Hormones and their Clinical Consideration in Orthodontics

Amit Prakash[1], Prabhuraj Sabarad[2], Sonali Rai[3]

Quick Response Code

doi: 10.5866/2013.511120

[1]Senior lecturer
[3]Lecturer
Department of Orthodontics and Dentofacial Orthopedics
Rishi Raj Dental College and Hospital, Bhopal

[2]Senior lecturer
Department of Orthodontics and Dentofacial Orthopedics
Mansarovar Dental College and Hospital, Bhopal

Article Info:
Received: October 14, 2012;
Review Completed: November 12, 2012;
Accepted: December 11, 2012
Available Online: March, 2013 (www.nacd.in)

Email for correspondence:
amitprakash30@gmail.com

ABSTRACT:

Hormones have an important influence on the rate of tooth movement, and information on their consumption is essential to adequately discuss treatment planning with patients. Orthodontic tooth movement results from the response of the periodontal tissue to orthodontic force, which leads to modeling and remodeling of the surrounding alveolar bone. The response is considered to occur through the activation of specific signaling pathways, many of which are known, all acting to ultimately result in tooth movement. The rate at which tooth movement occurs is dependent upon the ability of these pathways to effect metabolism of bone by the two main cell types responsible for tooth movement: osteoblasts and osteoclasts. GH can affect craniofacial growth and tooth formation and eruption. Therapeutic administration of eicosanoids resulted in increased tooth movement, whereas their blocking led to a decrease. Corticosteroid hormones, parathyroid hormone, and thyroxin have all been shown to increase tooth movement. Estrogens probably reduce tooth movement, although no direct evidence is available. Vitamin D3 stimulates tooth movement, and dietary calcium seemed to reduce it. Bisphosphonates had a strong inhibitory effect. This article covers each aspect of hormonal influence on orthodontics.

Key words: Hormone, Orthodontics, Tooth Movement, Craniofacial

INTRODUCTION

The activities of various organs in our body are controlled by two systems namely, nervous system and endocrine system.[1] Most of the functions of nervous system are executed by hormonal substances, and endocrine functions are controlled by nervous system. The endocrine system constitutes endocrine glands which are situated in different part of body.[2] The functions of these glands are mediated by chemical substances which are called chemical messengers or chemical mediators or first messengers or hormones. The endocrine glands are also called as ductless glands because the hormones secreted by them are directly release into blood.[3]

MECHANISM OF HORMONE ACTION[1-3]:

On the target cell, the hormone in combination with the receptor acts by any of the following mechanism.

By altering the permeability of the cell membrane

- Neurotransmitter substance

By activating the intracellular enzyme

- Protein hormones and catecholamine's

By activating the gene

- Thyroid and steroid hormones

GROWTH HORMONE (GH)

GH is a protein hormone, secreted by the acidophils of the anterior pituitary. GH secretion is pulsatile, and the secretory bursts occur especially at early hours of sleep and throughout the night. GH has no specific target organ. It's an anabolic hormone to which every organ system responds. It doesn't show any direct action on bones, but acts through a substance called somatomedin. GH stimulates liver to secrete somatomedin.[4,5]

Humans have 2 types of somatomedin. These are:

1. Insulin like growth factor I (IGF-I), also called somatomedin-C.
2. Insulin like growth factor II (IGF-II)

Among these two IGF-I act on the bones and cause the growth and other affects on bones. GH carries out most of the metabolic functions through the somatomedin IGF-I.

GH is the main regulator of childhood and adolescent growth. GH pulse amplitude is increased in growth spurt with simultaneous increase in plasma IGF-I concentrations.

DENTAL DEVELOPMENT

Dental delay is always less pronounced than delay in height or bone age. The dentition seems to be harmoniously delayed, so that all studied components of dental development (primary root resorption, secondary tooth formation, and eruptive movement) display the same degree of retardation. GH influence on growth starts after 9 months of age so their effect on growth of primary teeth is very little known.[4-7]

GH Deficiency

Children gave big skull with babyish face, but in contrast their intelligence is normal for their age. Cephalometric studies in such children have shown small size of anterior and posterior cranial bases and smaller mandibular dimensions, small posterior facial height, and small posterior mandibular height. This has been shown in a study done over 13 untreated patients with pituitary deficiency the cephalometric findings were low as compared to normal, N-ANS being the lowest.[6-8]

Hypersecretion of Growth Hormone

Hypersecretion leads to gigantism in the young and to acromegaly in the adults, caused usually by a pituitary adenoma.

Gigantism

A cephalometric study was done on two female patients suffering from gigantism, due to excess of GH. Anterior facial heights were relatively the largest cephalometric dimensions, followed by posterior facial height. Posterior cranial base were long, but anterior cranial base were normal. Face was broad with pronounced zygomatic arches but relatively normal dental occlusion.[2-3]

Acromegaly

Serum levels of IGF-I in these patients were very high; mean value 10-fold higher than normal adults. Mandibular growth is gradual and often noted by the dentist when crossbite has developed. The calvarium, hands and feet grow by bone apposition. The tongue grows, and general visceral growth has been documented. Cartilaginous tissue enlarges, ribs thicken and costochondral cartilage has been shown to be hypertrophic.[9.10] Hypertrophic articular cartilage and the growth of chondrocytes in the articular cartilage may give rise to acromegalic arthropathy. Mandibular growth in acromegaly results from both appositional growth and hypertrophic changes in the condylar cartilage.[4,5]

INSULIN

Insulin is a polypeptide hormone secreted by beta cells of the Islets of Langerhans of Pancreas. Its main function is to maintain the blood glucose level. Insulin deficiency produces a clinical state called diabetes mellitus, while its excess leads to hypoglycemia. Diabetes mellitus is diagnosed in 3-4% of the population and it is very common to find such patients in our day-to-day orthodontic practice. The orthodontic practitioner should have a basic knowledge and understanding of this disease and its impact on the oral cavity, and should understand the consequences of diabetes mellitus in relation to dental treatment.[1-2]

Diabetes mellitus is characterized by hyperglycemia and chronic cases are associated with long-term damage, dysfunction and failure of various organs. Patients show the symptoms of polyuria, polydipsia, weight loss, and susceptibility to infections. Long-term complications include retinopathy, cardiovascular disease and increased tendency for periodontal diseases. Once the diagnoses is established, the patient must control the disease to prevent have minimize complications.

Patients who require large doses of insulin can have periods of extreme hyperglycemia and hypoglycemia, even with the best medical management. When a hypoglycemic condition develops, patients might appear weak, nervous and confused, skin is moist and pale, with excessive flow of saliva, respiration is normal, pulse is full and bounding, and blood pressure is normal, sometime tremors are seen. Most patients are familiar with these symptoms and can tell the dentist in time. A conscious and co-operative patient who is developing such symptoms should be immediately given a high carbohydrate beverage, such as orange juice, etc. and kept under observation until the symptoms have disappeared.[3-4]

Orthodontic considerations

Having knowledge of the oral complications of diabetes mellitus, the dental practitioner should consider them when treating a DM patient; the key to any orthodontic treatment is a good medical control. Orthodontic treatment should not be performed in a patient with uncontrolled diabetes. There is no treatment reference with regard to fixed or removable appliances. It is important to stress good oral hygiene especially when fixed appliances are used. These appliances might give rise to increased plaque retention, which could more easily cause tooth decay and periodontal breakdown in these patients. Daily rinses with a fluoride-rich mouth rinses can provide further preventive benefits. Candida infections can occur in oral cavity, so it should be well monitored[10]. It is advisable to apply light forces and not to overload the teeth. In adults, before starting the orthodontic treatment, the orthodontist should obtain a full-mouth examination (periodontal) and evaluate need for periodontal treatment. The periodontal condition should be improved before starting the treatment and should be monitored regularly.

Maintaining strict oral hygiene is important. Proper use of toothbrush, interdental toothbrush and chlorhexidine mouthwash should be advised. DM patients with good metabolic control, without local factors such as calculus and with good oral hygiene, have a similar gingival status as healthy patients and thus can be treated orthodontically.

THYROID HORMONE

Thyroid is an endocrine gland situated at the root of the neck on either side of trachea. It is larger in female than in male. Its function is slightly increased during pregnancy and lactation and is decreased during menopause. Diseases of thyroid gland are more common in females than in males.[2]

Functions of thyroid hormone

- To increase the overall metabolic rate in the body
- To stimulate growth in children

Hypothyroidism

A failure of thyrotropic function on the part of pituitary or an atrophy or destruction of the thyroid gland leads to an inability of the thyroid to produce sufficient hormone to meet the requirements of the body.

- Cretinism
- Myxedema

Clinical features

- Stunting of growth
- Infantile skeletal proportions and naso-orbital configurations
- Delayed and defective tooth development
- Epiphyseal dysgenesis

Congenital hypothyroidism affects

1) Bone of cartilaginous origin.
2) Bone of intra membranous origin.

The cranial base which is of endochondral origin is disturbed and arrested in growth. Length of the cranial base is shortened. Retardation in normal rate of deposition of calcium in the bones and in the development of the tooth buds in the fetus. In the thyroid cretinism patient has a large head and face which dull and infant facial expression. Dental

retardation in hypothyroidism parallels endochondral ossifications, which is extremely delayed. Delayed ossification of tooth bud. Hypothyroidism after age 6 years and before puberty can manifest itself as a juvenile myxedema. It is characterized by decrease in rate of maturation which showed itself as follow:[9,11]

1) Retardation in normal rate of deposition of calcium in bones and in tooth buds.
2) Disharmonies in the eruption of the teeth.
3) Inadequate development of maxilla.
4) Prolonged retention of the deciduous teeth.
5) Permanent teeth are slow to erupt.
6) Mesio or disto-occlusion and crowding of teeth.
7) Malposed maxillary and mandibular incisors and canine with loss of proximal contact.
8) Openbite due to tongue enlargement.
9) Abnormal dental calcification and root resorption.
10) Alveolar bone becomes osteoporotic.
11) Dentoalveolar prognathism.

Hyperthyroidism

Produces increase in rate of maturation increase in BMR and exopthalmic goiter. Premature eruption of disturbed resorption of the roots of the deciduous teeth with early eruption of permanent teeth. Bones become fragile and secretion of saliva is increased. Acceleration of skeletal ossification. Hyperthyroidism is rare in children but when it does occur eruption of teeth accelerated; occasionally some of teeth may be present at birth. Acceleration of eruption of teeth permanent by as much as 2 years or more ahead of their normal time. Teeth may show bluish white colouring. Osteoporosis may be present. This would contraindicate orthodontic treatment.[11]

CALCITONIN

It is a peptide hormone secreted by the interfollicular or C-cells in the thyroid gland, also called thyrocalcitonin. Thyrocalcitonin flows into the bloodstream and attracts calcium to the bone, thus reducing serum calcium. It also inhibits bone resorption by reducing the number of osteoclasts. Calcitonin is used in the treatment of hypercalcemia and in osteoporosis; because of its physiological role, it is considered to inhibit tooth movement. consequently, a delay in orthodontic treatment can be expected.[9]

PARATHYROID HORMONE

The function of parathyroid is to maintain a normal level of diffuse calcium and phosphorus in the blood plasma and to keep constant the ratio of these minerals to each other. The act as a check on the thyroid gland parathyroids are important organs in ca metabolism and play a leading role in calcification of teeth. However, once the teeth are formed, there is no evidence found of calcium withdrawal from teeth due to parathyroid disturbances. The parathyroids are important in regulating blood ca level, but have little or no direct effect on growth or tooth eruption.

PTH affects osteoblasts' cellular metabolic activity, gene transcriptional activity, and multiple protease secretion. Its effects on osteoclasts occur through the production of RANK-L Recepor activator of nuclear factor kappa -B ligand), a protein playing a crucial role in osteoclasts' formation and activity. In 1970s, animal studies demonstrated that PTH could induce an increase in bone turnover that would accelerate orthodontic tooth movement. More recently, an increased rate of tooth movements has been observed in rats treated with PTH, whether administered systemically or locally. These results indicate that orthodontists should take note of patients being treated with PTH, as for example, in cases of severe osteoporosis.[12]

VITAMIN-D_3

Vitamin-D and its most active metabolite vitamin-D_3, together with parathyroid hormone and Calcitonin, regulate the amount of calcium and phosphorus in the human organism. It promotes intestinal Ca^{+2} and PO_4^{-3} absorption. Vitamin-D_3 increases bone mass and thus reduce fractures in osteoporosis patients. Because or there beneficial effects on bone tissue, we can assume that it can inhibit tooth movement.[13,14]

SEX-STEROIDS

A slight increase in growth rate is seen at the age of 6-8 years in most children. This is thought to be due to increased GH and IGF-I production, which is stimulated by adrenal androgens. At puberty an increase in GH production is seen in boys and girls.

Several lines of evidence indicate that this increase is sex-steroid dependent. The sex-steroids here are adrenal and ovarian androgens and ovarian and testicular estrogens. Plasma concentration of IGF-I shows a similar increase.

Role of sex-steroids in dental and craniofacial development

Estrogen directly stimulates the bone-forming activity of osteoblasts, so it is reasonable to expect a decrease of the velocity of orthodontic tooth movement. Androgens also inhibit bone resorption and modulate the growth of the muscular system. Thus, the excessive use of these drugs by athletes, in an attempt to achieve better athletic scores, may affect the length and the results of orthodontic treatment.[15-17]

CORTICOSTEROIDS

Hyperglucocorticoidism leads to a short stature and developed bone maturation but increases relative weight. Very small amounts given a medication can decrease growth rate. Skeletal IGF-I synthesis is decreased by cortisol, which has an inhibitory effect on bone collagen synthesis. In the process of tooth eruption, however, cortisone has a special effect. Eruption rate is accelerated.[15]

PROSTAGLANDINS (PGs)

The precursor, for PGs is arachidonic acid, which is metabolized by cycloxygenase (cox) enzymes resulting in its production. Experiments have shown that PGs may be important mediators of mechanical stress during orthodontic tooth movement. They stimulate bone resorption by increasing number of osteoclasts and activating already existing osteoclasts. Studies on the PGs administration in shortening the treatment time in orthodontic patients was done and it was found that administration of PGE_1 and PGE_2 in experimental models (rat) and in orthodontic patients accelerated bone resorption and orthodontic tooth movement. Systemic administration is reported to have a better effect than local administration.[18]

CONCLUSION

Most of the studies on hormones are done on rats, squirrels and monkeys and not on human beings and so still very little is known about the effects of hormone on development of face and craniofacial skeletal and rate of orthodontic tooth movement in humans. Role of endocrine in orthodontics is still a great mystery for an orthodontic practitioner and further research is required to understand it better.

REFERENCES:

1. Brasel JA, Blizzard RM. Textbook of Endocrinology. 5th ed. Philadelphia, Pa: WB Sauders; 1974.
2. Thilander B. Basic mechanisms in craniofacial growth. Acta Odontol Scand. 1995; **53**:144-151.
3. Pirinen S. Endocrine regulation of craniofacial growth. Acta Odontol Scand. 1995; **53**:179-185.
4. Funatsu M, Sato K, Mitani H. Effects of growth hormone on craniofacial growth. Angle Orthod. 2006; **76**:970-977.
5. Bevis RR, Hayles AB, Isaacson RJ, Sather AH. Facial growth response to human growth hormone in hypopituitary dwarfs. Angle Orthod. 1977; **47**:193-205.
6. Cantu G, Buschang PH, Gonzalez JL. Differential growth and maturation in idiopathic growth-hormone-deficient children. Eur J Orthod. 1997; **19**:131-139.
7. Cohen MM, Wagner R. Dental development in pituitary dwarfism. J Dent Res. 1948; **27**:445-458.
8. Schour I, Brodie AG, King EQ. The hypophysis and the teeth. IV. Dental changes in a hypopituitary condition: a case report. Angle Orthod. 1934; **4**:285-304.
9. Gameiro GH, Pereira-Neto JS, Magnani MB, Nouer DF. The influence of drugs and systemic factors on orthodontic tooth movement. J Clin Orthod 2007; **41**:73-78.
10. Bensch et al (2003). Orthodontic treatment consideration in patients with diabetes mellitus. Am J Orthod Dentofacial Orthop. **123**:74-78.
11. Shirazi M, Dehpour AR, Jafari F. The effect of thyroid hormone on orthodontic tooth movement in rats. J Clin Pediatr Dent 1999; **23**:259-264.
12. Potts JT, Gardella TJ. Progress, paradox, and potential. Parathyroid hormone research over five decades. Ann N Y Acad Sci 2007; **1117**:196-208.
13. Collins MK, Sinclair PM. The local use of vitamin D to increase the rate of orthodontic tooth movement. Am J Orthod Dentofacial Orthop 1988; **94**:278-284.
14. Suda T, Ueno Y, Fujii K, Shinki T. Vitamin D and bone. J Cell Biochem 2003; **88**:259-266.
15. Ashcraft MB, Southard KA, Tolley EA. The effect of corticosteroid- induced osteoporosis on orthodontic tooth movement. Am J Orthod Dentofacial Orthop 1992; **102**:310-319.
16. Kalia S, Melsen B, Verna C. Tissue reaction to orthodontic tooth movement in acute and chronic corticosteroid treatment. Orthod Craniofac Res 2004; **7**:26-34.
17. Miyajima K, Nagahara K, Iizuka T. Orthodontic treatment for a patient after menopause. Angle Orthod 1996; **66**:173-178.
18. Sandy JR, Harris M. Prostaglandins and tooth movement. Eur J Orthod 1984; **6**:175-182.