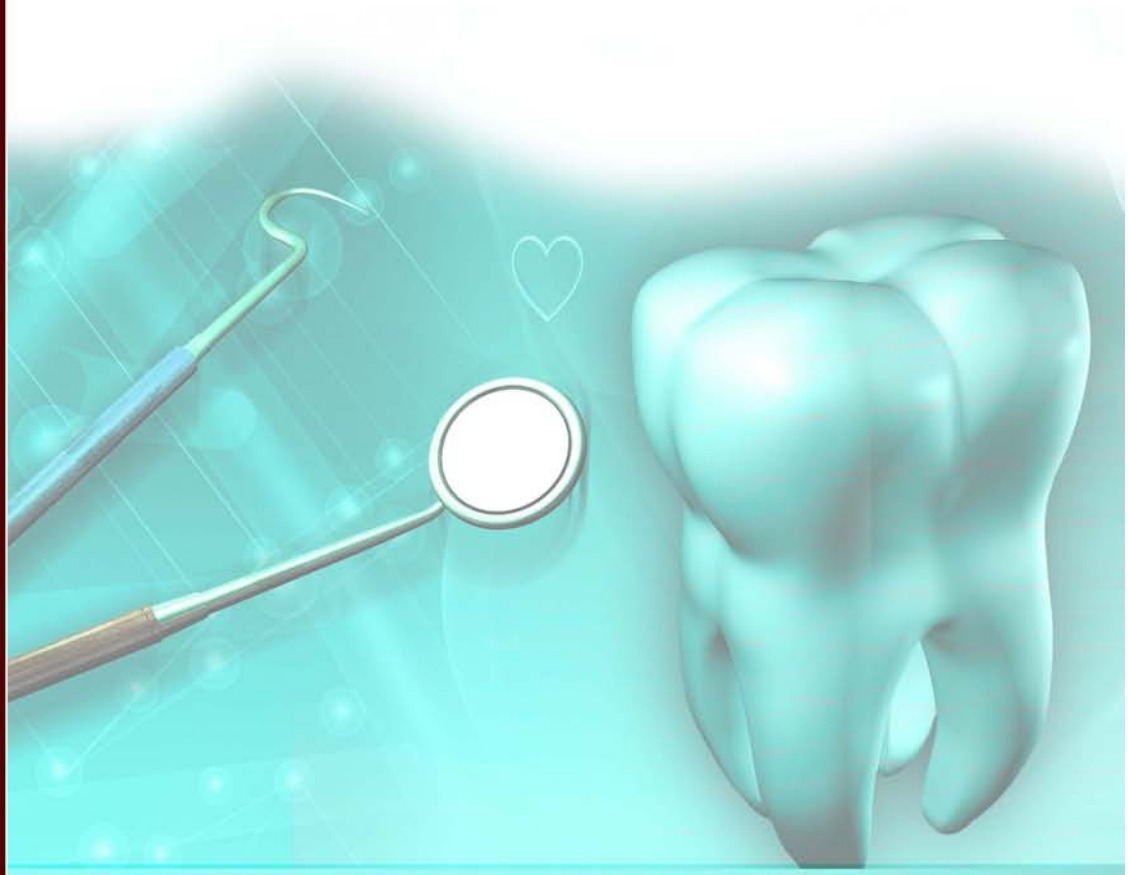
Print-ISSN-
E-ISSN-

# Journal of Interdisciplinary Dental Sciences

Vol. 1 No. 2, July-Dec 2012

An Official Publication of The SD MESOC Foundation Parbhani

JIDS



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Journal of Interdisciplinary Dental Sciences

# Contents

# Editorial

It gives me immense pleasure to write the editorial for the second issue of the journal. It has been a pretty tough job although but I sincerely want to thank all my colleagues for supporting me in this endeavor. Bringing out the second issue of the journal with multi-specialty articles has been an enlightening vista for readers and I am extremely delighted to say that we are getting huge response from several colleges. With this, I expect similar work-out from everyone in the future to make this journal a huge success and I wish that the journal proves to be a tremendous source of knowledge for all its readers.

**Dr. Vivekanand S. Kattimani**
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# Centuries of Endodontics

*Dr. V. Santi, MDS*, Dr. Vummidisetti V. Subbarao, MDS***

**Abstract:**

As far back as 1500 B. C., the Greeks, Romans and Chinese have been focused on remedies to relieve and treat tooth pain. The Chinese first described dental caries through the tooth worm theory. Medical literature depicts inscriptions of worms atop tooth structure and its subsequent damage. The history of Endodontics begins in the 17th century. Since then, there have been numerous advances and developments, and research has proceeded continuously. At that time, necessity was the mother of invention: experimenting with new techniques, materials, and instruments, even though very rudimentary. The aim of Endodontics has been to relieve pain, maintain exposed pulp, and preserve teeth. Often, these attempts were successful. The profession should be prepared to move on with knowledge of past. This article briefly revisits the history of endodontics.

**Key words:** history, endodontics.

**Introduction:**

As far back as 1500 B. C., the Greeks, Romans and Chinese have been focused on remedies to relieve and treat tooth pain. The Chinese first described dental caries through the tooth worm theory.[1] Pierre Fouchard, in the "Surgical Dentist" refutes the worm theory in 1728, as he describes a method of access and removal of the offending pulpal tissue with the consequent placement of lead fillings. This historic text of the 18th century truly marks the beginning of endodontics in medicine.[2] Leonard Koecher expanded upon this idea in 1820 as he used a heated instrument to effectively cauterize the infected pulpal tissue and protect the remaining tissue with lead foil.[2,3]

The techniques, technologies, materials, and practices continued to improve with dentists performing rudimentary root canals to relieve pain and restore teeth in their patients. The year 1910 marked a shift in the paradigm of this type of treatment. Decades earlier Miller had proposed the concept that general disease was influenced by oral infection, and by extrapolation the microorganisms that cause the oral infection could disseminate from the focus to the entire body via the bloodstream.[1] This theory did not gain acceptance until an English pathologist and physician, William Hunter, gave a lecture on focal infection in 1910. His lecture, "The Role of Sepsis and Antisepsis in Medicine" halted advancement in endodontics as he accused the dentist of covering "a mass of sepsis" with gold fillings.[4] This "focal infection theory" lead to the mass extraction of teeth and crippled the advancement of endodontics for more than 20 years.

It was not until the late 1940s or early 1950s that the cumulative laboratory research and clinical evidence was sufficient to confirm that the devitalized tooth did not play a role in the causation of systemic disease. Thus the theory of focal infection fell and faith was restored in endodontic treatment.[5] Johnson's colleague, Jasper, promoted conservation of teeth and focused on improving endodontic success as he advocated strict asepsis, standard treatment protocols, and precise root length measurement. He denounced the use of mummifying agents to fix diseased pulpal tissue and thus campaigned that all pulpal tissue must be removed. Mitchell et al.[6] As root canal therapy gained momentum in the 1940s, a group of 20 dentists seeking an organization that would serve as the steward of endodontic treatment met in Chicago in 1943. The result of their meeting was The American Association of Endodontists (AAE) and began to set the standard of endodontic treatment in dentistry. These efforts in 1943 would grow to create the American Board of Endodontics in 1956. The American Dental Association validated these early efforts as endodontics was officially recognized as a specialty in 1963.

The way in which medicine and dentistry was practiced would change drastically with the influence of x-rays and their discovery by Wilhelm Roentgen in 1895.[7] Edmund Kells, an entrepreneur and dentist, was the first to apply the use of x-radiation to a dental setting and in 1913 marketed and sold the first x-ray machine.[1] Within five years, dentists were using the technology to visualize and enhance endodontic treatment as well as evaluate the successes of treatment. The practice of endodontics has been around for hundreds of years dating all the way back to the first century A.D. when the concept of draining root canals emerged as a way to relieve pain and pressure. Since then, much has changed, but it is interesting to note how this specialty has progressed over the years, and who helped contribute to each advancement that eventually led to where it stands today. *Below is a timeline that highlights every major development in the practice of endodontics:*[8]

**1728:** In his book "Le chirugien dentiste", Pierre Fauchard precisely described the role of dental pulp, and dispelled the legend of the 'tooth worm'—which dated back to the Assyrians (612 BC) as the primary cause of decay.

**1746:** Fauchard went on to accurately describe the removal of pulp tissue.

**1756:** Phillip Pfaff used gold and lead for pulp capping.

**1766:** Robert Woofendale alleviated pain by cauterizing the pulp and stuffing the open canal with cotton.

**1800:** Frederick Hirsch used percussion as a diagnostic aid.

**1809:** Edward Hudson is credited with inserting the first root canal fillings.

**1819:** Charles Bew described pulp circulation through the apex, then the dental wall, and to the periodontal ligament.

**1820:** Leonard Koecker cauterized exposed pulp with a heated instrument and protected it with lead foil.

**1821:** Koecker went on to theorize the aim of pulp capping.

**1836:** Shearjashub Spooner recommended arsenic trioxide for pulp devitalization.

**1838:** Edwin Maynard created the first root canal instrument which was made by filing a watch spring.

**1847:** Edwin Truman introduced gutta percha as a filling material for endodontic therapy.

**1850:** Codman confirmed Koeker's theory from 1821 that pulp capping was intended for dentin bridge formation.

**1864:** S.C. Barnum prepared a thin rubber leaf to isolate a tooth for the purpose of filing.

**1867:** Magitot proposed the use of an electric current in order to test pulp vitality.

**1867:** Bowman used gutta percha as the sole filling material to obturate root canals.

**1873:** S.C. Barnum and G.A. Bowman introduced the first rubber dam forceps.

**1880:** J.N. Farrar introduced the radical treatment of alveolar abscess, while E.S. Talbot prepared nerve canals for treatment and filling for the first time.

**1882:** Arthur Underwood popularized the use of caustic antiseptic agents to sterilize the pulp chamber and canal.

**1885:** Lepkoski replaced formalin with arsenic to "dry" the non-vital pulp stumps that were left in canals after the removal of coronal pulp in order to prevent decomposition.

**1890-1900:** There was a surge in popularity of prosthetic restorations, including Richmond or Davis crowns, which required the use of canal posts, and therefore increased the need for endodontic therapy.

**1891:** Otto Walkhoff introduced camphorated chlorophenol as a medication to sterilize root canals.

**1892:** E.C. Briggs initiated the removal of tooth pulp with the use of cocaine.

**1894:** S.C. Barnum developed the rubber dam.

**1895:** Konrad Wilhelm von Roentgen accidentally discovered a new form of energy that could penetrate solid materials, which later became known as X-Rays.

**1895:** Otto Walkhoff took the first dental radiograph a few weeks after the discovery of X-rays.

**1900:** Periapical radiolucencies were recalled as "blind abscesses".

**1900**: Radiographs were proposed as a way to diagnose pulpless teeth.

**1904:** Frank Billings suggested a relationship between oral sepsis and bacterial endocarditis.

**1906:** J.P. Buckley introduced the rational treatment of putrescent pulps and their sequellae.

**1908:** Meyer Rhein introduced a technique for determining canal length and level of obturation.

**1908:** G.V. Black suggested a way to measure the length of canals and the size of the apical foramen to prevent overfilling.

**1909:** Rosenow developed the "focal infection theory"—which suggested a relationship between bacterial aspects and root canal therapy.

**1909:** Mayrhofer published an article that linked pulpal infections to specific organisms, and found that Streptococci were present in roughly 96% of cases studied.

**1912:** Guildo Fisher published the first comprehensive study of root canal anatomy.

**1916:** Carl Grover showed that toxic chemicals could produce apical lesions.

**1925:** UG Rickert recommended the use of sealer with a gutta percha cone.

**1929:** Balint Orban showed microscopic evidence that the pulp has the same cells as other connective tissues.

**1936:** Walker recommended the use of sodium hypochlorite as a canal irrigant.

**1938:** Zander and Teuscher used calcium hydroxide for vital pulp capping.

**1943:** A group of 20 men formed the American Association of Endodontics (AAE).

**1943:** Harry B. Johnston officially coined the term *endodontia*.

**1946:** Journal of Endodontics (JOE) was published as the first periodical dedicated exclusively to endodontics.

**1947-1954:** Following the "focal infection theory" paranoia, a group of men including Coolidge, Johnson, Reihn, Callahan, Grove, Prinz and others continued to improve procedures in order to preserve pulpless teeth—which eventually confirmed that devitalized teeth had no direct influence on systemic disease.

**1956:** The American Board of Endodontics (ABE) became established.

**1959:** Winkler and Van Amerogen studied root canal flora.

**1963:** The ADA officially recognized endodontics as a specialized area of dentistry.

**1963:** More than 200 dentists in the US were limiting their practice to endodontics.

**1965:** The first Diplomate exams were initiated.

**1967:** Herb Schilder introduced his technique on filling root canals in 3-D using thermo-softened gutta percha/sealer.

**1982:** The Touch-n-Heat electric heat carrier was introduced—eliminating the need for open flame in the operatory.

**1987:** Buchanan modifies Schilder's technique using a "continuous wave" of condensation.

Owing to the efforts of these researchers, patients today can be assured of predictably reliable and safe endodontic treatments, the success rate of which, as perhaps in no other branch of medicine, approaches 100%. Thanks to them, the number of people who specialize in Endodontics today is very high and continues to rise and Endodontics has assumed its precise role in the field of dentistry.

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REVIEW ARTICLE

# Tissue Engineering : Future of Pediatric Endodontics

*Dr. George Kurian Panampally, MDS*, Dr. Vivek M. Patil, MDS**,Dr. Rehan Khan, MDS***, Dr. Anil Kumar Patil, MDS*****

**Abstract:**

During the last 10–15 years, there has been a tremendous increase in our clinical "tools" (i.e. materials, instruments and medications) and knowledge from the trauma and tissue engineering fields that can be applied to regeneration of a functional pulp-dentin complex. Tissue engineering is a multidisciplinary approach that aims to regenerate functional tooth tissue structure based on the interplay of three basic key elements: Stem cells, morphogens and scaffolds. A number of recent clinical case reports have revealed the possibilities that many teeth that traditionally would be treated byapexification may be treated by apexogenesis. Pulpal regeneration after tooth injury is not easy to accomplish, because of the infected pulp requires tooth extraction or root canal therapy. Current treatment modalities offer high levels of success for many conditions; an ideal form of therapy might consist of regenerative approaches in which diseased or necrotic pulp tissues are removed and replaced with healthy pulp tissue to revitalize teeth. This review discusses fundamental concepts of stem cell biology and tissue engineering within the context of regenerative dentistry.

**Keywords :** Tissue engineering, pediatric endodontics, future of pedodontics

**Introduction :**

There is a high rate of success in retention of teeth by endodontic therapy. However, many teeth are not restorable because of apical resorption, fracture, incompletely formed roots or carious destruction of coronal structures. A novel approach to restore tooth structure is based on biology i.e. regenerative endodontic procedures by the application of tissue engineering. Tissue engineering is an emerging multi disciplinary field that applies the principles of engineering and life sciences for the development of biological substitutes that can restore, maintain, or improve tissue function.[1] Regenerative endodontic procedures can be defined as biologically based procedures, designed to predictably replace damaged, diseased, or missing structures, including dentin and root structures as well as cells of the pulp dentin complex with live viable tissues preferably of the same origin that restore the normal physiologic functions of the pulp dentin complex.[2]

Hermann (1952)[3] was the first to carry out regenerative endodontic procedure, when he applied calcium hydroxide in vital pulp amputation. Subsequent regenerative dental procedures included guided tissue or guided bone regeneration (GTR, GBR) procedures and distraction osteogenesis,[4] the application of platelet rich plasma (PRP) for bone augmentation,[5]emdogain for periodontal tissue regeneration,[6] recombinant human bone morphogenic protein (rhBMP) for bone augmentation,[7] and preclinical trials on the use of fibroblast growth factor 2 (FGF2) for periodontal tissue regeneration.[8,9] A counter argument to the development of regenerative endodontic procedure is that although the replaced pulp has potential to revitalize the teeth, it may also become susceptible to further pulp disease and may require retreatment. Over the last two decades, tissue engineering has evolved from science fiction to science. Indeed, isolated clinical case reports are consistent with the concept that certain clinical treatments might evolve into regenerative endodontic procedures.[10] However, additional translational research is needed to develop predictable clinical regenerative procedures. The purpose of this article is to review the biological principles of tissue engineering and the hurdles that must be overcome to develop regenerative endodontic procedures.

**Tissue Engineering**

The field of tissue engineering has literally exploded during the last decade, and extensive

reviews on dental applications are available for the interested reader.[2,11]Here we briefly review 3 major components of tissue engineering from the concept of developing regenerative endodontic treatment regimens.

Tissue engineering is the field of functional restoration of tissue structure and physiology for impaired or damaged tissues because of cancer, diseases, and trauma. The key elements of tissue engineering are stem cells, morphogens, and a scaffold of extracellular matrix.[12,13]

**Adult stem cells:**

All tissues originate from stem cells. A stem cell is defined as a cell that has the ability to continuously divide to either replicate itself (self replication) or produce specialized cells that can differentiate into various other types of cells or tissues.[14]

Types of stem cells are:

1. Early embryonic stem cells
2. Blastocyst embryonic stem cells
3. Fetal stem cells
4. Umbilical cord stem cells
5. Adult or postnatal stem cells

The plasticity of stem cell defines its ability to produce cells of different tissues. Stem cells are commonly subdivided into totipotent, pluripotent and multipotent categories according to their plasticity.

To accomplish endodontic regeneration, the most promising cells are autologous postnatal stem cells, because these appear to have the minimum disadvantages. Postnatal stem cells have been found in almost all body tissues, including dental tissues. Four types of human dental stem cells have been isolated:

(a). Dental pulp stem cells (DPSCs)

(b). Stem cells from Human exfoliated deciduous teeth (SCHED)

(c). Stem cells from apical papillae (SCAP)

(d). Periodontal ligament stem cells (PDLSCs)

Odontoblasts are post-mitotic terminally differentiated cells which cannot proliferate to replace subjacent irreversibly injured odontoblasts. The ability of both young and old teeth to respond to injury by induction of reparative dentinogenesis suggests that a small population of competent progenitor pulp stem cells may exist within the dental pulp throughout life.[15]

Information on the mechanism by which these cells are able to detect and respond to tooth injury is a scarce, but this information will be valuable for use in developing tissue engineering and regenerative endodontic therapies.[15]

Several, if not all, adult tissues have a subpopulation of stem cells. Examples of such tissues are the bone marrow, brain, skin, muscle, and adipose tissue.[16,17] Stem cells have also been found in several dental tissues. One of the first tooth-related stem cell types was found in the pulp of permanent teeth and was named dental pulp stem cells(DPSCs).[18] In addition, stem cells from human exfoliated deciduous teeth (SHED), stem cells from the apical papilla, dental follicle progenitor cells, and periodontal ligament stem cells have also been characterized. Mechanistic studies focused on these cells are certainly improving our understanding of tooth development. In addition, this knowledge has been applied in translational studies that aim at the use of these stem cells in clinical settings where the regeneration of dental and craniofacial tissues is indicated.[19]

The usefulness of stem cells in clinical applications depends on their proliferation rate, differentiation potential, and accessibility. For example, when bone marrow stem cells were compared with DPSCs, DPSCs presented favourable results with regard to odontogenic capability.[20] Stem cells of dental origin can certainly generate dentaltissues. It has been shown that SHED and DPSCs are capable of generating a tissue that has morphological and functional characteristics that closely resemble those of human dental pulp.[21] Other studies have expanded the potential of these cells in the treatment of diseases and conditions such as muscular dystrophies, critical size bone defects, corneal alterations, spinal cord injury, and systemic lupus erythematosus. Such studies clearly demonstrate the plasticity and the differentiation

potential of stem cells of dental origin. And finally, SHED cells have the unique advantage of being retrievable from naturally exfoliated teeth, which can be considered a "disposable" source of postnatal human tissue. Collectively, these studies suggest that the tooth constitutes an attractive source of stem cells that can potentially be useful in a wide spectrum of clinical scenarios.[19]

Recent evidence suggests that stem cells are localized in areas with low oxygen tension. Work on hematopoietic and neural stem cells showed that culturing progenitors in hypoxic conditions increases the number of multipotent clones when compared with normoxic cultures. In addition to effects on differentiation and cell fate, hypoxia promotes survival and increases the proliferation of multipotent precursors. This phenomenon may depict clinical situations in which pulp tissues are affected by noxious stimuli such as mechanical pulp exposure or trauma that leads to localized ischemia. The secondary dentin bridge that formed under the injury site is possibly the product of differentiated progenitors from deciduous pulp stem cell reservoir. Further studies are required to understand whether DPSCs react differently to signalling molecules after hypoxic treatment, which might alter their differentiation potential.[22]

**Signalling molecules and dental pulp stem celldifferentiation**

Growth factors and morphogenic factors are proteins that bind to specific membrane receptors and trigger a series of signalling pathways that coordinate all cellular functions. These molecules play a critical role during development, guiding processes that determine the fate of stem cells and regulate the generation of all tissues and organs in the developing embryo. Similarly, these morphogenic molecules play a critical role in physiological processes of tissue regeneration as, for example, wound healing in the skin or dental pulp responses to the progression of dentinal caries. The same growth factors that guide embryogenesis and physiological tissue regeneration can also be used therapeutically to guide stem cell differentiation toward specific cell fates and to coordinate cellular processes that result ultimately in the generation of a new tissue or organ via tissue engineering-based approaches. More specifically, there are many similarities between morphogenic factors regulating dentinogenesis and the factors that regulate reparative dentinogenesis.It can be easy to conclude that the field of dental tissue engineering can benefit tremendously from studies focused on the cellular and molecular mechanisms of odontogenesis. Growth factors have an important role in signalling reparative processes in dentin and pulp. Indeed, it is known that factors such as transforming growth factor, bone morphogenic proteins (BMPs), platelet-derived growth factor, fibroblast growth factor, and vascular endothelialgrowth factor (VEGF) are incorporated into thedentin matrix during dentinogenesis and are retained thereas "fossilized" molecules. Interestingly, when these moleculesare released from the dentin, they are bioactive andfully capable of inducing cellular responses, as for examplethose that lead to the generation of tertiary dentin and todental pulp repair.[23]

The application of recombinant human insulin like growth factor-1 together with collagen has been found to induce complete dentin bridging and tubular dentin format.[24]This indicates the potential of adding growth factors before pulp capping or incorporating them into restorative and endodontic materials to stimulate dentin and pulp regeneration. The therapeutic effect of calcium hydroxide may be because of its extraction of growth factors from dentin matrix.[25]Once released, these growth factors may play key roles in signalling many of the events of tertiary dentinogenesis, a response of pulp dentin repair.

FGF2 plays a role not only as a differentiation inducing factor in the injury repairprocess of pulpal tissue but also as a positive regulator of chemokine expression, which may help in tissue engineering and pulp regeneration using Human DPSCs. However, the fate of odotoblastic or osteoblastic differentiation, effective local delivery for FGF2

interaction of chemotactic and odontogenic factor limitations need to be overcome.[26] Ability of MTA to induce useful cellular response to achieve suitable tissue wound healing is by promoting by adhesion, supporting cellular proliferation and by inducing migration of human mesenchymal stem cells. Mesenchymal stem cells are usually involved in tissue and bone remodelling, and local environment is thought to play an important role in the commitment and differentiation of mesenchymal derived stem cells.[27]

**Scaffold:**

The scaffold provides a physico-chemical and biological three dimensional micro environments for cell growth and differentiation, promoting cell adhesion and migration. The scaffold serves as a carrier for morphogens in protein therapy and for cells in cell therapy.

Types of scaffolds:

(a). Biological or Natural e.g. Collagen, Glycosaminoglycan

(b). Artificial or Synthetic e.g. Poly lactic acid (PLA) Poly glycolic acid (PGA), Poly ethylene glycol (PEG), Arginine, Hydroxyapatite, Tricalcium Phoshate.[15]

**Gene therapy:**

New techniques involving viral or non viral vectors that can deliver genes for growth factors, morphogens, transcription factors and extracellular matrix molecules into target cell populations has been developed. The use of gene delivery in endodontics would be to deliver mineralizing genes into pulp tissues to promote tissue mineralization. Dr.Rutherford transfected ferret pulps with cDNA-transfected mouse BMP-7 that failed to produce a reparative response, suggesting that further research is needed to optimize the potential of pulp gene therapy. Because of the apparent high risk of health hazards, the development of a gene therapy to accomplish endodontic treatment seems very unlikely in the near future.[15, 29]

**Potential technologies for regenerative endodontics:**

Following are the areas of research that might have application in the development of regenerative endodontic techniques:[15]

1. Root canal revascularization via blood clotting
2. Postnatal stem cell therapy
3. Pulp implantation
4. Scaffold implantation
5. Injectable scaffold delivery
6. Three – Dimensional cell printing
7. Gene therapy

A study has found that inducing bleeding of pulp was easier and effective when an anestheticsolution did not contain a vasoconstrictor.[30]

**Challenges and future direction:**

Collectively, there has been a tremendous increase in our clinical tools (i.e. materials, instruments, and medications) and knowledge from the trauma and tissue engineering fields during the last decade. Despite the impressive progress in tissue engineering approaches to regenerative pulp therapy, numerous challenges remain. The associated broad spectrum of responses in pulp includes neural and vascular regeneration.[15, 19, 28]

**(a) Nerve regeneration:**

Dental pulp is richly innervated. The main nerve supply enters the pulp through apical foramen along with the vascular elements. They include both sensory and sympathetic nerves. Pulpal nerves play a key role in regulation of blood flow, dentinal fluid flow, and pressure. The innervation of the pulp has a critical role in the homeostasis of the dental pulp. The pulpal nerve fibers contribute to angiogenesis, extravasation of immune cells and regulate inflammation to minimize initial damage, maintain pulp tissue, and strengthen pulpal defense mechanisms. The increasing interest in tissue engineering of tooth must take into account neuro-pulpal interactions and nerve regeneration.[31]

**(b) Vascular regeneration:**

Pulp vasculature plays an important role in regulating inflammation and subsequent repair and regeneration of dentin. There is an intimate association of the neural elements with vascular supply of the dental pulp suggesting the interplay of neural and vascular elements and involvement in pulp homeostasis. The vascular endothelial growth factor (VEGF) is an excellent regulator of

angiogenesis and is known to increase vascular permeability. VEGF induces chemotaxis, proliferation and differentiation of human dental pulp cells. The utility of gene therapy in stimulation of vascular growth permits local stimulation of vascularization during regeneration.[32]

The recent advances in vascular biology and VEGF and techniques of gene transfer and gene therapy will be of potential clinical utility in dentistry, especially in endodontics. Statin, 3-hydroxy-3-methy glutayl coenzyme A reductaseinhibitor is known to promote bone formation.

Pulp tissue contains a large amount of blood vessels and peripheral nerves. Statin is known to induce angiogenesis and to regulate the survival and increase neurogenesis of neuronal cells, indicating the possible effectiveness of statin in pulp regeneration along with dentin regeneration. Furthermore, statin has an anti-inflammatory effect in various tissues. This could help to restore the inflamed pulp tissue. Taken together, results suggest that statin might be an ideal active ingredient in pulp capping material to accelerate reparative dentin formation. However, at the same time attention has to be paid to the cell death observed in the cells treated with high concentration of statin. Therefore, a careful evaluation of the suitable concentration is required before its use in pulp regeneration.[33]

**(c) To measure appropriate clinical outcomes we have to find out the following:**

1. Vascular blood flow
2. Mineralizing odontoblastoid cells
3. Intact afferent innervations
4. Lack of signs or symptoms

**Conclusion:**

Tissue engineering using the triad of dental pulp stem cells, morphogens and scaffolds may provide an innovative and biologically based approach for generation of clinical materials and treatment of dental diseases. The challenges of introducing endodontic tissue engineered therapies are substantial; the potential benefits to patients and the profession are ground breaking.

Better understanding of cell interactions and growth along with further research can make endodontic tissue engineering a reality in the near future. Progress will depend on the collaboration between clinicians and researchers from diverse fields (e.g., biomaterials, stem cell biology and endodontics) working together toward the goal of developing biological approaches to regenerate dental and craniofacial tissues.

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REVIEW ARTICLE

# Centuries of Oral Radiology

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**Abstract**

In today's scenario, can we imagine dental profession without radiology or treatment without diagnosis? The beginning of radiology was when Roentgen discovered X-rays and dental radiology began with Edmund Kell's first intraoral radiograph. Since then many experiments were done for development of newer techniques and machines like panoramic machine, CT scan, MRI etc. The best part in radiology is mistakes done in past gives us path for improvising future with more precise diagnosis and hazard free radiology.

**Key words:** Cathode ray, radiant matter, emissive, X-rays, radiology, X-ray machine, darkroom.

**Intoduction:**

The discovery of a 'fragile substance, fair in color and fine-looking in transparency' from Baltic shores by Phoenician voyager Thales of Miletus began history who named it as electron or amber and noted that it attracted light particles of matter when rubbed.[1] The term electricity was coined by Gilbert(1600) who also categorized all things into electric or nonelectric. Stephen Gray ascertained that current would flow over conductor for large distances.[2] Charles DuFay (1730) discovered two variants of electricity i.e. 'vitreous' and 'resinous' which were called by Franklin as 'positive' and 'negative' electricity.[3] Otto von Guericke (1648) devised the first air-pump used to create vacuum.[4] The first permanent vacuum situated in empty space above the mercury column was formed by Evangelista Toricelli's mercury barometer in 1643.[1]

**Discovery of X-rays:** Sir William Morgan (1785), while examining the discharge of current in perfect vacuum, obtained a vacuum so high that there was no discharge. During one of his experiments, the glass cracked and he visualized a display of colors. Serendipitously, he was the first man to produce X-rays.[5] Michael Faraday in 1821 conducted his foremost experiment on electric discharge in partial vacuum and expressed fluorescence as 'radiant matter' and deemed it as the fourth state of matter. Wilhelm Hittorf (1870) improvised vacuum pumps and introduced the term 'cathode ray'. Sir William Crooke in 1880s considered 'radiant matter' to be the 'ultra gaseous stage'. He referred to a 'molecular' and 'emissive' ray from his tube which could only be seen when a fluorescent screen was placed in path of the rays beyond the tube. He unknowingly produced X-rays.[6] The 'Inverse square law' was proposed by Lenard.[6] Jead Perrin in 1895 declared that cathode rays are negatively charged particles.[7]

While experimenting in Wurzberg, Bavaria, Professor Wilhelm Conrad Roentgen discovered rays that could penetrate substances opaque to light, and the degree of penetration was proportional to the density of the substance. The same could also not be reflected or refracted. These were unchanged by magnetic or electric fields similar to cathode rays He termed these rays "X-rays" as X in American way stands for unknown and eventually called them 'Roentgen Rays'.[8] While investigating he accidentally positioned his hands between the tube and the fluorescent screen and was astounded to see image of bones within his hand on the screen and he consequently demonstrated that images of the body could be recorded on photographic plates. He took the first radiograph of human body, by placing his wife Bertha's hand on photographic plate and exposed it to the mysterious rays for 15 minutes and when developed, he found that outline of bones in her hand could be seen.[8] Roentgen was awarded the first Noble Prize in Physics in 1901.[7]

The first bite-wing image was made by Dr Friedrich Otto Walkhoff who used a regular photographic glass plate wrapped in rubber dam

placed between the teeth and tongue. When the image was obtained, the crowns of the maxillary and mandibular teeth were seen. In 1898, he accomplished making extra-oral pictures with an exposure time of 30 minutes. In 1896 Walkhoff along with Fritz Giesel set-up the first dental Roentgenologic laboratory in the World.[7]

The 'Father of Dental Radiology', Dr C Edmund Kells (1880) took first intraoral radiograph on a live person in the US in 1896 in his dental office.[7] He fabricated a film holder made from a thin aluminium plate and gutta percha, for patient to bite into occlusion, thereby holding the film in place during swallowing.[2] He was the foremost to promote right-angle or paralleling technique in taking intraoral radiographs.[3] He was also the first dental surgeon to utilize radiographs in endodontic therapy on May 10, 1899.[5]

Dr. John Daniel intimated loss of hair from the head of a colleague who was exposed to radiation.[8] The first microscopic study of the effect of radiation on tissues about a case of severe skin reaction after prolonged exposure was published by W Marcuse.[7] Leonard recommended placing aluminium sheet between the tube and the patient to avoid static charges at the skin. He proposed that the same also eliminated many of the needless X-ray beams and saved several patients from serious injury.[3]

Four fundamental pieces of apparatus were employed by the radiological chemist -

1. X-ray machine
2. X-ray tube
3. Adjustable tube stand
4. The darkroom.[3]

The X-ray machine comprised of three essential components - the induction coil, the interrupter and the rheostat.[6]

Induction coil offers a source of high potential current for the X-ray tube.[6] Interrupter is of two basic types-

1. Mechanical

a. Vibrating type

b. Mercury type

2. Electrolytic [8]

**Gas Tubes**: Early vacuum tubes had partial vacuum as source of electrons at cathode, but with the gas tubes the gas molecules were trapped by vaporized residues from the anode and cathode which amplified the vacuum. When the vacuum became too high no X-rays were produced and this 'cranky' tube could be heated by alcohol lamp to drive gas molecules from its walls which sustained continued production of X-rays.[4]

**Regulator Tubes**: To augment the durability of X-ray tube, automatically self regulating and regenerative tubes were developed in 1896 by Queen and Company, in which a degree of vacuum, when changed repeatedly, by the operator permitted variation of the penetrability of X-rays.[5] It used the principle that certain chemicals (caustic potash and potassium permaganate) liberated gases upon heating (that cause the vacuum in the main tube to be lowered sufficiently to produce X-rays again) and absorbed them upon cooling. To assist alteration of vacuum an accessory bulb (filled with chemicals which produced gas on heating) was fitted with the chief bulb.[5]

**Evolution of X-ray machine**: In 1907, Clyde Snook established his first X-ray machine rated at 110kVp and 200mA.[7] A phenomenal day in radiology was the development of the hot cathode tube by William David Coolidge, (1913).[7] It permitted-

1. Enhanced flexibility in the quality and quantity of X-rays produced.
2. Superior tube stability during the production of X-rays.
3. Lesser tube size.
4. Longer tube life.
5. Direct operation from a transformer.[6]

With the invention of the autotransformer, flexibility and dependability of the tube producing high voltage current was augmented.[8] A tungsten anode backed by copper was found to be the most agreeable method of dispelling heat rapidly; heat was conducted to the radiation fins at the end of the tube or by circulating cold water through anode stem.[1]

In 1916, Coolidge self rectified his previous model which had good competence when unrectified AC

voltage was applied between the anode and cathode.[7] Until 1918, all X-ray tube cooling was by means of air and water.[6]

Hirsh patented the thought of submerging the X-ray bulb in oil for superior cooling of anode and tube.[4] In 1919, Harry Waite submerged the tube and transformer as a single unit in same oil bath.[7]

**Insulation of Dental X-ray Unit**: The high voltage wires used formerly were un-insulated, open and unguarded due to which many dentists and patients were unfortunately burned.[2] The principle of this design was to position the tube and high voltage components in an oil filled grounded compartment which acted as an electric insulator, coolant and radiation shield. The benefit of this tube was that the electrical and fire peril was eliminated.[2]

**Remodeling of the Tube Stand**: Dr William Rollins (1896) designed an apparatus that featured a protective screen and an adaptable diaphragm to prevent unnecessary irradiation of the patient.[1] He was first to bring out harmful effects of X-rays and build a protective device to shield the X-ray tube.[6]

**X-ray films:** The first dental radiograph was taken on a small glass plate which had been cut to size from bigger ones.[7] Kells and Rollins used photographic film.[5] In the early days, films were hand-made and comprised of glass plates or roll films, cut to proper length and wrapped in black paper and rubber dam material to prevent moisture escape.[8]

In 1920's cellulose nitrate base was used. In 1924, non-inflammable cellulose triacetate was used. In the same year the emulsion was placed on either side which doubled the speed of film, reduced exposure time by half and lessened the tendency to curl when dried. In 1940s ultra speed films became obtainable.[8] In 1960s a film base of polyester was introduced so thinner films could be made. In 1980s Ekta speed film reduced the exposure by fifty percent.[7]

Evolution of the dark room

Early darkroom was a closet with or without running water, of the size three and a half feet by five feet.[2]The steps involved in development were-

1. Covering the sensitive material with a developing agent.
2. Adding a preservative.
3. Adding an accelerator.
4. Adding bromide.[2]

Automatic processors were introduced in the 1910s.[7]

**Development of Dental Radiograph**: William Herbert Rollins (1852-1929) also known as the 'Father of radiation protection'.[3] In 1896, he invented an X-ray arm and bracket for the dental office.[6] He was first to advocate the use of radium for treatment of cancers.[7] He gave three defensive measures to dental and medical X-ray users-

1. Wear radiopaque (leaded) glasses.
2. Enclose the X-ray tube in a leaded (or other non-radiable) housing and
3. Irradiate only the area of interest of the patient and cover all adjacent areas with radiopaque materials.[3]

He introduced collimator to reduce the beam size and recommended a long target film distance to improve image quality.[6] He advised draping of patient in non-radiable material.[2] He pioneered inserting the film between two intensifying screens to reduce exposure. He suggested use of filtration of the X-ray beam to remove low energy X-rays.[2]

The two techniques for film positioning in the oral cavity- the paralleling and the bisecting angle technique were introduced by Weston A Price in 1904. He held the film in the patient's mouth and used opaque (leaded) rubber gloves.[8]

In 1910, Franklin W McCormack opened the first dental X-ray laboratory.[7] He used extended target film distance.[3]

Franklin W McCormack hand wrapped his dental films in black paper, adding a flat metal plate to give the film firmness and then wrapped both in waxed paper for use in the patient's mouth; this blocked the penetration of the roentgen rays and prohibited entry of backscattered radiation on the film.[5]

Dr Gordon Fitzgerald (1907-1981) designed long cone for the dental X-ray machine.[6]

In 1949, the American Academy of Oral Roentgenology (now known as American Academy of Dental Radiology) was formed.[7]

**History of Panoramic Radiography**: Dr. H Numata was the first to suggest (1933) and experiment(1934) with the method of panoramic radiography.[7] Numata placed a curved film in the mouth lingual to teeth and used a thin slit or narrow X-ray beam that rotated around the patient's jaw to expose the film.[3] Y.V.Paatero proposed, experimented and demonstrated a slit beam method of panoramic radiography for dental arches.[4] In 1960s S.S. White and Company marketed the first Panoramic machine (Panorex).[6]

**History of Computed Tomography**: Radon described the concept that two dimensional or three dimensional objects could be reconstructed from infinite set of all its projections.[5]In 1969, Godfrey Hounsfield developed the prototype scanner.[1] In 1970, with the aid of computer technology, this concept was clinically applied by Hounsfield and it was known as computed tomography(C T).[1] In 1971 the first scanner installed and in 1972, the first commercially viable CT scanner was invented by G.N.Hounsfield. In 1987, first dental computed tomography reformatting package called Denta Scan was planned.[8] Interactive CT was introduced in 1993.[6]

This sectional imaging technique provided diagnostic radiology with better insight into the pathogenesis of the body, thereby increasing the chances of recovery.[2] It delivers non-superimposed, cross-sectional images of the body, which can show slighter contrast differences than conventional X-ray images.[3] The first CT scanner was a siemens SOMATOM Plus system.[6] In 1974, scanning time was 2.5 min while in 1975, it was 18sec.[8] In 1976, the Whole body CT image started.[6] In 1980 Electron Beam CT was introduced by Andrew Castagnini.[5] In 1990, Helical also called spiral CT was introduced. In 1992, Integrated CT angiography was introduced.[7]

**History of Magnetic Resonance Imaging**: Felix Bloch and Edward Purcell, both were awarded the Nobel prize in 1952, discovered magnetic resonance phenomenon independently in 1946.[7] In 1971, Raymond Damadian showed that the nuclear magnetic relaxation times of tissues and tumors differed thus motivating scientist to consider magnetic resonance for the detection of disease.[4] In 1973, the X-ray based computerized tomography (CT) was introduced by Hounsfield.[6] On July 3, 1977 first MRI exam performed on human being in 5 hrs but now it takes seconds.[7]

In same year Peter Mansfield developed the echo-planar imaging(EPI) technique.[3] In 1987, Charles Dumoulin was perfecting magnetic resonance angiography (MRA) which allowed imaging of flowing blood without the use of contrast agents.[5] In 1991, Richard Ernst was rewarded for his achievement in pulsed Fourier Transformer NMR and MRI with Nobel prize in Chemistry.[1] In 1992, functional MRI was developed which allows mapping of function of various regions of brain.[6] In 2003, Paul C.Lauterbur and Sir Peter Mansfield were awarded the nobel prize in medicine for MRI.[7]

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REVIEW ARTICLE

# Effect of growth hormone therapy on craniofacial bones - A Comprehensive Review

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**Abstract**

Growth hormone (GH) has significant effects on linear bone growth, bone mass and bone metabolism. The primary role of GH supplementation in children with GH deficiency, those born small for gestational age or with other types of disorders in somatic development is to increase linear growth. However, GH therapy seems to elicit varying responses in the craniofacial region. Whereas the effects of GH administration on somatic development are well documented, comparatively little is known of its effects on the craniofacial region. The purpose of this review was to search the literature and compile results from both animal and human studies related to the impact of GH on craniofacial growth.

**Keyword:** craniofacial growth, growth hormone, growth hormone deficiency.

**Introduction:**

Growth hormone (GH) is a peptide hormone produced and secreted mainly by the somatotroph cells of the anterior pituitary gland. Its secretion occurs in a pulsatile pattern and its serum level varies greatly throughout the day. Secretion is low in the prepubertal period, rises at puberty (0.4–0.5 mg per 24 h) and decreases in old age. GH acts on tissues directly or through other growth factors. The somatomedin theory introduced the concept that GH stimulates skeletal growth by stimulating insulin-like growth factor-I (IGF-I), which in turn stimulates longitudinal bone growth and exerts a negative feedback on GH secretion by action on the hypothalamus and pituitary. Circulating GH is bound to a GH-binding protein, which is the extracellular domain of GH receptor (GHR). GHR is ubiquitously expressed, and GH has direct effects on most tissues. GH is an important for the regulation of longitudinal bone growth, whereas GH deficiency (GHD) or other defects in the GH signal-transduction pathway such as Laron syndrome lead to growth disturbances, mainly short stature, as a result of inhibition of pituitary gland hormones.

The skeleton is formed via two mechanisms: intramembranous and endochondral ossification. Endochondral ossification accounts for the formation of the vertebrae and long bones, whereas the cranial bones are formed through intramembranous ossification. Although an association between craniofacial and somatic development has been clearly established, growth of the craniofacial region is complex because it involves interaction of adjacent growth sites, each with a different pattern and timing of growth maintained that the splanchnocranium follows the general growth curve and the neurocranium the neural growth curve, whereas cranial base was considered to follow a combination of the general and neural growth curves. Growth of the craniofacial skeleton intergraded between Scammon's neural and general growth curves, according to concept of a 'craniofacial growth maturity gradient'. According to this concept, the craniofacial skeleton expresses a continuous pattern of variation in maturity, which manifests between structures as relative maturity for a particular structure at a given time. A 'maturity gradient' runs from head height through the anterior cranial base, posterior cranial base and maxillary length, upper facial height, corpus length and ramus height.

Growth hormone influences skeletal growth primarily by stimulating the growth of cartilage in areas of endochondral ossification. The condylar cartilage is a secondary type of cartilage, and is unique among ossifying cartilages in the skeleton in that it is derived from cells of periosteal origin of the neural crest. In the mandibular condyle, chondrogenesis is activated when external stimuli (e.g. condylar repositioning) cause the

differentiation of mesenchymal cells in the articular layer of the cartilage into chondrocytes, which proliferate and then progressively mature into hypertrophic cells.

Humans with GHR mutations (P56IT variant) had a significantly smaller mandibular ramus length than did those without P56IT. Furthermore, the mandibular linear parameters tended to be smaller in subjects carrying the heterozygous rather than the homozygous P561T mutation. Growth hormone also influences skeletal craniofacial growth by stimulating the growth of cartilage of the cranial base synchondrosis.

GH enhances cranial base growth directly by stimulating prechondrocytes in the synchondrosis in a similar manner to growth plates. The synchondroses of the cranial base are similar to long bone growth plates morphologically, in that the chondrocytes are distributed into resting, proliferating and hypertrophic zones. However, a major difference is that the growth of the cranial base has the unique characteristic of being bidirectional. As GH has become more readily available via recombinant DNA techniques, the therapeutic benefit of GH supplementation in improving height in children of idiopathic short stature (ISS) and GHD children as well as in those born small for gestational age (SGA) has been widely recognized.

An ISS patient grows <4 cm a year without discernible cause, and the bone age is usually retarded 2 years compared with chronological age[1]. These children are characterized byconstitutional delay of growth and puberty or GHD. In SGA individuals, the bone age is usually retarded 1–2 years compared with chronological age, whereas the pubertal growth spurt occurs early and is reduced in magnitude. Children with GHD display significant maturational delays and reduced somatic growth. Skeletal age may be delayed by up to 2 years. In clinical practice, we should know both the characteristic craniofacial features of these children and the subsequent effects of GH administration on craniofacial growth. However, information about the craniofacial effects of GH is limited because ethical considerations make it impossible to perform human prospective clinical trials to evaluate the effects of GH on craniofacial growth. The purpose of this article was to review the literature on animal and human studies related to the impact of GH therapy on craniofacial bones.

**Craniofacial characteristics in GHD children**

Single case reports of the effects of GHD on facial structuresstarted to appear in the literature almost 70 yearsago and studies of larger size havedescribed well the facial characteristics of children withGHD. Most cephalometric studies demonstratedshort ramus height as well as small linear dimensionsin the posterior cranial base and mandibular andmaxillary lengths.

Thegreater reductions in posterior compared with anterior cranialbase length may be explained by the early, higher rateof growth of the anterior cranial base [during the first2 years of life[42]] as well as the earlycessation of growth in the synchondroses ofthis skeletalcomponent.The cranial base angle and gonial angle as well as the angle between the maxillary and mandibular planes are larger than normal.

**Influence of GH administration in children**

The effect of GH supplementation on craniofacial growth has been studied in children with ISS or GHD and those born SGA, as well as in children with syndromes or hypo pituitary deficiency. Poole et al (1982) demonstrated that GH therapy increases mandibular length and lower face height while minimally affecting the cranial base length in five of eight treated cases. With maxillary length increased disproportionately during treatment. This is influenced by the small number of samples, because variability in response to hGH must be considered when estimating treatment effects. Moreover, it has been observed that GH supplementation provokes proportional increases in facial height, but narrower facial widths and disproportionate increases in head circumference.

Cantu et al (1997) evaluated growth in 40 GHD children during treatment with replacement therapy (0.3 mg kg_1 per 3–6 times per week). The sample was divided into three groups based on the duration of GH therapy (<0.2 years, 0.2–2.0 years, >2.0 years). Posterior facial height, posterior cranial base length and anterior facial height showed greater improvement among the three groups than any other craniofacial measurement. Age at the start of GH administration positively influenced most measurements except the cranial base length, anterior facial height and maxillary length. Interestingly, ramus height failed to display any significant catch-up. These results showed that therapy should start as early as possible to take advantage of the differential growth potentials of facial structures. High-dose GH supplementation (0.2 or 0.3 IU kg_1 per day) over a 2-year period in 21 SGA children led to significant craniofacial catch-up growth in posterior total facial height, cranial base length and mandibular length. The higher the dose (100 kg_1 per day compared with 67kg_1 per day) and the younger the child at the beginning of treatment, the more pronounced were the effects. However, linear measurements 2 years after the cessation of GH administration showed a significant 'catch-down' growth effect, although the measurements remained larger than those of untreated children.

Investigationof GH supplementation in 28 Turner's syndrome patients yielded no significant effects on growth. Although the maxillary length and the mandibular ramus height had increased, the higher cranial base angle, decreased posterior facial height and decreased mandibular length continued to create the retrognathic face characteristic of the syndrome.

Long-term (3.3 years) but not short-term (1.2 years) GH therapy in Japanese GHD children resulted in significantly higher values for upper facial height, maxillary length and ramus height compared with the untreated group. The mean s.d. scores for mandibular length tended to increase with the duration of therapy, even if no significant differences were observed.

GH administration on craniofacial morphology in GHD and ISS boys were studied prospectively. All linear variables showed significant catch-up growth approaching normal values. The earlier the GH administration, the larger were the positive effects on craniofacial structures such as the posterior cranial base. All sagittal angles improved significantly, whereas growth rates beyond the norm were observed for the mandibular corpus length and total and lower anterior faceheights.

**Discussion**

A catch-up growth phase could be defined as a recovery phase after the removal of a growth-inhibiting condition such as GHD. Tanner (1986) has suggested that catch-up growth can occur in two patterns. In the first, the individual shows early growth acceleration that reduces the deficit rapidly. In the second pattern, the individual remains at a low growth percentile for years and grows at a normal velocity beyond the usual age. Completely mature measures do not have the potential to 'catch-up'. Based on findings in GHD adults, a 'biphasic model' ofGH action in bone remodeling has been suggested. According to this model, GH administration initially increases bone resorption with bone loss that is followed by a phase (12–18 months after treatment) of increased bone formation. The transition point (6 months) occurs when bone formation proceeds at a higher rate than bone resorption.

In the cranial base, the intersphenoidsynchondrosis fuses around the time of birth and the spheno-ethmoidal around 6–7 years of age, whereas the spheno-occipital synchondrosis fuses shortly after puberty. Human investigationshave shown that earlier GH administration results in larger positive effects on craniofacial structures such as the cranial base. Humans with idiopathic GHD showed catch-up growth in the posterior cranial base in response to GH treatment, whereas in SGA children both the anterior and posterior cranial bases showed significant growth catch-up. The different treatment effects could be attributed to individual growth

spurts, sex differences and variations in response to GH treatment.

Defects that disrupt the normal growth and development of the cranial base result in craniofacial malformation deformities such as achondroplasia, Apert and Crouzon syndrome, cleidocranial dysplasia and mandibulofacialdysostosis.

The condylar cartilage, a secondary type of cartilage, is structurally distinct from the limb growth plate. It differs primarily in that its superficial layers comprise a perichondrium in which prechondroblastic cells secrete type I collagen rather than type II collagen, which is secreted by the chondrocytes. When there is excess GH, local IGF-I synthesis is stimulated; mitotic activity and activity of the mature cells of the mandibular cartilage increase, leading to more endochondral ossification . Conversely, a lack of GH decreases mitotic activity because there is less IGF-I synthesis, leading to less endochondral ossification. In children with GHD, ISS and SGA, posterior and anterior facial height was increased by GH supplementation because of both spheno-occipital synchondrosis and condylar cartilage growth

Moreover, high doses (up to 100 mg kg_1 per day) of GH therapy resulted in a pronounced growth response for both total posterior and anterior face heights. Regarding the maxillary bone, some investigators reported that GH administration could increase the maxillary length as well others found no significant differences. Moreover, GH stimulates periosteal bone apposition and muscle mass enhancement, resulting in further development of the soft tissue complex that pulls the maxilla and associated structures forward.

Craniofacial growth is a complex interaction between genes and hormones. In dentofacial orthopedics, control of craniofacial growth is essential for determining treatment goals and predicting stability during the retention period. The increased availability of recombinant GH has resulted in more conditions being treated with this therapy, and clinicians are therefore becoming more likely to treat a child who is under GH therapy.

**Conclusion**

The benefits of GH therapy in improving skeletal maturation and somatic growth in short children are widely recognized. Despite the relatively widespread use of GH to augment stature, the effects of this practice on the growth of the craniofacial complex have not been extensively investigated. Craniofacial growth in GHD, both in animals and humans, supports the concept that certain regions of the head are affected more severely by the deficiency and respond more strongly to GH supplementation.

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REVIEW ARTICLE

# Surgical Management of Massive Adult TMJ Ankylosis - A Case Report

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**Abstract:**

Ankylosis may be defined as the fusion of articular surfaces with bony or fibrous tissue. The treatment of temporo-mandibular joint ankylosis poses a significant challenge because of the high incidence of recurrence. The etiologies include trauma, arthritis, infection, previous TMJ surgery, congenital and Idiopathic[1]. The most common etiology of TMJ ankylosis is trauma, with the second being infection[2]. The major sequence of TMJ ankylosis treatment includes complete resection of ankylotic block, creation of a new joint lining with an interpositional substance, and reconstruction of skeletal deformity. This protocol was first established by Kabanet al[3]. Majority of the cases reported in previous published articles were cases in children. In cases of Adult TMJ ankylosis more vigorous post operative physical therapy is mandatory, and it is difficult to use mandibular growth potential to adjust the occlusion. We present a case of a patient with unilateral massive TMJ ankylosis treated with gap arthroplasty along with interpositioning of temporalis myofascial flap.

**Key words:** Massive, TMJ, Ankylosis, Adult ankylosis, Temporalis myofascial flap

**Introduction:**

Ankylosis may be defined as the fusion of articular surfaces with bony or fibrous tissue. The treatment of temporo-mandibular joint ankylosis poses a significant challenge because of the high incidence of recurrence. The etiologies include trauma, arthritis, infection, previous TMJ surgery, congenital and Idiopathic[1]. Temporo mandibular joint ankylosis might include fibrous or bony ankylosis in the TMJ that limit functional opening. Incidence of Adult TMJ ankylosis is low and only few articles are quoted in literature compared to TMJ ankylosis in children which makes it challenging to treat. Multiple procedures are used to manage TMJ ankylosis, but none have been universally accepted. The operative procedures include 1) gap arthroplasty with or without interpositional grafts and 2) resection of the ankylotic mass and reconstruction of the ramus-condyle unit (RCU) with autogenous or alloplastic grafts.

**Case Report:**

A 45 yr old female patient was referred to department of oral & maxillofacial surgery with a chief complaint of inability of opening mouth and facial asymmetry*fig(1)* since 25 years. There was a history of trauma in her adolescence when she was 17 yr old, for which she was not treated. Since then, the mouth opening gradually reduced*fig(2)* and developed a swelling on left side of her face. The patient did not seek any medical advice for the problem. Now, due to peer pressure and siblings pressure she is psychologically ready to consult and undergo necessary treatment.

The clinical examination of the patient revealed facial asymmetry due to hard swelling in left TMJ region*fig(1)*, slight dental midline deviation towards right side, reduced mouth opening with maximum interincisal opening (MIO) being 2 mm*fig(2)*. TMJ movements are not felt in left side and mildly palpable in right side. On contrary to findings of a classic ankylosis patient, this patient did not have retrogenia, malocclusion, prominent antegonial notch, elongated coronoid processes and hypoplastic mandible.

Several radiographic investigations likepanaromic view, pre and post contrast axial and coronal CT, 3D CT *fig(3)*were performed. Panoromic radiography revealed complete loss of TMJ architecture in left side, the right side was normal. Around 4.5x4 cm bony ankylotic mass was seen in left side TMJ region. In axial CT, ankylosis appeared to extend from lateral aspect of zygomatic arch medially as far as foramen spinosum. In coronal CT*fig(4)*, elongated lateral pterygoid plate was fused

to mandible and relation of maxillary artery is medial to ankylotic segment. 3D CT reconstruction *fig(3)* shows the sigmoid notch obliterated and ankylosis as a bilobed mass.

A diagnosis of severe sawhney's class IV type left bony TMJ ankylosis was made. Treatment plan included surgical removal of ankylotic mass, interpositional arthroplasty with temporal myofascial flap and post operative aggressive physiotherapy. Surgical access to left TMJ region was achieved through Al-kayat and Bramley incision[4]. The ankylotic mass was removed in two separate pieces, first one*fig(5)* by using surgical bur and later by using chisels with adequate protection on the medial aspect to protect the vital structures. After achieving a mouth opening of 2.5 cm*fig(6)*, temporal myofascial flap is raised and passed over zygomatic arch to be interpositioned in the gap created by removal of the ankylotic mass and secured in place by sutures. Post operatively mouth opening was reduced to 2 cm. Then aggressive physical therapy was carried on for 3 weeks to achieve a maximum interincisal opening (MIO) of 2.7 cm. Regular follow-up of patient is done for 2 yrs with adequate mouth opening and no signs of reankylosis.

Discussion:

Ankylosis may be defined as the fusion of articular surfaces with bony or fibrous tissue. The most common etiology of TMJ ankylosis is trauma, with the second being infection[2]. Incidence of Adult TMJ ankylosis is low and only few articles are quoted in literature compared to TMJ ankylosis in children which makes it challenging to treat.The clinical and radiographic findings of this case are in agreement with those of sawhney. Long standing, early onset ankylosis in childhood results in marked facial asymmetry, where as bony changes are minimal when the problem occurs during adolescence. For precision in surgical treatment planning and to reduce the incidence of recurrence, adequate pre operative evaluation of the type and extent of deformity is necessary. According to classification given by IE El-Hakim et al[5], based on post contrast axial and coronal CT scans the ankylosed joints can be grouped as:

Class I: includes unilateral and bilateral fibrous ankylosis. The condyle and glenoid fossa retain their original shape and the maxillary artery is in normal anatomical relation to the ankylosed mass

Class II: there is unilateral or bilateral by fusion between condyle and temporal bone. The maxillary artery lies in normal anatomical relation to the ankylosed mass

Class III: the distance between maxillary artery and medial pole of manibular condyle is less on ankylosed than in normal side or the maxillary artery runs with in the ankylotic bony mass

Class IV: the ankylosed mass appeared fused to the base of the skull and there is extensive bone formation especially from the medial aspect of the condyle to the extent that the ankylosed bony mass is in close relationship to the vital structures at the base of the skull such as pterygoid plates, the carotid, jugular foramina and foramen spinosumans no joint anatomy can be defined.

This present case falls into the category of class IV of the above classication. Treatment of TMJ ankylosis can be divided into 3 groups; gap arthroplasty, interpositional arthroplasty and joint reconstruction with autogenous or alloplastic materials[6]. Gap arthroplasty is similar compared to other technical procedures and takes shorter operation time. The disadvantages of gap arthroplasty include creation of pseudoarthrosis and short ramus and an increased risk of reankylosis[3]. Interpositional arthroplasty is widely accepted treatment of TMJ ankylosis. Dermis fat graft can be used as interpositioning material to fit the gap that is created by callus resection. The disadvantages of dermis fat graft consist of atrophy under pressure and possibility of epidermoid cyst formation[7]. Temporal myofascial flap can be a good choice for interpositional graft. The advantages of such flap are as follows: (1) close proximity to surgical area, which can be used from same incision; (2) good blood supply; (3) easy preparation and harvesting; and (4) minimal cosmetic and functional morbidity of the donor site[8]. Some disadvantages are as follows: (1) fibrosis and scar contracture of temporalis muscle,

which may cause trismus (some clinician suggested coronoidectomy to prevent this complication); (2) depresson in temporal region; and (3) chronic headache. Su-Gwan[9] treated seven patients with TMJ ankylois and used temporalis myofascial flap as an inter positional flap. He reported that post operative mean MIO was 36 mm and there was no reankylosis in patients[9]. After considering various treatment options suggested by several authors[10-15], the treatment plan should be mainly based on the finding in a specific patient. In this case, contrary to the findings[16] seen in a classic ankylosis patients.,retrogenia, malocclusion, prominent antegonialnotch,elongated coronoid processes and hypoplastic mandible are absent. So treatment modalities like reconstruction with costo-chondral graft and /or distraction osteogenesis are excluded. The treatment plan included Gap arthroplasty with temporal myofascialinterpositioning flap. According to Sawhney[17], in classI and II ankylosis, the fibrous adhesions were excised and the condylar head rounded and smoothed until free movement was achieved. In cases of class III and IV ankylosis, where there is close relationship of a vital structure, the resection was started using a surgical bur and then completed with chisels. As the ankylotic mass is a massive one, around 4.5x4 cm in dimensions, Al-kayat and Bramley[4] incision was preferred over a regular preauricular incision to get a wide exposure. The ankylotic mass was removed in two separate pieces and then interpositioning the temporalis myofascial flap after achieving MIO of 2.5 cm. The challenging aspect of this case is that mouth opening reduced to 2 cm post operatively which needed aggressive physical therapy post operativelyfor three weeks, finally achieving MIO of 2.7 cm. Because of the age of the patient, the consistency and nature of muscles is different compared to children necessitating vigorous physiotherapy. Patient has adequate mouth opening with no signs of reankylosis for about 2 yrs till now and is regularly followed-up which is most important in TMJ ankyosis.

Conclusion:

Management of Adult TMJ ankylosis is a challenging one.The advantages of arthroplasty are its simplicity and shorter operating time[3]. The disadvantages of arthroplasty include shortening of RCU, malocclusion, anterior open bite deformity, increased recurrence rate, formation of pseudoarthrosis, possible damage to the internal maxillary artery and difficulty in resecting bone from the medial aspect of the condyle[3]. In adult TMJ ankylosis patient, the RCU has already achieved its desired vertical dimension there by excluding the necessity of distraction osteogenesis or reconstruction with a costo-chondral graft. That favours the gap arthroplasty as a ideal surgical treatment for TMJ ankylosis in adult patients with a interpositioning material to prevent reankylosis.

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Fig 1. Showing Zero mouth opening due to ankylosis

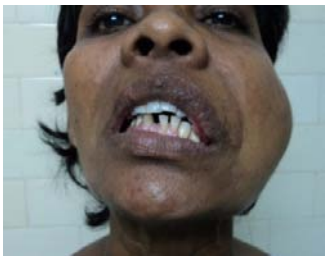
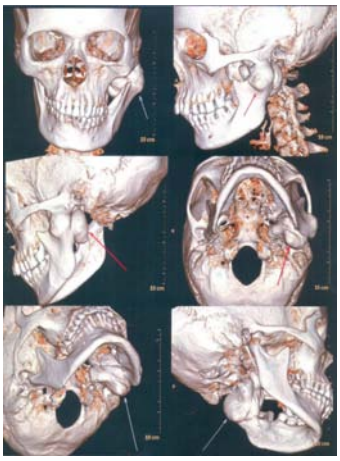


Fig 2.Showing ankylotic mass.
(a)



(b)

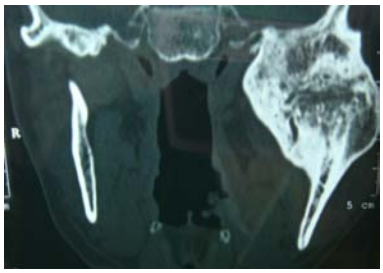


Fig 3.Intra operative photo showing huge ankylotic mass.

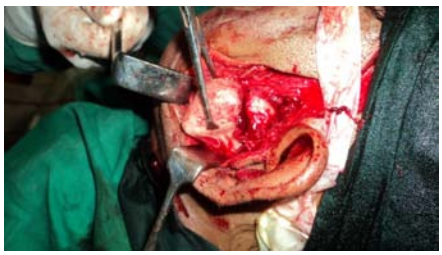
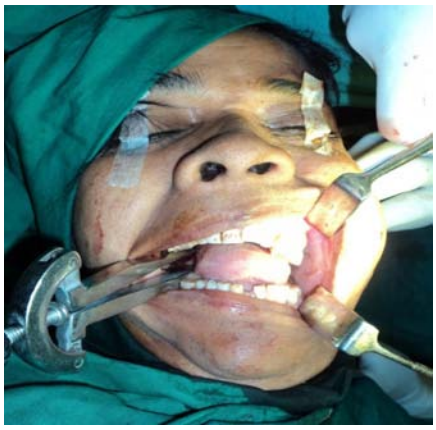


Fig 4.Intra operative photo showing mouth opening immediately after resection of ankylotic mass.



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