

## CASE REPORT(S): UNCOMPLICATED CROWN FRACTURES

Tooth fractures can be classified as follows:

Uncomplicated crown fracture = fracture limited to the crown of the tooth with dentin exposure but no pulp exposure.

Uncomplicated crown/root fracture = a fracture that involves the crown of the tooth and extends below the gum line to also affect the root of the tooth, with dentin exposure but without pulp exposure.

Complicated crown fracture = fracture limited to the crown of the tooth that does involve pulp exposure.

Complicated crown/root fracture = a fracture that involves the crown of the tooth and extends below the gum line to also affect the root of the tooth, and involves pulp exposure.

Treatment planning for any complicated tooth fracture (involves pulp exposure) gives us only two options; endodontic therapy or extraction.

Treatment planning for many crown/root fractures only give us one option: extraction.

Treatment planning for uncomplicated crown fractures pose a real challenge as there are a variety of possible outcomes and some pretty wide grey zones.

Before diving into the case report, I suggest the following papers from previous issues of The CUSP:

<http://www.toothvet.ca/PDFfiles/endo.pdf>

[http://www.toothvet.ca/PDFfiles/endo\\_dx.pdf](http://www.toothvet.ca/PDFfiles/endo_dx.pdf)

<http://www.toothvet.ca/PDFfiles/Chevron.pdf>

The highlights from those papers that apply to this discussion include the following:

- ❖ All of the enamel forms prior to tooth eruption, it is hard, impervious and insensate. Once it is lost, there is no mechanism to repair or replace it.
- ❖ The dentin wall of a tooth is very thin when the tooth erupts, the pulp tissue inside the tooth produces dentin internally as long as it

is alive so over time the pulp chamber gets smaller as the wall of the tooth grows thicker. Dentin produced prior to and during eruption is primary dentin. Dentin produced as the normal maturation process of the tooth is secondary or physiologic dentin.

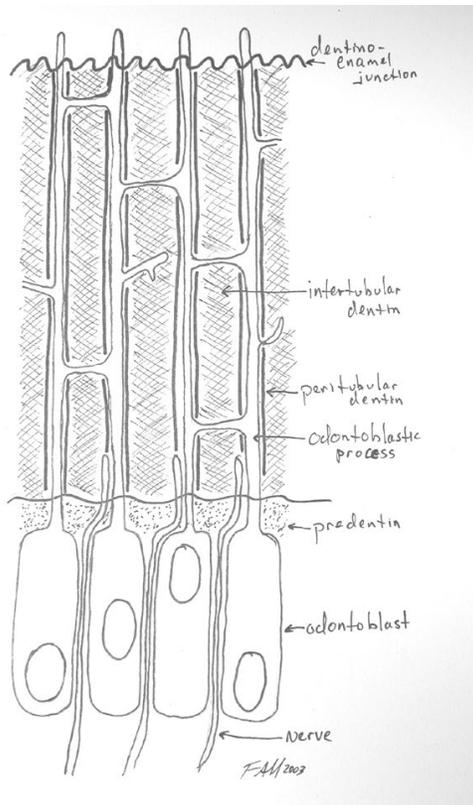


The lower canines of a German short haired pointer at 6, 14 and 32 months of age. Note how the pulp chambers got smaller and the walls of the tooth grew thicker over time.

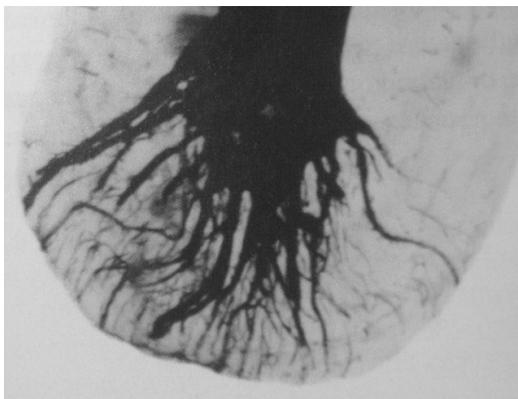
- ❖ Dentin is porous, with thousands of tiny tubules per  $\text{mm}^2$  in direct communication with the pulp chamber and these tubules contain cytoplasmic processes from the pulp producing cells (odontoblasts) as well as nerve endings.



Freshly cut dentin surface showing the open dentin tubules. While these pores are very small, they are larger than bacteria and so can act as open pathways for bacteria to gain entry to the pulp chamber inside the tooth.



- ❖ Exposed dentin will be sensitive to heat, cold, touch, osmotic gradients and chemical assault.
- ❖ Irritated odontoblasts may respond by producing tertiary or reparative dentin which is darker in colour than normal dentin.
- ❖ Normally the only way in or out of the pulp chamber is through a collection of tiny channels at the root tip known as the apical delta.



An apical delta stained with India ink. These delta channels are too small to be seen radiographically.

We are now going to look at the case of Breeze, a female, spayed Elkhound born in August of 2004.

I first saw Breeze in October of 2008 (just over four years of age) for assessment of uncomplicated crown fractures of the right and left upper 4<sup>th</sup> premolar teeth.



These were quite superficial fractures, but I have seen such damage result in septic pulp necrosis as shown in the following three examples.

Here is the one from the JRT in the Endodontic Diagnosis paper.

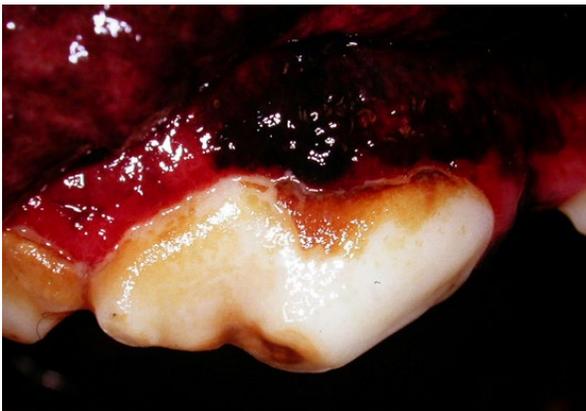


Not much damage to the crown or anything visually to suggest endodontic disease...



...but radiographically, there is massive bone loss around all three root tips indicating chronic infection.

And here is a mature Newf with a long history of a chronic purulent ocular discharge.



Again, pretty unimpressive damage to the right upper 4<sup>th</sup> premolar...



...but radiographically, lots of apical bone loss.

And finally, here is the left upper 4<sup>th</sup> premolar of a 5.5-year-old golden retriever with very superficial damage...



...but very obviously the victim of septic pulp necrosis and chronic periapical periodontitis.



Now, back to Breeze. Knowing that superficial coronal damage does have the potential to allow bacterial ingress through the porous dentin, resulting in septic pulp necrosis and apical periodontitis, Breeze was admitted for a detailed examination including whole mouth intra-oral dental radiographs, transillumination etcetera.



And I could find no clinical or radiographic evidence of endodontic disease at that time. The transillumination suggested blood flow through the crown of the tooth meaning the pulp was alive then, but this does not mean it was healthy. There was insufficient evidence of pulp pathology to warrant extraction or root canal treatment at that time, but this is no guarantee that endodontic disease would not develop in the future. So, treatment involved a thorough oral hygiene procedure followed by coating the fracture sites with a fluoride-releasing bonding agent to seal the exposed dentin tubules and offer some protection for the underlying pulp tissue. Then we planned to see Breeze again a year or so later for follow-up.

When next we saw Breeze (March 2010), she had done more damage to these teeth.

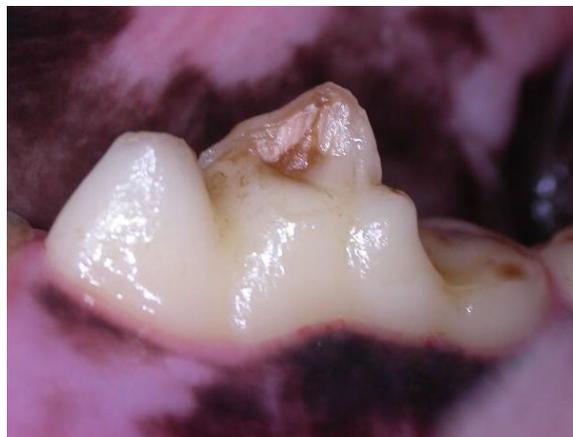
My assessment was the same – ‘no clinical or radiographic evidence of endodontic disease at this time’. I smoothed and bonded the fracture sites and sent Breeze on her way.

Next time we saw Breeze was April of this year (2011). I could detect no further damage to the upper 4<sup>th</sup> premolars (did not bother photographing them this time as there was no change).

Radiographically, I think they are still doing well.



Transillumination also showed no signs of trouble. No treatment for these teeth this year. But Breeze always has something going on and this year it was the lower right 1<sup>st</sup> molar.



Here is a pre-op photograph from the lingual aspect showing a significant uncomplicated crown fracture. Fortunately, I had prior year's radiographs as a baseline and here they are. The first is from October, 2008 and the second from March 2010.



In those images we can confirm that there was no significant damage to the crown of the tooth at prior visits and that the chevron signs have been there and unchanged for some years (ie normal for this dog). Even the little density mesial to the tip of the mesial root has been there for a long time without change (again, likely of no significance). Below is this year's radiograph showing the obvious coronal damage but otherwise, no changes to indicate endodontic disease.



I was worried about the tall, thin spike of the central cusp being a weak point and prone to further fracture so I did some odontoplasty to create a mechanically more sound profile to the crown of the tooth and then sealed the exposed dentin with a bonded resin as shown in the following images.



Breeze's owners have figured out where the damage is coming from, but there is little they can do to stop it. They do not give Breeze hard chew toys, but when they visit Mrs. Owner's parent's farm, Breeze finds bones scattered about the property to chew on. Since they cannot scour the 200 acres for all risks, we best assume Breeze will continue to find things to chew on and so is at risk of further dental damage. So the plan is to see Breeze again next year for further monitoring of the situations and to look for any new damage.

In another case, a 1.5-year-old Australian kelpie was presented with an uncomplicated crown fracture of the left upper 4<sup>th</sup> premolar in which the evidence was that the pulp was alive but that the fracture was so close to the pulp (near pulp exposure) that it would likely not survive much longer without treatment.



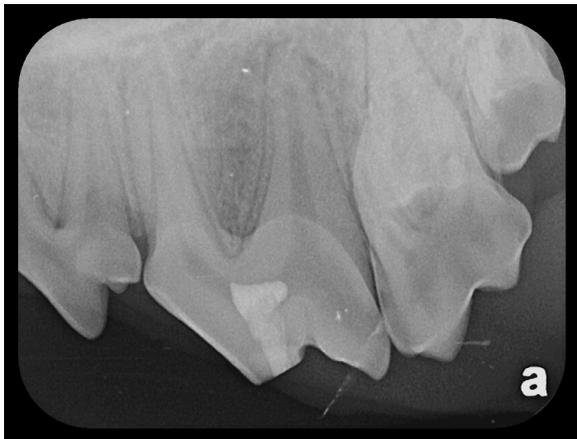
Simply putting a bonding agent on the exposed dentin was not going to be enough protection for this tooth. And any solid restoration I might have bonded over the area as a bandage would have popped off within weeks. Restorations can do well in holes when surrounded by tooth tissue but do not last well in a dog mouth when simply glued on to an exposed surface. On the other hand, it was an immature tooth with vital pulp.

The immaturity meant (among other things) that the pulp chamber would be large and the wall of the tooth thin.



Here is another paper to review now: [http://www.toothvet.ca/PDFfiles/endo\\_dx.pdf](http://www.toothvet.ca/PDFfiles/endo_dx.pdf).

I did not want to extract this tooth, I did not want to do root canal (total pulpectomy) on this tooth and I could not protect the pulp by gluing a restoration on to the exposed fracture site, so I did vital pulp therapy. I have done this procedure many times on single-root canine teeth but not often in multirooted teeth. None-the-less, it seemed the best option in this case and so we proceeded.



The images above were taken immediately post-op. The images in the next column were taken at follow-up eight months later.



The pulp chambers are smaller than pre-op, the same size as the right 4<sup>th</sup> premolar and there are dentin bridges adjacent to the pulp capping material. As well, the tooth transilluminated well and there are no signs or periapical changes. All

indications are that the tooth survived the injury and the treatment and should do well in the long-run, providing the dog does no further damage to it.

### **Take Home Messages:**

Dentin is porous and so any damage that removes the thin enamel covering on the crown opens up thousands of potential pathways for bacteria to colonize and gain access to the pulp tissue resulting in septic pulp necrosis.

The amount of visible coronal damage is often a poor predictor of whether or not the pulp is going to be compromised. Some serious-looking fractures result in no pulp pathology and some very subtle ones result in septic pulp necrosis.

Fractured and worn teeth require careful clinical and radiographic evaluation to determine what treatment, if any, is indicated.