

Typical pathological processes in oral cavity

Disorders of odontogenesis

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Typical pathological process: definition, signs, examples

• The stereotype reactions of the body to various external and internal stimuli. Their appearance causes a cascade of reactions, which are aimed at maintaining the constancy of the internal environment of the body.

The main characteristics of the typical pathological process

- Stereotype. The presence of the features of a typical process, regardless of the cause of its appearance and localization.
- Versatility. Typical pathological process can be in the composition of different nosological units.
- Polyetiologic. The etiologic factor of the disease performs only a starting role and is not permanent.
- Autochthonism. The process's ability to develop independently, even when the etiologic factor ceases to function.
- Equifinality. Different ways to implement the pathological process, which lead to the same development and resolution.

- Inflammation
- Hypoxia
- Fever
- Pain
- Disorders of consciousness
- Edema
- Thrombosis
- Tumors

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Edema

Oedema (dropsy, hydropsy) an abnormal accumulation of fluid in the interstitium or in body cavities

Classification of edemas according to localization

✓Generalized

🗸 Cardial

✓ Renal, ✓ Hepatal

- ✓ Hypoproteinemia
- ✓ Idiopathic

√Local

✓ Face✓ Eyes✓ Feet

 \checkmark Genitals



✓ Cavities ✓ Ascites ✓ Hydrothorax ✓ Hydropericardium ✓ Inner organs ✓ Lungs ✓ Brain ✓ Larynx





Classification of edemas according to etiology

✓ Edemas caused by increased hydrostatic pressure
 ✓ Edemas caused by decreased oncotic pressure
 ✓ Edemas caused by increased permeability
 ✓ Lymphedemas

↑ hydrostatic pressure



arterial

venous obstruction
heart failure

left - pulmonary oedema
right - peripheral oedema

postural oedema







\downarrow oncotic pressure

hypoproteinaemia
 liver diseases

 cirrhosis
 nephrotic syndrome
 protein malnutrition - kwashiorkor
 some metabolic diseases









\uparrow permeability



arterial

inflammation - mediators
allergy - histamine
hypoxia
toxic







Lymphatic obstruction

lymphoedema (woody oedema)

- •lymfatic obstruction
 - parasites (filariasis)
 - cancer of lymph nodes
 - surgery or radiation therapy (breast cancer)
 inflammatory changes lymphangitis

arterial







Angioedema

Quincke's edema Angioneurotic edema



Sounde: shattine RK Sandh MA, Hawwan KS, Churteev HS, The Color Alass of Energy Multiums, Sanotal Estimetry services assessments the control Cooperget & The ReGener HM Companies, Inc. Ad rights coursed

- repetitive episodes of swelling face, lips, tongue, limbs, and genitals
- edema of the gastrointestinal mucosa severe abdominal pain; in the upper respiratory tract
- edema of larynx life-threatening

Classification

Acquired angioedema

- Immunologic allergy
- Nonimmunologic medication (ACE inhibitors)
- Idiopathic

Hereditary angioedema - AD

- Types I mutation in the SERPING1 gene diminished levels of the C1-esterase inhibitor protein
- Types II mutation in the SERPING1 gene dysfunctional forms of the same protein
- Type III mutation in the F12 gene coagulation protein factor XII
- Abnormal activation of the complement system, activation of the contact pathway by the initial generation of kallikrein and/or clotting factor XII by damaged endothelial cells - production of bradykinin increased vascular permeability and vasodilation - swelling.

C1 esterase inhibitor

- A protease inhibitor inhibition of the complement system, also inhibits proteases of the fibrinolytic, clotting, and kinin pathways.
- In hereditary angioedema deficiency of C1-inhibitor permits plasma kallikrein activation, which leads to the production of the vasoactive peptide bradykinin → edema.



Signs & Symptoms



- Sudden appearance of red welts, near eyes & lips, also hands, feet, and inside of throat
- Burning, painful, swollen areas; sometimes itchy
- Discolored patches or rash on the hands, feet, face, or genitals
- hoarseness, tight or swollen throat, breathing trouble





Angioedema



Lumps and swellings in oral cavity

Main conditions which may present as lumps or swellings in the mouth.

	Normal Anatomy	Parotid papillae Lingual papillae	Martin Contraction
	Developmental	Unerupted teeth Developmental cysts Haemangioma Lymphangioma Maxillary and mandibular tori	
	Inflammatory	Cellulitis Sialadenitis Pyogenic granuloma Chronic granulomatous disorders - Orofacial granulomatosis - Crohn's disease - Sarcoidosis Insect bites	
	Traumatic	Denture-induced hyperplasia Epulis Fibro-epithelial polyp Haematoma Mucocele Surgical emphysema	

Main conditions which may present as lumps or swellings in the mouth.		
Neoplasms	Carcinoma Leukaemia Lymphoma Myeloma Odontogenic tumours Minor salivary gland tumours	
Fibro-osseous	Cherubism Fibrous dysplasia Paget 's disease	
Hormonal	Pregnancy epulis/gingivitis Oral contraceptive pill gingivitis	
Metabolic	Amyloidosis Other deposits	
Drugs	Phenytoin Calcium channel blockers Ciclosporin	
Allergic	Angioedema	
Infective	HPV	
	1 Contracts	

Hypoxia

Hypoxia - condition in which body or part of body is deprived of oxygen supply at tissue level

Classification of hypoxia according to localization

Generalized

- ✓ hypoventilation
- ✓ low oxygen values
- ✓ airway obstruction
- ✓ respiratory diseases
- ✓ shock
- cardiovascular diseases
- 🗸 anemia
- hemoglobin dysfunction

- Localized (tissue/organ ischemia)
 - ✓ compression of vessel
 - ✓ vascular occlusion
 - ✓ vasoconstriction
 - < compartment syndrome</pre>
 - ✓ Tissue/Organ
 - ✓ cerebral ⇒ stroke
 - ✓ intrauterine ⇒ fetal hypoxia
 - ✓ heart ⇒ myocardial infarction

Tumor hypoxia

 hypoxia triggers important changes in tumor metabolism

https://en.wikipedia.org/wiki/Hypoxia-inducible_factor

activation of HIF

normoxia

- subunits HIF-1alfa and HIF-1beta
- > prolyl-hydroxylases
- ✓ VHL
- ✓ ubiquitination

hypoxia

- stabilization
- transfer to nucleus
- ✓ creation of HIF
- creation of dimer with HIF-1beta
- trigers expression of lot of genes

Nobel Prize in Physiology or Medicine 2019: How Cells Sense and Adapt to Oxygen Availability
 Awarded to William G. Kaelin, Sir Peter J. Ratcliffe, and Gregg L. Semenza



Tumor hypoxia

Activation of genes for:

https://en.wikipedia.org/wiki/Hypoxia-inducible_factor

✓ GLUT 1

glycolytic enzymes

- ✓ hexokinase
- ✓ phosphoglucose isomerase
- ✓ phosphofructokinase
- fructose-biphosphate aldolase
- glyceraldehyde-3-phosphate dehydrogenase
- ✓ phosphoglycerate kinase
- > phosphoglycerate mutase
- ✓ enolase 1
- v pyruvate kinase
- pyruvate dehydrogenase mutase (isoenzyme 1)
- lactate dehydrogenase A
- carbonic anhydrase 9
- ✓ VEGF
- hyaluronan
- ✓ ↑ EPO



Tumor hypoxia

https://en.wikipedia.org/wiki/Hypoxia-inducible_factor



Classification of hypoxia according to etiology

✓ hypoxic hypoxia
✓ anemic hypoxia
✓ circulatory hypoxia
✓ histotoxic hypoxia



https://www.facebook.com/photo.php?fbid=151687524215 7232&id=363518240826277&set=a.363587157486052

Hypoxic hypoxia <pr

✓ Causes:

✓ hypoventilation
 ✓ depression of respiratory
 center
 ✓ low oxygen partial pressure
 ✓ airway
 obstruction
 ✓ pulmonary and airway diseases





https://en.wikipedia.org/wiki/Altitude_sickness

Hypoxic hypoxia

- cyanosis \checkmark
- headache \checkmark
- decreased reaction time \checkmark
- disorientation \checkmark
- affected cognitive function \checkmark
- euphoria \checkmark
- visual impairment \checkmark
- lightheadedness \checkmark
- fatigue \checkmark
- drowsiness \checkmark
- dyspnea \checkmark
- palpitations \checkmark
- nausea and vomiting \checkmark
- fluctuations in blood pressure \checkmark
- seizures (severe hypoxia) \checkmark
- tingling in fingers \checkmark
- numbness \checkmark



NEWBORN CYANOSIS



https://www.theraspecs.com/blo g/does-light-make-you-tiredfatigue-and-light-sensitivity/ https://www.osmosis.org/learn/ Approach to cyanosis %28ne wborn%29: Clinical sciences https://www.health.harvard.edu/ diseases-and-conditions/top-7reasons-you-have-a-headache



https://www.osmosis.org/notes/Methemoglobinemia https://en.wikipedia.org/wiki/Sickle_cell_trait https://en.wikipedia.org/wiki/Bleeding

Symptoms:

✓

 \checkmark

 \checkmark

 \checkmark

 \checkmark

 \checkmark

 \checkmark

 \checkmark

 \checkmark

 \checkmark

 \checkmark

blue lips (depends of

severity of anemia)

lack of coordination

confusion

dizziness

headaches

hypertension

fainting

lethargy

dysphoe

legs

tachycardia

 \checkmark

visual blurrines ✓

> https://www.youtube.com/watch?v=xAfj5AHxC2I https://clinicalproblemsolving.com/deutscheschemata/pulmologie/dyspnoe/ https://www.urban-optics.com/blog/5-common-causes-ofblurry-vision-and-what-to-do.html

Hypoxia

https://www.facebook.com/photo.php?fbid=1146547822523311&id=3635 18240826277&set=a.363528530825248&locale=ur_PK

Changes of colour

Changes of skin colour

- ✓ Red erythema
 ✓ Pale ischemia
 ✓ Yellow jaundice
 ✓ Blue cyanosis
 ✓ Black necrosis
- ✓ Depigmentation
 ✓ Hyperpigmentation
 ✓ Local pigment accumulation

Erythema

- > Hyperemia UV radiation, ↑ temperature, inflammation
- > Flushing fever, anger, alcohol...

Alcohol intolerance

• East Asia - China, Taiwan, Japan, Korea

Cause

- Alcohol dehydrogenase variant 1C ADH1C
 - Higher production of acetaldehyde
- Deficiency of aldehyde dehydrogenase 2

Signs:

- Anaphylactic reaction
- Flushing of the face
 - It starts a few minutes after drinking alcohol, peaks in 30-40 minutes, disappears after 1-2 hours.
 - A little alcohol is enough, but the intensity also depends on the dose
- Hives, red itchy rashes
- Stuffy nose, runny nose as in rhinitis
- Hypotension
- Nausea, vomiting, diarrhea
- Headaches, anxiety, drowsiness
- Anaphylactic reaction

Pallor

- > Anemia
- Emotional shock, panic attack, stress
- > Cold environement, frostbite
- > Fever
- > Leukemia
- Medications (amphetamines, ethanol, cannabis)
- Heart disease
- > Hypotension
- > Peripheral vascular disease
- Sleep deprivation

Jaundice



- Hyperbilirubinemia
 - Prehepatic hemolytic anemia
 - Hepatic liver diseases, virus infection, alcohol, drugs...
 - Posthepatic posthepatic obstruction





Classification of cyanosis

Hemoglobin cyanosis

- reduced hemoglobin > 50 g/l
- Central and periferal cyanosis

Hemiglobin cyanosis

-abnormal hemoglobins (methemoglobin, sulfhemoglobin)
 Methemoglobin cyanosis

 -Fe³⁺ (hemiglobin)
 -metHb 1,5% of total Hb
 clinical signs - > 35% of total hemoglobin
 -inborn (defects of methemoglobin reductese, G-6-PD)
 -acquired (drugs-antipyretics, lidokain, nitrites)

Central cyanosis

Hemoglobin cyanosis

- > total, arterial, anoxic
- visible: mucosa, lips, tongue, nail beds, ears
- > arterial blood is not enough saturated bu oxygen
- \rightarrow \uparrow reduced Hb

Inborn errors with right-left shunt

-Fallot tetralogy, Eisenmenger sy., Ebstein sy. Lung diseases

- -obstructive lung diseases
- -pulmonary edema, pulomonary embolia





Peripheral cyanosis

- > acral, venous, stagnation
- > normal saturation of arterial lood
- \succ stagnation \uparrow oxygen extraction
- \succ \uparrow reduced Hb
- > tongue pink







- Peripheral cyanosis
- Cyanotic skin, lips
- Tongue not cyanotic

Central cyanosis Cyanotic skin, mucosa Tongue cyanotic

Acanthosis nigricans

- a condition that causes areas of dark, thick velvety skin in body folds and creases
- ✓ usually affects:
 - posterior and lateral neck
 - ✓ axilla
 - ✓ groin
 - ✓ navel
 - ✓ forehead
 - $\checkmark\,$ and lot of other parts

https://www.researchgate.net/publication/321269616_Facial_Acanthosis_Nigricans_A_Morph_ological_Marker_of_Metabolic_Syndrome/figures?lo=1

https://www.researchgate.net/publication/369413720_A_comparison_of_the_efficacy_and_saf ety profiles of 10_salicylic_acid_and_10_urea_creams_in_treating_acanthosis_nigricans_in_ adolescents_a_randomized_double-blinded_study/figures?lo=1

✓ Symptoms:

 dark brown-black, poorly defined, velvety patches of skin of different intensity





Acanthosis nigricans - causes

- ✓ Type I familial
 - ✓ autosomal dominant
 - ✓ present from birth
- ✓ Type II endocrine
 - insuline resistance (DM, metabolic syndrome)
 - ✓ excess of androgens
 - ✓ Cushing disease, acromegaly, PCOS
 - Addison disease
- Type III obesity and pseudoacanthosis nigricans
 - associated with insuline resistance
 - mostly affected face areas forehead, perioral, periorbital or generalized

- ✓ Type IV drug-related
 - nicotinic acir
 - ✓ glucocorticoids
 - ✓ oral contraceptives
 - ✓ growth hormone
- Type V malignancy
 - ✓ paraneoplastic syndrome
- ✓ Acral acanthotic anomaly
 - ✓ not known origin

Acanthosis nigricans – mechanism

- ✓ activation of growth factor receptor
- most commonly activation of IGF receptors on keratinocytes
- activation of fibroblast growth factor receptor (in hereditary forms)
- ✓ activation of epidermal growth factor receptor (EGFR)
 - ✓ by increased TGF

https://www.drniveditadadu.com/blog/how-tomanage-acanthosis-nigricans/



Pseudocyanosis

Chemical compounds accumulated in the skin

Argyria (argyrosis)

- Gray-violet colour of skin
- Silver intoxication (antibacterial creams) Arsenic
- Arsenic melanosis





Local pigment accumulation



Alkaptonuria

ochronosis - accumulation of the ochronotic pigment in:

- joints ochronotic artropathy
- endocardium
- renal and prostatic stones
- other (eyes, teeth, CNS)







Definition

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage

(IASP – The International Association for the Study of Pain, 1979)

- damage (danger) signal
- indication and localization of damage
- prevention of more serious damage

Pain in orofacial region - oral cavity (teeth, gingiva, oral mucosa), face, jaw bone, temporomandibular joint

Orofacial Pain Pathway

Orofacial pain pathways includes primary afferent neurons, trigeminal ganglion, brainstem nociceptive neurons, and higher brain function regulating orofacial nociception.

- Nociceptors in the orofacial region
- 1st order neurons in the trigeminal nerve
- 2nd order neurons in the trigeminal nucleus caudalis located in the brainstem
- 3rd order neurons in the thalamus via the ventral trigeminothalamic tract
- The descending pathway sends signals to the trigeminal nucleus caudalis – serotonin, norepinephrine and opioid peptides are produced - this process leads to pain reduction



Classification of orofacial pain

	Acute orofacial pain	Chronic orofacial pain			
Duration	Onset	Sustained, persistent >3 months in humans			
Cause	Inflammation or injury of tissue	Inflammation, nerve damage			
Physiologic response	Increased blood pressure, tachycardia via sympathetic response	Adaptation behaviors or psychological responses such as depression and anxiety			
Examples in the orofacial area	Dental pain: pulpitis Mucogingival pain	Neuropathic pain: trigeminal neuralgia, peripheral trigeminal nerve injury, postherpetic neuralgia Chronic inflammatory pain: chronic pulpitis and apical lesions, temporomandibular disorder pain Neurovascular pain: migraines, tension-type headaches			

Classification of orofacial pain

	Nociceptive orofacial pain	Inflammatory orofacial pain	Neuropathic pain
Causes and mechanism of pain pathway	Noxious stimulation at the peripheral nerve and transmitted by normal components of the sensory trigeminal nerve	Strong noxious stimulus causes lesions in the tissue leading to local inflammation responses and increased inflammatory mediators	Caused by nerve damage or injury and increased peripheral sensitization, structure change by increased sodium activation, calcium activity of nerves leading to ectopic discharges, and glia cell activation
Stimulation	Response to noxious stimulus	Response to noxious stimulus	Response to non-noxious and noxious stimulation Spontaneous pain without stimulation occurred in damaged nerves
Example	E.g. response to hot drink	Pulp necrosis Temporomandibular joint inflammation	Peripheral trigeminal nerve injury - facial trauma accident or trigeminal neuralgia



Pain in oral cavity

"Toothache" is pain typically around a tooth, teeth or jaws.

Causes

- dental problems dental cavity, a cracked or fractured tooth, an exposed tooth root, or gum disease.
- diseases of the jaw joint (temporomandibular joint)
- spasms of the muscles
- cancers
- teeth implants

The severity of a toothache can range from chronic and mild to sharp and excruciating. It can be a dull ache or intense.

The pain may be aggravated by chewing or by foods and liquids which are cold or hot, sweet.



Heart pain can radiate to the jaw and teeth

Sometimes, pain in oral cavity may be caused by a problem not originating from a tooth or the jaw at all.

Pain around the teeth and the jaws can be symptoms

- of diseases of the heart (angina pectoris, myocardal infarction)
- ears (inner or external ear infections)
- sinuses (sinusitis)
- neuralgias and other nerve ailments

Some problems that cause oral pain

Sensitivity to hot or cold foods without recent dental work.

- loose filling,
- decay,
- fracture in the tooth,
- minimal gum recession which exposes small areas of the root surface.

Prolonged sensitivity to hot or cold foods without recent dental work.

- pulp irreversibly damaged by deep decay,
- crack/fracture,
- periodontal disease or trauma.

Sensitivity to hot or cold foods after recent dental treatment.

• inflammation of the pulp, inside the tooth, causing temporary sensitivity.

Some problems that cause oral pain

Dull ache near a tooth and/or biting sensitivity <u>after recent dental</u> <u>treatment</u>

• inflammation

Sharp pain when biting down on food

- loose filling
- decay
- cracked or split tooth
- cuspal fracture
- vertical root fracture

Constant and severe pain with pressure, swelling of the gum, and sensitivity to touch

• absces

A tooth hurts after taping on it with finger from the side.

inflammation of periodontal ligament

Some problems that cause oral pain

Dull ache and pressure in upper teeth and jaw

- sinus problems (sinusitis)
- grinding of teeth (bruxism)

Chronic pain in head, neck, or ear

• sometimes pulp-damaged teeth cause pain in other parts of the head and neck, but other dental or medical problems may be responsible.

Touching a specific spot in or near mouth triggers a sharp, jabbing pain lasting a few seconds. Sometimes talking may also cause this to occur.

trigeminal neuralgia

Clicking or pop is heard when opening mouth. Opening/closing of mouth may be painful

temporomandibular dysfunction

Trigeminal neuralgia





https://my.clevelandclinic.org/health/disea ses/15671-trigeminal-neuralgia-tn https://en.wikipedia.org/wiki/Trigeminal_ neuralgia

Trigeminal pain

- long-term pain disorder that affects trigeninal nerve
 - responsible for transfer of pain, touch and temperature
- ✓ it is type of neuropathic pain
 - caused by lesion or disease of somatosensory NS
- ✓ Two types:
 - ✓ typical (paroxysmal)
 - atypical (with continous pain)
- ✓ Cause:
 - ✓ unknown
 - ✓ some theories exist:
 - primary pressure of enlarged or lenghtened blood vessel (usually a. cerebri superior) on part of n. trigeminus root
 - ✓ can demage myelin of n. trigeminus
 - ✓ results to erratic and hyperactive functioning of the nerve
 - triggers attack of crucial pain after slightest stimulation of innervated regions







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Trigeminal pain

Symptoms:

 \checkmark

- usually paroxysmal attacks
- last from few seconds to several minutes or hours
- ✓ 4-10 attacks daily
- like stabbing electric shocks
- ✓ burning, sharp, pressing, crushing, exploding or shooting
- ✓ can migrate to other branches
- ✓ unilateral

✓ triggers:

- ✓ touch
- blow of wind
- ✓ chewing
- ✓ spontaneously
- ✓ eating
- ✓ talking
- ✓ shaving



Atypical trigeminal neuralgia

- ✓ continual pain
- ✓ less serious as typical type
- \checkmark over 50% of the time
- ✓ burning, prickling

https://www.osmosis.org/answers/trigeminal-neuralgia

Bleeding in oral cavity

Causes of bleeding in oral cavity

Gingivitis

• Edema, pain, bleeding

Periodontitis

- Inflammation of gums, jawbone and supportive tissues
- Bleeding, inflammation, pain, loss of teeth.

Vitamin deficiencies

- Vitamin C
- Vitamin E

Other

- Leukemia
- Hemophilia
- Chronic irritation, injury
- Surgery
- Pregnancy hormonal changes



Disorders of odontogenesis





https://x.com/DrLindseyFitz/status/613 433381137842176

https://www.reddit.com/r/oddlyterrifying/co mments/17bfwvs/my_5_year_olds_dentist_ xray_with_adult_teeth/

• The process of development of teeth is a very complex process resulting from interactions between the ectoderm of the oral cavity, which gives rise to cells that produce enamel, and the neural crest ectomesenchyme which gives rise to the tooth structures other than enamel.



	There are four stages i 1. Bud stage 2. Cap stage 3. Early bell stage 4. Late bell stage	in the development of the tooth germ:		
S	TAGE/TIME SPAN	DESCRIPTION		
In si	itiation stage/ xth to seventh week	Ectoderm lining stomodeum gives rise to oral epithelium and then to dental lamina; adjacent to deeper ectomesenchyme, which is influenced by the neural crest cells. Both tissue types are separated by a basement membrane		
Bud stage/ eighth week		Growth of dental lamina into bud shape that penetrates growing ectomesenchyme		
C ni	ap stage/ nth to tenth week	Formation of tooth germ as enamel organ forms into cap shape that surrounds inside mass of dental papilla, with an outside mass of dental sac, both from the ectomesenchyme.		
B el	ell stage/ eventh to twelfth week	Differentiation of enamel organ into bell shape with four cell types and dental papilla into two cell types		
A Va	pposition stage/ aries per tooth	Dental tissue types secreted in successive layers as matrix		
M Va	aturation stage/ aries per tooth	Dental tissue types fully mineralize to mature form		



Radiograph of lower right (from left to right) third, second, and first molars in different stages of development

r d



X-ray of teeth of a boy aged 5 years showing left lower primary molar and developing crowns of left lower permanent premolar (below primary molar) and permanent molars



Histologic slide showing a tooth bud. A: enamel organ B: dental papilla C: dental follicle https://en.wikipedia. org/wiki/Human_too th development

https://en.wikipedia. org/wiki/Human_too th_development

	Maxillary (upper) teeth							
Primary teeth	Central incisor	Lateral incisor	Canine	First molar	Second molar			
Initial calcification	14 wk I.U.	16 wk I.U.	17 wk I.U.	15.5 wk I.U.	19 wk I.U.			
Crown completed	1.5 mo	2.5 mo	9 mo	6 mo	11 mo			
Root completed	1.5 yr	2 yr	3.25 yr	2.5 yr	3 yr			
	Mandibular (lower) teeth							
Initial calcification	14 wk I.U.	16 wk I.U.	17 wk I.U.	15.5 wk I.U.	18 wk I.U.			
Crown completed	2.5 mo	3 mo	9 mo	5.5 mo	10 mo			
Root completed	1.5 yr	1.5 yr	3.25 yr	2.5 yr	3 yr			

https://en.wikipedia. org/wiki/Human_too th_development

Teeth development

	Maxillary (upper) teeth							
Permanent teeth	Central incisor	Lateral incisor	Canine	First premolar	Second premolar	First molar	Second molar	Third molar
Initial calcification	3–4 mo	10–12 mo	4–5 mo	1.5– 1.75 yr	2–2.25 yr	at birth	2.5–3 yr	7–9 yr
Crown completed	4–5 yr	4–5 yr	6–7 yr	5–6 yr	6–7 yr	2.5–3 yr	7–8 yr	12–16 yr
Root completed	10 yr	11 yr	13–15 yr	12–13 yr	12–14 yr	9–10 yr	14–16 yr	18–25 yr
	Mandibular (lower) teeth							
Initial calcification	3–4 mo	3–4 mo	4–5 mo	1.5–2 yr	2.25– 2.5 yr	at birth	2.5–3 yr	8–10 yr
Crown completed	4–5 yr	<mark>4–5</mark> yr	6–7 yr	5–6 yr	6–7 yr	2.5–3 yr	7–8 yr	12–16 yr
Root completed	9 yr	10 yr	12–14 yr	12–13 yr	13–14 yr	9–10 yr	14–15 yr	<mark>18–25 y</mark> r

https://en.wikipedia. org/wiki/Human_too th_development

Eruption times for primary and permanent teeth [51]

	Primary teeth							
Teeth	Central incisor	Lateral incisor	Canine	First premolar	Second premolar	First molar	Second molar	Third molar
Maxillary teeth	10 mo	11 mo	19 mo	-	-	- <mark>16 m</mark> o	-29 mo	-
Mandibular teeth	8 mo	13 mo	20 mo	-		-16 mo	-27 mo	-
			~	Perman	ent teeth			
Teeth	Central incisor	Lateral incisor	Canine	First premolar	Second premolar	First molar	Second molar	Third molar
Maxillary teeth	<mark>7–</mark> 8 yr	<mark>8–</mark> 9 yr	11–12 yr	10–11 yr	10–12 yr	6–7 yr	12–13 yr	17–21 yr
Mandibular teeth	6–7 yr	7–8 yr	9–10 yr	10–12 yr	11–12 yr	6–7 yr	11–13 yr	17–21 yr

Disorders of tooth development and eruption

- Anodontia
 - Absence of teeth Hypodontia
 - Oligodontia





• Hyperdontia - supernumerary teeth






Hyperdontia



https://www.sciencedirect.com/science /article/pii/S088954061100583X

Disorders of tooth development and eruption

- Abnormalities of size and form
 - Fusion of teeth
 - Dens evaginatus
 - Dens in dente
 - Enamel pearls
 - Macrodontia
 - Microdontia
 - Peg-shaped (conical) teeth
 - Supernumerary roots
 - Taurodontism













Disorders of tooth development and eruption

- Mottled teeth
 - Dental fluorosis



- Disturbances of tooth formation
 - Aplasia and hypoplasia of cementum
 - Enamel hypoplasia
 - Hypocalcification of teeth
- Hereditary disturbances in tooth structure
 - Amelogenesis imperfecta
 - Dentinogenesis imperfecta
 - Odontogenesis imperfecta





Disorders of tooth development and eruption

• Disturbances in tooth eruption

- late
- obstructed
- premature



Disorders of tooth development and eruption

neonatal teeth

natal teeth

• at birth

• up to 1st month



https://www.researchgate.net/publication/285588021_Natal_and_neonatal_teeth_Literature_review_and_report_of_seven_cases_in_a_Nigerian_Tertiary_Hospital/figures?lo=1