**ROOT RESORPTION**: INVASIVE CERVICAL RESORPTION

* **Root Resorption**: loss of dental hard tissues as a result of clastic activities.
  + **BEWARE:** Do not confuse open apices in immature roots with root resorption.

Physiologic Root Resorption

Pathologic Root Resorption

* Under normal conditions, permanent teeth are resistant to resorption. **Odontoclasts** do not adhere to the non-mineralized layers covering the **external root surface – precementum** and the **root canal wall – predentin.**
  + Damage to the precementum, with a resultant breach in its integrity is the precipitating factor in all types of **external** resorption.
    - Pressure on the root surface during orthodontic treatment and from impacted teeth, cysts, and tumors may also denude the protective precementum from the root surface and therefore initiate **external root resorption**.
* Root resorption of permanent teeth does not occur naturally and is inflammatory in nature. Thus, root resorption in permanent dentition is a **pathologic event**. If left untreated, it may result in loss of the affected teeth.

# Root Resorption: classification

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| **Root Resorption** | | | | | | |
| **Internal** | | **External** | | | | |
| Inflammatory | Replacement | **Invasive Cervical Resorption** | Replacement Restoration | Transient Apical Breakdown | Surface Resorption | Inflammatory (Infection-Related) Resorption |

**External Inflammatory Resorption vs. Surface Resorption**EIR differs from surface resorption in that it is a progressive event that relies on microbial stimulation from the infected necrotic pulp of the affected tooth for its progression

**Also known as**External Cervical Resorption  
Supraosseous extracanal invasive resorption  
Subepithelial external root resorption  
Peripheral Inflammatory Root Resorption

# diagnostic features of external vs internal root resorption

* It is difficult to distinguish external from internal root resorption so misdiagnosis and incorrect treatment may occur. Most misdiagnoses are made between subepithelial external root resorption (Invasive cervical resorption) and internal root resorption.

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| Features | Internal Root Resorption | External Root Resorption |
| **Radiographic Features**  A change of angulation of x-rays gives an indication of whether a defect is internal or external | * Appears close to the canal, whatever the angle of the x-ray. * Uniform enlargement of the pulp space as an oval radiolucency * Outline of canal cannot be seen in resorptive defect * Does not involve the bone and as a rule, **radiolucency** is confined to root.   + However, in rare occasions, the internal defect perforates the root and bone adjacent to it is resorbed 🡺 appears radiolucent on radiograph | * A defect on the external aspect of the root moves away from the **canal** as the angulation changes. * By using the **Buccal Object Rule** it is usually possible to distinguish whether the external root defect is buccal or lingual. * Outline appears normal and usually seen running through the radiolucent defect * External root resorption is always accompanied by resorption in bone and the radiolucencies will be apparent in the root and adjacent bone. |
| **Sensibility Testing** | * Respond within normal limits to pulpal testing (they contain both vital and necrotic pulp tissues). * It is common to have -ve to sensitivity testing because often the coronal pulp has been removed or is necrotic, and the active **resorbing cells** are more apical in the canal. | * External inflammatory resorption in the apical and lateral aspects of the root involved an infected pulp space, so **no response** to sensitivity supports the diagnosis. |
| **Clinically** | * Advanced internal resorption involving pulp chamber is often associated with pink spots in the crown. |  |
| **Etiology** | Inflammation and secondary infection: caries, attrition, abrasion, erosion, cracked tooth, trauma, Ca(OH)2 pulpotomy, crown resorption, extreme heat, chronic pulpitis, idiopathic |  |
| **Treatment** | Immediate removal of the inflamed tissue and completion of RCT |  |

# External inflammatory resorption (EIR)

* EIR differs from surface resorption in that it is a progressive event that relies on **microbial stimulation** from the infected necrotic pulp of the affected tooth for its progression

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| **Definition** | Most common form of external root resorption after luxation injuries (5—18%) and avulsion injuries (30%) |
| **Pathogenesis** | Response to moderate or extensive injury to the PDL and associated pulpal infection. The initial injury to the root surface triggers a macrophage and osteoclast attack on the root surface. Osteoclasts are exposed to toxins from microorganisms located in the root canal and dentinal tubules. These **toxins** serve as direct **activators** of osteoclastic activity |
| **Treatment** | Removal of the causal agent; namely, infected necrotic pulpal tissue in root canal. |

# Invasive cervical resorption (External cervical resorption)

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| Location | Develops on the external root surface immediately apical to the epithelial attachment of the tooth |
| Stages | **Class 1:** Small lesion with shallow penetration into dentin  **Class 2**: Well-defined lesion that penetrates close to the pulp chamber with little extension into radicular dentin  **Class 3:** Deeper invasion, not only involving coronal dentin but also extending into the coronal 1/3rd of the root  **Class 4:** Large invasive processes that extends beyond the coronal 1/3rd of the root |
| Etiology | **Orthodontic** treatment, dental **trauma**, oral **surgery**, **periodontal** therapy, bruxism, intracoronal restorations, delayed eruption, enamel stripping, dental developmental defects, **internal bleaching**.  It might also be caused by a **combination** of predisposing factors (**in bold**) but no definitive cause-and-effect relationship has been established. **(cont. In page 4)** |
| Pathogenesis | **Hyperplastic resorptive tissue**, apparently derived from precursor PDL cells, invade the hard tissues of the tooth in a destructive and invasive fashion. Initially, the resorptive tissue is fibrovascular in nature. As the resorptive process extends more deeply into radicular root structure, it becomes fibro-osseous in character. |
| Clinical Features | In earlier stages it is asymptomatic, with no clinical signs and symptoms. The diagnosis is commonly made as result of a **chance radiographic finding** or a **pink discoloration** (due to granulation tissue undermining enamel). Pulp necrosis and periapical periodontitis develop as the process progresses.  **NOTE:** in apical and lateral external root resorption, pulp is nonvital, and granulation tissue is deep in the apical part of the root canal 🡺 pinkish discoloration can **not** be seen clinically.  **BEWARE**: Pulpal injury may cause discoloration even after only a few days. Initially changes tend to be **pink**, but subsequently, if the pulp does not recover and becomes necrotic, there may be a **grayish darkening** of the crown, often accompanied by **loss in translucency.** |
| Sensibility Testing | Since it does not involve the pulp (the bacteria are thought to originate from the tooth sulcus), a **normal response** is expected to sensitivity testing.  In advanced cases, it extends into the pulp causing irreversible pulpal changes followed by **necrosis**. |
| Radiographic Features | CBCT was significantly **more accurate** than periapical radiographs at diagnosing the presence and nature of root resorption. CBCT helps in:   * Eliminate the need for exploratory treatment * 3D assessment of the nature position and extent of resorptive defect * Determine whether ECR has perforated the root canal wall.   **Radiographic Features of ECR:**   * Very similar to those of Internal Root Resorption (IRR), but in ECR, the outline of the canal wall should be visible, intact, and should maintain its course as it passes through defect **(not true in advanced cases)** * Present as a radiolucency, often in but not confined to cervical region of the affected teeth. * **Irregular margins** are more common, but some have smooth and/or well-defined margins. |
| Management | **Objectives:**   1. Excavate the resorptive defect 2. Halt the resorptive process 3. Restore the hard tissue defect with an aesthetic filling material 4. Prevent and monitor the tooth for recurrence   **Trichloroacetic Acid:**   * The topical application of 90% aqueous solution of trichloroacetic acid (TCA) causes coagulation necrosis of the highly active and invasive resorptive tissue. * Precautions: Apply glycerol to adjacent soft tissues and insert glycerol-impregnated cotton roll into labial and buccal sulcus 🡺 Apply RD using a cuff technique 🡺 TCA should be absorbed into a small cotton pellet, then dampened on a gauze to remove excess.   **Treatment Steps: \***  Protective application of glycerol to adjacent soft tissues 🡺 Dental dam “cuff” placement for protection and isolation 🡺 TCA (on a small cotton pellet) is applied so that the resorptive tissue within the cavity undergoes coagulation necrosis 🡺 Curettage of the avascular tissue 🡺 Smoothening of the incisal margin with high-speed bur under water spray 🡺 GIC is placed in cavity. |
| Endodontic Implications | **Class 1 & 2**: Pulp is generally not involved and no indication for endodontic therapy. **Class 3 & 4**: Pulp will ultimately be encroached. |

Bleaching: External Cervical Root Resorption

* Risk Factors:
  + Deficiency of cementum in the cervical area
  + Injury to PDL (trauma)
  + Infection
  + Lack of seal over GP
  + High Heat
  + High concentration of H2O2
  + Patient younger than 25 years
* A **protective layer** of at least 2mm thickness should be placed over the root filling material. Ideally, vertical outline of protective barrier should be 1mm coronal to the CEJ to cover lingual, labial, and proximal CEJ areas.
  + Use a periodontal probe to determine level of epithelial attachment from incisal edge which will serve as guide for placement of the barrier.
  + It is good practice to add a coronal extension to the barrier which matches the contour of epithelial attachment and increases safety during bleaching procedure by sealing a wider area against the leakage of peroxide to periodontal tissue.

