

ECR ≠ Exo

The cold test is not false. Surprisingly, the pulp is rarely necrotic even when an External Cervical Resorptive (ECR) defect is colossal. The pulp is protected from perforation by a pericanalar resorptive-resistant sheet (PRRS). It consists of predentine, dentine, and eventually reparative (bonelike) tissue. Despite the presence of the demiurgic PRRS, the pulp is irritated. Diffuse calcifications and pulp stones aplenty are par for the course. The resorptive process does not resemble the destructive pattern of a typical carious lesion in the slightest. Numerous resorptive channels extend haphazardly from the portal or portals of entry. Large sections of the pulp can indeed become enveloped by the resorptive channels, yet vitality is preserved because of the ever-present PRRS. Interestingly, a small external defect may indeed be the proverbial tip of the iceberg for ECR.

There are three stages to ECR, initiation, progression, and repair. Local disruption of the PDL and cementum leads to the eventual exposure of dentine. For a variety of reasons, the normal physiological reparative processes are conspicuous by their absence. This initiation phase creates a window of opportunity for invasive osteoclastic cells to start tunnelling through dentine (progression). Repair is not an apt description for the final stage. An ingrowth of bonelike material extends into the defect, spot welding (fusing) the root to the local alveolar bone. This new mineralised tissue starts to fill the voids of the resorptive channels. The progression and repair processes occur in tandem throughout the extent of the defect. If left untreated, ECR can effectively decoronate a clinical crown. However, the rate of progress is almost impossible to predict. Some sizable defects may have such a glacial rate of progress that the most predictable long-term prognosis is best achieved with no further intervention.

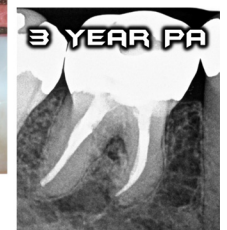
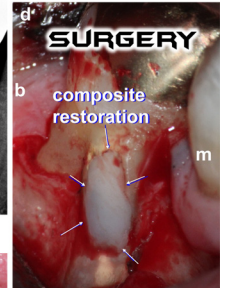
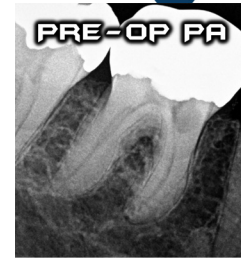
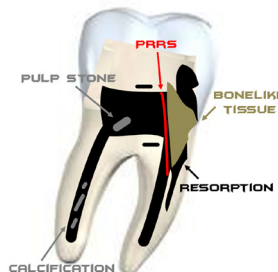
Arresting the resorptive process and maintaining the pulp vitality may be possible if the portal of entry is sealed with bioactive restorative materials. However, at its maximum the PRRS is only 500µm thick, offering scant protection for the frail pulp. Thus, prophylactic endodontic treatment is, more often than not, necessary to complement the cessation of ECR. That was indeed the case for the 46 of a pleasant 80-year-old lady I treated several years ago. She was motivated to save the tooth. Use of a Silker-Glickman clamp on the 47 enhanced efforts to isolate the buccal defect of the 46. The canal space, just apical to the defect, was sealed with bonded PermaFlo Purple. This ensured the coronal seal of the MB canal remained intact during the surgical defect repair. Removing a small section of coronal alveolar bone eliminated any direct hard tissue link (i.e. fusion) between the alveolar bone and root. Finally, a bonded white flowable composite was placed to seal the preparation.

A three-year recall examination confirmed the 46 responded favourably to our treatment. The tooth is asymptomatic and functional and it is expected to remain so. ECR defects are attracting more and more attention in endodontics. Advances in clinical endodontics and restorative materials have greatly enhanced our ability to predictably treat ECR, free of any sense of unbridled fearful tohubohu.

Regards,



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Please support my 2021 B.C. Ride to Conquer Cancer. This will be my tenth year in the Ride and with your support, it will be the best yet! Thank you to the superheroes that have donated so far.

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