Review Article

"Orocrinology": Seven Easy Steps!

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Abstract

A complete examination of the oral cavity is a neglected part of physical examination and is not taught in both undergraduate and postgraduate medical training. We believe that a thorough oral examination helps in the identification of a variety of endocrine disorders and so to emphasize this, we have proposed the term "orocrinolgy." Orocrinology is the art of using a Thorough oral cavity examination to diagnose a variety of adult and pediatric endocrine disorders. Under "orocrinology," we have highlighted an easy to perform a seven-step technique to perform a complete examination of the oral cavity. The common endocrine-related abnormalities that you might encounter during each of these seven steps is summarized along with the steps. The seven steps start with the examination of the salivary glands, followed by the lips. This is followed by the examination of labial, buccal, alveolar, and gingival mucosa in two steps. The fifth step is the Inspection of the tongue and the base of the mouth followed by the sixth step, which is the evaluation of the palate, uvula, and tonsils. The final seventh step is the examination of the hard structures in the oral cavity, which includes the teeth, mandible, and the maxilla.

Keywords: Endocrine disorders, endocrinology, oral cavity, oral examination, orocinology, seven steps

Introduction

A thorough and comprehensive examination of the oral cavity is an area of physical examination that receives less emphasis in undergraduate and postgraduate medical education. A variety of systemic disorders can be diagnosed by a detailed stepwise examination of the oral cavity. We propose the term "orocrinology" to highlight the importance of thoroughly examining the oral cavity in endocrinology to help in identifying primary hormonal (endocrine) disorders. An understanding of the science and art of "orocrinology" would be of benefit to general practitioners, internists, and practicing endocrinologists. We suggest an easy *seven-step technique* in this review to complete a thorough oral examination and not miss any findings that may lead to an endocrine diagnosis.

Anatomy of the oral cavity

Anatomically the lips anteriorly limit the oral cavity. The cheeks form the lateral boundary, the floor of the mouth the inferior boundary, the oropharynx the posterior boundary and the palate the superior boundary. A thorough examination of this cavity by a physician requires about few minutes, a good light source, and a pair of gloves for palpation. The mobile phone with its torchlight function is fast replacing the trusty clinical flashlight in providing a ready light source in the clinics and wards! A mouth mirror maybe a useful tool to access hard to visualize areas but is not essential. If the patient is wearing a removable denture, then it should be removed prior to examination.[1]

Step-1. Examination of the salivary glands

Before examining the oral cavity, it is important to start with the salivary glands that are intimately associated with the oral cavity. The parotid gland can be found on the lateral surface of the mandible and it folds itself around the posterior border of the maxilla. Palpation can be done bidigitally from the front or behind the patient. Generally, the gland is soft and not palpable discretely unless the teeth are clenched when the anterior border maybe felt [Figure 1]. When the gland is enlarged, it is important to check for tenderness and to look at the opening of the parotid (Stensen's) duct, which enters into the cheek opposite the maxillary second molar teeth.

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Submandibular glands are found below the inferior border of the mandible and are best palpated bimanually with one hand in the floor of the mouth and the other hand pressing on the gland from outside. Bilateral painful enlargement of the salivary glands is seen with mumps infection and large unilateral enlargement should signal an alarm for a neoplasm.[1,2]

Orocrinology of the salivary glands

1. Causes of bilateral painless enlargement are shown in Table 1 and include the following endocrine causes.
   a. Growth hormone excess (Acromegaly and Gigantism)
   b. Untreated hypothyroidism
   c. Poorly controlled diabetes mellitus
   d. Sjogren's syndrome associated with renal tubular acidosis (Metabolic bone disease and hypokalemic periodic paralysis).
2. Intermittent painful enlargement is rarely seen with parotid stones associated with primary hyperparathyroidism.[3]
3. Inflammation of the salivary glands (sialadenitis) maybe a side effect of high-dose radioactive Iodine 131 therapy for differentiated thyroid cancers.

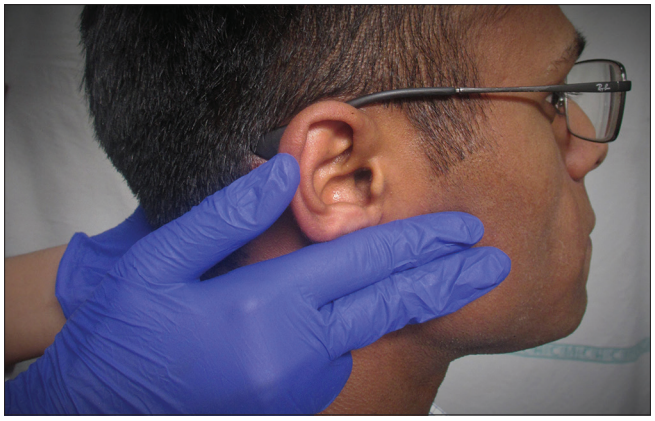

Figure 1: Bidigital palpation of the parotid glands from behind the subject

Table 1: Causes of painless bilateral parotid enlargement (Sialosis)

Nutritional	Chronic Alcoholism
	Eating Disorders: Anorexia nervosa/Bulimia
	Malnutrition
Infections (commonly painful)	HIV-related Sialosis
	Tuberculosis (rarely painless)
Endocrine	Diabetes mellitus
	Primary hypothyroidism
	Acromegaly and gigantism
Autoimmune	Sjogren's syndrome
	Wegner's granulomatosis
	Sarcoidosis
Iatrogenic	Radioactive iodine therapy (usually painful)
	Following general anesthesia
	Kimura Disease
Idiopathic	

Step-2. Examination of the lips

Generally, lips are homogenously pink in color, symmetrical, smooth with a distinctly defined vermillion border. In women, it may be useful to remove any lip coloring before examining the lips. After inspection, both the upper and the lower lips maybe palpated gently using the thumb and the forefinger and rolling downward [Figure 2]. During palpation observe for irregularities, edema, nodularity, tenderness, and growths.[1]

Orocrinology of the lips

1. Large, thick lips are seen in patients with growth hormone excess, and untreated hypothyroidism.
2. Thin lips with fine wrinkling at the labial edges can be seen in patients with hypopituitarism.[4]
3. Aphthous ulcers on the inner lips maybe seen in patients with poorly controlled diabetes mellitus (a complete list of causes is given in Table 2).
4. Angular cheilitis is also common with poorly controlled diabetes mellitus.
5. The orocrinology of cleft lips are discussed below with cleft palate.
6. Papules or nodules on the lips may represent mucosal neuromas which are seen in MEN-2B.

Step-3. Examination of the labial and buccal mucosa

This consists of the mucosa covering the lips and the inner cheeks that is nonkeratinized. Inspection of these areas is done directly using a good light source. Palpation is done again bimanually with one hand inside the mouth the other outside as for mandibular gland examination [Figure 3a and b]. The mucosa should appear pink and have uniform consistency. Any alterations in color or consistency may require help from an oral medicine professional to accurately characterize the disease.

Orocrinology of the labial and buccal mucosa

1. Burning mouth syndrome by definition is characterized by normal oral mucosa with symptoms of continuous burning in the mouth accompanied by gustatory abnormalities like dysgeusia, parageusia, and subjective xerostomia. The endocrine disorders associated with these are given as follows:[5]
   a. Estrogen deficiency in post menopausal women
   b. Associated with diabetic peripheral neuropathy
   c. Hyperthyroidism[6]
   d. Side effects of common drugs like angiotensin convertase enzyme inhibitors (ACEi).
2. Candidial infections of the mucosa may present as white lesions over the oral mucosa and tongue and maybe seen in the following endocrine disorders.
   a. Poorly controlled diabetes mellitus[7]
   b. Cushing's syndrome of any etiology including exogenous use of steroids
   c. As part of Autoimmune PolyEndocrinopathy-Candidiasis-Ectodermal Dysplasia (APECED) or Autoimmune Polyglandular Syndrome-1 (APS-1).[5]

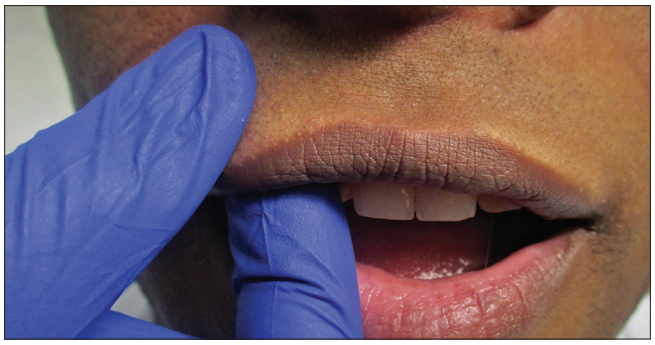

Figure 2: Palpation of the upper lips, after holding the lips between the thumb and the forefinger gently roll it downwards

3. Lichenoid reactions are common in patients who are on antihyperglycemic medications, especially when combined with antihyperglycemics. It may be difficult to differentiate from oral lichen planus and change of medication may alleviate the symptom.
4. Aphthous ulcers can also be seen over the oral mucosa and may represent poorly controlled diabetes mellitus. A complete list of nonendocrine causes of recurrent aphthous ulcers is given in Table 2.
5. Small but numerous pink papules or nodules over the buccal mucosa and the tongue may represent mucosal neuromas which are characteristic of multiple endocrine neoplasm-2B (MEN-2B).
6. Diffuse or patchy pigmentation of the oral mucosa with sparing of the lips is seen with adrenocorticotropic hormone (ACTH) excess. The pigmentation may not be a direct action of ACTH hormone. This is commonly encountered in Addison's disease, ectopic ACTH producing tumors, and rarely in Nelson's syndrome. Other endocrine and nonendocrine causes of pigmentation are summarized in Table 3.
7. Xerostomia, a dull, lusterless mucosa may signify dry mouth or xerostomia that can be seen with uncontrolled hyperglycemia, Sjogren's syndrome, and conditions with chronic hypercalcemia. When no cause is found, it is important to consider drugs, especially oral antihypertensives. A complete list of endocrine and nonendocrine causes is summarized in Table 4.

Step-4. Examination of the gingival and the alveolar mucosa

The gingiva (pink or coral) and the alveolar mucosa (red) are examined for color, texture (usually smooth), swelling or edema, ulceration, recession, spontaneous or bleeding on touch and enlargement. The color of the gingiva in the Indian scenario may not be pink or coral pink, but pale pink with pigmentation. Physiologic pigmentation is restricted to coronal portion of attached gingiva. Palpation is done using a gloved hand to ascertain if the visible enlargement is due to mucosal edema or an underlying bony or fibrous process.[1]

Orocrinology of the gingival and alveolar mucosa

1. Gingival edema and easy bleeding are seen in patients with Cushing's syndrome of any etiology.

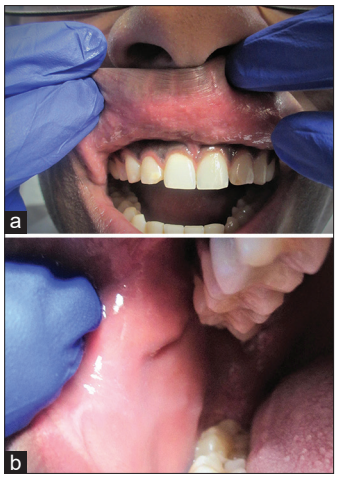

Figure 3: (a and b) Inspection of the labial and buccal mucosa

Table 2: Causes of recurrent aphthous ulcers

Idiopathic	RAS (recurrent aphthous stomatitis)
Nutritional	Iron deficiency
	Folate deficiency
	Vitamin B12 deficiency
	Vitamin B1, B2, B6 deficiency
	Zinc deficiency
Drug induced	Fixed drug eruptions
	Drug-induced bullous pemphigoid
	Steven-Johnsons syndrome
	Toxic epidermal necrolysis
Hematological	Anemia
	Neutropenia
	Hypereosinophilic syndrome
Autoimmune	Crohn's disease
	Behcet's disease
	SLE
	Wegner's granulomatosis
	Coeliac disease
Infectious diseases	Viral infections
	Herpes simplex
	Coxsackie A virus
	HIV
	Epstein-Barr virus
	Tuberculosis
	Syphilis
	Fungal infections

2. A peculiar type of gingivitis with proliferation of exuberant tissue from gingival margins can accompany poorly controlled type 1 and type 2 diabetes mellitus. The swellings are soft, well demarcated, red, irregular, and hemorrhagic.[5]
3. High estrogen levels may induce gingivitis in pregnant women. Hyperplasia of the gingiva and pyogenic granuloma affect 1%–5% of pregnant women toward the end of pregnancy and recovers with delivery.[5]
4. Gingiva becomes sensitive to sex hormones in puberty and can have exaggerated response to local factors

which in turn can cause pubertal gingivitis and gingival hyperplasia.
5. Gingival mucosal pigmentation is seen with ACTH excess syndromes like Addison's disease.

Step-5. Examination of the tongue and base of the mouth

The tongue is best examined with the patient's oral cavity at the examiners eye level and the examiner in front of the patient. After inspection, the tip of the tongue is grasped with a small piece of gauze and slightly moved out [Figure 4a]. This allows you to examine the lateral part of the tongue and the floor of the mouth. During inspection, check color of all the three sides of the tongue (dorsal, ventral, and lateral). In addition, check for plaques, ulcers, thickenings, and changes in papillae. During palpation, check for indurations and tenderness in the tongue.

The floor of the mouth is visualized with a good light source after asking the patient to lift his tongue up [Figure 4b]. No keratinization is seen in the mucosa of the floor of the mouth. Observe the lingual frenum in the middle of the floor attached to the tongue. In addition, the submandibular salivary gland ducts open on both sides of the frenulum.[1,2]

Orocrinology of the tongue and the floor of the mouth

1. Macroglossia is a visibly enlarged tongue. Indentations or crenations on the lateral border of the tongue may also signify an enlarged tongue. It is seen in the following conditions:
   a. Growth hormone excess states like Acromegaly and Gigantism
   b. Untreated primary hypothyroidism, especially in children
   c. Beckwith–Wiedemann Syndrome, which also may show macrosomia and hypoglycemia.
2. Glossitis or inflammation of the tongue with malnutrition and vitamin deficiencies and with Sjogren's syndrome.
3. Diabetes can cause depapillation of tongue and glossopyrosis; depapillation may be localized or can affect the whole tongue rendering it red and glossy.
4. Pink papules or nodules over the tongue, especially over the lateral borders may represent mucosal neuromas which are seen in MEN-2B.
5. A midline soft tissue mass at the back of the tongue may represent an ectopic lingual thyroid.

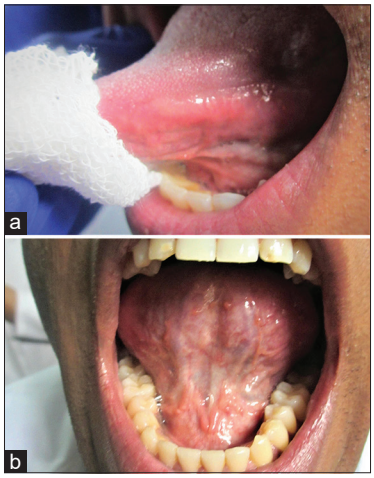

Figure 4: (a) Inspection of the lateral side of the tongue is done by gripping the tip of the tongue with a small piece of gauze and pulling it to one side. (b) The floor of the mouth is visualized after asking the subject to lift his tongue up

Step-6. Examination of the palate, uvula, and tonsils

The hard palate is covered with keratinized pink mucosa, which can be directly inspected with good light. During inspection, attention is paid to check for any color changes, ulcerations, and presence of any swelling. Structures to be visualized include the midline raphe and the corrugated ridges that extended from the raphe called the palatal rugae. Palpation is done to confirm findings from inspection. The soft palate is generally only inspected under good lighting for color (pink), presence of ulcerations, thickening, exudates, and petechiae [Figure 5]. When the patient says "ah," the movement of the soft palate and the uvula is assessed. The tonsils are located on either walls of the pharynx and are generally inspected under light. These areas are generally not palpated unless indicated.[1,2]

Orocrinology of the palate, uvula, and tonsils

1. Cleft lip or cleft palate is associated with a large variety of endocrine disorders. A paper from Turkey suggested that over 70% of neonates born with cleft lip/palate have a one or more endocrine deficiency.[8] The commonest endocrine syndrome associated with cleft lip/palate is DiGeorge's syndrome associated with hypoparathyroidism.[9] Isolated midline defects are associated with the idiopathic growth hormone deficiency, multiple congenital pituitary hormone deficiencies (including the recently described RAX and GLI2 mutations),[10,11] and Kallman's syndrome. Other syndromes associated with clefting and short stature include Apert's syndrome, CHARGE syndrome, Cornelia de Lange syndrome, Pierre Robin syndrome and the rare Abruzzi-Erickson syndrome. Bamforth–Lazarus syndrome is associated with congenital thyroid

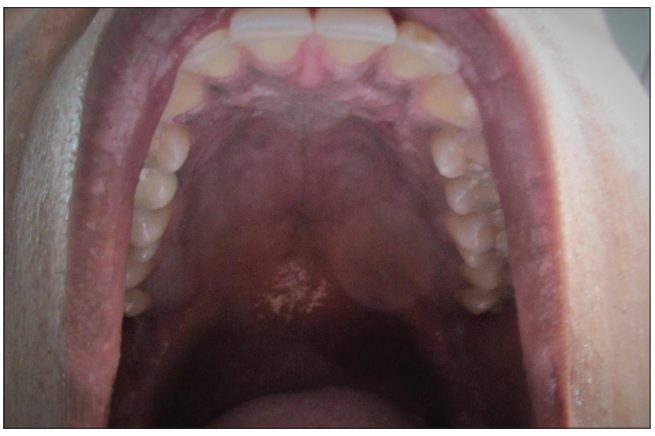

Figure 5: Inspection of the hard palate

agenesis, clefts, and choanal atresia.[9] A large list of over 200 specific syndromes are associated with clefting and are labelled syndromic cleft lip and palate. However, over 90% of children with cleft lip and palate have a nonsyndromic variety associated with environmental and genetic risk factors. The environmental factors are given in Table 5.

2. High arched palate (Gothic palate) defined as a narrow, tall hard palate is seen in Kallman's syndrome, Turner's syndrome, Bardt Beidl's syndrome, and FGF8 deficiency associated with congenital pituitary hormone deficiency and central diabetes insipidus.[12] A more complete list is given in Table 6. It is important to remember that a high arched palate can lead to sleep disordered breathing and obstructive sleep apnea.
3. Palatal hypertrophy maybe seen in growth hormone excess leading to sleep apnea.

Step-7. Examination of the teeth, bones of the oral cavity, and the bite relationship.

The final step in "orocrinology" is the evaluation of the hard structures in the oral cavity. The teeth should be inspected for number, position, spacing between teeth, the color of the teeth, and the intact surfaces [Figure 6]. Dental occlusion is determined by asking the patient to close his mouth and observing the relationship between the upper and lower jaw. A normal bite relationship implies that the anterior teeth on upper jaw are positioned in front of the anterior teeth of the lower jaw. Malocclusion usually implies that either the lower jaw is retrognathic (type 2 malocclusion) or is prognathic (type 3 malocclusion).[1] Masses arising from the mandible and maxillae should be noted and could include a variety of bone tumors. Destruction of the bones of the oral cavity can be secondary to infections and osteonecrosis seen with bisphosphonate use.

Orocrinology of teeth, bones of the oral cavity, and the bite relationship

1. Delayed eruption of teeth is seen in children with congenital hypothyroidism, hypoparathyroidism, isolated growth hormone deficiency, and with congenital multiple

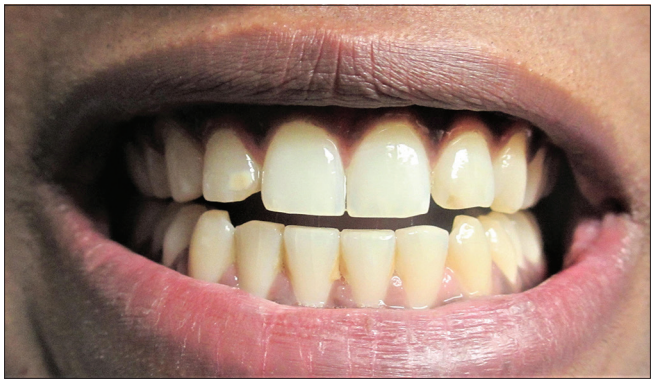

Figure 6: Inspection of the teeth should concentrate on number, position, spacing, and color of the teeth

Table 3: Causes of oral mucosal hyperpigmentation

Physiological	Racial	
Pathological		
Exogenous		
	Drugs	
	Tobacco chewing	
	Heavy metal poisoning	
	Amalgam tattoo	
Endogenous	Endocrine	Addison's disease
		Ectopic ACTH producing tumors
		Nelson's syndrome
		Diabetes
		Hyperthyroidism
	Syndrome Associated	Peutz-Jeghers syndrome
		MaCune-Albright syndrome
		Hemochromatosis
		Leopard syndrome
	Infections	Tuberculosis
		HIV
		Candidiasis
	Chronic Irritation	Post Inflammatory
		Post Traumatic
Idiopathic		

Table 4: Causes of xerostomia

Nonsalivary causes	Dehydration	
	Cognitive alterations	
	Neurological dysfunction	
	Mouth breathing	
	Oral sensory dysfunction	
	Eating disorders	
Salivary causes (associated with hyposalivation or altered salivary composition)		
Drug induced	Tricyclic antidepressants	
	Antihypertensives like diuretics and alfa adrenergic agonists	
	Antispasmodic agents	
	Gabapentin and pregabalin	
	Antipsychotics like clozapine	
	Opioids	
Pathological	Endocrine	Diabetes mellitus
		Hypothyroidism
		Triple A syndrome
	Autoimmune	Sjogren's syndrome
		SLE
		Scleroderma
		Amyloidosis
		Primary biliary cirrhosis
	Infections	Hepatitis C infection
		HIV
		Viral infections like EBV/CMV
	Others	End-stage renal disease
		Sarcoidosis
		Chronic alcoholism
Post External Beam Radiation Therapy		
Past Radioactive Iodine Therapy		
Graft versus host disease		

Table 5: Environmental risk factors for nonsyndromic cleft palate and lips

Maternal Exposure during early pregnancy to
Smoking
Tobacco chewing
Viral infections
Alcohol
Drugs
Other teratogens

Table 6: Causes of high arched palate

Congenital (syndromic) with possible endocrine association	Cornelia de Lange syndrome
	Smith-Lemli-Optiz syndrome
	Turner's syndrome
	Kallman's syndrome
	Bardt-Beidl's syndrome
	Downs syndrome
	Treacher's syndrome
	Apert's syndrome
	Cruzon's syndrome
	Soto's syndrome
Congenital (Syndromic) without known endocrine association	Saethre-Chotzen syndrome
	Derivative 22 syndrome
	Ramon syndrome
	SAT-B2 associated syndromes
	Emanuel syndrome
	Duchene's muscular dystrophy
	Bainbridge-Ropers syndrome
	Incontinentia Pigmenti
	Jarcho-Levin syndrome
	Bosma syndrome
	Schwartz-Jampel syndrome
	Wolf-Hirschhorn syndrome
	Hereditary ectodermal dysplasia
	Marfan's syndrome
	Muenke's syndrome
	Sheldon-Hall Syndrome
	Job's syndrome
	Proteus syndrome
	Lujan-Fryns syndrome
	Lenz microphthalmia syndrome
Acquired conditions	Thumb sucking
	Chronic rhinitis
	Mouth breathing
Isolated high arched palate	

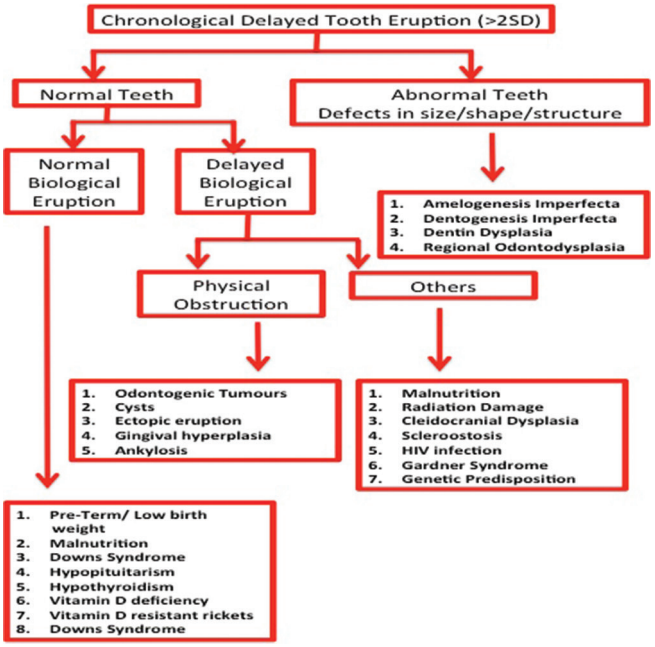

Figure 7: A diagnostic algorithm for delayed tooth eruption (DTE)

pituitary hormone deficiencies. Definition of delayed tooth eruption and common causes are summarized in Figure 7.

2. Accelerated eruption of teeth is seen in hyperthyroidism.
3. Delayed shedding of deciduous teeth is also seen in both congenital or acquired hypopituitarism and hypothyroidism.
4. Early shedding of deciduous teeth is seen in hyperthyroid children.[3]
5. Enamel hypoplasia is a defect where enamel is deficient leading to differing manifestations. It can present as a pitting, planar, linear, and localized varieties. Endocrine causes for enamel hypoplasia include chronic hypocalcemia seen in primary hypoparathyroidism, "calcipenic" rickets, osteomalacia, and undiagnosed coeliac disease. It may also accompany bone disease associated with fluorosis.[13] More details of dental disease in fluorosis are given in Table 7.
6. Dental abscesses maybe seen with "phosphopenic" rickets.
7. Dentinogenesis imperfecta (DI) is a dentin dysplasia that causes the teeth to be discolored (blue-gray or yellow-brown) and translucent giving it an opalescent sheen. Type 1 DI is associated with osteogenesis imperfecta and type 2 maybe isolated without any bone disease.[14]
8. Widely spaced teeth in children maybe a manifestation of untreated hypothyroidism. However, in adults, acromegaly should always be considered.
9. Crowding of teeth maybe seen in patients with hypopituitarism.[4]
10. Mandibular prognathism with or without macrognathism (Lantern jaw) is seen in adults with acromegaly. This leads to class 3 malocclusion with a cross bite.
11. Buccally tilted teeth due to macroglossia also may be seen with acromegaly.
12. Maxillary prognathism is seen in Cornelia de Lange syndrome. However mandibular retrognathism may be falsely diagnosed as maxillary prognathism. Mandibular retrognathism is associated with Class 2 malocclusion and is seen in congenital hypopituitarism and in patients with Noonan's syndrome. Patients with idiopathic retrognathism are at a higher risk of obstructive sleep apnea syndrome. Retrognathia maybe associated with multiple syndromic causes of short stature including Pierre Robin sequence, Goldenhar syndrome, and Treacher

Table 7: Dean's fluorosis index

A clinical scoring system of the severity of dental fluorosis was first developed by Dean in 1934 and subsequently modified in 1942

Score	Criteria
0. Normal	The enamel represents the usual translucent semivitriform type of structure. The surface is smooth, glossy, and usually of a pale creamy white color.
1. Very Mild	Small opaque, paper white areas scattered irregularly over the tooth but not involving as much as 25% of the tooth surface. Frequently included in this classification are teeth showing no more than about 1-2 mm of white opacity at the tip of the summit of the cusps of the bicuspids or second molars.
2. Mild	The white opaque areas in the enamel of the teeth are more extensive but do not involve as much as 50% of the tooth.
3. Moderate	All enamel surfaces of the teeth are affected, and the surfaces subject to attrition show wear. Brown stain is frequently a disfiguring feature.
4. Severe	Includes teeth formerly classified as "moderately severe and severe." All enamel surfaces are affected and hypoplasia is so marked that the general form of the tooth may be affected. The major diagnostic sign of this classification is discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded-like appearance.

Collins syndrome.[15]

13. Jaw tumors can be seen with primary hyperparathyroidism as part of bone disease (brown tumors) both in the maxillae or mandible.[16]
    A rare type of autosomal inherited endocrine neoplasia syndrome called hyperparathyroidism jaw tumor syndrome is associated with ossifying fibromas of the jaw along with parathyroid adenomas/carcinoma, and renal and uterine tumors.[17]
14. Fibrous dysplasia involving the maxillae and the mandible can be present either alone or in combination with endocrinopathy in McCune–Albright syndrome.
15. Cherubism is a rare genetic condition that causes prominence of the lower portion of the face. The prominence of the lower jaw is related to loss of bone in the mandible where the bone is replaced by expanded fibrous tissue. Usually this is an isolated disorder but may be rarely associated with Noonan's syndrome and Fragile X syndrome.

Conclusions

This review summarizes a useful seven-step approach to "orocrinology." Orocrinology is the art of using a through oral cavity examination to diagnose a variety of adult and pediatric endocrine disorders. Following the above seven steps will help in keeping a mental checklist during the examination and hence not lead to missing any findings.

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Conflicts of interest

There are no conflicts of interest.

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