Hormonal Influence on Orofacial Structure

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Introduction:

The endocrine system consists of several glands which secrete hormones.

Hormones are biologically active substances produced by these glands,

directly released into the blood-stream in which it circulate continuously

and exert their biological effect on different systems of our body. Hormones

have a vital role in growth and development of orofacial structures and their

functional activities and therefore altered levels of these can cause variety of

manifestations in orofacial structures.



THYROID HORMONE

Thyroid hormone is secreted by thyroid glands situated in the lower part of anterior region of neck. The hormone secreted by thyroid gland, i.e. thyroxin plays an essential role in regulation of metabolic activities of the body and also in physical and intellectual development. Calcitonin produced by 'c' cells of thyroid gland is also important in maintaining serum calcium level. Abnormal functioning of the thyroid gland may cause hyperthyroidism or hypothyroidism, which can adversely affect growth by accelerating or retarding the growth.

THYROID HORMONES



Hyperthyroidism causes increased metabolic activity. Affected persons are abnormally energetic. This condition, if occurs in early stages of life can lead to formation of large teeth, accelerated eruption of deciduous and permanent teeth and premature loss of deciduous teeth.



Experimental studies have shown that excessive thyroid hormone can have a toxic effect on odontoblasts resulting in disturbed dentin formation. In advanced cases alveolar atrophy occurs. Hyperthyroidism in adults does not show any orofacial manifestations, but they may have increased sensitivity to epinephrine and due to hyperthyroidism they become poor dental patients.

Hypothyroidism: This condition results from decreased functioning of thyroid gland. Congenital hypothyroidism or cretinism affects the mental and physical developments of a child depending upon severity of deficiency. Affected children have a characteristic facial appearance with depressed nasal bridge and flared nose. Face is wide and fails to develop in a longitudinal direction. The mandible is underdeveloped while maxilla is overdeveloped. Tongue is enlarged due to edema fluid and it protrudes out. Enlarged tongue exerts pressure on the teeth leading to malocclusion.







Congenital hypothyroidism Mother-Top.com pictures

In hypothyroidism, generalized retardation of skeletal growth takes place which also affect the jaw bones. Poor development of the jaw bones leads to anterior open bite and receded chin. In addition to skeletal development, teeth development is affected leading to decreased size of the tooth, delayed eruption, delayed exfoliation of deciduous teeth, etc.

Myxedema

Hypothyroidism in children and adults leads to myxedema, a condition characterized by subcutaneous edema. Clinical and orofacial findings of myxedema are limited to soft tissues of face and mouth. Tongue is large and edematous, interfering with speech and mastication. Lips, nose, eyelids and suborbital tissue also show edema.





Parathyroid hormone (PTH)

 Parathyroid hormone is secreted by parathyroid gland situated on the posterior aspect of thyroid gland. This hormone has a significant role in maintaining the serum calcium level and therefore plays a vital role in orofacial development.

Parathyroid hormone



Hypoparathyroidism: Dental changes can be observed in teeth which have formed during the time of PTH deficiency such as defective matrix deposition and mineralization of enamel and dentin. Delayed eruption also has been reported in individuals suffering from hypoparathyroidism which could be due to inhibitory effect on osteoclasts. A tooth cannot erupt without osteoclastic bone resorption, Exaggerated incremental lines and areas of interglobular dentin also have been reported in teeth of hypoparathyroid patients.

 Hyperparathyroidism: Parathyroid hormone can mobilize the calcium from bone causing bone resorption. This effect is applicable only on bone and not in fully formed teeth. Therefore in hyperparathyroidism, no visible changes occur in dental tissue. But the alveolar bone undergoes resorption.



Loss of lamina dura is a very important observation in this condition. Alveolar bone becomes osteoporotic and soft leading to drifting of teeth and malocclusion. The jaw bones also may show areas of bone resorption which may be evident in the radiographs as large areas of radiolucencies. The areas of bone resorption will be filled with highly vascular connective tissue. These lesions are termed as brown tumor.





HYPERPARATHYROIDISM

Giant Cell Granuloma Epulis Loss of lamina dura, pathognomonic oral change in hyperparathyroidism



In this panoramic image the loss of bone. The radiopaque teeth standing out in contrast to the radiolucent jaws

Pituitary Hormones

Pituitary gland is the master endocrine gland, secretions of which control the functioning of many other glands and many body functions. Anterior pituitary produces at least six hormones: The somatotropic, the thyrotropic, the adrenocorticotropic and the lactogenic hormone. The posterior pituitary produces antidiuretic hormone. Decreased activity of this hormone results in excessive production of urine and general dehydration of the body. The main effect of pituitary hormones on teeth and orofacial structures are mainly through the effect of growth hormone and partly by thyroid stimulating hormone.



Hypopituitarism can occur due to congenital defects or due to destructive diseases. If it occurs before puberty it leads to a condition called dwarfism. In pituitary dwarf the eruption of teeth is delayed and the shedding time of deciduous teeth is also delayed, as is the growth of body in general. The size of the crown and root of the tooth is smaller than normal. The supporting structures of the teeth also show retardation of development. Because of incomplete eruption, clinical crown of the teeth may be smaller.

Decreased growth hormone also causes retardation of development of maxilla and mandible. The dental arch is smaller than normal, therefore results in crowding of teeth and malocclusion. Pituitary dwarfs are reported to have a decrease in caries rate. Hypopituitarism in adults does not show any specific dental changes.

Hyperpituitarism that occurs before the closure of epiphysis of long bones leads to a condition called gigantism and if it occurs later in life after epiphyseal closure, leads to acromegaly. Gigantism is characterized by symmetric overgrowth of the body. As a part of general overgrowth of bones, both mandible and maxilla are larger than normal. The teeth, both crown and root are larger and is in proportion to the size of the jaws. The eruption of both deciduous and permanent teeth is accelerated with premature shedding of deciduous teeth.

In acromegaly, mandible continues to grow leading to abnormally long face and mandibular prognathism. Supra-eruption of teeth may occur leading to overgrowth of alveolar bone. Increase in length of mandibular arch may lead to malocclusion. The lips become thick and the tongue enlarged with indentations on the sides of the tongue. The enlarged tongue exerts pressure on the teeth leading to buccal or labial displacement of teeth and malocclusion.







Various hormones have been secreted by adrenal cortex and medulla. The main secretions from adrenal medulla are epinephrine (adrenaline) and norepinephrine. Adrenaline is very essential for a quick physiological response to crisis situations. Adrenal cortex is concerned with liberation of steroids which involve in carbohydrate, mineral, fat and protein metabolisms and fluid electrolyte balance. Hydrocortisone also has a marked antiinflammatory effect.

Chronic insufficiency of adrenal cortex leads to a condition called Addison's disease which is characterized by pigmentation of oral mucous membrane involving buccal mucosa, tongue, gingiva and lip. Hyperfunctioning of adrenal cortex causes Cushing's syndrome. The changes in orofacial region could be related to osteoporosis. Cortisone causes osteoporosis by suppressing the activity of osteoblasts resulting in defective matrix deposition.

