How might Operative Dentistry be a Threat to the Pulp?

Reshma Seeburrun

INTRODUCTION

Operative dentistry is the art and science of diagnosis, treatment and prognosis of defects of teeth that do not require full coverage restorations for correction. Such treatment results in restoration of proper tooth form, function and aesthetics while maintaining physiologic integrity of the adjacent teeth and soft tissues, all of which should enhance the general health of the patient.[1] Examples of such procedures include simple amalgam fillings, inlays, onlays and preliminary periodontal surgery. The pulp is a viscous connective tissue of collagen fibres and ground substance supporting the vital cellular, vascular and nerve structures of the tooth (see figure 1). Pulpal responses to dental treatment depend on many factors including thermal injury, injury to odontoblastic processes, desiccation of dentin, vibration, pulp exposure, smear layer, remaining dentin thickness, restorative materials used and microleakage.

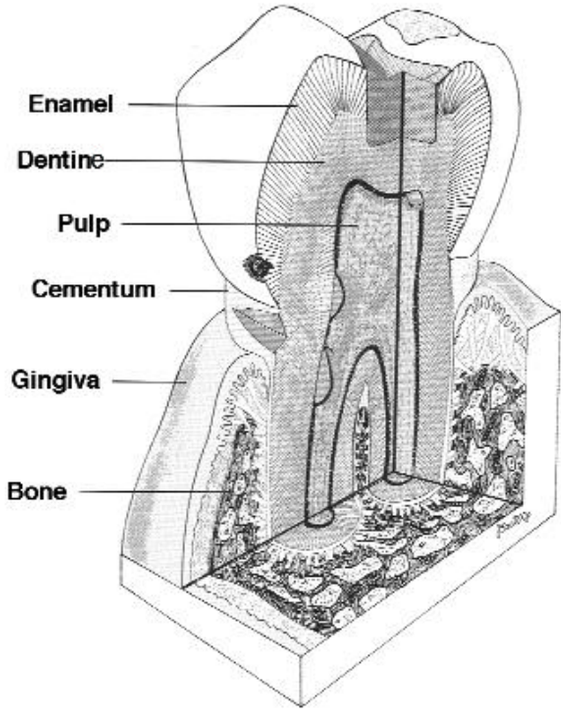

Figure 1.[6]

THERMAL INJURY

Tooth preparation with a rotating bur or stone produces a considerable amount of frictional heat. The amount of intrapulpal heat generated is determined by many factors, including the drill rotation speed, size, type and shape of cutting instrument, length of time the instrument is in contact with dentine, the amount of pressure exerted on the handpiece, the cutting technique and the use of coolants.

Studies have shown that high-speed cutting with copious water coolant and a reduced force results in minimal histological alteration of the pulp.[2] In fact, Stanley showed in 1976 that given the same conditions of comparable remaining dentine thickness (RDT) similar cutting instrument and adequate water cooling, the intensity of the pulpal response will be less with high speed rather than lower speed cutting. This is due to the lower application force required by the high speed handpiece.[3] Wittrock *et al.*[4] have shown that the cutting technique also greatly influences heat generation. It is not advisable to start removing amalgam by making a deep internal slot within the restoration as this results in the localised concentration of heat. Preparation for restoration close to the pulp may generate substantial frictional heat causing a significant and detrimental temperature increase in the pulp. Repair will usually occur but the formation of reparative dentine can be extensive and render the pulp vulnerable to repeated injury. In fact, clinical follow-ups of teeth restored with cast restorations (full crowns and teeth included as abutments in bridgework) have shown that pulpal necrosis may occur with a frequency of 10-15% over a period of 5-10 years.[5] Often one will find that the coronal portion of the pulp in such teeth is obliterated by reparative dentine, making endodontic therapy precarious.

Another complication to cavity and crown preparation is internal bleeding. In rare cases it may be so extensive that pulpal necrosis occurs almost instantaneously. The tooth structure of such teeth may turn red and later a grey colour.

INJURY TO ODONTOBLASTS

Odontoblasts are exposed to a variety of insults, including frictional heat, amputation of processes, displacement of the cell body, vibration and exposure to bacterial toxins and other chemical irritants. The odontoblastic cells are packed closely together with both permanent and temporary junctions between the cellular membranes. The integrity and spacing of the odontoblastic layer mediates the passage of molecules and tissue fluids between the pulp and dentine. Routine dental procedures can

temporarily disrupt the odontoblastic layer and may sometimes inflict permanent cellular damage.[7] Depending on the depth of the cavity preparation, odontoblastic processes are amputated at various points along their distal segment. If the processes are not amputated close to the cell body then repair of the cell membrane occurs. However if the odontoblasts do die they undergo autolysis and are replaced by new cells derived from odontogenitor cells. This replacement occurs provided that the underlying cell-rich zone of the pulp has not been injured.[8]

DESSICATION OF DENTINE

The use of compressed air to dry a cavity preparation for a prolonged period can result in a delayed healing response. Drying surface dentinal fluids activates strong capillary forces which cause a rapid outflow of fluid in the dentinal tubules. This rapid outflow stimulates mechanoreceptors, which not only cause post-operative pain, but can even cause the displacement of the odontoblast from the odontoblastic layer up the tubule. The displaced cells undergo autolysis and disappear. These can be replaced with cells from the uninjured underlying pulp. Contrary to popular belief, vigorous drying alone does not cause major irreversible pulpal reactions.[9] This is because the products of degradation are so diluted by the dentinal fluid as to induce no inflammation in the pulp. Also, it could be due to the fact that too few cells are involved to cause a significant reaction. Tertiary dentine or reparative dentine is then laid down in about 1 to 3 months by the odontoblasts to wall off the pulp from the site of injury.

VIBRATION

The effects of the vibratory movement of cutting burrs on the pulp have not been thoroughly researched. According to Holden the shock waves generated occur beneath the point of application of the bur in the pulp.[10] They appear to be more pronounced when the bur is stalled by digital pressure. Stalling not only decreases cutting efficiency by clogging up but also leads to an increase in temperature.[2]

PULP EXPOSURE

Exposure occurs most often in the process of removing deep carious dentine. Accidental mechanical exposure may result during placement of pins and retention points in dentine. Friction-locked pins often produce microfractures, which establish communication between the pulp and dentine. In all these cases the pulp appears to be affected primarily by bacterial contamination. Frank showed that exposure of the pulps in germ-free rats was followed by complete healing without any inflammatory reaction.[11] No such published studies were found in the review of the literature.[11]

SMEAR LAYER

The use of rotary instruments leaves an amorphous layer of microcrystalline debris on the enamel and dentine surfaces, known as the smear layer. Some controversy exists regarding the value of removing or maintaining this layer. One school of thought purports that the debris occludes the dentinal tubules, decreasing dentine permeability and preventing the ingress of bacteria.[12] However, Pashley suggested that this does not prevent the ingress of bacterial toxins and by-products that can lead to pulpal inflammation.[13] Moreover, Brannström believes that most restorative materials do not adhere to dentinal walls and bacteria from the smear layer may invade the contraction gaps.[14]

MICROLEAKAGE

In spite of substantial efforts over the years to improve the composition of restorative materials, including resin composites and the techniques for their use, the shrinkage of these materials after setting is critical.[15,16] Shrinkage builds up strains in the filling that later may result in gaps at the interface between tooth and restoration. This may allow bacteria and bacterial products in the oral environment to affect the pulp. The term microleakage is used to imply this form of pulpal irritation.

Research in recent years has indeed demonstrated that bacterial leakage in restoration margins is a major threat to the vital functions of the pulp subsequent to restorative therapies.[5,17] In particular, on deep and extensive exposures of dentine, the infectious load on the pulp can be substantial. In principle, inflammation of the pulp in response to these bacterial exposures is similar to that for caries but there are some distinct differences. Neutrophils play an important role in the initial responses owing to the more sudden and extensive bacterial exposure than that in the relatively slowly progressing caries lesion. These cells accumulate in areas of the pulp that correspond to the involved dentinal tubules. Chemotactic stimuli also prompt neutrophils to migrate into the tubules. This is probably the most significant defence factor that, in addition to the protective effects of the dentinal fluid, helps to block further penetration of bacteria and bacterial elements into the pulp. Collectively these mechanisms are likely to explain why pulpal repair and healing are still possible even when a restoration does not completely seal the margins.

TOXIC EFFECTS OF RESTORATIVE MATERIAL

In addition to the trauma from preparing teeth for restoration and the subsequent leakage of bacterial elements, constituents of restorative materials may have an adverse influence on the pulp. For many years the toxicity of restorative materials was regarded as the major cause of adverse pulpal responses in restorative procedures. Some of the properties of restorative materials that are believed to cause or contribute to pulpal damage include chemical cytotoxicity, exothermic reaction on setting, acidity, absorption of water during setting and poor marginal adaptation leading to bacterial microleakage.

However, research in recent years has shown that, contrary to previous beliefs, toxic components in restorative materials are a lesser threat to the pulp than previously anticipated.[5] This has been best demonstrated in experimental studies where dental materials in common use, such as amalgam, zinc phosphate cement and resin composites, were applied directly on pulpal tissue in circumstances where the surface of the restoration was sealed bacterial-tight.[8,18] These experiments demonstrated that the pulp around the sealed restorations often resumed a healthy state. However without a bacterial-tight surface seal severe inflammation developed in the pulp. The risk of severe pulpal complication is even less when a dentine barrier remains. Dentine seems to serve as a detoxifying tissue in that highly toxic materials may be absorbed to the inner walls of the dentinal tubules. It has been shown that dentine buffers the effects of acids and bases.[19] Experiments in vitro and in vivo have demonstrated that catatonic components of resin monomers (e.g. triethylene glycol dimethacrylate (TEGDMA) and 2-hydroxyethyl methacrylate (HEMA)) readily penetrate thin dentine walls upon topical application.[20] The effect of such penetration is not well understood. However observations in animals suggest that the toxic effect on the pulp of this agent is short lasting. Furthermore, it has been shown that most leachable substances from resin composites are released within the first few days after placement and that then little will be discharged.[21] Therefore the threat to the pulp resulting from restorative procedures does not seem to be from the material *per se* but more from the improper seal that often results.

In addition, the exothermic reaction of some luting cements on setting has been thought to cause pulpal injury. However, Plant (1976) demonstrated that zinc phosphate, the most exothermic cement, caused an intrapulpal increase in temperature of just 2^{o}C.[22] This is insufficient to cause any injury to the pulp. Changing the amount of powder and liquid ratios when mixing cements however was shown to cause a marked rise in temperature.[23]

REMAINING DENTINE THICKNESS

Over the years, the degree of remaining dentine thickness (RDT) believed to be required in order to maintain a healthy pulp has greatly decreased. Stanley suggested that a RDT of 2mm is necessary to protect the pulp from most restorative procedures whereas Pameijer reported that a RDT of 1mm or more would protect the pulp tissue from the cytotoxic effect of zinc phosphate and resin-modified glass ionomer materials during the luting process.[24] Murray suggested that deeper cavities carefully cut down to 0.5mm appeared to have only a limited effect on underlying odontoblast survival.[25]

OTHER FACTORS

Root Planning

Pulp response to root planning is negligible unless dentine removal is excessive. Depending upon the remaining thickness of dentine the dentinal tubules are capable of repair and healing despite being exposed to microorganisms. However, if the apical foramen is involved periodontically, curettage could sever the blood vessels resulting in pulp damage. In this case these teeth are prophylactically root canal treated. It can be summarised that unless the root apices are involved, the effects of periodontal therapy on the pulp appears to be negligible.

Local Anaesthetics

Though intraligamentary injections have often been put forward as a cause for pulpal damage, clinical and animal studies have shown no adverse effects on the pulp.[26,27] Physiologic changes such as rapid and marked reduction in blood flow caused by adrenaline do occur.[28] However this vascular impairment has not shown any damaging effects on the pulp even in conjunction with restorative procedures.[29]

Electrosurgery

Electrosurgery, used in conjunction with operative dentistry to remove gingival tissue for enhanced access during tooth preparation and impression making, may affect the pulp. If the probe contacts a metallic restoration then adverse and often severe reactions occur.[30] These adverse reactions occur whether the restoration is based with a metallic or a non-metallic material.[31] The pulpal response is more severe with increased contact time and decreased RDT. Contact of more

than 0.4 sec has been shown to lead to irreversible damage.[32]

Bleaching of Vital Teeth

Vital teeth can be bleached using the "in-office" technique or the "at-home" technique. In-office techniques include the application of a bleaching agent, usually 35% hydrogen peroxide, to teeth isolated by a rubber dam. They may include activation of the hydrogen peroxide using heat or light in order to enhance or activate the release of peroxide. At-home bleaching uses a different bleaching agent, usually a 10% solution of carbamide peroxide, applied in a custom fitted tray that the patient wears at home, usually while sleeping. Many studies have shown that although penetration of the peroxide through the tooth to the pulp can produce sensitivity the pulp remains healthy and the sensitivity is completely reversible.[33,34] The rate of oxygen release, and therefore the rate of colour change, is proportional to the temperature. An increase of 10°C doubles the rate of chemical reaction.[33,35] However temperatures elevated to an uncomfortable level may result in tooth sensitivity or irreversible pulpal inflammation. Bleaching materials should always be administered without anaesthesia to avoid overheating the tooth.

CONCLUSION

Many operative procedures can be traumatic to the pulp and the effects are at least somewhat cumulative. The dental practitioner should be fully aware of the methods and materials that can jeopardise the pulp. Knowledge of proper cavity preparation and its application can greatly reduce pulpal injury. Many materials including cavity varnishes, liners and bases are widely available on the market to reduce the occurrence of pulpal damage. However other important factors that can determine a good pulpal prognosis are patient factors, which include age, previous treatment history, dental characteristics and diet.

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