

EMBERTIOLOGY

LEC 1+2

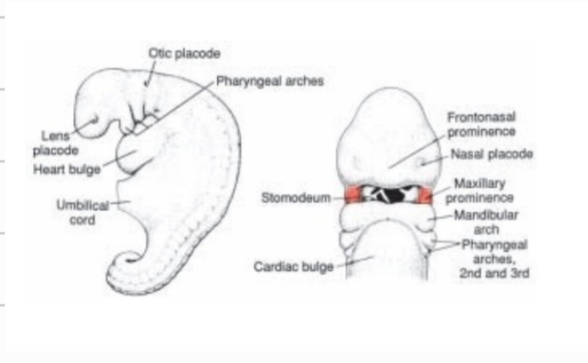


LEC-1

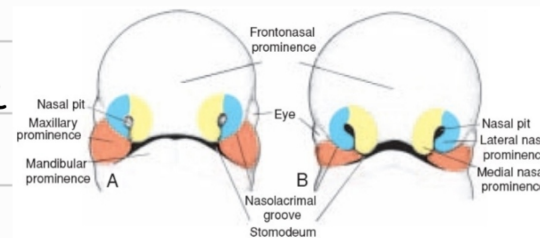
Nose and Palate

- **Olfactory**
- Nasal placode: the point where the nose and nasal opening will start their development
- Below forehead

- Origin of placodes: Ectoderm



- ① Nasal placode will make invagination and form nasal pits (Nostril).
- ② More invagination → forms the vestibule



Facial prominences: Mesenchyme

- 1- Frontonasal prominence from frontal bone and descends to form the nasal septum + medial and lateral nasal prominences

Nasal prominences: lateral and medial

- Medial: Growth near the midline → participate in formation of septum + tip of the nose.
- + Philtrum of upper lip + medial $\frac{1}{3}$ of upper lip. → while the lateral is made from the maxillary prominence.

- The maxillary grow upward to meet the medial nasal and fuse with it to form the upper lip.

☆ If did not fuse → Cleft lip & the uni or bilateral

- lateral: Form the Ala of the nose + lateral wall of the nose

TABLE 15.2 Structures Contributing to Formation of the Face

Prominence	Structures Formed
Frontonasal ^a	Forehead, bridge of nose, medial and lateral nasal prominences
Maxillary	Cheeks, lateral portion of upper lip
Medial nasal	Philtrum of upper lip, crest and tip of nose
Lateral nasal	Alae of nose
Mandibular	Lower lip

^a The frontonasal prominence is a single unpaired structure; the other prominences are paired.

Nasal Cavity

- ☆ The nasal pits → More invagination → Mesenchymal cells undergo more proliferation → then canalization → form the cavity above the the oronasal membrane.
- ☆ The posterior part of the cavity is the primitive Choana.
- Oronasal membrane: separates the oral cavity from nasal cavity then ruptures and the palate will form in its place (primary palate).
- The definitive choana and secondary palate will form
- ☆ The lateral wall is also formed.

Paranasal Air Sinuses

- Their formation starts from the lateral wall of nasal cavity; from the opening of the sinus starts the proliferation of cells the proliferation will form the duct → when reaching the bone will form rudimentary cavity that are small in size → with the growth of facial bones the cavities will grow and become in the Adult size. (sinuses)
- Same for all the paranasal sinuses.

Palate

Primary palate

- Hard palate: formed of two parts. ① palatine process of Maxilla
② Horizontal process of palatine
- During Development: the maxillary process and medial nasal prominence grow toward the midline and they fuse together and form

Intermaxillary segment

- ☆ Inter maxillary segment gives: ① Labial components (upper lip + philtrum)
② primary palate
③ upper jaw components (upper 4 incisors).

☆ Primary palate is triangular in shape with an apex and communicate with the septum to form the 2 nasal cavities.

Secondary palate

☆ From the maxillary prominence (Palatine shelflike outgrowth).

- A shelf originates from the maxillary prominence from the side to midline
- At the sixth week.
- Directly above the tongue to form the oral cavity.
- 2 shelves one from right and one from left.

☆ Then the secondary fuses with the primary to form the hard palate.

- Incisive foramen: After the infusion between the primary and the secondary a small opening will remain persistent for passage of greater palatine nerve and artery.

- The posterior edge of Secondary palate; the edge of palat process will grow backward to form the Soft palate and uvula.
two sides right and left fuse together

- Soft palate 8 week 8 uvula: week 11

- Rarely: people may have a separated uvula due to failure of fusion



Primitive Gut

- 1- Pharyngeal gut: from the buccopharyngeal membrane and forms the pharynx.
- 2- Foregut: Esophagus $\rightarrow$ liver buds (middle of duodenum). **Celiac Trunk**
- 3- Midgut: liver buds $\rightarrow$ Junction between proximal $\frac{2}{3}$ and distal $\frac{1}{3}$ of transverse colon. **Superior Mesenteric A.**
- 4- Hindgut & distal $\frac{1}{3}$ of transverse colon $\rightarrow$ upper $\frac{1}{2}$ of anal canal. **Inferior Mesenteric A.**

Respiratory Diverticulum

- Depends on fibroblast growth factor (FGF).
- FGF released in the 4th week of gestation stimulates the endodermal anterior wall of the foregut $\rightarrow$ proliferation of cells $\rightarrow$ form the Respiratory diverticulum $\rightarrow$ continue to grow to form the trachea then primary bronchi then secondary $\rightarrow$ Alveoli.

- Origin: Endoderm

- Origin of cartilages and muscles: splanchnic Mesoderm.

☆ ^{4th week} Trachioesophageal ridge: Makes constriction between respiratory and digestive $\rightarrow$ Increase in growth become Tracheo-esophageal Septum and the trachea is anterior and esophagus posterior (5th week).

$\rightarrow$ The only remain communication between trachea and esophagus is through laryngeal pharynx and Inlet of larynx.

- The larynx at first: it's a slit like opening.

$\rightarrow$ when the cartilages are formed (Cricoid and thyroid mainly) it become T-shaped.

$\rightarrow$ when the epiglottis is formed $\rightarrow$ The permanent adult shape is formed.

Esophagus

- when it's formed it's short
- with the development of the heart and lungs it will elongate and descend.
- Muscular tube : upper part : striated muscles — innervated by Vagus
lower part : smooth muscles — innervated by splanchnic plexus of nerves (Autonomic)

Anomalies of Trachea : Tracheoesophageal Fistula (TEF). And Atresia

* Atresia : blind end of esophagus usually proximal.

* Fistula : connection between esophagus and trachea.

- The most common form : proximal Atresia with distal fistula
↳ 1/3000 births.

- There are other forms, see the slides.

☆ Clinical picture of proximal Atresia with distal fistula :

① During pregnancy : polyhydramnios (Excess of Amniotic fluid).

- Must be a constant amount and undergo secretion and absorption
→ It's absorbed into the GI tract then secreted again through urination. In case of Atresia all the fluid will go back to the amniotic cavity → Excess Amniotic fluid.

② After delivery : Each time the mother tries to feed the baby the milk will come back out (regurgitation). (Atresia)

③ When the baby cries : Air will enter the stomach → Abdomen will become distended and bloated. (Fistula)

④ Chronic infections in the Respiratory tract : when a material from the GIT enters the Respiratory.

→ Treatment: urgent operation closes the fistula and reconnect the esophagus.

☆ Common Anomalies combined with Atresias

1- Cardiac Anomalies : - Intra-Atrial Septal defect
- Intra-Ventricular Septal defect
- Fallots tetralogy.

2- Vertebral Anomalies

3- Anal Atresia

4- limb defect

5- Renal Anomalies.

Tracheal Atresia and Stenosis

- Block in the trachea due to formation of an abnormal tissue
- Very rare

Larynx

- Internally : Endoderm
- Cartilage and Muscles : splanchnic mesoderm.

☆ Formed from the 4th and the 6th pharyngeal arches

- The opening when it's firstly formed it's split like then when the cartilages are formed it become T-shaped. (Inlet of larynx)

- Proliferation of epithelial cells → Occlusion of larynx

(It was a cavity → then became occluded with cells).

→ Then vaculation and Recanalization (the cells that have been formed will be reabsorbed). Why did all that happen?

- To form the ventricles.

* When ventricles are formed that means :

① True vocal cords ② False vocal cords ③ The Sacule
are all formed

- Muscles of larynx : All are internal except the cricothyroid, External.
↳ All innervated by recurrent laryngeal nerve except Cricothyroid by External laryngeal → Why? due to their origin from pharyngeal^{arches}

→ Superior laryngeal Nerve divides into internal and external laryngeal.

The external laryngeal go to the 4th pharyngeal arch that gives the Cricothyroid.

While the recurrent laryngeal nerve go to the 6th pharyngeal that will give rise to all the muscles.

- Cricothyroid : 4th pharyngeal Arch.

- All muscles : 6th pharyngeal Arch.

Anomalies of Larynx

- Atresia (Rare) → Obstruction of upper airway passage.

↳ Known as : Congenital High Airway Obstruction Syndrome.

→ Enlargement of lung to compensate.

→ production of abnormal tissue, echoes (echogenic)

→ The diaphragm won't have two cupula → It will be flattened → Ascites will form which is accumulation of fluid in the abdomen.

- Diagnosis : Ultrasonography.

Trachea, Bronchi and Lungs

- Formed by the growth of lung buds.
- Trachea will be formed first and when it reaches T₄ it will divide into two main bronchi.

→ More development will form the secondary bronchi: ↗ 3 in right
↘ 2 in left

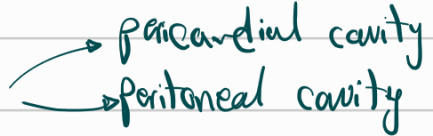
→ Then the tertiary 10 in Right + 8 in Left.

↳ Remember Anatomy Lec-5.

LEC-2

Dichotomous division of bronchioles means:

Each bronchiole splits into two smaller branches, and each of those branches divides again in the same way, repeatedly.

- o After the development of the segmental bronchi the bronchioles will develop. (conducting + respiratory).
- o Bronchiole develop through (Dichotomous division). نزي أفرع الشجر
 - when going distally → more branches with smaller diameter.
 - These divisions occur in a space around the lung called **pericardioperitoneal canal**.
 - ☆ **pericardioperitoneal** will form  **pericardial cavity** and **peritoneal cavity**.
 - ↳ then will have three cavities & peritoneum will go toward the abdomen to form peritoneal cavity
 - ☆ **pericardium** will go toward the chest and become **pleuropericardial cavity**.
 - ☆ **pleuropericardial** will divide into **pleural** and **pericardial** cavities.

- Pleura is made of visceral and parietal pleura.
 - Visceral : Splanchnic pleura from splanchnic mesoderm.
 - Parietal : Somatic pleura from somatic mesoderm.

Terminal Bronchioles

- ☆ by the end of sixth month approximately 17 generations of subdivisions have formed.
- ☆ In post natal life 6 additional divisions will form.
- 23 divisions
- Divisions occur due to fibroblast growth factor.

Maturation of the lung

→ Means the formation of respiratory bronchioles + Alveolar duct and Alveolar sac + Alveoli

☆ Maturation of lungs is in 4 stages:

TABLE 12.1 Maturation of the Lungs

Pseudoglandular period	5-16 weeks	Branching has continued to form terminal bronchioles. No respiratory bronchioles or alveoli are present.
Canalicular period	16-26 weeks	Each terminal bronchiole divides into 2 or more respiratory bronchioles, which in turn divide into 3-6 alveolar ducts. → no respiratory membrane
Terminal sac period	26 weeks to birth	Terminal sacs (primitive alveoli) form, and capillaries establish close contact. → Cells will change from cuboidal to simple squamous. Respiratory membrane
Alveolar period	8 months to childhood 10 years	Mature alveoli have well-developed epithelial endothelial (capillary) contacts. → well developed respiratory membrane.



→ few years before any baby born before the 6th month will die directly. nowadays babies can live even when born at age 5½ months in the premature incubator with oxygen supply and surfactant until completing the 9th month.

→ The number of Alveoli at birth is only 1/6 of its number in adults and increase till reaching its maximum at the age of 10.

→ Role of Surfactant & It decreases the surface tension so during Expiration inflation of alveoli will occur

? If no Surfactant is present $\Rightarrow$ Infant Respiratory Distress Syndrome ^{IRDS}
 $\rightarrow$ During expiration alveoli will shrink $\rightarrow$ Atelectasis of lungs.

$\rightarrow$ Nowadays artificial surfactant can be given to the baby with IRDS.

or we may stimulate its synthesis using ^{Cortizone} Betamethazone or by ^{thyroxine} from thyroid gland.

$\rightarrow$ After the secretion of surfactant a fluid will form in the lung: formed from surfactant and amniotic fluid.

☆ Amniotic fluid is very important in the development of the respiratory tract

$\rightarrow$ In some cases when the baby is born the amniotic fluid and surfactant are blocking the respiratory tract.
 $\rightarrow$ So we use a tube from the oral cavity to inlet of larynx for suction of the fluids

Anomalies of the respiratory tract

☆ **RDS**: Common cause of death in premature infants (30% of neonatal diseases / 20% of neonatal deaths).

$\rightarrow$ Cause: type II cells secrete low amount of surfactant.
 $\rightarrow$ Atelectasis with expiration.

☆ Also known as **Hyaline membrane disease**

☆ **Agenesis of one lung** : Born with one lung only while the other is not formed *very rare*.

☆ **Supernumerary lobules** : The lung is divided into more lobules.

☆ **Ectopic lung** : The lung is not in the chest → may be in the abdomen.

☆ **Congenital Cysts of the lung** : Multiple cysts filling the lung with honeycomb appearance.

- *Complications*: 1- Chronic infections
2- Insufficiency of breathing

- *Treatment*: through excision

☆ **Lung Hypoplasia** : Shrinkage of lung due to 2 causes.

① Congenital diaphragmatic hernia : Common in left side.

→ Small intestine herniate through the diaphragm and compresses the left lung → hypoplasia

→ Decreased Amniotic fluid

② Oligohydramnios : Amniotic fluid is essential in lung Development → Decreased amniotic fluid will cause retardation of lung development.

Lungs of Newborn Infants

- Fresh and healthy lungs contain some air so pulmonary samples float in water

- The lungs of the stillborn infants are firm and sink in water because they contain fluids not air.