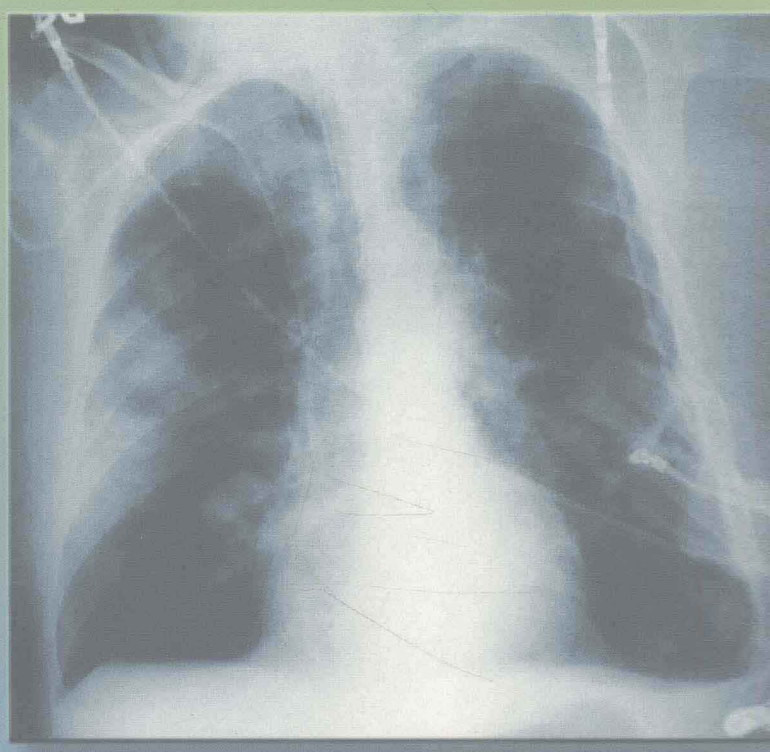
HIGH-YIELD SYSTEMS

High-Yield™ *Lung*

RONALD W. DUDEK



High-Yield Lung is designed to:

- Provide coverage of basic science concepts using a systems-based approach
- Help medical students integrate material from first two years and prepare for clinical years
- Present consistent, easy review for USMLE Step 1
- Appeal to the student seeking consistently formatted, highly illustrated coverage

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High-Yield Systems

High-Yield Lung

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High-Yield Lung

Preface

A focused curriculum is a curriculum whereby students are immersed in one basic science discipline (e.g., histology) for a concentrated period of time, during which histology is covered from A to Z. A systems-based curriculum is a curriculum whereby students are immersed in one system (e.g., respiratory system) for a concentrated period of time, during which all basic science disciplines of the respiratory system are covered (e.g., embryology, histology, physiology, pharmacology, and so forth).

The High-Yield Systems Series addresses a problem endemic to medical schools in the United States and medical students using a focused curriculum. After completing a focused curriculum, the medical student is faced with the daunting task of integrating and collating all the basic science knowledge accrued from the focused curriculum into the various systems. For example, a medical student wanting to review everything about the lung will find the information scattered in his/her embryology notes, histology notes, physiology notes, pharmacology notes, and so forth. The High-Yield Systems Series eliminates this daunting task for the medical student by bringing together the embryology, gross anatomy, radiology, histology, physiology, microbiology, and pharmacology of the lung in one clear and concise book.

The High-Yield Systems Series is useful for the following:

1. First-year medical students in a focused curriculum who want to get a head-start on the inevitable integration and collation process of all the information learned in a focused curriculum into systems.
2. First-year medical students in a systems-based curriculum will find this series a natural textbook for a systems-based curriculum.
3. Medical students preparing for Step 1 of the USMLE, in which the questions are becoming increasingly more systems- than discipline-based.
4. Second-year medical students in which the curriculum is much more systems-based, as pathology covers the pathology of each system as a block (e.g., pathology of the lung, pathology of the heart, pathology of the kidney, etc.).
5. Senior medical students may want to quickly review all aspects of lung function before starting a rotation in pulmonology, for example.
6. Recent medical graduates may want to quickly review all aspects of lung function before starting a residency in pulmonology, for example.

In the High-Yield Systems Series, the student will find the same painstaking attention given to include high-yield information as found in other High-Yield books. However, the breadth of information has been expanded somewhat to cover some baseline information without which a complete understanding of the system would be difficult.

The High-Yield books, based on the presentation of high-yield information that is likely to be asked on the USMLE, have clearly been an asset to the medical student. However, after writing many High-Yield books, I have found that high-yield information can also be presented in a high-efficiency manner. In the High-Yield Systems Series, the student now gets the benefit of both high yield and high efficiency in their studies.

I appreciate any feedback and can be contacted at dudekr@mail.ecu.edu.

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Embryology of the Lung

I General Features

The respiratory system is divided into the **upper and lower respiratory systems.** The upper respiratory system consists of the **nose, nasopharynx,** and **oropharynx** and is typically discussed with head and neck embryology, namely the pharyngeal apparatus. The lower respiratory system (*Fig. 1-1*) consists of the **larynx, trachea, bronchi,** and **lungs.** The first sign of development is the formation of the **respiratory diverticulum** in the ventral wall of the primitive **foregut** during week 4, both of which are lined by **endoderm** and surrounded by a bed of **mesoderm.** The distal end of the respiratory diverticulum enlarges to form the **lung bud.** The lung bud divides into two **bronchial buds** that branch into the **main (primary), lobar (secondary), segmental (tertiary),** and **subsegmental bronchi.** The respiratory diverticulum initially is in open communication with the foregut, but eventually they become separated by indentations of mesoderm, the **tracheoesophageal folds.** When the tracheoesophageal folds fuse in the midline to form the **tracheoesophageal septum,** the foregut is divided into the trachea ventrally and the esophagus dorsally. The **Hox-complex, FGF-10** (fibroblast growth factor), **BMP-4** (bone morphogenetic protein), **N-*myc*** (a proto-oncogene), **syndecan** (a proteoglycan), **tenascin** (an extracellular matrix protein), and **epimorphin** (a protein) appear to play a role in development of the respiratory system.

II Development of the Larynx

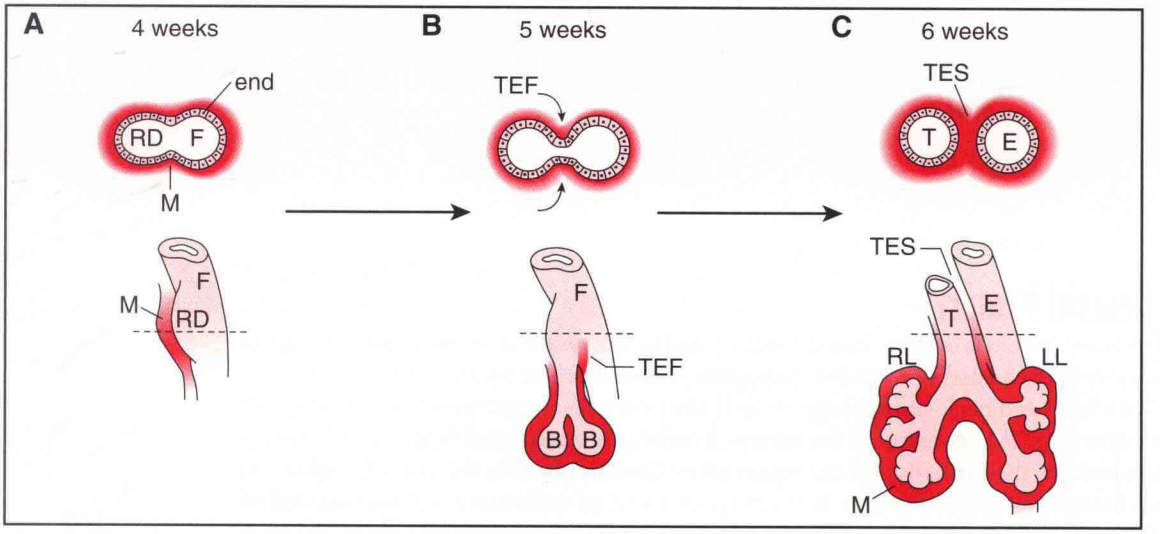
The opening of the respiratory diverticulum into the foregut becomes the **laryngeal orifice.** The laryngeal epithelium and glands are derived from endoderm. The laryngeal muscles are derived from somitomeric mesoderm of pharyngeal arches 4 and 6 and therefore are innervated by branches of the vagus nerve (CN X); that is, the superior laryngeal nerve and recurrent laryngeal nerve, respectively. The laryngeal cartilages (thyroid, cricoid, arytenoid, corniculate, and cuneiform) are also derived from somitomeric mesoderm of pharyngeal arches 4 and 6.

III Development of the Trachea

A. **Sources.** The tracheal epithelium and glands are derived from endoderm. The tracheal smooth muscle, connective tissue, and C-shaped cartilage rings are derived from mesoderm.

B. **Clinical considerations of the trachea**

1. A **tracheoesophageal fistula** is an abnormal communication between the trachea and esophagus that results from improper division of foregut by the tracheoesophageal septum. It is generally associated with **esophageal atresia** and **polyhydramnios.** Clinical features include: excessive accumulation of saliva or mucus in the nose and mouth; episodes of gagging and cyanosis after swallowing milk; abdominal distension after crying; and reflux of gastric contents into the lungs, causing pneumonitis. Diagnostic features include: inability to pass a catheter into the stomach and radiographs demonstrating air in the infant's stomach.
2. **Tracheal agenesis** is a rare congenital abnormality that may occur in three forms. **In Type I,** the upper trachea is absent while the lower trachea is connected to the esophagus. **In Type II,** the trachea is absent while a common bronchus that branches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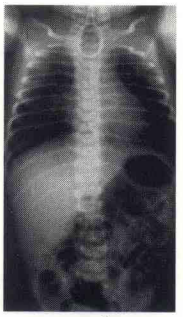

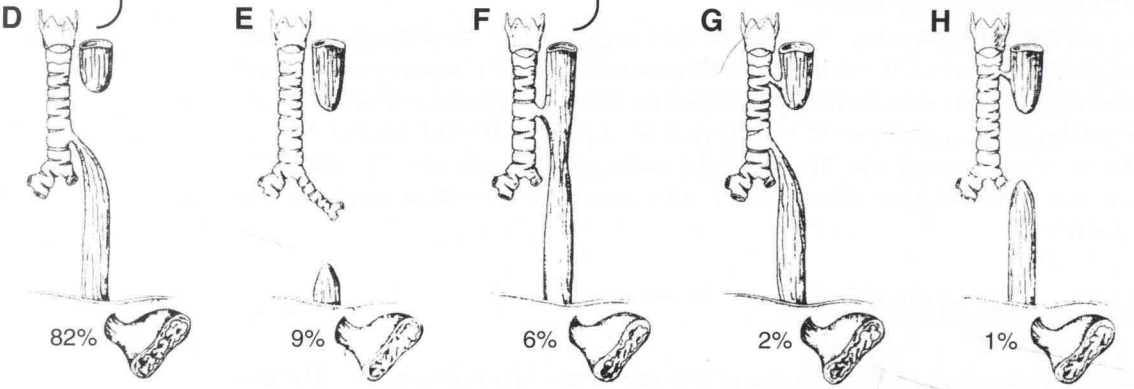
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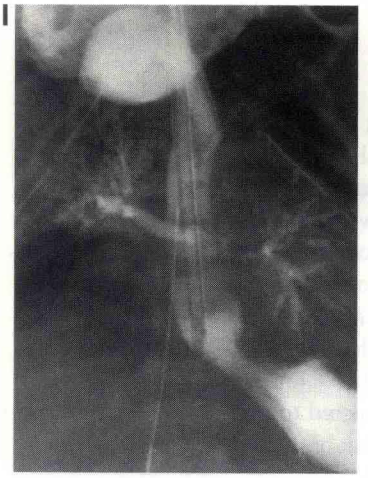
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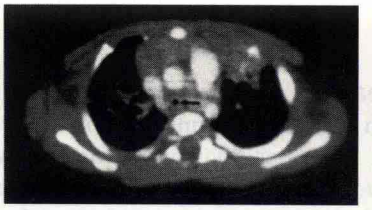
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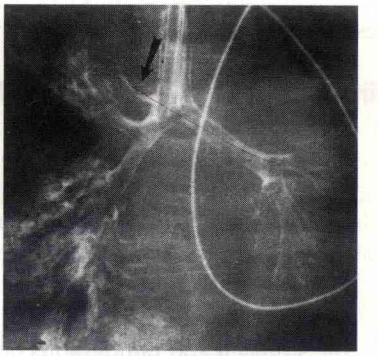
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into a right and left main bronchus arises from the esophagus. **In Type III,** the right and left main bronchi arise from the esophagus. Clinical features include: immediate, severe respiratory distress, absence of a cry, and inability to intubate below the larynx.

3. **Tracheal stenosis** is a rare congenital abnormality caused by the complete lack of cartilaginous rings in the tracheal wall resulting in a narrowing of the tracheal lumen. Acquired tracheal stenosis usually is caused by intubation or traumatic tracheobronchial suctioning.
4. **Tracheal bronchus** occurs when an ectopic bronchus branches directly from the trachea. This generally occurs on the right so that the tracheal bronchus supplies the entire right upper lobe or an apical bronchopulmonary segment. Sometimes, a supernumerary segment may be present.
5. **Tracheomalacia** is a rare congenital abnormality caused by the softening of the cartilaginous rings in the tracheal wall. Clinical features include: expiratory stridor (wheezing) during crying, reflex apnea ("dying spells"), and sometimes cyanosis/bradycardia after feeding.

IV Development of the Bronchi

A. Stages of development. The lung bud divides into two **bronchial buds.** In week 5 of development, bronchial buds enlarge to form **main (primary) bronchi.** The right main bronchus is larger and more vertical than the left main bronchus. This relationship persists throughout adult life and accounts for the greater likelihood of foreign bodies lodging on the right side than on the left. The main bronchi further subdivide into **lobar (secondary) bronchi** (3 on the right side and 2 on the left side, corresponding to the lobes of the adult lung). The lobar bronchi further subdivide into **segmental (tertiary) bronchi** (10 on the right side and 9 on the left side), which further subdivide into **subsegmental bronchi.** The segmental bronchi are the primordia of the **bronchopulmonary segments,** which are morphologically and functionally separate respiratory units of the lung. As the endoderm-lined bronchi continue to branch into the bed of mesoderm, the airways become progressively smaller and expand into a space known as the primitive pleural cavity. The mesoderm covering the surface of the lung develops into **visceral pleura,** and somatic mesoderm covering the inside of the body wall develops into **parietal pleura.** The space between the visceral and parietal pleura is called the **pleural cavity.**

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FIGURE 1-1. Development of the respiratory system at (A) 4 weeks, (B) 5 weeks, and (C) 6 weeks. Both lateral views and cross-sectional views are shown. Note the relationship of the respiratory diverticulum (*RD*) and foregut (*F*), both of which are lined by endoderm and surrounded by a bed of mesoderm (*M*). Note that as the endoderm-lined bronchi continue to branch into the bed of mesoderm, the airways become progressively smaller. This branching is controlled by cell interactions between the endoderm and mesoderm called epithelio-mesenchymal interactions. *Curved arrows* indicate the movement of the tracheoesophageal folds (*TEF*) as the tracheoesophageal septum (*TES*) forms between the trachea (*T*) and esophagus (*E*). B—bronchial buds; RL—right lung; LL—left lung. **(D–H) Five different anatomical types of esophagus and trachea malformations. (D)** Esophageal atresia with a tracheoesophageal fistula at the distal one-third end of the trachea. This is the most common type, occurring in 82% of cases. The AP radiograph of this malformation shows an enteric tube (*arrow*) coiled in the upper esophageal pouch. The air in the bowel indicates a distal tracheoesophageal fistula. **(E)** Esophageal atresia only, occurring in 9% of cases. **(F)** H-type tracheoesophageal fistula only, occurring in 6% of cases. The barium swallow radiograph shows a normal esophagus (*E*), but dye has spilled into the trachea (*T*) through the fistula and outlines the upper trachea and larynx. **(G)** Esophageal atresia with a tracheoesophageal fistula at both proximal and distal ends, occurring in 2% of cases. **(H)** Esophageal atresia with a tracheoesophageal fistula at the proximal end, occurring in 1% of cases. **(I) Type II tracheal agenesis.** Postmortem AP esophagogram of a term newborn with severe respiratory distress. Note the common bronchus arising from the esophagus. **(J) Tracheal stenosis.** A CT scan shows severe narrowing of the lumen of the trachea. Note that the lumen of the trachea is a "perfect circle" rather than oval-shaped. **(K) Tracheal bronchus.** A tracheogram shows a right tracheal bronchus (*arrow*) along with a collapsed right upper lobe.

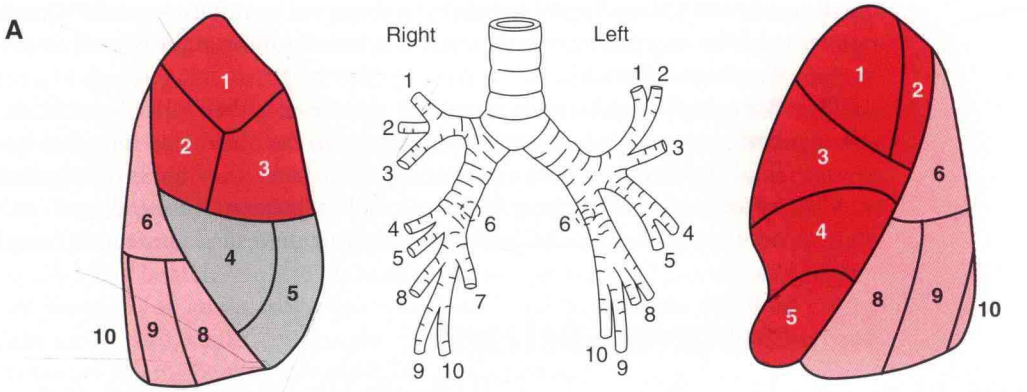
B. Sources. The bronchial epithelium and glands are derived from endoderm. The bronchial smooth muscle, connective tissue, and cartilage are derived from mesoderm.

C. Clinical considerations of the bronchi *(Fig. 1-2)*

1. The **bronchopulmonary segment** is a segment of lung tissue supplied by a **segmental (tertiary) bronchus, a branch of the pulmonary artery, a branch of the bronchial artery, and a branch of the bronchial vein,** all of which run through the **center** of the bronchopulmonary segment. **Branches of the pulmonary veins** are found at the **periphery** between two adjacent bronchopulmonary segments (i.e., intersegmental location), which form **surgical landmarks** during segmental resection. Surgeons can resect diseased lung tissue along bronchopulmonary segments instead of removing the entire lobe.
2. **Congenital lobar emphysema (CLE)** is characterized by progressive overdistension of one of the upper lobes or the right middle lobe with **air.** The term emphysema is a misnomer, as there is no destruction of the alveolar walls. Although the exact etiology remains unknown, many cases involve **collapsed bronchi** as a result of **failure of bronchial cartilage formation.** In this situation, air can be inspired through collapsed bronchi but cannot be expired. During the first few days of life, fluid may be trapped in the involved lobe, producing an opaque, enlarged hemithorax. Later, the fluid is resorbed and the classic radiological appearance of an emphysematous **(air-filled)** lobe with generalized radiolucency (hyperlucent) is apparent.
3. **Congenital bronchogenic cysts** represent an abnormality in bronchial branching and may be found within the mediastinum (most commonly around the carina, upper trachea, or main bronchi) or intrapulmonary. Intrapulmonary cysts are round, solitary, sharply marginated, **fluid-filled,** and do not initially communicate with the tracheobronchial tree. Cysts are lined by a ciliated epithelium and have smooth muscle and cartilage in its wall. Because intrapulmonary bronchogenic cysts contain fluid, they appear as water-density masses on chest radiographs. These cysts may become air-filled as a result of infection or instrumentation.
4. **Congenital cystic adenomatous malformation** is a hamartomatous proliferation of terminal bronchioles at the expense of alveoli, which results in both cysts and solid masses. The cysts are lined by an adenomatous epithelium and communicate with the rest of the tracheobronchial tree.
5. **Pulmonary sequestration** occurs when a mass of pulmonary tissue forms that is not connected to the tracheobronchial tree or pulmonary artery. If the pulmonary mass is located within the lung, it is called an **intralobar sequestration (ILS).** An ILS usually occurs in the lower lobes, is supplied by an anomalous artery branching from the aorta, and is drained by the pulmonary veins. If the pulmonary mass is located outside the lung, it is called an extralobar sequestration (ELS). An ELS usually occurs on the left side between the lower lobe and the diaphragm, is supplied by an anomalous artery branching from the aorta, and is drained by the azygous vein.

▶

FIGURE 1-2. **(A) Distribution of bronchopulmonary segments and their relationship to the tracheobronchial tree.** Segmental bronchi of the right and left lungs are numbered. *Right Lung:* 1,2,3: segmental bronchi that branch from the upper lobar bronchus; 4,5: segmental bronchi that branch from the middle lobar bronchus; 6,7,8,9,10: segmental bronchi that branch from the lower lobar bronchus. Note that bronchopulmonary segment #7 is not represented on the outer costal surface of the right lung (#7 is located on the inner mediastinal surface). *Left Lung:* 1,2,3,4,5: segmental bronchi that branch from the upper lobar bronchus; 6,8,9,10: segmental bronchi that branch from the lower lobar bronchus. Note that there is no #7 segmental bronchus associated with the left lung. **(B) Congenital lobar emphysema.** Expiratory AP radiograph shows a hyperlucent area in the emphysematous right upper lobe caused by air trapping. The affected lobe is overdistended and the mediastinum may be shifted to the contralateral side. Secondary ipsilateral compression of the lower lobe or herniation of the upper lobe into the mediastinum may occur. **(C) Congenital bronchogenic cyst.** AP radiograph shows a large, opaque area in the right upper lobe, caused by an intrapulmonary fluid-filled cyst. **(d) intrathoracic bronchogenic cyst.** CT scan shows an intrathoracic bronchogenic cyst (*C*) located near the carina. **(E) Congenital cystic adenomatous malformation.** CT scan shows a complex cystic mass in the right lower lobe with a reticulated, bubbly appearance. **(F, G) Pulmonary sequestration.** CT scan shows an extrapulmonary sequestration whereby a pulmonary mass (*arrowheads*) is present, associated with the left lower lobe. A pulmonary arteriogram shows an anomalous artery branching from the aorta to supply the sequestration.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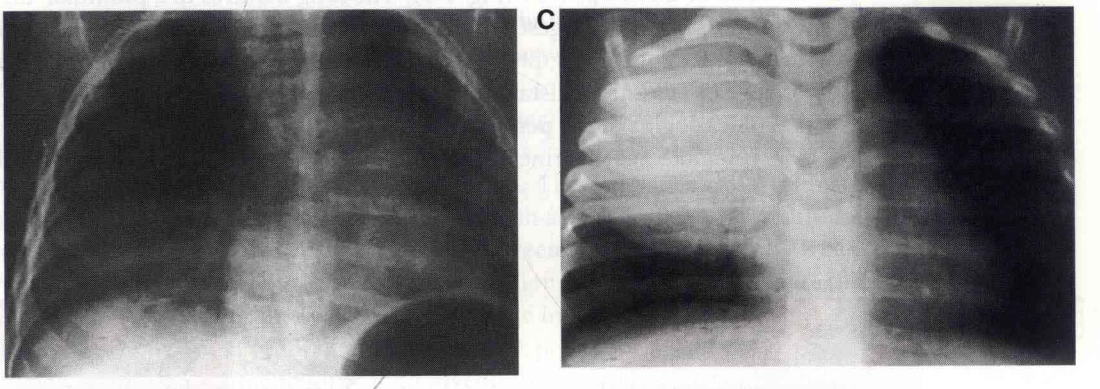
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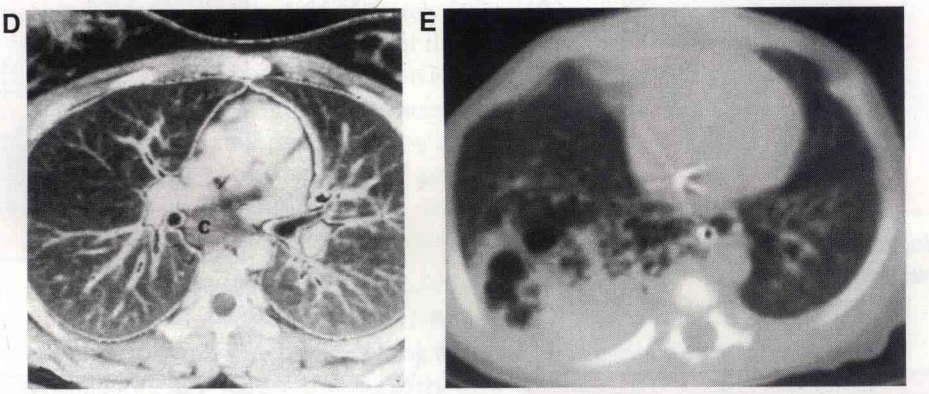
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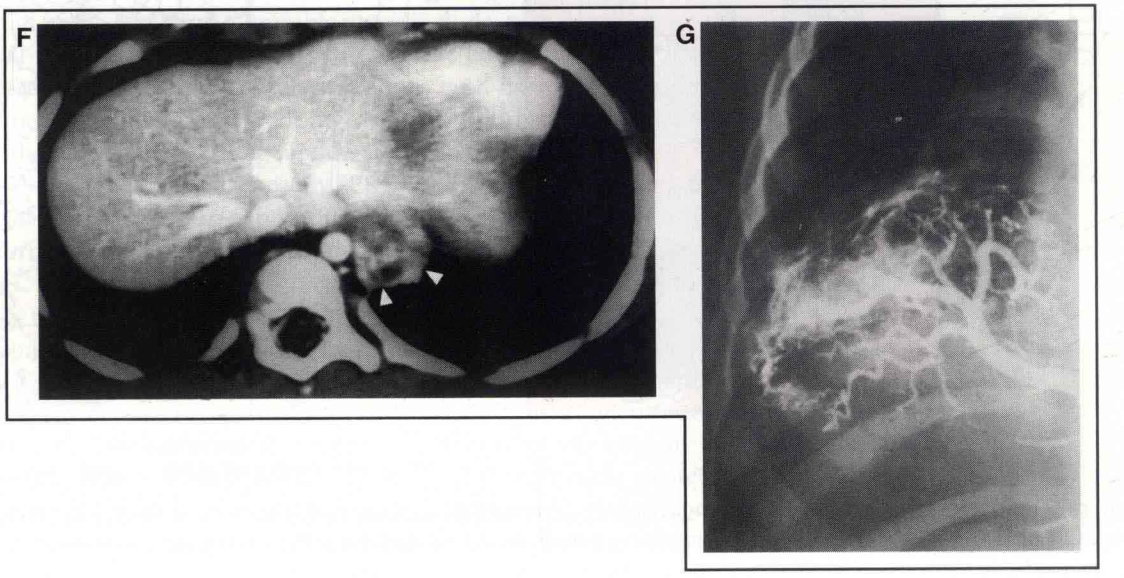
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6. A **bronchoesophageal fistula** is an abnormal communication between a bronchus and the esophagus, which usually is found on the right side in association with esophageal atresia.
7. **Bronchial atresia** is an abnormal closure of either lobar bronchi or segmental bronchi usually of the upper lobe. At birth, the obstructed lung may be filled with fluid and thus appear as a pulmonary mass. Later, the fluid is resorbed and replaced by air such that the lung becomes emphysematous. A central, oval, or fan-shaped accumulation of mucus distal to the obstruction (called a **mucocele**) may be present.

V Development of the Lungs

A. Periods of development *(Fig. 1-3)*. The lung matures in a proximal–distal direction, beginning with the largest bronchi and proceeding outward. As a result, lung development is heterogeneous; proximal pulmonary tissue will be in a more advanced period of development than distal pulmonary tissue.

1. **Pseudoglandular period (weeks 7–16).** During this period, the developing lung resembles an exocrine gland. The numerous **endodermal tubules** are lined by **sim-**

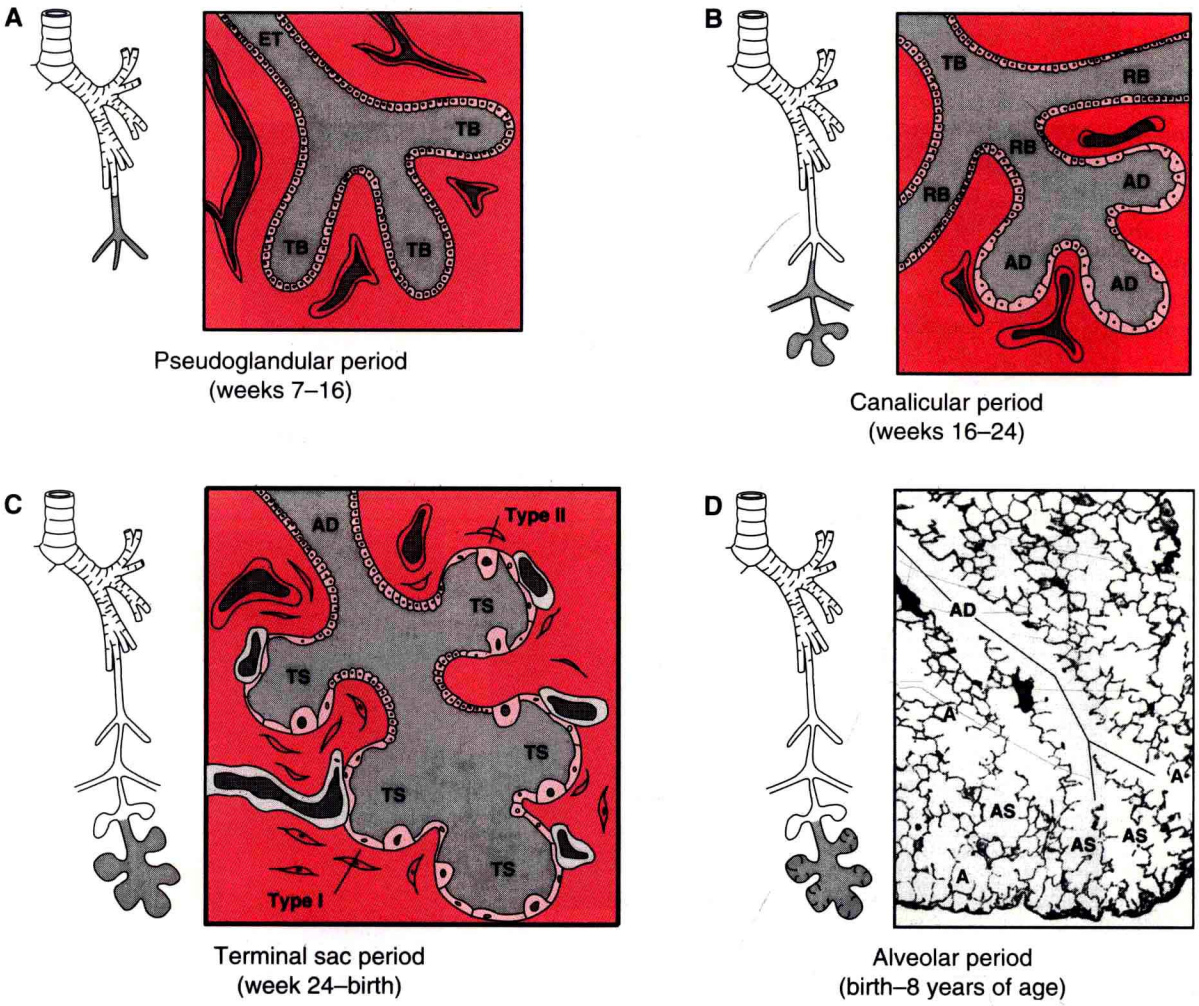


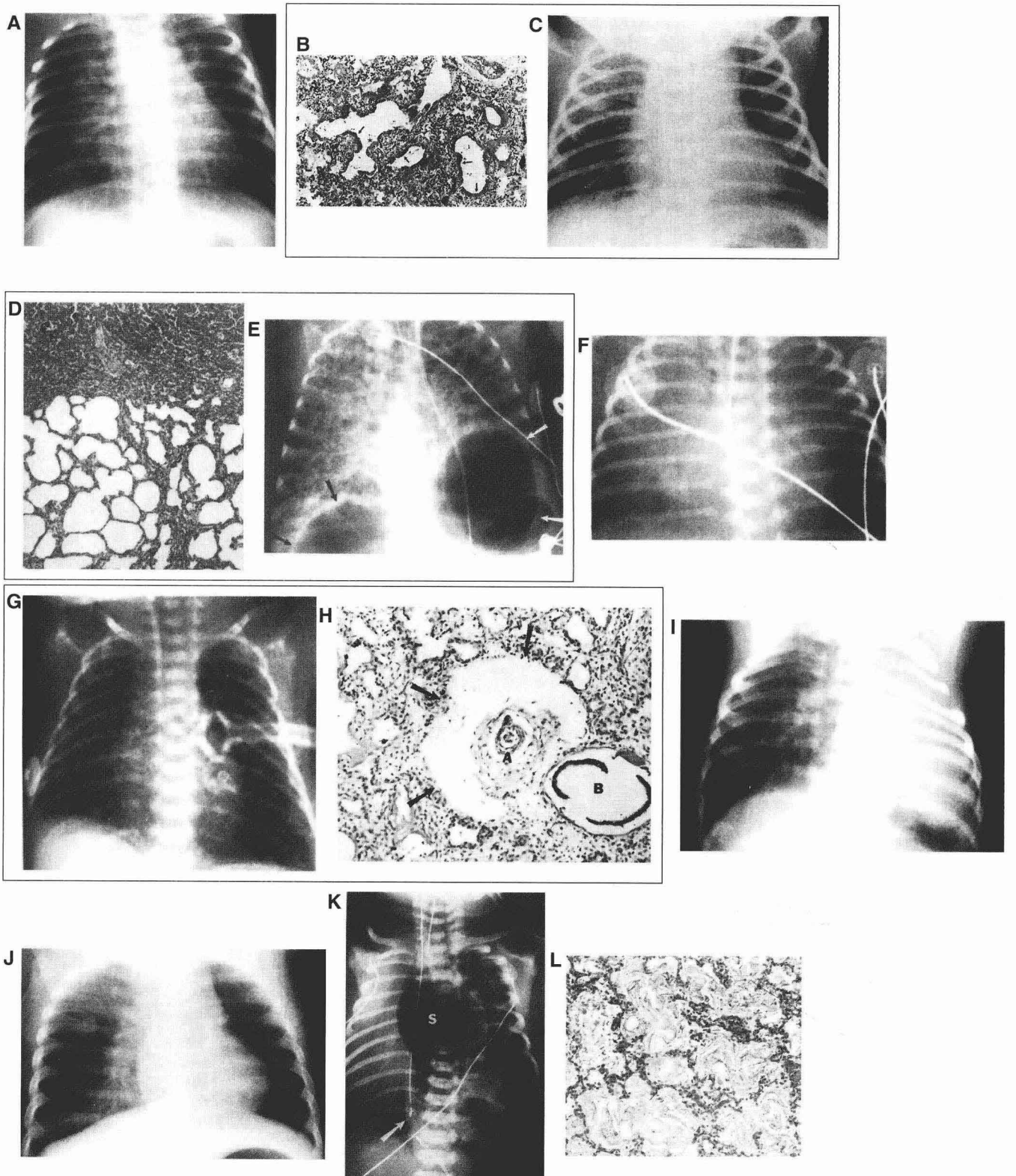
FIGURE 1-3. Time periods of lung development. (A) Pseudoglandular period, **(B)** Canalicular period, **(C)** Terminal sac period, **(D)** Alveolar period. ET—endodermal tubules; TB—terminal bronchiole; RB—respiratory bronchiole; AD—alveolar duct; TS—terminal sac; AS—alveolar sac; A—alveoli.

ple columnar epithelium that give rise to the epithelium of the future airways and are surrounded by **mesoderm** that give rise to the future connective tissue components of the lung (i.e., cartilage, collagen and elastic fibers, smooth muscle and vasculature). At this time period, the mesoderm forms only a **modest capillary network.** Each endodermal tubule branches into **15–25 terminal bronchioles.** During this period, respiration is not possible and premature infants cannot survive.

2. **Canalicular period (weeks 16–24).** During this period, the terminal bronchioles branch into three or more **respiratory bronchioles.** The respiratory bronchioles subsequently branch into 3 to 6 **alveolar ducts.** The terminal bronchioles, respiratory bronchioles, and alveolar ducts are now lined by a **simple cuboidal epithelium** and are surrounded by mesoderm containing a **prominent capillary network.** Premature infants born before week 20 rarely survive.
3. **Terminal sac period (week 24–birth).** During this period, **terminal sacs** bud off the alveolar ducts and then dilate and expand into the surrounding mesoderm. The terminal sacs are separated from each other by **primary septae.** The simple cuboidal epithelium within the terminal sacs differentiates into **type I pneumocytes** (thin, flat cells that make up part of the blood-air barrier) and **type II pneumocytes** (which produce surfactant). The terminal sacs are surrounded by mesoderm containing a **rapidly proliferating capillary network.** The capillaries make intimate contact with the terminal sacs and thereby establish a **blood-air barrier** with the type I pneumocytes. **Premature infants born between week 25 and week 28 can survive with intensive care.** Adequate vascularization and surfactant levels are the most important factors for the survival of premature infants.
4. **Alveolar period (birth–8 years of age).** During this period, terminal sacs are partitioned by **secondary septae** to form adult **alveoli.** Approximately 20 to 70 million alveoli are present at birth. Approximately 300 to 400 million alveoli are present by 8 years of age. The major mechanism for the increase in the number of alveoli is formation of secondary septae that partition existing alveoli. After birth, the increase in the size of the lung is the result of an **increase in the number of respiratory bronchioles.** The diameter of the pulmonary acinus (all airways distal to the terminal bronchiole) is **1 to 2 mm** at birth and gradually increases to its adult diameter of **6 to 10 mm** by adolescence. On chest radiographs, lungs of a newborn infant are denser than those of an adult lung because of the fewer number of mature alveoli.

B. Clinical considerations of the lung *(Fig. 1-4)*

1. **Retained fluid syndrome (or "wet lung disease")** is caused by delayed resorption and clearance of lung fluid and is one of the most common causes of respiratory distress in the newborn. Clinical features include: tachypnea, nasal flaring, grunting, and retractions. Radiographic findings include: fluid within the lungs, parahilar radiating congestion, vascular markings with hazy borders, and fluid within fissures. The lungs generally begin to clear within 10 to 12 hours. **Aeration at birth** is the replacement of lung liquid with air in the newborn's lungs. In the fetal state, the functional residual capacity (FRC) of the lung is filled with liquid secreted by fetal lung epithelium (up to 500 mL/day) via Cl^- transport using **CFTR (cystic fibrosis transmembrane protein)** and **CIC-2 (volume-activated chloride channel.** At birth, lung liquid is eliminated by a reduction in lung liquid secretion via Na^+ transport (H_2O follows) by type II pneumocytes and resorption into pulmonary capillaries (major route) and lymphatics (minor route). Lungs of a stillborn baby will sink when placed in water because they contain fluid rather than air.
2. **Respiratory distress syndrome (RDS)** is caused by a deficiency or absence of **surfactant.** This surface active agent is composed of **cholesterol** (50%), **dipalmitoylphosphatidylcholine** (DPPC; 40%), and **surfactant proteins A, B, and C** (10%) and coats the inside of alveoli to maintain alveolar patency. RDS is prevalent in: premature infants (accounts for 50–70% of deaths in premature infants),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infants of diabetic mothers, infants who experienced fetal asphyxia or maternofetal hemorrhage (damages type II pneumocytes), and multiple birth infants. Clinical features include: dyspnea, tachypnea, inspiratory retractions of the chest wall, expiratory grunting, cyanosis, and nasal flaring. Treatments include: administration of betamethasone (a corticosteroid) to the mother for several days before delivery (i.e., antenatal) to increase surfactant production, postnatal administration of an artificial surfactant solution, and postnatal high-frequency ventilation. RDS in premature infants cannot be discussed without mentioning **germinal matrix hemorrhage (GMH).** The germinal matrix is the site of proliferation of neuronal and glial precursors in the developing brain, which is located above the caudate nucleus, in the floor of the lateral ventricles, and the caudothalamic groove. The germinal matrix also contains a rich network of fragile, thin-walled blood vessels. The brain of the premature infant lacks the ability to autoregulate the cerebral blood pressure. Consequently, increased arterial blood pressure in these blood vessels leads to rupture and hemorrhage into the germinal matrix. This leads to significant neurologic sequelae, including cerebral palsy, mental retardation, and seizures. Antenatal corticosteroid administration has a clear role in reducing the incidence of GMH in premature infants.

3. **Bronchopulmonary dysplasia (BPD)** occurs when infants with respiratory distress syndrome are placed on O_2 and positive-pressure ventilation. High concentrations of O_2 (i.e., O_2 toxicity) damage the basement membrane of pulmonary arterioles, leading to leaky lung syndrome. Positive-pressure ventilation causes mechanical damage leading to "bubbly lungs." The "bubbly lungs" result from hyperaeration of some alveoli and atelectasis of other alveoli. Radiographic findings include: haziness of blood vessel margins during week 1–2 of life which progresses to linear densities that persist into week 3–4 of life; gradual appearance of "bubbly lungs," whose appearance is quite variable but generally pronounced at the bases; some infants may develop large pneumatoceles. Long-term BPD leads to submucosal fibrosis of bronchi, septal fibrosis, chronic inflammation, and squamous metaplasia of terminal bronchioles and alveoli.
4. **Wilson-Mikity syndrome** is a condition very similar to BPD (see above) and indeed some clinicians consider them to be the same disease. Classic Wilson-Mikity syndrome occurs when infants with initially clear lungs (i.e., no apparent respiratory distress) develop "bubbly lungs."
5. **Leaky lung syndrome** occurs when infants with respiratory distress syndrome are placed on O_2 and positive-pressure ventilation. O_2 toxicity damages the basement

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FIGURE 1-4. (A) Retained fluid syndrome (RFS). AP radiograph shows pronounced bilateral parahilar infiltrates with some fluid in the minor fissure. **(B and C) Respiratory distress syndrome (RDS).** *Light micrograph.* The pathological hallmarks are acinar atelectasis (i.e., collapse of the respiratory acinus, which includes the respiratory bronchioles, alveolar ducts, and alveoli), dilation of terminal bronchioles (*), and deposition of an eosinophilic hyaline membrane material (*arrows*) that consists of fibrin and necrotic cells. *AP radiograph.* The radiological hallmarks are a bell-shaped thorax caused by underaeration and reticulogranularity of the lungs caused by acinar atelectasis. **(D and E) Bronchopulmonary dysplasia (BPD).** Light micrograph shows alternating areas of hyperaeration and atelectasis causing a "bubbly lung" appearance. AP radiograph shows predominately the "bubbly lung" appearance along with two large pneumatoceles (*arrows*). **(F) Leaky lung syndrome.** AP radiograph shows that the lungs are opaque and hazy. The lungs are larger in size as a result of the edema than when the respiratory distress occurred initially. **(G and H) Pulmonary air leaks.** AP radiograph shows a typical salt-and-pepper appearance of pulmonary interstitial emphysema (PIE) as a result of the radiolucent air surrounding bronchovascular sheaths. Note the left chest tube that was placed to treat the pneumothorax. Light micrograph shows the escaped air (arrows) surrounding the bronchovascular sheath. A—pulmonary arteriole; B—bronchiole. **(I) Unilateral pulmonary agenesis.** AP radiograph shows complete absence of the left lung with a shift of the mediastinum to the left hemithorax. In addition, the right lung shows hypervascularity and overdistension. **(J) Unilateral pulmonary hypoplasia.** AP radiograph shows decreased size of the left lung with a shift of the mediastinum to the left hemithorax. In addition, the right lung shows hypervascularity. **(K) Congenital diaphragmatic hernia.** aP radiograph shows air-filled stomach (*S*) and air-filled loops of intestine in the left hemithorax with a shift of the mediastinum to the right hemithorax. Note the tip of the nasogastric tube is in the distal esophagus (*arrow*). **(L) Meconium aspiration syndrome.** Light micrograph shows meconium with a large number of epithelial squames within the terminal airways and alveoli.