THE REPARATIVE CAPACITY OF PULP TISSUE

Opinions regarding the reparative capacity of pulp tissue are contrasting. Several maintain that the pulp cannot sustain any insult, while others are convinced that because it is very resistant any attempt to preserve it is justified. These are two extreme positions. Neither is correct; rather, the truth lies somewhere in between.

Histologically, the pulp, apart from the odontoblasts, is a fibrous connective tissue of mesenchymal origin, has most of the characteristic cells of this tissue, and responds in an analogous manner to various irritant stimuli. Its responses are thus typical of connective tissue of any other part of the organism: an irritation produces damage, and this stimulates an inflammatory process whose final end is the “restitutio ad integrum”.

Nonetheless, the responses of the pulp are different from those of the other tissues. This is due to topographical and anatomical conditions that cannot be modified.

In 1939, Gottardi noted that the pulp was enclosed in a mineralized, rigid, and inextensible theca. Other authors have even said that the pulp is already enclosed within its own coffin!

The source of its blood supply is at a considerable distance from the large mass of coronal pulp tissue, and the volume of the pulp is relatively large if compared with the transverse diameter of its foramen or foramina, except in the case of teeth with an immature apex. This means that the blood supply of the pulp is abundant (Fig. 7.1). Unfortunately, however, all the arteries enter the tooth through a relatively restricted apical opening, and the veins exit through the same foramen.

For this reason, the pulp, having to react to external stimuli and pathological processes, both inflammatory and infectious, is unfavorably situated. Indeed, when the pulp is in some way injured at the coronal level, it responds with inflammation and a consequent increase of vascular permeability and exudation of fluid into the surrounding tissues, as in other connective tissues. In contrast to the latter, however, the pulp has no room to expand and swell as it manifests the five pathological signs of inflammation: “rubor, calor, tumor, dolor, and functio lesa”.

Thus, the pulp draws no advantages from the inflammatory process. On the contrary, the increased volume of its inflamed tissues and the simultaneous inability to swell, the compromised efferent circulation and rapid reabsorption of the exudate induce necrosis of a large number of cells.

Further aggravating the situation, the pulp lacks collateral circulation, since its circulation is of the terminal type, especially at the coronal level, where there is a greater amount of tissue.

Fig. 7.1. A, Blood vessels in a healthy, adult pulp. B, Detail (courtesy of Dr. N. Perrini).
Moreover, the coronal pulp is the first to sustain insults of any sort and the furthest from the entrance of the blood supply in the apical foramen. When the pulp sustains an insult sufficient to create true inflammation, its anatomy makes it highly susceptible to necrosis and gangrene. Indeed, the pressure caused by edema is frequently sufficient to interrupt the circulation at the level of the apical foramen. The stasis of blood flow provokes destruction of this peduncle of soft tissue. In conclusion, when the pulp is exposed to external pathogenic agents, in particular bacteria, following an accidental insult or pathological process, it is incapable of reacting to them. Rather, in spite of opposing them with its reactive and defensive processes, it is fatally destined to succumb to necrosis.

On the other hand, this does not mean that the pulp dies no matter what! Many studies have demonstrated the reparative capacity of the pulp tissue. Nonetheless, one must be very prudent when undertaking so-called “vital pulp therapies” and must determine very carefully whether the compromise has caused reversible or irreversible damage in the pulp that one is about to treat.

Many have confirmed that, in the absence of bacterial infection, the most important parameter to be taken into consideration is the blood supply. If the pulp has an ample blood supply, its reparative capacity may be considerable.

A pulp exposed by trauma to a tooth with an immature apex can retain its vitality. (Proper maintenance will maintain the vitality of the pulp tissue until it has carried out its primary, formative function, and thus has led to maturation of the root and of the apex. From this point of view, the pulp could be considered a supporting tissue of the odontoblasts. The pulp of a tooth that has sustained a radicular fracture to its middle one third, where there is a certain mass of pulp tissue and a discrete blood circulation, and the fracture is close enough to the entrance of the blood vessels in the root, can maintain its vitality (Fig. 7.2). The apical portion of the pulp of an horizontally fractured tooth can remain vital (Fig. 7.3). The pulp of an immature tooth that is extracted with its follicle and reimplanted in the bleeding alveolus of another just-extracted tooth can remain vital (Fig. 7.4). Even the pulp of a tooth that is accidentally injured by a dental burr in the course of apicectomy can retain its vitality (Fig. 7.5).

Revascularization is a function of the diameter of the apical foramen, of the mass of the exposed tissue, and therefore it is a function of its blood supply. The case of pulp exposure in a tooth with a mature apex, however, is very different, especially if it is complicated by bacterial infection. In the long term, the possibility of recovery is quite low in this case. For these reasons and for the reasons that will be analyzed in detail in the paragraph on vital pulp therapy, Schilder and his entire school is definitely opposed to the various types of pulp capping and considers pulp exposure to be an indication for endodontic therapy.