Pathology and diagnosis of traumatically injured teeth

Traumatic damage to teeth represents a significant cause of chronic inflammation and pain in our domestic animal species. This article will look at how the pathology associated with traumatic damage develops and how this can be assessed using dental radiography. Treatment options will also be discussed.

**Key words:** tooth fracture, pulp, periapical necrosis, lucency, root canal treatment

**Introduction**

The teeth of domestic dogs and cats can be damaged in a number of ways, with an incidence of up to 25% of dogs and 10% of cats. Initially owners may be aware of the trauma – for example we commonly see dogs presenting with acute dental fractures following incidents where owners have thrown stones which the dog has then caught, resulting in the damage. In these cases, the exposed pulp is likely to be obvious, often with pulpal haemorrhage at the fracture surface (Figure 1), and the animal is likely to be showing signs of oral discomfort. However, as time progresses, and pulp tissue becomes necrotic and less sensitive, pain will often wain and the impetus for treatment can often be lost with what appears to be an innocuous injury (Figure 2). It is however at this stage that the unseen pathology is developing, which can make salvage of these teeth impossible, resulting in the need for tooth extraction.

**The pathology of pulp exposure**

In common with many oral pathologies, traumatic damage can be seen as contest between the oral bacterial flora and the host’s immune response, which works to prevent those bacteria from reaching the underlying structures. In the initial stages of tooth trauma, when the pulp is first exposed and oral bacteria gain access, the pulp will mount an inflammatory response in order to combat this invasion. This inflammatory response releases inflammatory cytokines which, along with bacterial endotoxins, stimulate a pain response from the tooth. In humans this commonly manifests itself as an increased sensitivity to hot and cold stimulation, a test commonly used to assess pulp vitality and inflammation in human patients.

This is a response that is very difficult to quantify in our animal patients. At this stage, the only diagnostic test for assessing disease is by assessing the fracture surface with a sharp explorer, which will identify the exposed pulp chamber (Figure 3).

This dynamic situation of bacterial infection and the inflammatory response from the pulp of the tooth can be sustained for some time, although eventually the pulp becomes necrotic owing to the continuous bacterial invasion (Figure 4). Bacteria at this stage will digest the pulp tissue and establish colonies within the pulp and dentinal tubules (in the dentine lining the pulp chamber). Pulp sensitivity *per se* is lost as the pulp tissue is destroyed. However the bacteria will now start to release endotoxins and other waste products which exit the tooth root via the canals of the apical delta (Box 1). These products stimulate an inflammatory response in the periapical tissues. At this stage the only diagnostic test for assessing disease is still the use of a sharp dental explorer as no radiographic evidence of periapical bone loss will be seen. The patient is however likely to now be feeling a dull ache associated with periapical inflammation.

Inflammation in the periapical tissue – caused by ongoing leakage of material from the necrotic pulp, stimulates the release of prostaglandins and leukotrienes in the periapical area. This results in an influx of osteoclasts which in turn leads to periapical bone loss – seen on dental radiography as a periapical lucency (Figure 5a). The lysed bone is replaced by a periapical granuloma (Figure 5b) which serves to provide a buffer zone around the tooth apex, preventing bacteria present within the root canal from penetrating the surrounding bone.

Eventually, osteoclast and odontoclast activity results in apical root resorption (Figures 6a, 6b), allowing bacteria to now directly escape from the root canal into the periapical space; this pathology can now be termed a tooth root abscess (Figure 6c).

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Figure 1: A freshly fractured left mandibular canine tooth showing active haemorrhage from the fracture site with exposed pulp.

Figure 2: The pulp of this tooth is necrotic and there is little outward sign of pathology other than the exposed necrotic pulp. Teeth such as these are commonly ignored during general examination.

Figure 3: The sharp explorer should be run over the surface of a tooth and this will allow identification of the exposed pulp as the instrument will catch here.

Figure 4: Black necrotic pulp being removed from a tooth during endodontic treatment.

Figure 5a: Periapical osteolysis characteristic of periapical inflammation following a complicated crown fracture of this left lower 1st mandibular molar tooth in a dog.

Figure 5b: Periapical granuloma (arrowed) surrounding the apex of an extracted tooth root.

Figure 5c: Box 1: Tooth anatomy

Pulp is a specialised connective tissue containing blood vessels, lymphatics and nerves. In the crown, the section containing the pulp is called the pulp chamber and in the root it is called the root canal.

Dentine consists of microscopic channels, called dentinal tubules, which radiate outward through the dentine from the pulp to the exterior cementum or enamel border. The tubules contain tiny outpouches of pulp.

Figure 6a, b and c: Osteoclast and odontoclast activity results in periapical bone loss and resorption of the tooth root. This opens the infected pulp to the periapical tissues, allowing the escape of bacteria as a tooth root abscess now forms.

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