Original Article

PULPAL RESPONSE TO MINERAL TRIOXIDE AGGREGATE (MTA) IN DECIDUOUS AND PERMANENT DENTITION - A REVIEW

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Abstract

Mineral trioxide aggregate (MTA) is a calcium-silicate cement that reliably preserves pulp vitality in both primary and permanent teeth. On hydration, MTA releases calcium and hydroxyl ions, creating a sustained alkaline, antibacterial microenvironment that supports hemostasis, dampens acute inflammation, and liberates dentin growth factors (e.g., TGF-β). These signals drive odontoblast-like differentiation, laying a collagen scaffold that mineralizes into a continuous, often tubular dentin bridge formation. Clinically, this yields symptom resolution, maintained sensibility where appropriate, and radiographic evidence of a stable hard-tissue barrier with normal periapical/furcal outlines. In primary teeth, MTA pulpotomy demonstrates high success with fewer adverse sequelae than traditional agents. In permanent dentition, MTA performs predictably for direct/indirect pulp capping, partial pulpotomy, and apexogenesis, and serves as a dependable apical plug for apexification. Outcomes are seal-dependent and technique-sensitive, emphasizing meticulous hemostasis, isolation, and an immediate coronal seal. In primary teeth, the pulp typically shows rapid resolution of superficial inflammation, preservation of a healthy radicular pulp, and formation of a uniform dentin bridge with minimal internal resorption and physiologic exfoliation maintained. In immature permanent teeth, MTA fosters a mild, transient inflammatory phase followed by recruitment of odontoblast-like cells and stem/progenitor cells, enabling structured dentin bridge formation and continued root development (apexogenesis). In mature permanent teeth, the pulpal response is characterized by controlled hemostasis, a low-grade inflammatory infiltrate that subsides quickly, and deposition of a well-sealed mineralized barrier with few tunnel defects. Collectively, these tissue-level responses underpin the high clinical success of MTA across both primary and permanent dentitions.

Key words- Maternal oral health, Emineral trioxide aggregate; vital pulp therapy; dentin bridge; pulpotomy; direct pulp capping; apexogenesis; apexification; calcium silicate cement.

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INTRODUCTION

Preservation of a healthy, sensate pulp is central to contemporary conservative dentistry, especially when carious or mechanical exposures occur in teeth with the capacity to heal. Mineral trioxide aggregate (MTA), a calcium-silicate–based cement, has emerged as a biologically active material capable of promoting

predictable pulpal repair and dentin bridge formation when used in vital pulp therapy. Upon hydration, MTA releases calcium hydroxide, establishing a sustained alkaline environment that is inhospitable to bacteria and conducive to hemostasis, attenuation of acute inflammation, and activation of signaling pathways that drive odontoblast-like differentiation and mineralized tissue deposition [1–3]. Its fine particle size, radiopacity, dimensional stability, and sealing ability help maintain a bacteria-tight interface—arguably the decisive determinant of long-term pulpal health [1,3,4].

At the tissue level, the early response beneath an MTA interface commonly includes resolution of neutrophil-predominant inflammation, macrophage-mediated cleanup, and preservation or rapid repopulation of an odontoblastic layer at the wound margin. In the subsequent weeks, matrix remodeling and angiogenesis support the emergence of odontoblast-like cells that elaborate a tubular, mineralized barrier at the exposure site. Histologically, this barrier often displays continuity and increasing thickness over time, shielding subjacent pulp from irritants and restoring structural integrity [2,4,5]. Clinically, these cellular events translate to resolution of symptoms, recovery of sensibility where appropriate, and radiographic evidence of a well-defined hard-tissue bridge with stable periapical or furcal outlines [5,6].

Because these biologic effects are material-driven and seal-dependent, case selection and execution are critical. Atraumatic exposure management, meticulous hemostasis, moisture control, and an immediate coronal seal are prerequisites for the favorable pulpal trajectory associated with MTA. Follow-up at structured intervals enables verification of symptom resolution, functional stability, and radiographic maturation of the dentin bridge. Collectively, mechanistic, histologic, and clinical data support a reproducible sequence under MTA—early inflammatory control, cell recruitment and differentiation, angiogenesis, and hard-tissue barrier formation—consistent with durable pulpal vitality and function across clinical scenarios [2–6]. This focused review presents the pulpal response to MTA without comparative analyses, emphasizing the biologic underpinnings and clinical endpoints that inform everyday decision-making in vital pulp therapy.

THE PULPAL HEALING SEQUENCE UNDER MTA

1) Immediate tissue events (0–72 hours)

Following placement, MTA hydrates to form calcium-silicate hydrates and calcium hydroxide, creating a high-pH interface (≈11–12) that is bacteriostatic and favors stable clot formation at the exposure site. The alkaline milieu denatures bacterial endotoxins and reduces matrix metalloproteinase activity, limiting early tissue breakdown. Neutrophil influx peaks and then subsides rapidly, while macrophages transition from a pro-inflammatory to a pro-resolving phenotype, clearing debris and releasing mediators that temper inflammation. Odontoblasts at the cut margins are often partially preserved; where they are lost, the subodontoblastic Höhl's cell layer and resident pulp progenitors remain viable, setting the stage for subsequent differentiation. Early ion release (Ca^{2+}, OH^-, and soluble silica species) and a nascent carbonated apatite layer at the MTA–dentin interface support biocompatibility and signaling to underlying pulp cells [4,5].

2) Matrix reorganization (days to weeks)

With hemostasis secured and bacterial ingress minimized, fibroblasts proliferate and remodel extracellular matrix (ECM) beneath the capping site. Dentin matrix–bound growth factors—particularly TGF-β1 liberated from the cut dentin—combine with calcium-rich microenvironments to promote odontoblast-like differentiation of pulp stem/progenitor cells. Up-regulation of odontogenic markers (e.g., ALP, DSPP, and Runx2) accompanies deposition of a collagenous scaffold that seeds mineralization. Concomitantly, angiogenesis increases microvessel density at the interface, improving oxygen and nutrient delivery and facilitating waste clearance—prerequisites for orderly tissue repair and mineral deposition [4,5].

3) Hard-tissue formation (reparative dentin bridge)

Mineralization initiates at the MTA–tissue boundary and progresses into the organized ECM, yielding a continuous reparative dentin bridge. Histologically, early globular mineral foci coalesce into a more homogeneous barrier that often exhibits tubular or tubule-like architecture, reflecting odontoblast-lineage activity rather than dystrophic calcification. Over ensuing weeks to months, the bridge thickens and matures, reducing permeability and creating a physiologic barrier that protects the subjacent pulp from thermal, chemical, and microbial challenges. The quality of this bridge—its continuity, thickness, and tubular features—

correlates with long-term clinical success and pulpal stability [5].

4) Long-term pulpal homeostasis (months to years)

After barrier maturation, the residual pulp typically demonstrates low-grade or absent chronic inflammation, with preserved neural and vascular architecture compatible with normal sensibility responses. Sustained sealing ability of the set MTA—owing to slight hygroscopic expansion, marginal adaptation, and continued apatite formation—limits microleakage, which is arguably the decisive determinant of durability. Radiographically, a well-defined dentin bridge with stable periapical or furcal outlines is expected; clinically, the tooth remains asymptomatic and functional at recall. When executed with meticulous hemostasis, moisture control, and an immediate coronal seal, this biologic sequence is reproducible across clinical scenarios and underpins the predictability of MTA in vital pulp therapy [4,5].

BIOACTIVE MECHANISMS UNDERPINNING MTA USE IN VITAL PULP THERAPY AND APICAL BARRIER FORMATION

Mineral trioxide aggregate (MTA) is uniquely positioned to serve both as a pulp capping agent (direct/indirect) and as an apical barrier material for apexification, because its core physicochemical behaviors map directly onto the biologic requirements of each indication. Upon hydration, MTA releases calcium and hydroxyl ions, generating a sustained alkaline microenvironment and promoting precipitation of a carbonated apatite layer at material–dentin interfaces. These events are coupled with a high-quality seal in moist fields, dimensional stability, and documented biocompatibility—together reducing bacterial survival, attenuating early inflammation, and enabling cell recruitment and differentiation (odontoblast-like lineage) [1–4].

In pulp capping (IPC in Primary & IPC & DPC in Permanent;), the clinical objective is to resolve acute inflammation rapidly and induce an organized reparative dentin bridge while maintaining pulp vitality. MTA's alkalinity and ion release stimulate growth-factor liberation from dentin (e.g., TGF-β1), support angiogenesis, and up-regulate odontogenic markers, leading to a continuous, tubular hard-tissue barrier with low residual inflammation on histology and favorable clinical success in prospective and retrospective series [2,5–7]. Critically, MTA's sealing ability minimizes microleakage—arguably the strongest

determinant of long-term success—thereby aligning material behavior with the biologic healing sequence already outlined in this manuscript (early hemostasis and inflammation control → matrix reorganization → mineralized bridge formation) [3,5].

In pulpotomy (Cvek's partial pulpotomy) for mechanically/traumatically or shallow cariously exposed pulps, limited removal of the superficial inflamed tissue followed by MTA placement leverages the same mechanisms—alkaline antibacterial milieu, calcium-mediated bioactivity, and a durable seal—to maintain vitality and induce a tubular dentin bridge with minimal inflammation, preserving proprioception and long-term function [2,5–7]. Histologically, primary teeth exhibit a broadly similar pattern—rapid resolution of superficial inflammation, preservation of a healthy radicular pulp, and a continuous dentin bridge—tempered by the context of physiologic root resorption; with an intact coronal seal, pathologic internal resorption is uncommon. In secondary (permanent) dentition, the response likewise features a transient low-grade inflammatory phase followed by organized tubular bridge deposition; in immature teeth this supports apexogenesis, and—under proper isolation—shows a lower propensity for resorptive sequelae than inadequately sealed primary pulpotomies.

In apexogenesis (immature permanent teeth with a vital pulp), MTA placed over a vital pulp stump sustains odontoblast-lineage activity, enabling root wall thickening and physiologic apical maturation while the coronal seal limits reinfection—again reflecting concordance between material behavior and biologic goals of continued root development [2,5–7].

In apexification (immature permanent teeth with necrotic pulp), the therapeutic target is an immediate, biocompatible apical stop that permits definitive obturation and function. Here the same properties—hydraulic set in moisture, marginal adaptation, and ion-mediated bioactivity—allow MTA to function as a predictable apical plug (typically 3–5 mm), producing a dense barrier against which the canal can be filled [4,8–10]. Clinical reports and series demonstrate reliable radiographic barrier formation and timely case completion, while avoiding the prolonged treatment times and structural compromises historically associated with long-term calcium hydroxide dressings [8–11]. When used after appropriate canal disinfection, MTA's apical seal and bioactivity facilitate

periapical healing and reduce the risk of reinfection.

Critical considerations across these indications include meticulous hemostasis, strict isolation, and an immediate bacteria-tight coronal seal; failures cluster around microleakage rather than intrinsic material biology. Known limitations—handling, set time, potential discoloration depending on formulation—are practical (not biologic) and can be mitigated with current operative protocols and material variants [1,3,4]. Taken together, convergent mechanism + histology + clinical data justify MTA as a first-line material for vital pulp therapy (IPC/DPC, Cvek's pulpotomy, apexogenesis) and for apexification, within a coherent, biology-consistent framework.

DISCUSSION

The introduction of mineral trioxide aggregate (MTA) into clinical practice has reshaped the therapeutic approach to vital pulp therapy (VPT) in both pediatric and adult dentistry. The unique biological and physicochemical properties of MTA have facilitated a paradigm shift away from devitalizing agents toward regenerative and pulp-preserving strategies. The pulpal responses elicited by MTA in both deciduous and permanent dentition reinforce its role as a cornerstone in conservative endodontics. While substantial progress has been achieved, ongoing questions regarding its long-term performance, alternatives, and integration into bioactive and regenerative endodontics continue to be areas of scientific scrutiny [12].

In pediatric practice, the maintenance of primary molars until their natural exfoliation is critical for mastication, speech development, and arch length preservation. MTA has demonstrated clinical advantages over ferric sulfate, zinc oxide–eugenol, calcium hydroxide and formocresol in this context. Several randomized controlled trials and systematic reviews have reported significantly higher long-term success rates of MTA pulpotomy in primary teeth, with reduced rates of internal resorption and pathological root resorption [13,16]. Histological analysis further supports these clinical observations, demonstrating a decrease in chronic inflammation and an increased likelihood of dentin bridge formation compared with conventional medicaments. The reduced incidence of pulp canal obliteration observed with MTA is particularly relevant for radiographic follow-up and differential diagnosis in pediatric populations [17].

In immature permanent teeth with open apices, the preservation of pulp vitality is indispensable for continued root development, apical closure, and reinforcement of dentinal walls. MTA has proven superior to calcium hydroxide in apexogenesis procedures, demonstrating a more predictable apical barrier and fewer long-term complications such as cervical root fracture [18]. Direct pulp capping with MTA, particularly in traumatic exposures in young permanent teeth, results in histologically superior dentin bridges with odontoblast-like cell alignment and minimal necrosis [14]. Clinical follow-ups extending up to five years reveal stable pulpal vitality and functional retention of treated teeth [18].

Moreover, in mature permanent dentition, MTA has clear applicability for partial pulpotomy and direct pulp capping, where the therapeutic aim is to preserve vitality and establish a durable hard-tissue barrier under a bacteria-tight restoration. By virtue of its moisture-tolerant seal, sustained alkalinity, and ion release, MTA limits microleakage and bacterial penetration at the restoration–dentin interface, attenuates early inflammatory activity, and supports odontoblast-like differentiation with formation of a tubular reparative dentin bridge rather than a porous, necrosis-adjacent barrier [12,14]. Clinically, when meticulous hemostasis and isolation are achieved and an immediate high-quality coronal seal is placed, MTA-based partial pulpotomy or direct pulp capping in mature teeth is associated with stable sensibility, symptom resolution, and radiographic normality on follow-up—outcomes that align with the material's mechanism (seal first, bioactive interface) and with the broader VPT paradigm you outline [12,14,18]. In short, for carefully selected mature permanent teeth, MTA's sealing ability and bioactivity work in tandem to reduce bacterial ingress and sustain pulp vitality after partial pulpotomy or direct pulp capping, consolidating its role within contemporary conservative endodontics [12,14,18].

The pulpal response to MTA involves a cascade of cellular and molecular events that promote repair and regeneration. MTA stimulates the release of bioactive dentin matrix proteins, such as transforming growth factor-beta (TGF-β), which in turn promote odontoblastic differentiation [15]. It also provides a stable alkaline environment conducive to angiogenesis, fibroblast proliferation, and stem cell recruitment. Molecular studies have confirmed the upregulation of markers like dentin sialoprotein and osteocalcin in pulp cells exposed to MTA, corroborating its role in reparative dentinogenesis [9]. Additionally, the material's excellent

marginal seal limits microleakage and bacterial ingress, a critical determinant of long-term pulpal survival [12].

Despite these advantages, MTA is not devoid of limitations. Its extended setting time (ranging from 2 to 4 hours) poses clinical inconveniences, particularly in pediatric patients with limited cooperation. Handling difficulties, including its sandy texture, can compromise placement and marginal adaptation [20]. Discoloration of crowns, especially in anterior teeth, has been attributed to the bismuth oxide content used as a radiopacifier. This is a significant esthetic concern in young patients, leading to the development of modified formulations and alternative calcium silicate-based materials [17]. Furthermore, MTA's cost remains higher than that of traditional medicaments, which may restrict its widespread adoption in resource-limited settings [16].

A notable frontier for MTA lies in its integration into regenerative endodontic procedures. Its ability to serve as a coronal seal and scaffold for cell homing has been demonstrated in revascularization protocols of immature permanent teeth with necrotic pulps. Studies indicate that MTA placement over a blood clot scaffold results in continued root development in apexogenesis and apical closure in apexification. The success of such procedure's underscores MTA's adaptability and its potential role in advancing biologically centered treatment paradigms [18].

For clinicians, the choice of pulp therapy material directly influences long-term outcomes. In primary teeth, MTA pulpotomies offer reduced postoperative complications, fewer failures, and longer retention compared with formocresol, ferric sulphate or calcium hydroxide [2]. In permanent dentition, MTA enables both preservation of pulp vitality in young permanent teeth and long-term functional retention in adults [14,18]. The decision to employ MTA should account for factors such as cost, esthetic considerations, and patient compliance, but its biological predictability makes it the material of choice where pulp preservation is feasible [17].

Future Directions

Despite extensive validation, several areas warrant further research. First, efforts to shorten setting time and improve handling properties are critical for enhancing clinical efficiency. Second, alternatives to bismuth oxide radiopacifier must be further investigated to eliminate the issue of discoloration. Third, head-to-head randomized clinical trials with newer bioactive cements are necessary to

determine whether they offer incremental improvements over MTA or merely equivalent outcomes. Finally, as regenerative endodontics evolves, MTA's role in conjunction with stem cell-based and scaffold-mediated therapies may redefine pulp therapy protocols in the coming decades. Because MTA works well with primary pulps in pulpotomies, it stands to reason that we can also consider direct pulp capping with MTA for small, clean, iatrogenic exposures in primary teeth—provided hemostasis, isolation, and a good coronal seal are achievable. In short: if the exposure is accidental and the pulp is healthy, MTA-based DPC is a reasonable option. However, this indication remains underexplored in large pediatric cohorts, and long-term follow-up data are sparse. Future controlled trials are therefore required to validate safety, durability of dentin bridge quality, and physiological exfoliation patterns following MTA-based DPC in primary teeth.

CONCLUSION

The alkaline pH of Mineral trioxide aggregate (MTA) causes a short, self-limiting inflammatory phase, releases calcium ions, and forms hydroxyapatite at the dentin–material interface, which seals and bioactivates the pulp. It up-regulates growth factors (e.g., TGF-β) from dentin, driving stem/progenitor cells to differentiate into odontoblast-like cells. These cells lay a collagen scaffold that mineralizes into a tubular dentin bridge with a continuous, well-formed hard-tissue barrier. The result is stable hemostasis, tight seal, and durable vitality preservation.

Mineral trioxide aggregate (MTA) has revolutionized vital pulp therapy in both deciduous and permanent dentition. Its ability to provide an excellent seal, stimulate odontoblastic differentiation, and promote consistent dentin bridge formation makes it superior to conventional agents. In deciduous teeth, MTA has demonstrated higher success rates and reduced pathological complications, establishing itself as the material of choice for pulpotomy. In permanent dentition, particularly immature teeth requiring apexogenesis /Apexification, MTA has proven invaluable in preserving vitality and enabling continued root development / apical seal formation.

Despite limitations related to handling, discoloration, and cost, MTA remains the benchmark material against which newer calcium silicate-based cements are compared. With ongoing refinements and emerging bioactive alternatives, MTA

continues to play a central role in advancing evidence-based endodontics and pediatric dentistry. Ultimately, understanding the pulpal response to MTA across dentitions enhances our ability to preserve natural dentition, improve patient outcomes, and uphold minimally invasive treatment philosophies.DR

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