoronitis**

The common complaint of the patient with **pericoronitis** is severe radiating pain in the posterior mouth and the inability to comfortably open or close the mandible. Not only is it painful to close against the inflamed operculum distal to the erupting mandibular molar, but the pain of muscle trismus limits translation of the mandible as well. The tissue distal to the erupting molar is most painful to touch, especially during eating. The pain radiates through the region, down into the neck, and up into the ear and can easily be confused with pulp pain. Occasionally, an erupting third molar elicits the same deep, spreading pain well before the tooth breaks through the oral epithelium.

**Etiology.** **Pericoronitis** is caused by injury and infection of the pericoronal tissue associated with erupting molars, usually mandibular third molars. The tissue may be injured during eating by trauma from food such as peanuts or bread crust. The infection begins under the operculum and extends with attendant swelling around the entire unerupted crown. This area is frequently a source of primary infection with *Borrelia vincentii* and *Fusiformis dentium*.

**Examination.** The history of trismus and discomfort on opening or closing the mandible is indicative of **pericoronitis**. When the operculum is palpated or probed, it is found to be swollen and exquisitely painful. The patient usually assumes that it is the tooth that is painful.

**Pericoronitis** must sometimes be differentiated from a periodontal abscess commonly occurring along the distal aspect of the second molars. Again, the periodontal abscess is not nearly as painful as **pericoronitis**.

**Treatment.** The reader is referred to an oral surgery text for information on treatment of **pericoronitis**.

**REFERRED PAIN**

**Referred Pulp Pain**

One of the most frequently encountered and most baffling phenomena with which the dental diagnostician must deal is the problem of referred pulp pain. Texts and articles discussing this subject frequently give “pat rules” of pain reference with the implication that if pain is to be referred from a tooth, it is always referred in a particular pattern. This is not so, as anyone active in diagnosis soon discovers. Quite bizarre reference pathways are frequently encountered, and the clinician soon comes to realize that almost any reference, except across the midline, is possible.

**Symptoms.** Glick has well illustrated referred pain from pulpalgia—from tooth to tooth and from tooth to nearby cutaneous and deep structures. Figures 7-9 and 7-10 illustrate this information to facilitate diagnosis by visual association. The leading published scientific study on pulp pain and referred pain is that of Robertson et al. These authors produced toothache by placing stimulating electrodes into defects in the enamel of their own teeth. They found that by delivering up to 10.0 volts to the teeth, severe pain could be induced. Moreover, they discovered that when they maintained the shock for 10 minutes, the pain would be referred out of the teeth and over the entire distribution of the involved division of the nerve. Systematically and cleverly, these researchers mapped and described the reference pain from a number of teeth, mandibular and maxillary alike (Figures 7-11 and 7-12).

What Roberston et al. described from experimentation has been seen countless times by dentists—the patient with advanced acute pulpalgia who is suffering localized and referred pain with all of the attendant systemic signs and symptoms. They further experimented with the reduction of referred pain first by injecting anesthetics into the area of referred pain on the face and scalp and, second, into the area of the original “noxious stimulation,” that is, the tooth. As Figure 7-13 demonstrates, only partial relief from pain developed following procaine injection into the referred area; however, complete relief from pain was experienced when the region of the original source of pain was anesthetized.

Robertson et al. also found that protracted pain, as found in osteomyelitis of the mandible, led to sustained contraction and pain of the muscles of the face, head, and neck. In this case (Figure 7-14), when the source area of pain in the mandible was anesthetized, the referred pain area involved with the third division of the fifth nerve was abolished. On the other hand, the pain areas owing to spasm of the muscles of the neck continued. The phenomenon of myofascial trigger point pain and dysfunction developing as a result of pulpalgia is exactly the reverse of referred tooth or jaw pain from spasm of the trapezius muscle or the muscles of mastication as described by Travell (see Figures 8-28, 8-29, and 8-30).

The interesting research of Ray and Wolff would seem to confirm the reference of deep pulp pain into more superficial and cutaneous associated regions but could hardly be construed to explain the pain referred...
Figure 7.9  Referred pain pathways from teeth involved with pulpalgia to other teeth as well as to the immediate area.
Black signifies tooth with pulpalgia; stippled areas, the site of referred pain. A, The maxillary canine may refer to the maxillary first or second premolars and/or the first or second molars, as well as to the mandibular first or second premolars. B, Maxillary premolars may refer pain to the mandibular premolars. The reverse is also true. C, Mandibular incisors, canine, and first premolar may refer pain into the mental area. D, The mandibular second premolar may refer pain into the mental and midramus area. E, Mandibular first or second premolars may also refer pain into maxillary molars. F, Mandibular molars may refer pain forward to the mandibular premolars. Adapted with permission from Glick DH.64
Figure 7-10  Pain referred from pulpalgia to structures remote from the involved tooth. Black indicates teeth involved in pulpalgia; stippled areas, remote areas of referred pain. A, Maxillary incisors may refer pain to frontal area. B, Maxillary canine and first premolar may refer pain into the nasolabial area and orbit. C, The maxillary second premolar and first molar may refer pain to the maxilla and back to the temporal region. D, Maxillary second and third molars may refer pain to mandibular molar area and occasionally into the ear. E, Mandibular first and second molars may commonly refer pain to the ear and to the angle of the mandible. F, The mandibular third molar may refer pain to the ear and occasionally to the superior laryngeal area. Adapted with permission from Glick DH.64
Figure 7-11  Pain referred from a maxillary second premolar. Pain was initially experienced locally in the tooth following noxious stimulation by 10 volts of electricity. Within 5 minutes, numbness, fullness in the ear, and muscle stiffness had developed, in addition to steady pain along the homolateral temporal, zygomatic, and supraorbital areas. The headache reached its maximum distribution and intensity within 20 minutes after cessation of toothache. Reproduced with permission from Wolff HG. Headache and other head pain. New York: Oxford University Press; 1950.

Figure 7-12  Electrical stimulation of a lower first molar maintained severe toothache for 10 minutes. Pain was referred into the ear canal and throughout the upper and lower jaws, over the zygoma and temple, to the top of the ear; a sensation of fullness and ache persisted in the ear. Numbness and stiffness of the masseter muscle developed. Twenty minutes after stimulation, severe “lower-half” headache developed throughout the region. Reproduced with permission from Wolff HG. Headache and other head pain. New York: Oxford University Press; 1950.
from one tooth to another or from the paranasal tissue into the teeth.67 This type of referred pain has been discussed by Ruch and Fulton as habit reference.68 “Evidence that reference of sensation is a learned phenomenon,” they stated, “can be found in the clinical observation that a pain may be referred not to its usual point of reference but to the site of a previous surgical operation, trauma, or localized pathological process.”68

“Habit reference” was verified by Hutchins and Reynolds, who demonstrated that teeth filled without benefit of local anesthesia could be made to ache when the homolateral nasal wall was stimulated by a needle prick in the vicinity of the maxillary sinus ostium.69 Ruch and Fulton, in reviewing the research of Hutchins and Reynolds, looked on the traumatized sensory nerves of involved dental pulps as a “learned response.”68 They
stated further, “The pain impulses from the sinus, conducted in an overlapping pathway, were simply given the previously learned reference for impulses in that path.”

This same thought may be projected to explain how pain in one tooth (possibly even in the opposing homolateral arch) could be referred there from pulpitis in another tooth some distance away. The pulp of the referred tooth might well have been previously traumatized by caries, a blow, or a dental procedure without anesthesia. This injured pulp then becomes the fertile ground on which the seeds of referred response could be sown in the future. This might also explain why pain is referred to teeth when the patient has mumps or inflammation of the temporomandibular joint.

Continuing further with their studies, Reynolds and Hutchins found that they could virtually eliminate referred pulp pain by procaine block anesthesia. This was done by repeating their previous experiment in which they performed traumatic dental work without anesthesia on both sides of the maxillary arch and later demonstrated that pain was referred to these teeth from a pinprick of the maxillary ostium in the nose. After this fact was well established, the teeth on the right side were all anesthetized. Two weeks following these injections, the right side was again tested with stimulation of the ostium. Amazingly, no referred pain could be elicited on any tooth that had been previously anesthetized. On the left side, however, which had not been anesthetized, the teeth still responded with referred pain when the left maxillary ostium was stimulated. Therefore, the importance of rendering dental treatment under local anesthesia is emphasized by Reynolds and Hutchins since pain was not referred to teeth treated under local anesthesia.

EXTRAORAL PAIN

As explained above, pain from sources outside the oral cavity may refer into the oral cavity. The reverse is also true.

These diagnostic and treatment problems will be dealt with in some depth in chapter 8. To complete the record, however, these sites will merely be mentioned at this point.

Atypical Toothache

Rees and Harris described a disorder that they called atypical odontalgia. Patients present themselves with all of the typical features of an acute toothache—severe, throbbing, continuous pain starting in one quadrant but spreading even across the midline. Also referred to as “dental migraine” or “phantom tooth pain,” this condition is often associated with patients suffering from unipolar, or common, depression. A recent review of 28 cases, followed since 1979 from a cohort of 120 cases, revealed that 81% of the patients were female and ranged from 13 to 80 years of age (mean 42.6 years). The pain was located in the teeth, jaws, or gingiva 93% of the time. However, 14% said that the pain affected areas of the face—the cheek and around the eyes and ears. Only 4% reported tongue pain. The onset of the pain was precipitated by dental procedures 31% of the time. Tricyclic and monoamine oxidase inhibitor antidepressant therapy relieved pain in many of the cases. Because it falls within the descriptive area of “atypical facial neuralgia,” this type of pain will be discussed further in that section of chapter 8.

Salivary Gland Disorders

The salivary glands can be affected by many diseases, including obstruction, infection, degeneration, and tumor growth. Pain and tenderness, however, are usually found in association with inflammation of the gland itself. In Sjögren’s syndrome, parotitis is also accompanied by diminished salivation and lacrimation and some other connective tissue disorder, such as lupus erythematosus or rheumatoid arthritis. Pain from any of these conditions will refer to the teeth. For further details, see chapter 8.

Ear Pain

Pain within the ear can be caused by a disease within the ear and related structures as with otitis media or mastoiditis. Pain may also be referred to the ear from many other head and neck structures including the teeth, temporomandibular joint, tonsils, tongue, throat, trachea, and thyroid. This is because the ear is innervated by cranial nerves V, VII, VIII, IX, X, XI, and C1, C2, and C3.

Sinus and Paranasal Pain

Sinusitis is a common cause of dull, constant pain. The location of this pain can vary from the maxilla and maxillary teeth in maxillary sinusitis to the upper orbit and frontal process in frontal sinusitis, between and behind the eyes in ethmoid sinusitis, and at the junction of the hard and soft palate, occiput, and mastoid process in sphenoid sinusitis. Pain from the sinuses may be referred into the oral cavity, the teeth in particular. The reverse is also true; that is, pain from the teeth or from periapical lesions may be discerned as sinus pain or may be the source of maxillary sinus disease. Selden has pointed out, in discussing the oral-antral syndrome, that about 25% of chronic maxillary sinusitis is secondary to dental infections. Because of the extraoral nature of sinus and paranasal pain and disease, it will be considered in greater depth in chapter 8.
Myocardial Infarction, Coronary Thrombosis, Angina Pectoris, and Thyroid Disease.

It is hard to imagine that a site as distant from the oral cavity as the heart may refer pain into the teeth and jaws. But refer it does, in a most confusing manner. The dentist must be quite astute to recognize that the “toothache” described by the patient is actually reflecting serious cardiac disease.

Other sources of referred pain to the jaws are cardiopasm (spasm of the esophageal cardiac sphincter associated with hiatal hernia of the diaphragm) and thyroid disease. In addition to these pain references, women have reported oral discomfort associated with the menopause—43% of peri- and postmenopausal women reporting oral pain compared with only 6% of premenopausal women.77 Two-thirds of the menopausal women reporting discomfort were relieved of their symptoms after hormone replacement therapy.77

Also reported is pain from non-Hodgkin’s lymphoma disguised as odontogenic pain. There is a higher incidence of this disease in patients with acquired immune deficiency syndrome (AIDS).78

Because of the extraoral nature of these sources of referred oral pain, they will be discussed further in chapter 8.

CONCLUSION

In conclusion, one could state that the vast majority of patients who present themselves in pain are suffering acute pain. It would also be fair to state that most of these patients are suffering from a toothache (dental pulp algia).

The dentist who is experienced in pain diagnosis will systematically examine and test to narrow down the suspected source of the pain. The inexperienced often push ahead and blunder into a serious error in misdiagnosis and treatment. One must deal with certainty, and if one is uncertain of the tooth or condition involved, the perplexing case should be referred to an expert diagnostician.

Chronic pain is an entirely different matter. Diagnosis and treatment of these cases might involve a team of experts at a tertiary care pain center. For this reason, an entire chapter, chapter 11, is devoted to these baffling cases.

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