Endodontic flareup

- Flareup is described as the occurrence of pain, swelling or the combination of these during the course of root canal therapy, which results in unscheduled visits by patients

Gerald W Harrington,1992

- Flareups may occur with the best of the therapy, but most flareups occur when improper treatment is rendered or when insufficient time is allowed for specific modalities in therapy according to Franklin S Weine

- Inter appointment flare up causes
- 1.pain
- 2.swelling
- 3. occurs within hours/ days following procedure
- 4. requires unscheduled visit for emergency treatment
- Incidence- 1.4-16% (siqueira 2002)

Predisposing factors

- Age
- Gender
- Tooth type
- Pulpal status
- Pre-operative pain
- Allergies
- Sinus tract

Morse *etal* 1986. Torabinejad 1988, Siqueria 2002

Predisposing factors

- ✓ Flareup and post-operative sensitivity rarely occur in older patients due to the narrowing of the diameter of the root canal and decreased blood flow in the alveolar bone resulting in weaker inflammatory response
- ✓ post-operative pain is more common among women than men
- ✓ Flare rate after endodontic treatment procedures is low in patients using systemic steroids as treatment for systemic diseases
- ✓ It is established that 47-60% of patients having asymptomatic necrotic pulp experience pain defined from medium to acute during the first 24 hours after endodontic treatment

- Bone destruction which is visible in dental radiograph is said to be a risk factor of post-operative pain and flare-up
- 80% of patients who feel tooth pain before the beginning of the treatment usually feel the pain after treatment
- Glennon etal study results show that temporary pain is felt 17 times more often when the canals of the molar teeth are treated compared to other teeth types
- Yold etal study summarizes that flare-up rate is 4-9 times higher after one visit endodontic retreatment compared to retreatment by two – visits

Hypothesis for flare ups

- Dr Seltzer discussed a number of hypothesis thought to be related to the etiology of flareups

✓ Alteration of the local adaption syndrome .

✓ Changes in periapical tissue pressure .

✓ Microbial factors.

✓ Effects of chemical mediators.

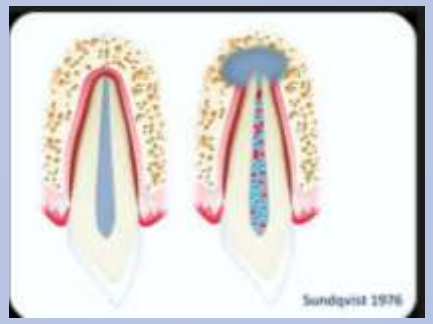
✓ Changes in cyclic nucleotides.

✓ Immunological phenomena.

✓ Various psychological factors

- Alteration of local adaptation syndrome
- There is local tissue adaptation to the environment and violent reaction occurs due to change in new irritant
- When endodontic therapy is performed a new irritant in form of medicament, irrigating solution, or tissue proteins may be introduced in the granulomatous lesion and then a violent reaction follows leading to liquefaction necrosis indicating of an alteration.

- Microbial Factors
- Sundquivst in his study concluded that in all flare up cases *Bacteroides melanninogenicus,* a gram negative anaerobic rod was present which was also endorsed by Grifee etal saying that symptomless infected teeth did not contain Bacteroides melanninogenicus



- This gram negative rod produces collagenolytic , fibrinolytic enzymes and endotoxins which activates Hageman factor releasing bradykinin , a potent pain mediator.

- Effects of chemical mediators
- Cell mediators Like histamine, serotonin, prostaglandins, platelet activating factors, leukotrienes etc. are capable of producing severe pain, which are released from cells.
- Changes in periapical tissue pressure
- Mohorn et al showed that endodontic therapy causes pressure changes in periapical area in both directions
- A positive periapical pressure i.e excessive exudate not absorbed by lymphatic system, presses on nerve endings causing pain.
- In contrast a negative periapical pressure leads to aspiration of microbes and altered tissue proteins from root canal to periapical area resulting in increased inflammatory response and pain. In such cases no drainage occurs when root canal is opened

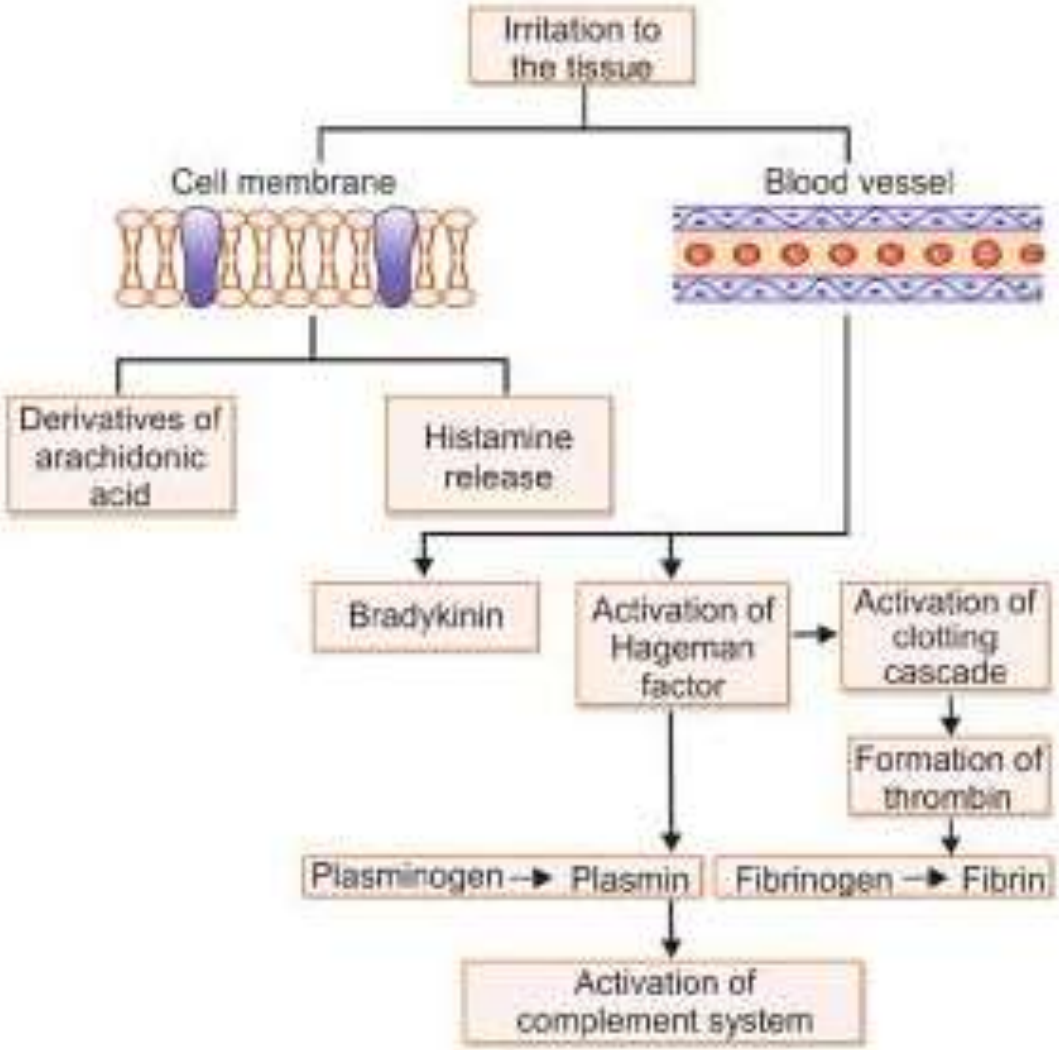


Fig. 21.12 Tissue response to irritation

- Immunological phenomenon

1. Pulp has capacity to produce antibodies like immunoglobulin IgG, IgM, IgA against dental caries
2. Immunoglobulins were detected in periradicular cysts & granulomas
3. Amongst immunoglobulin, IgG is the most commonly present(70-74%) in periradicular lesions
4. Cymrrman et al observed cytotoxic and helper T lymphocytes in excised periapical granulomas.

- Psychological factors
- Anxiety, fear, psychosis, apprehension & previous traumatic dental experience means a lot to dental patients especially during root canal procedures
- These anxieties aggravate and intensify painful episodes

Propagation of pain

Periradicular injury

Release of chemical substances

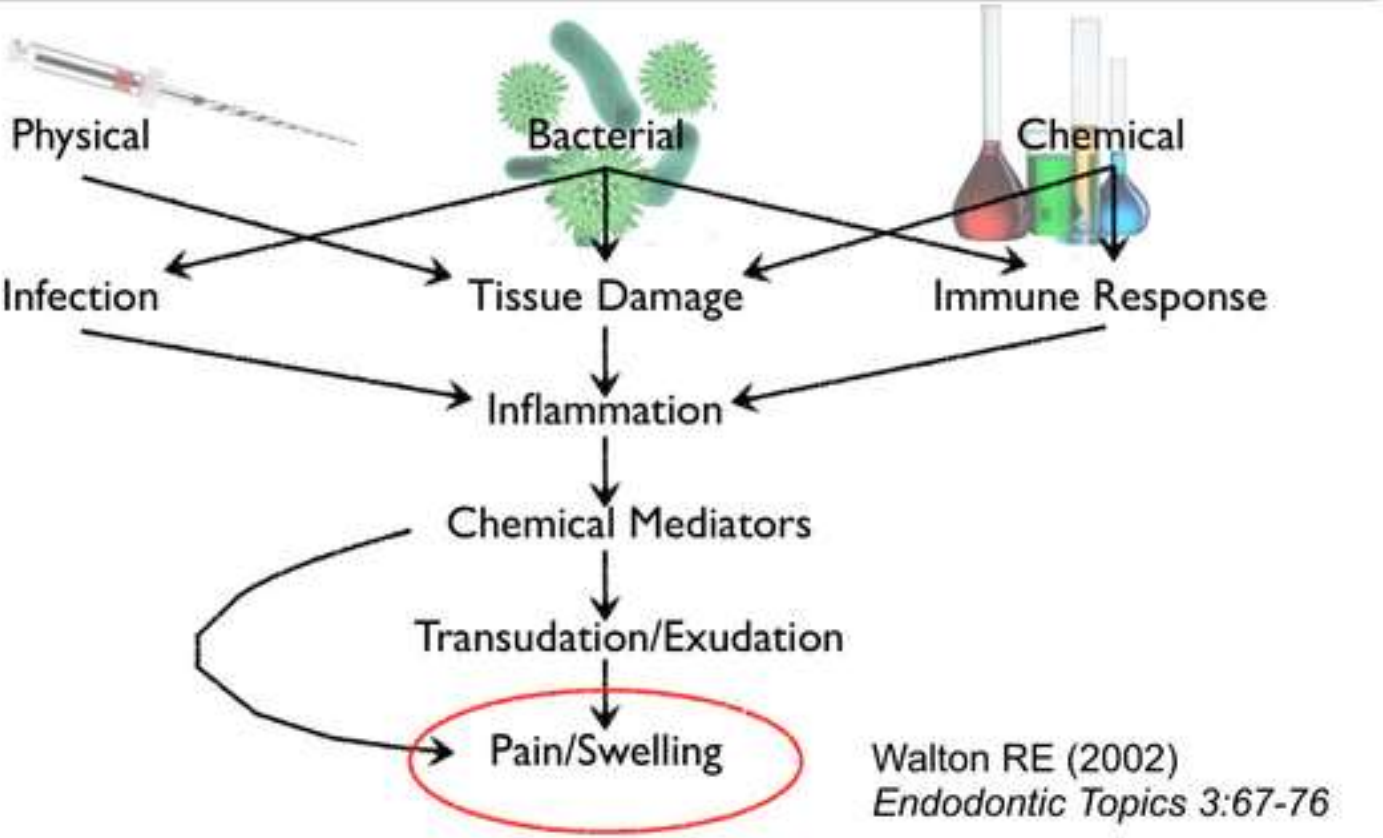
Vasodilation, vascular permeability, chemotaxis

Vascular permeability causes Oedema and exudation

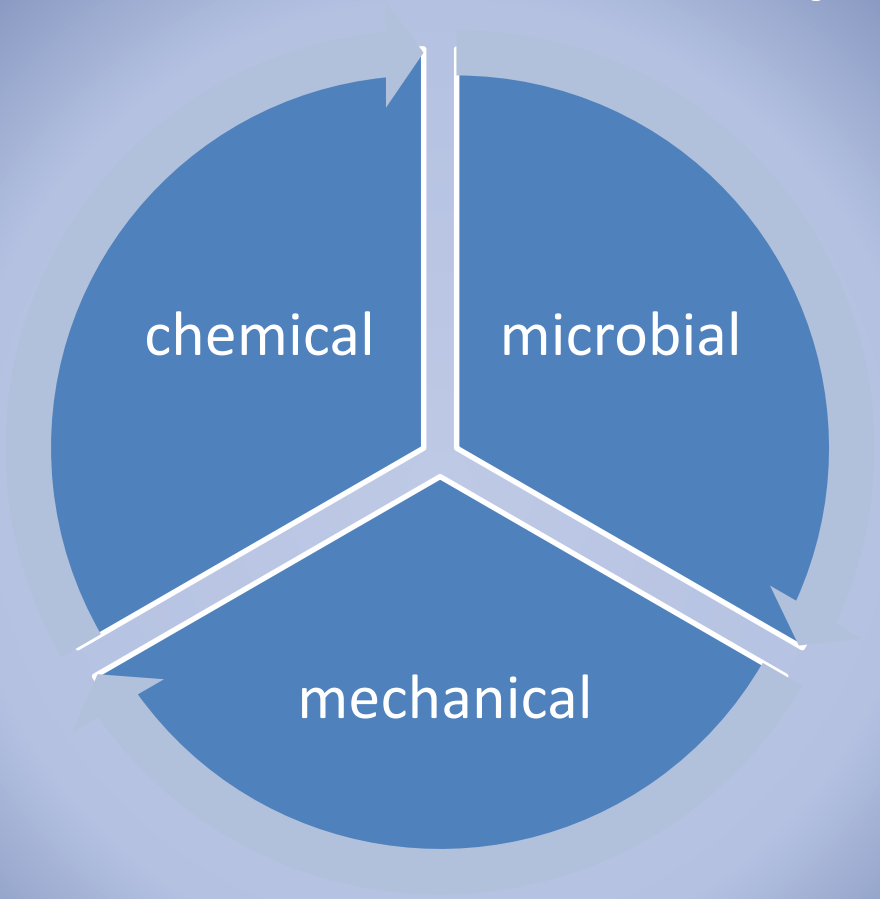
some chemical mediators directly stimulate nerve fibres

Increase in tissue hydrostatic pressure-compression of nerves

Endodontic flareup



Causes of flare up



Most of the flare ups occur as a result of acute periradicular inflammation secondary to intracanal procedures

- **Mechanical cause**

✓ overinstrumentation of root canal

✓ extrusion of root canal filling materials

✓ incorrectly measured working length

✓ Inadequate removal of pulpal tissue

- Chemical causes

✓ Irrigation solutions

✓ Intracanal medicaments

✓ over extended Root fillings

Fig. 21.4 Periapical extrusion of debris

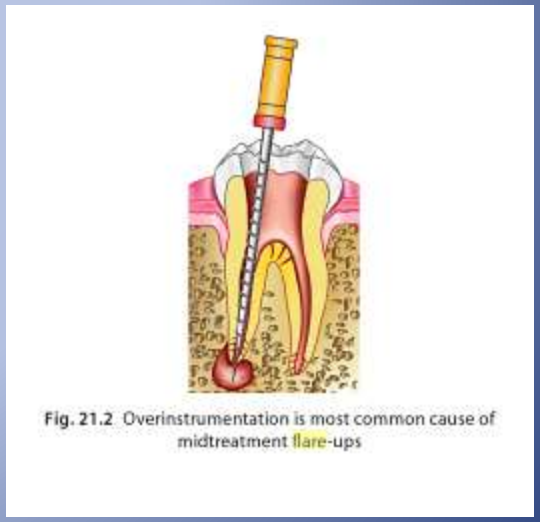


Fig. 21.2 Overinstrumentation is most common cause of midtreatment flare-ups

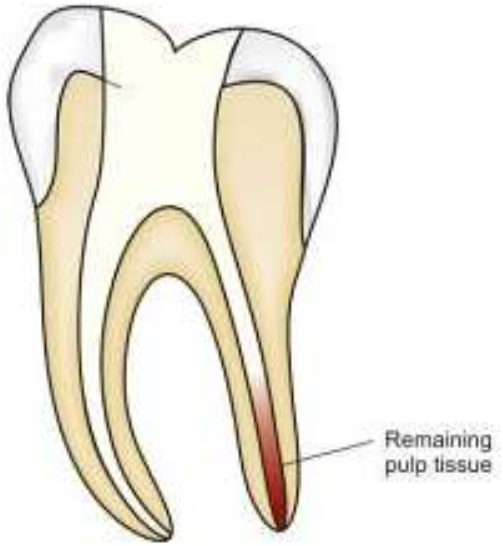


Fig. 21.3 Inadequate debridement of pulp tissue

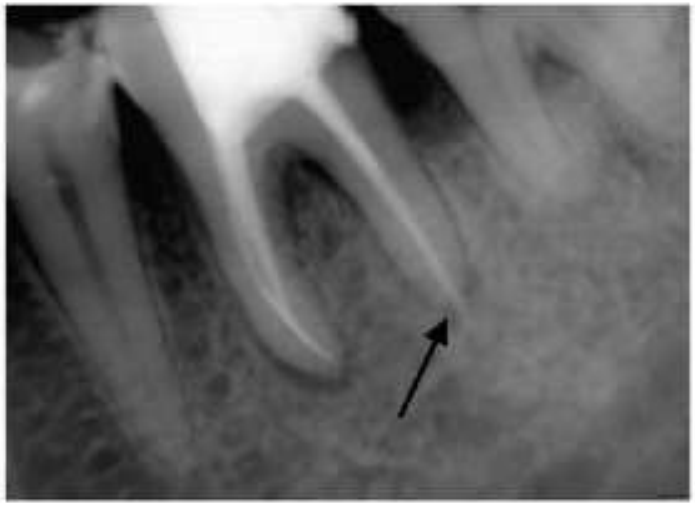


Fig. 21.5 Overextended filling material may result in severe postoperative pain

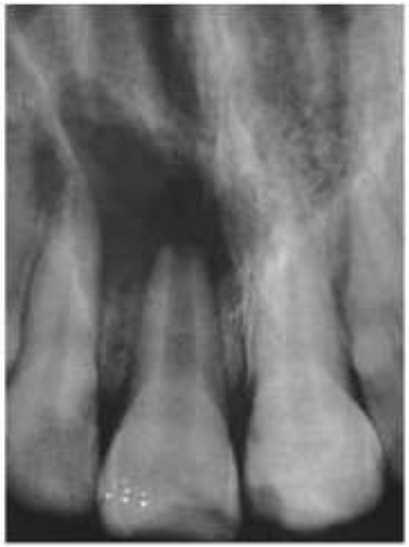


Fig. 21.6 A tooth with necrotic pulp and periapical radiolucency shows more incidence of flare-up than a tooth with vital pulp

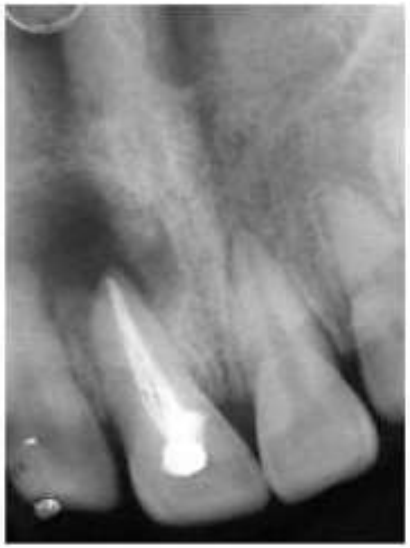


Fig. 21.7 Chances of flare-ups are more in retreatment cases

Microorganisms as cause for flareups

- ✓ Necrotic tissue in root canal is conducive for strict anaerobes with nutritional demands
- ✓ Porphyromonas is associated with symptomatic periradicular lesions
- ✓ Percussion pain frequently displayed peptostreptococcus. Prevotella species, porphyromonas endodontalis
- ✓ Gram negative anaerobic bacteria- associated with symptomatic lesions
- ✓ More virulent clonal types are found in symptomatic cases
- ✓ Pathogen must achieve sufficient numbers to initiate and maintain disease

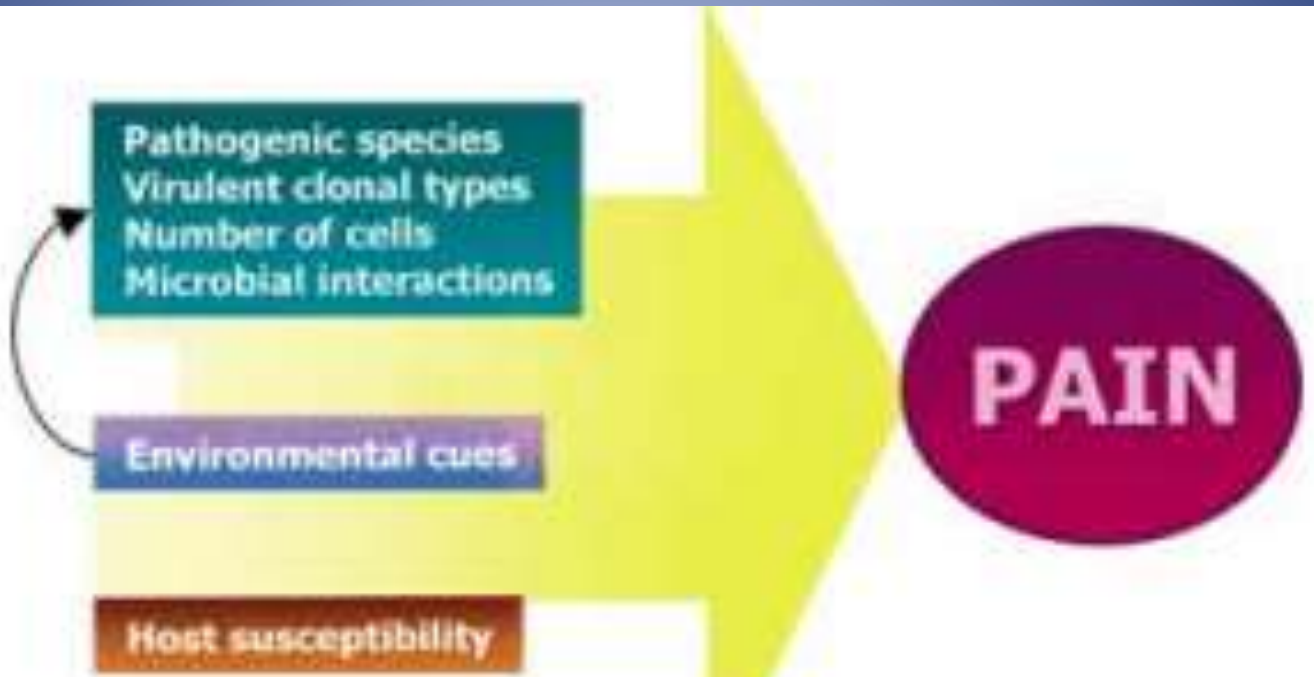
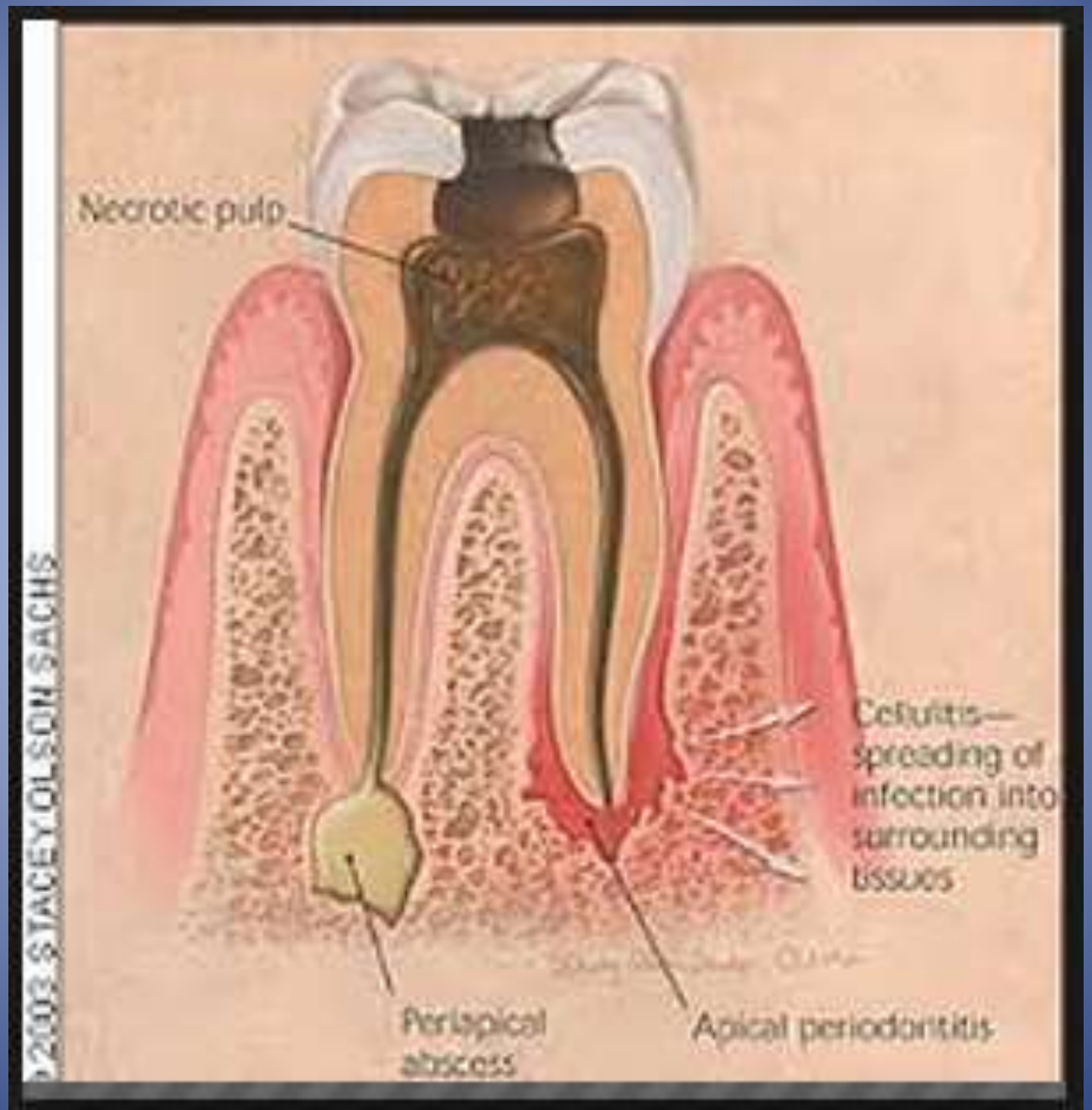


Figure 1 Factors influencing the development of pain associated with endodontic infections. In addition to the pathogenic species, other microbial and host-related factors are also highly likely to be involved in the pathogenesis of symptomatic periradicular diseases (see text for more discussion).

Clinical conditions

Apical periodontitis secondary to treatment

- Causes for this condition most frequently are over instrumentation or over medication or forcing debris into the periapical tissues.
- *Balance between the microbial aggresion and host defence of the periradicular tissues is disrupted*
- *Host will mobilize a acute inflammation to re establish the equilibrium*
- Apical foramen enlargement increases influx of exudate and blood in to root canal


Necrotic pulp
Cellulitis—spreading of infection into surrounding tissues
Periapical abscess
Apical periodontitis
©2003 STACEY OLSON SACHS

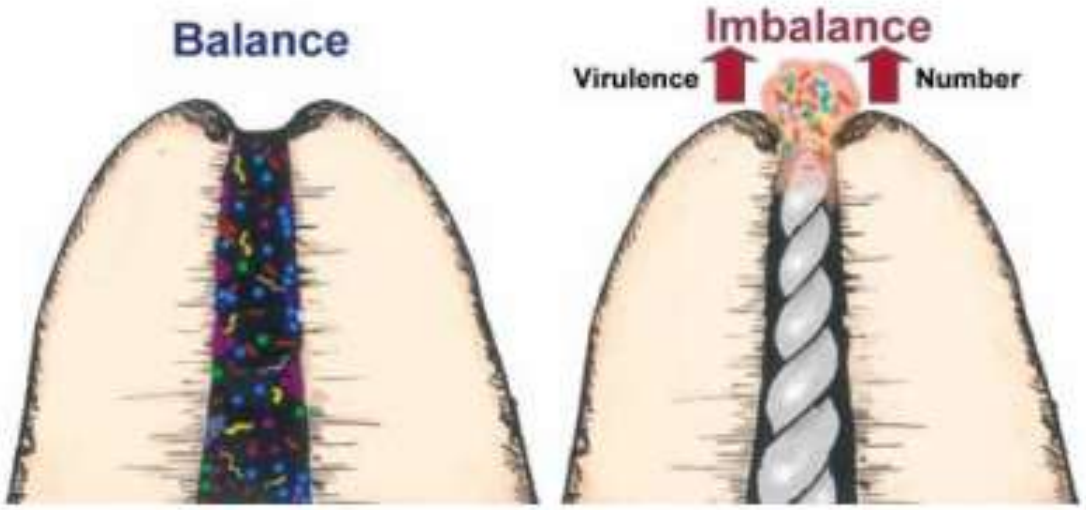
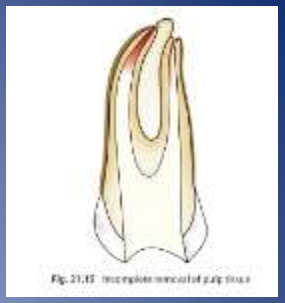


Figure 2 Apical extrusion of microorganisms and/or their products during chemo-mechanical procedures may induce acute periradicular inflammation to re-establish the balance between aggression and defence. Such response depends on both the number and virulence of extruded microorganisms.

Virulency , Polymicrobial nature and imbalance between human immune system and microorganisms contributes to flare up



- Incomplete removal of pulp tissues during the intial appointment-
- In some instances due to lack of time factor the endodontic therapy may consist of incomplete pulpectomy after a diagnosis of acute or chronic pulpitis. This situation generally occurs when the radicular pulp is already inflamed.

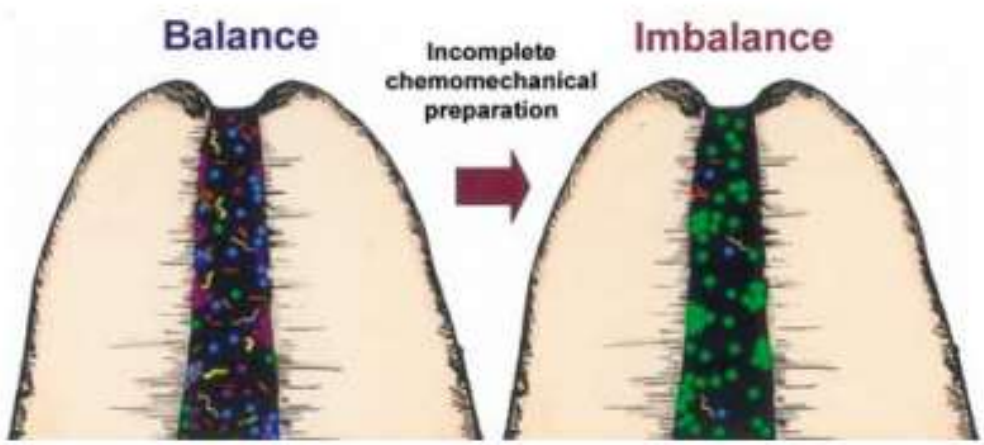


Figure 4 Incomplete chemo-mechanical preparation induces changes within the root canal system that may favour the overgrowth of certain species. If overgrown bacteria reach sufficient number and express virulence genes, they can induce damage to the periradicular tissues, and a flare-up may ensue.

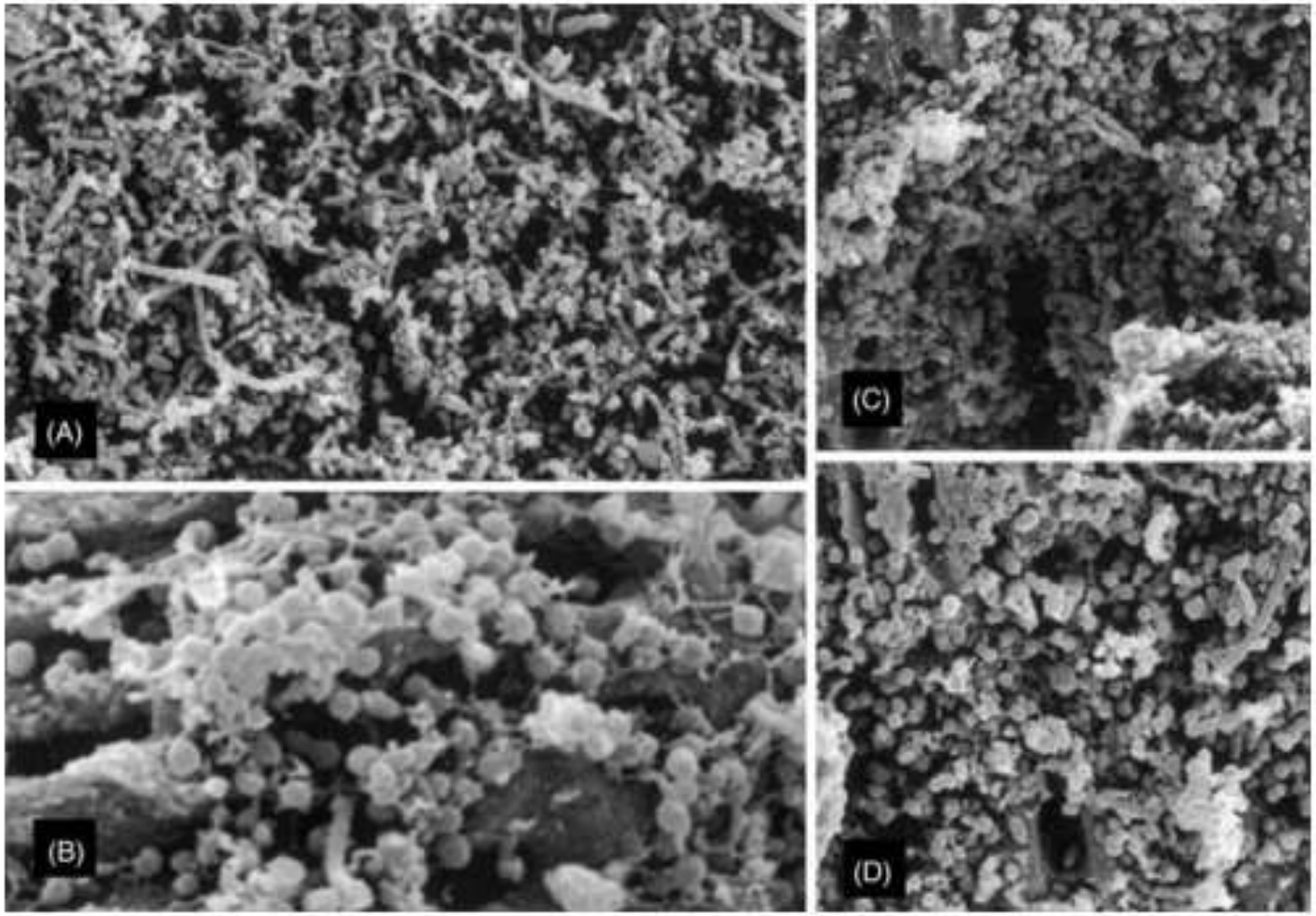
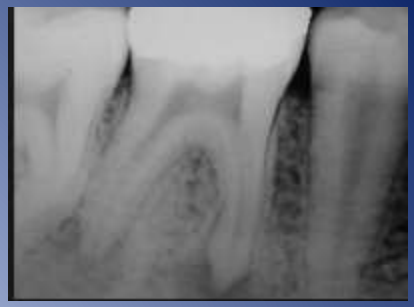
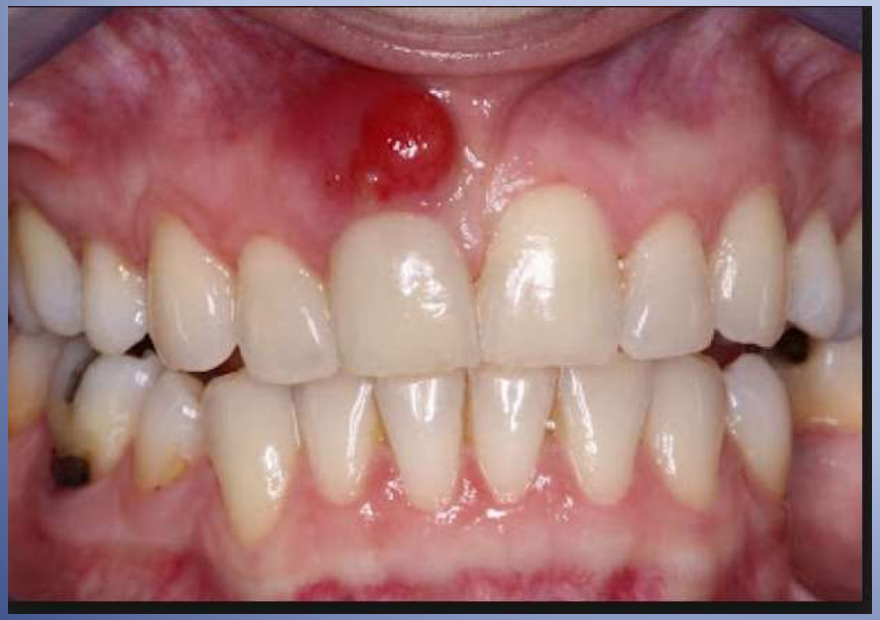


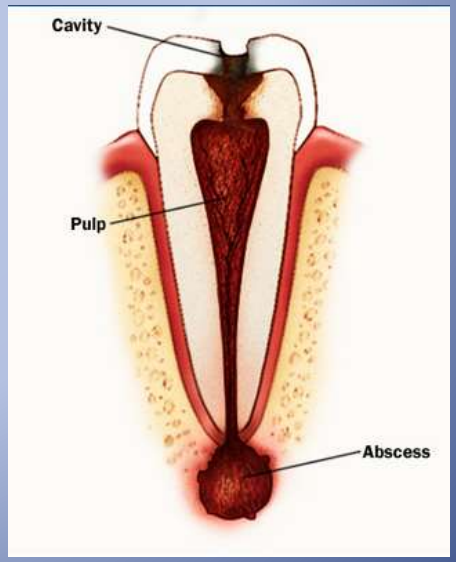
Figure 3 Scanning electron micrographs showing bacterial organizations within infected root canals associated with periradicular lesions. (A) Bacterial mixed community, composed of different morphotypes, resembling climax communities (original magnification ×3300). (B) Colony composed mainly by cocci and also by scarce bacilli, adhered to dentine. Some cells are invading dentinal tubules (original magnification ×4000). (C and D) Mixed bacterial communities predominated by coccal forms adhered to the dentinal walls at the apical part of the root canal (original magnifications ×1700 and ×1800, respectively).



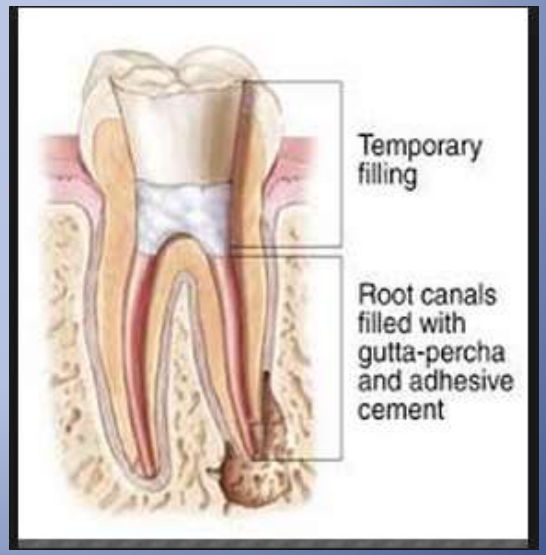
- Phoenix abscess-It is a condition that occurs in teeth with necrotic pulps and apical lesions that are asymptomatic.
- There is a exacerbation of a previously symptomless periradicular lesion.
- The reason for this phenomenon is thought to be due to the alteration of the internal environment of the root canal space during instrumentation which activates the bacterial flora
- Acute periapical peridontitis occurs due to overinstrumentation ,extrusion of canal contents through the apex ,leaving the tooth in traumatic occlusion or placing too much of intracanal medicament

- Recurrent periapical abscess - It is a condition where a tooth with an acute periapical abscess is relieved by emergency treatment after which the acute symptoms return. In some cases the abscess may recur more than once, due to micro organism of high virulence or poor host resistance





- **Secondary radicular infection**
- Introduction of new microbes occurs due to breach of aseptic chain
- **Sources of contamination**
- Dental plaque, calculus, leaking rubber dam
- Contaminated instruments
- Loss of temporary restoration
- Fracture of tooth structure
- Recurrent decay



Secondary radicular infection

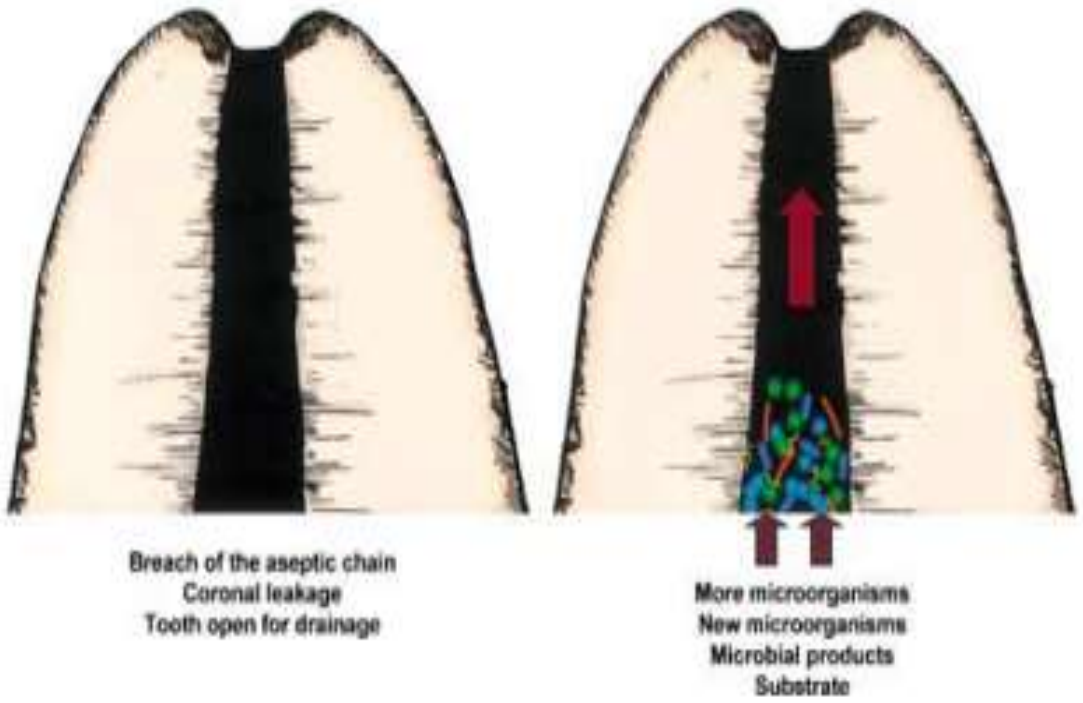


Figure 5 New microbial species, more microbial cells and substrate from saliva can be carried into the root canal system during treatment, between appointments or following treatment. If a secondary infection establishes itself, a flare-up may occur.

Increase in oxidation - reduction potential of microbes

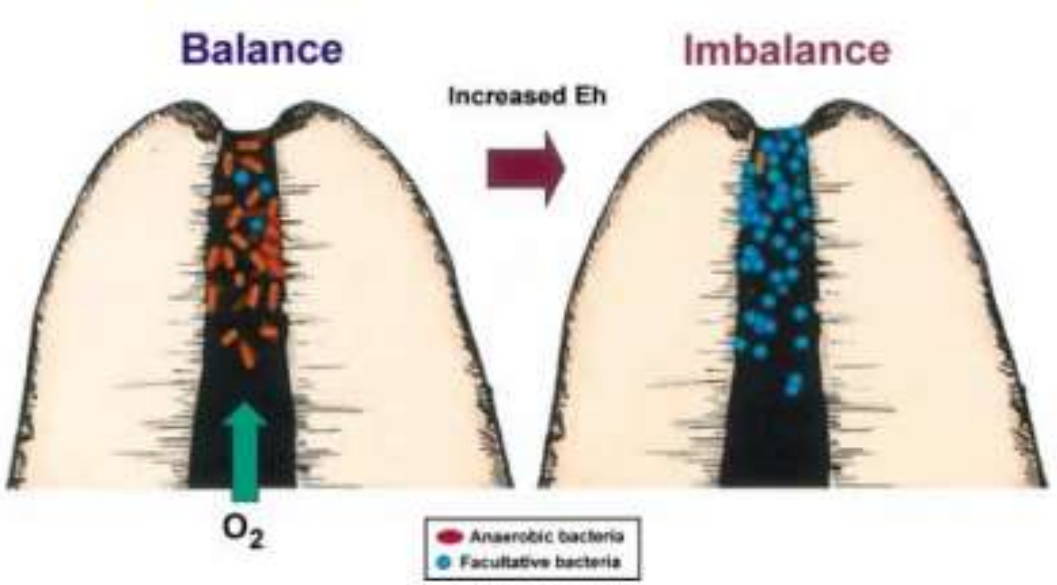
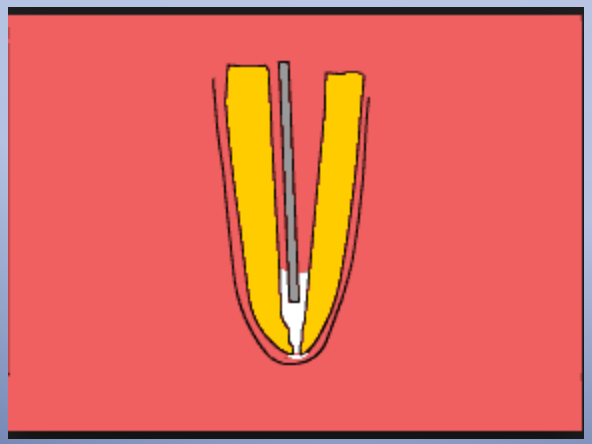


Figure 6 Entrance of oxygen into the root canal during treatment may favour the overgrowth of facultative bacteria that resisted chemo-mechanical procedures. This mechanism is only conjectural, and there is no clear evidence substantiating this theory.

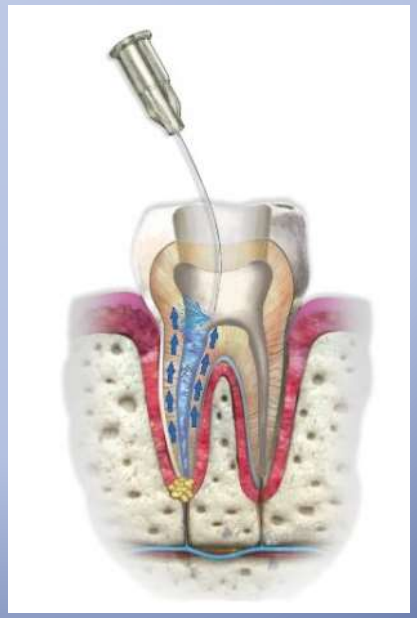
Prevention of flareups

- 1. selection of instrumentation techniques that extrude less amount of debris apically
- 2.completion of chemo-mechanical procedures in a single visit
- 3.use of antimicrobial intracanal medicaments
- 4.not leaving teeth open for drainage
- 5. maintaining the aseptic chain during intracanal procedures

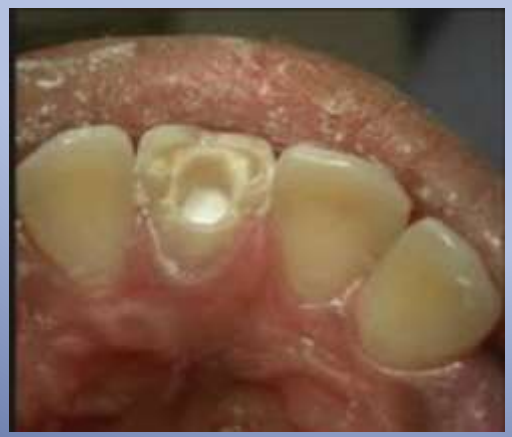
- selection of instrumentation techniques that extrude less amount of debris apically
- Crown down technique has demonstrated lesser amount of debris extrusion
- Copious and frequent irrigation enhances the removal of excess dentine, microbial cells and pulpal debris from root canal



- completion of chemo-mechanical procedures in a single visit
- Maximum removal of irritants from root canal relieves post operative discomfort
- Virulency and growth of microbial species can be restricted due to change in the environmental conditions



- use of antimicrobial intracanal medicaments
- Intracanal medicaments are required for maximum elimination of microbes not reached by instruments and irrigants
- Icm deny the space for microbial proliferation
- Post operative pain by persistent microbes and secondary microbial invaders can be prevented



- Do not leave the tooth open for drainage
- Leaving tooth open is the most direct way to permit the reinfection of the root canal
- If the tooth is left open microbial cells, products and substrate are allowed to gain access to the root canal and the periradicular tissues
-

- Maintain asepsis
- Clinician should be aware of the need to perform clinical procedures in aseptic condition

Table 1 Microbial mechanisms in the induction of flare-ups and respective preventive measures

Microbial mechanisms	Preventive measures
Apical extrusion of infected debris	Crown-down instrumentation techniques Instruments used with some sort of rotation action Copious and frequent irrigation
Changes in the endodontic microbiota and/or in environmental conditions	Completion of chemo-mechanical preparation in a single visit Placement of an antimicrobial intracanal medication that temporarily fills the root canal between appointments
Secondary intraradicular infections	Strict aseptic measures Proper coronal sealing Do not leave teeth open for drainage
Increase of the oxidation–reduction potential	Completion of chemo-mechanical preparation in a single visit Placement of an antimicrobial intracanal medication that temporarily fills the root canal between appointments

Management of flareup

- **Incision and drainage (I and D)**
- The rationale for an I and D procedure is to facilitate the evacuation of pus, microorganisms, and toxic products from the periradicular tissues. Moreover, it allows for the decompression of the associated increased periradicular tissue pressure and provides significant pain relief.
- **Intracanal medicaments**
- Clinical studies have demonstrated that post-treatment pain is neither prevented nor relieved by medicaments such as formocresol, camphorated paramonochlorophenol, eugenol, iodine potassium iodide, Ledermix, or calcium hydroxide.
- The use of intracanal steroids, nonsteriodal anti-inflammatory drugs (NSAIDs), or a corticosteroid–antibiotic compound has been shown to reduce post-treatment pain.

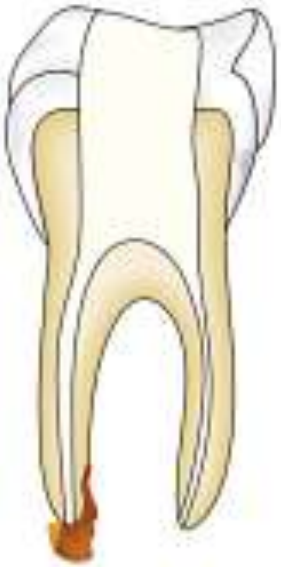


Fig. 21.20 Opening of pulp chamber to allow drainage

Fig. 21.19 Placement of $Ca(OH)_2$ in canal

- **Occlusal reduction**
- There appears to be minimal agreement in the dental literature as to the benefit of reducing the occlusion to prevent post-endodontic pain. Sensitivity to biting and chewing is perhaps due to increased levels of inflammatory mediators that stimulate periradicular nociceptors.
- Occlusal reduction may therefore alleviate the continued mechanical stimulation of the sensitized nociceptors

- **Non-narcotic analgesics**
- Non-narcotic analgesics, NSAIDs and acetaminophen, have effectively been used to treat patients with endodontic pain. These drugs produce analgesia by their actions on both the peripherally inflamed tissues as well as on certain regions of the brain and spinal cord
- Behavioral management- providing information about treatment can alleviate anxiety

Management of flare-ups

- Drainage through coronal access opening.
- Incision and drainage.
- Proper instrumentation.
- Trephination.
- Intracanal medicaments.
- Analgesics and antibiotics (when indicated).

Precautions to be taken by clinician

- ✓ Proper diagnosis
- ✓ Identify the correct tooth causing pain. Ascertain whether tooth is vital or non vital. Identify if tooth is associated with periapical lesion.
- ✓ Determine correct working length.- Radiographs. Apex locaters
- ✓ Complete extirpation of vital pulp.
- ✓ Irrigation - Preferably with combination of irrigants such as sodium hypochlorite and chlorhexedine.
- ❖ Avoid filing too close to the radiographic apex.
- ✓ Perform apical trephination only if necessary.
- ✓ Reduce tooth from occlusion especially if apex is severely violated by overinstrumentation.
- ✓ Placement of intracanal medicaments.
- ✓ Prescription of mild analgesics and antibiotics whenever condition warrants it .