INTERNATIONAL JOURNAL OF HEALTH & MEDICAL RESEARCH

ISSN(print): 2833-213X, ISSN(online): 2833-2148

Volume 04 Issue 12 December 2025

DOI : 10.58806/ijhmr.2025.v4i12n01

Page No. 647 - 652

Pain in Pediatric Endodontics

Büşra Almas

Necmettin Erbakan University, Faculty of Dentistry, Department of Pediatric Dentistry

ABSTRACT:

Today, with the increase in living standards, easier access to information and the integration of technology into our lives, the interest in teeth in terms of aesthetics, function and phonetics is increasing. Although it is not at a sufficient level yet, the importance of protecting dental health is beginning to be understood day by day. Understanding dental health also increases the care and attention given to primary teeth. Early loss of primary teeth can lead to malocclusion or cause aesthetic, phonetic and functional problems that can be temporary or permanent. Early loss of primary teeth can lead to malocclusion or cause aesthetic, phonetic and functional problems that can be temporary or permanent. Preventing the early loss of primary teeth, providing space maintenance for a permanent tooth, maintaining lifelong health in the mouth for permanent tooth deficiencies, and fulfilling all their functions in a healthy way as long as they remain in the mouth, our primary goal is protection, and our next goal is to treat teeth that have lost their health. The aim is to help the patient understand the need for treatment in their primary teeth, to interpret the pain correctly and to explain treatment options that are appropriate to the characteristics of the pain.

KEYWORDS: Endodontics, Pain, Pediatric Dentistry

1. PULPAL DISEASES

Since the enamel and dentin thickness in primary teeth is less than in permanent teeth (approximately half as much), the width of the pulp chamber relative to the entire size of the tooth is relatively greater than in permanent teeth [1]. Therefore, there is a risk that the lesion will reach the pulp in a much shorter time and at a much shorter distance. This risk is higher in the first primary molars. For this reason, the risk of perforation should be taken into account, especially in deep dentin caries, when the caries is being cleaned [2]. The pulp horns of primary teeth, especially the mesial horns, are higher, which means that caries on the mesial surfaces can reach the pulp tissue much earlier and affect the pulp in a much shorter time. Different perspectives have been introduced in the literature regarding the classification of pulpal pathologies [3].

2. CLASSIFICATION OF PULPAL DISEASES

Pulpal diseases are classified according to histopathological, clinical, and inflammatory criteria. From a histopathological perspective, pulpal pathologies are examined under four main categories: acute and chronic pulp inflammations, conditions in which the pulp has lost its vitality, and degenerative changes. Acute pulp inflammations are observed as pulp hyperemia, serous pulpitis, and purulent pulpitis, whereas chronic cases are characterized by hyperplastic and ulcerative pulpitis. Conditions in which the pulp is non-vital include pulp necrosis and pulp gangrene, while pulpal degeneration encompasses structural changes such as pulp atrophy and pulp calcification. Clinically, pulpal conditions are divided into three primary groups: normal pulp, reversible pulpitis, and irreversible pulpitis. Reversible pulpitis may present with or without symptoms, while irreversible pulpitis can be symptomatic or asymptomatic and is associated with ulcerative or hyperplastic pulpitis [4]. Non-vital pulp conditions are clinically expressed as pulp necrosis and gangrene; internal resorption and pulpal degenerations constitute additional elements of this classification.

Pulpitis types are further evaluated based on the course of inflammation and whether the pulp is exposed or intact. Three main categories are recognized: hyperemic pulpitis, acute pulpitis, and chronic pulpitis. Acute pulpitis can manifest as primary (open) or secondary (closed), whereas chronic pulpitis may appear in an open form—hyperplastic or ulcerative—or a closed form, known as simple chronic pulpitis [5]. Pulp necrosis results from the interruption of the pulpal blood supply due to acute or chronic inflammation, or trauma, leading to the loss of pulp vitality. Edema exerts pressure on the apical vessels, causing cell death, and hydrolitic enzymes subsequently break down the tissue through autolysis. Necrosis is categorized into two types: coagulation necrosis, characterized by a cheese-like consistency, and liquefaction necrosis, in which proteolytic enzymatic activity leads to tissue softening and liquefaction. Pulp gangrene develops when necrotic or devitalized pulp becomes infected by microorganisms. Bacterial degradation and putrefaction produce foul odor, which is a typical clinical finding. The treatment of necrosis and gangrene

generally involves multistep root canal therapy, with biomechanical preparation playing a crucial role, particularly in gangrene cases [6].

3. PAIN IN PULPAL DISEASES

3.1. Pain

The purpose of a dental history is to learn the patient's primary complaint, known signs and symptoms, and the time of onset. If the complaint is pain, the characteristics and nature of the pain are determined through various questions. When did the pain begin? Can you identify the aching tooth? Because a tooth with irreversible pulpitis experiences referred and radiating pain, the patient may not be able to distinguish which tooth is aching. Pulpitis pain does not cross the midline but can be felt in the opposite jaw or in other teeth within the same jaw. Localization difficulties arise because only nociceptors are present in the pulp. When inflammation spreads to the periapex, where both pain and pressure receptors are located, the pain can be localized. Because pain occurs with biting or chewing pressure in a tooth with acute apical periodontitis, the problematic tooth is more easily identified. "What factors increase and decrease the pain?" "Do cold, heat, or chewing cause pain?" If so, when cold or heat causes pain, does the pain last for a few seconds, minutes, or longer? Short-term pain following any stimulus occurs in reversible pulpitis, whereas pain that begins especially following a thermal stimulus and continues for a long time suggests irreversible pulpitis. "Does cold or heat reduce the pain?" In irreversible pulpitis, intrapulpal pressure is increased. Cold, because it causes vasoconstriction, reduces the intrapulpal pressure and thus relieves the pain. "Does the pain begin spontaneously or with a stimulus (provoked)?" Spontaneous pain occurs without any stimulus. Spontaneous pain is usually present when there is irreversible pulpitis or acute or subacute inflammation in the periapical tissues. Teeth with chronic inflammation are usually asymptomatic. "Is the pain mild, moderate, or severe?" As the severity of the pain increases, the likelihood of irreversible pathology increases. All of these questions lead to a subjective history. However, pain is a complex phenomenon. Many aspects of pain provide diagnostic clues to the clinician, but they are insufficient to determine the extent of the problem. A definitive diagnosis can only be made after all clinical examinations have been completed [7].

Spontaneous pain occurs without any external stimulus and often indicates irreversible pulpal damage. Sensitivity to pressure may indicate that the pulpal damage has extended to the periodontal ligament, resulting in tooth extrusion. However, it may also be caused by a more benign condition, such as an overcontoured or high restoration leading to hyperocclusion. Children often complain of "toothache" during the eruption of permanent first molars. In such cases, the clinician should carefully determine whether the symptoms are attributable to pulpal pathology, pericoronitis, or trauma caused by biting on the operculum. Food impaction may also mimic the symptoms of irreversible pulpal damage. In cases of trauma, both the patient and the parents should be questioned regarding the timing and nature of the incident, as well as any previous history of traumatic events [8].

3.2. The Trigeminal Pain Pathway

Nerve endings in the pulp and periradicular tissues, when activated by an adequate stimulus, begin transmitting signals perceived as pain to the central nervous system through the release of inflammatory mediators. The anatomical structures responsible for this transmission are highly organized. Researchers have observed that the pain system is a multilayered and complex mechanism that starts with the detection of a tissue-damaging stimulus in the periphery, continues with the processing of information at the spinal cord level, and is ultimately perceived in higher brain centers such as the cerebral cortex [9].

The trigeminal system contains various types of peripheral neurons, including large-diameter, heavily myelinated Aα, Aβ, and Aγ fibers, which are responsible for motor activity, touch, pressure, proprioception, and muscle stretch. The fibers found in the pulp that conduct pain-related information consist of thinner, unmyelinated C fibers and smaller, lightly myelinated Aδ fibers. The conduction velocity of unmyelinated C fibers is 3–8 times slower than that of Aδ fibers [10]. A stimulus capable of inducing pain in healthy pulpal and periradicular tissues can depolarize nociceptors via voltage-gated sodium channels, thereby generating an action potential. Voltage-gated sodium channels play a critical role in the altered sensitivity of pulpal and periradicular nociceptors during inflammation, enabling even non-noxious stimuli to be perceived as painful [11].

Following repeated exposure to a noxious stimulus, both A- and polymodal C-fiber nociceptors undergo a sensitization process, with three distinct changes in their response patterns. First, the firing threshold decreases, and thus a previously innocuous stimulus can trigger a discharge and contribute to the patient's perception of pain (allodynia). Second, local tissue damage or increased tissue concentrations of certain chemicals lower the threshold of pain receptors, leading to the generation of pain signals. This can lead to afterdischarge and increased pain intensity (hyperalgesia). Third, firing occurs spontaneously and can lead to the development of pain [12].

Patients experiencing these distinct changes in the periradicular tissues should be informed. The sensitization process that results from alterations in these mediators is called peripheral sensitization, and these changes often occur in patients with endodontic pain [13]. These include substances produced in the inflammatory pulp and tissues, agents of vascular origin, and peptides released from nerve fibers. Mediators have been reported to act on or sensitize nociceptors. Increased concentrations of mediators such as bradykinin and interleukin 1-α have been reported in samples taken from inflamed dental pulps [14]. Following activation of peripheral nociceptors, nociceptive signals reach the spinal trigeminal tract nuclear complex located in the medulla in the trigeminal

pain system. Three distinct nuclei are present in the complex. These are referred to as the oral, interpolar, and caudal nuclei due to their anatomical positions. Although many rostral subnuclei (oralis, interpolaris) receive some nociceptive input from oral tissues, most of this input is received at the level of the caudal subnucleus [15]. Because of its organizational similarities to the dorsal horn of the spinal cord, the caudal subnucleus is referred to as the dorsal horn of the medulla. This serves as the primary nociceptive processing center for signals from primary afferent sensory nerve fibers and transmits nociceptive input to higher centers in the brain. Nociceptive signaling can be facilitated (hyperalgesia), inhibited (analgesia), or misinterpreted (referred pain) [16]. Through the release of transmitters such as excitatory amino acids, glutamates, neuropeptides, and substance P, primary afferent fibers (whose bodies are located in the trigeminal ganglion) transmit signals to projection nerves. There is evidence that referred pain occurs through the convergence of afferent signals from different regions onto the same projection neurons. In the trigeminal pain system, the cell bodies of second-order (projection) neurons are located in the medullary dorsal horn; their projections cross the midline and reach the thalamus via the trigeminothalamic tract. Third-order neurons from the thalamus send information to the cerebral cortex via a thalamocortical tract. This is the final anatomical step in the trigeminal pain pathway. Once the signal reaches the cortex, it can be perceived as pain [17].

3.3. Pain History And Characteristics

It is crucial to establish a clear definition of pain based on its pathophysiological mechanism. When we review the literature, we encounter numerous definitions of pain developed at different times. Pain is defined as a personal, distinct sensory perception of noxious stimuli, a signal confirming ongoing tissue damage, and a theoretical concept that functions as a system of stimuli that protect the organism from damage. Another definition states that pain is an aversive sensory and emotional experience originating in a specific area of the body, usually resulting from a noxious stimulus, and also originating from potentially damaged body tissues. Bonica's definition is that pain is a subjective perception that persists even after it has been relieved. Summers, emphasizing the individual nature of pain, defines it as "the perception of pain is subjective, and pain is how the person experiencing it describes it, and when they say it is happening." [18].

The history and characteristics of pain are often important in determining whether the pulp is treatable. However, if there are extensive carious lesions and drainage, the child may not have a history of significant pain. Or, if dental problems begin very early (e.g., early childhood caries, baby bottle tooth decay), the child may lack conscious awareness of pulpal symptomatology. A dentist should be aware of these limitations and be able to distinguish between types of pain. Pain can be classified as chronic, provoked, or spontaneous [19].

Chronic pain: If the child reports intermittent odontogenic pain in the past, this is chronic pain and indicates widespread pulp degeneration or pulp necrosis. The infection may even have spread to the periodontal tissues. Endodontic intervention is generally indicated for these teeth based on radiographic evaluations [20].

Provoked (causative) pain: If pain results from thermal or chemical changes in the oral environment or is elicited during mastication, it is classified as provoked pain. However, provoked pain should be evaluated separately as transient or persistent. Transient pain represents the pulp's temporary response to temperature or pH changes occurring in teeth with deep carious lesions near the pulp or when dentin becomes exposed due to the loss of restoration. The pain subsides once the oral environment returns to normal. In such cases, the pulp is considered vital, the existing infection is confined to the coronal pulp, and the condition is reversible. Therefore, pulp capping or pulpotomy can preserve the tooth in a healthy and functional condition [21].

Spontaneous pain: Spontaneous pain is a type of pain that begins without any specific stimulus and is throbbing and persistent, often preventing the patient from sleeping at night. In such cases, the pulp is no longer considered treatable. It is assumed that widespread inflammation is present within the pulp, and the pulpal and even periodontal tissues have been largely destroyed. Root canal treatment is indicated; however, based on a combined assessment of clinical and radiographic findings, tooth extraction may be required [22].

3.4. **Pulpal Pain**

Acute pulpal pain has many causes, the most important of which is infection. Bacteria enter the pulp via dental caries and fractures, following the periodontium and systemic blood circulation. Direct trauma to the tooth may induce pulpitis, acute pulpitis-associated pain, incomplete fractures, or complete fractures that expose the pulp. As a result of trauma, the tooth can fracture partially or completely, interrupting the apical blood supply, leading to pulpitis and necrosis. Repetitive microtrauma, as in chronic bruxism, can also predispose to pulpal necrosis followed by pulp inflammation [23].

Pulpal pain is a spontaneous and severe pain caused by pulpal damage. Temperature changes, fermentable carbohydrates, caries, and pressure on the tooth can trigger the pain. The pain may be stimulus-evoked or occur spontaneously. Pulpal pain, similar to dentinal pain, is difficult to localize, and localization becomes even more difficult as the pain intensifies. Pain may radiate from adjacent areas to the ear, temporomandibular joint, or teeth in the maxillary or mandibular arch. Pain may increase when the patient assumes a supine position, potentially awakening the patient from sleep [24]. Pulpal pain can originate from either vital or nonvital pulps. In vital pulps, pain can be reversible or irreversible. In reversible cases, the pulp can restore pulpal vitality with the removal

of the irritant because it has the capacity to repair itself. In irreversible cases, the pulp cannot cannot recover its physiological vitality even if the irritants are removed [25].

Although the pain associated with pulpal pathology can be severe, there are also cases of pulpitis in teeth with pulpal necrosis that are asymptomatic. Acute pulpal pain is characterized by the pulp being a visceral tissue. Pulp pain is described as mild tenderness, moderate dull ache, or severe throbbing or burning pain. Pulp pain typically lacks prolonged persistence, and infection can lead to dental abscess formation. The pain may transition to chronicity or diminish in intensity. Acute pulpitis must be distinguished from periapical pain such as trigeminal neuralgia, maxillary sinusitis, otitis media, masseter muscle dysfunction, orofacial and myofascial pain, acute periapical inflammation, and acute periodontal pain [26].

3.5. **Dental Pain İn Children**

In dentistry, pain is one of the critical diagnostic parameters for understanding the condition of a decayed tooth and determining treatment options. It's crucial to interpret the clinical significance of pain, and and, if present, identify its etiological origin. Pain history and the characteristics of the pain are crucial in pulp therapy. However, identifying pain in children is not as consistent or clinically reliable as it is in adults. We often observe that children with multiple carious lesions don't complain of pain. This can be explained by factors such as conceal symptoms due to dental anxiety, reduced symptom perception secondary to hyperactivity, and higher pain thresholds [27].

Even if pain is recognized, children are likely to provide unreliable symptom descriptions. Sometimes, parents misrepresent or simplify their child's toothache to avoid being accused of negligence by the dentist. Despite all this, the dentist must first determine whether there is pain and, if so, the nature of the pain. The questions to be asked should be at a level the child can understand. For example, to determine whether the tooth has been affected by thermal or chemical changes, ask, "Does your tooth hurt when you eat ice cream?" Questions such as "Do you experience pain when eating hot foods?" or "Do you experience pain when eating sweet things like chocolate or candy?" can be asked to determine whether the tooth is affected by percussion. Questions such as "Do you flinch when the toothbrush touches your teeth while brushing, or when a fork or spoon touches your teeth while eating?" can be asked to determine the duration of the pain. Because children have a unique concept of time, the duration of the pain can be determined by comparing it with the child's daily activities. For example, questions such as "Did the pain last throughout the lesson? Did the pain that started at meal period disappear after starting the lesson?" can be used to determine the duration of the pain [28].

The response to the question of whether tooth pain awakens the child at night assists in estimating the potential severity of the condition. However, responses to these questions may not always reflect a pathological condition analogous to that of the permanent dental pulp. The absence of pain is not a reliable indicator of pulpal status. Since any form of toothache may be associated with pulpal inflammation, the lack of pain does not necessarily imply that the pulp is healthy and vital, particularly in deeply carious primary teeth. Indeed, it has been reported that the pulp may become completely necrotic in many children without producing any symptoms. Therefore, if a child experiences no pain during cavity preparation in deeply decayed primary teeth, the dentist should consider the possibility of pulp devitalization devital [28].

4. PULP TESTS

4.1. Thermal Tests

It is an clinically feasible and demonstrably reliable method. Thermal applications affect the hydrodynamic mechanism and, consequently, the A nerve fibers in the superficial pulp, producing pain. The ability of the pulp to react to sudden temperature changes in inflammatory conditions is a determining factor in the evaluation. This application not only helps identify the affected tooth but also provides insight into the pathological condition of the pulp. In teeth with physiologically intact pulpal status, pain disappears immediately upon removal of the stimulus during thermal testing. Cold and heat cause a temporary reaction in hypersensitive and hyperemic pulps. The pain subsides once the application is discontinued. In cases where pain persists after the stimulus is removed, irreversible pulpal inflammation is suspected [29].

4.2. Electric Pulp Tests

It is a diagnostic test that provides results based on the response of nerve fibers in the superficial pulp tissue stimulated by an electric current. Electrical or battery-powered devices (vitalometers) are utilized for this purpose. All contemporary electric pulp testers operate using high-frequency alternating current. The exact numerical value displayed in digitally equipped electric pulp testers is clinically insignificant and does not reflect the degree of pulpal vitality. The electric pulp test determines whether the pulp is vital or non-vital; it does not evaluate the inflammatory status of the pulp [30].

CONCLUSION

Primary teeth exfoliate between the ages of 6 and 12 to make way for permanent teeth. Any tooth loss that occurs before the physiological resorption of the primary tooth roots and their spontaneous exfoliation without intervention is defined as premature primary tooth loss. Preventing premature loss, maintaining the necessary space for the eruption of permanent teeth, preserving lifelong oral health in cases of permanent tooth absence, and ensuring that primary teeth can perform all their functions efficiently

while present in the oral cavity constitute our primary objective of preservation. The subsequent objective is to treat teeth that have lost their integrity. In this context, understanding and accurately interpreting the pain that alerts the patient to the need for treatment is of critical importance.

REFERENCES

1) Fuks, A. B., Kupietzky, A. R. I., & Guelmann, M. (2019). Pulp therapy for the primary dentition. In Pediatric dentistry (pp. 329-351). Elsevier.
2) Fuks, A. B. (2000). Pulp therapy for the primary and young permanent dentitions. Dental Clinics of North America, 44(3), 571-596.
3) Brosnan, M. G., Natarajan, A. K., Campbell, J. M., & Drummond, B. K. (2014). Management of the pulp in primary teeth–an update. NZ Dent J, 110(4), 119-23.
4) Rossman, L. E. (2009). American Association of Endodontists. Journal of the American College of Dentists, 76(1).
5) Fuks, A. B., Peretz, B., & Teeth, Y. P. (2016). Pediatric Endodontics. Current Concepts in Pulp Therapy for Primary and Young Permanent Teeth. Ed. Israiel: Editorial Springer.
6) Thakkar, T. K., Naik, S., & Ghule, K. (2019). Advances in rotary endodontics in pediatric dentistry. EC Dent Sci, 18(6), 1320-30.
7) Aminoshariae, A., & Kulild, J. C. (2021). Current concepts of dentinal hypersensitivity. Journal of endodontics, 47(11), 1696-1702.
8) Enabulele, J. E., Sede, M. A., Chukwumah, N. M., & Ehizele, A. O. (2024). Endodontic Treatment Need Index (ETNI): A Comprehensive Index for Assessing Endodontic Treatment Needs in Traumatized Anterior Teeth.
9) KEISER, K., & BYRNE, B. E. (2011). Endodontic pharmacology. In Cohen's Pathways of the Pulp (pp. 671-690). Mosby.
10) Dionysopoulos, D., Gerasimidou, O., & Beltes, C. (2023). Dentin hypersensitivity: etiology, diagnosis and contemporary therapeutic approaches—a review in literature. Applied Sciences, 13(21), 11632.
11) Rechenberg, D. K., Galicia, J. C., & Peters, O. A. (2016). Biological markers for pulpal inflammation: a systematic review. PloS one, 11(11), e0167289.
12) Diogenes, A., & Hargreaves, K. M. (2017). Endodontic Infections and Pain. Endodontic Microbiology, 251-267.
13) Maslamani, M., & Sedeqi, F. (2018). Antibiotic and analgesic prescription patterns among dentists or management of dental pain and infection during endodontic treatment. Medical Principles and Practice, 27(1), 66-72.
14) Yu, C. Y., & Abbott, P. V. (2018). Pulp microenvironment and mechanisms of pain arising from the dental pulp: From an endodontic perspective. Australian Endodontic Journal, 44(2), 82-98.
15) Takemura, M., Sugiyo, S., Moritani, M., Kobayashi, M., & Yonehara, N. (2006). Mechanisms of orofacial pain control in the central nervous system. Archives of histology and cytology, 69(2), 79-100.
16) Marriott, D., Wilkin, G. P., Coote, P. R., & Wood, J. N. (1991). Eicosanoid synthesis by spinal cord astrocytes is evoked by substance P; possible implications for nociception and pain. Advances in prostaglandin, thromboxane, and leukotriene research, 21, 739-741.
17) Levin, L. G., Law, A. S., Holland, G. R., Abbott, P. V., & Roda, R. S. (2009). Identify and define all diagnostic terms for pulpal health and disease states. Journal of endodontics, 35(12), 1645-1657.
18) Riepenhausen, A., Wackerhagen, C., Reppmann, Z. C., Deter, H. C., Kalisch, R., Veer, I. M., & Walter, H. (2022). Positive cognitive reappraisal in stress resilience, mental health, and well-being: A comprehensive systematic review. Emotion Review, 14(4), 310-331.
19) Okamoto, C. B., Bussadori, S. K., Prates, R. A., da Mota, A. C. C., Horliana, A. C. R. T., Fernandes, K. P. S., & Motta, L. J. (2020). Photodynamic therapy for endodontic treatment of primary teeth: A randomized controlled clinical trial. Photodiagnosis and photodynamic therapy, 30, 101732.
20) Agarwal, S. R., Bendgude, V. D., & Kakodkar, P. (2019). Evaluation of success rate of lesion sterilization and tissue repair compared to Vitapex in pulpally involved primary teeth: a systematic review. Journal of Conservative Dentistry and Endodontics, 22(6), 510-515.
21) Igna, A. (2021). Vital pulp therapy in primary dentition: Pulpotomy—A 100-year challenge. Children, 8(10), 841.
22) Dean, J. A. (2015). Treatment of deep caries, vital pulp exposure, and pulpless teeth. Dentistry for the Child and Adolescent, RE Mc Donald and DR Avery, Eds, 221-242.
23) Sharav, Y., & Benoliel, R. (Eds.). (2008). Orofacial pain and headache. Elsevier Health Sciences.
24) Lenz, F. A., Dougherty, P. M., Meeker, T. J., Saffer, M. I., & Oishi, K. (2024). Neuroscience of the human thalamus related to acute pain and chronic “thalamic” pain. Journal of neurophysiology, 132(6), 1756-1778.
25) Sáez, F. J., & Badiola, I. (2025). Dental Pulp. In Essential Oral Histology (pp. 325-341). Cham: Springer Nature Switzerland.

26) D'Orto, B., Baldi, C., Bonafede, V., Pacifici, A., Ianniceli, I., & Nagni, M. (2023). Analgesic drugs in dentistry: a narrative review. Annali di stomatologia, 14(4), 23-29.
27) Pentapati, K. C., Yeturu, S. K., & Siddiq, H. (2021). Global and regional estimates of dental pain among children and adolescents—systematic review and meta-analysis. European Archives of Paediatric Dentistry, 22(1), 1-12.
28) Miroshnychenko, A., Azab, M., Ibrahim, S., Roldan, Y., Martinez, J. P. D., Tamilselvan, D., ... & Brignardello-Petersen, R. (2023). Analgesics for the management of acute dental pain in the pediatric population: a systematic review and meta-analysis. The Journal of the American Dental Association, 154(5), 403-416.
29) Fan, L., Zeng, J., Ran, L., Zhang, C., Wang, J., Yu, C., & Zhao, N. (2023). Virtual reality in managing dental pain and anxiety: a comprehensive review. Frontiers in Medicine, 10, 1285142.
30) Carrasco-Labra, A., Polk, D. E., Urquhart, O., Aghaloo, T., Claytor Jr, J. W., Dhar, V., ... & Moore, P. A. (2023). Evidence-based clinical practice guideline for the pharmacologic management of acute dental pain in children: A report from the American Dental Association Science and Research Institute, the University of Pittsburgh School of Dental Medicine, and the Center for Integrative Global Oral Health at the University of Pennsylvania. The Journal of the American Dental Association, 154(9), 814-825.