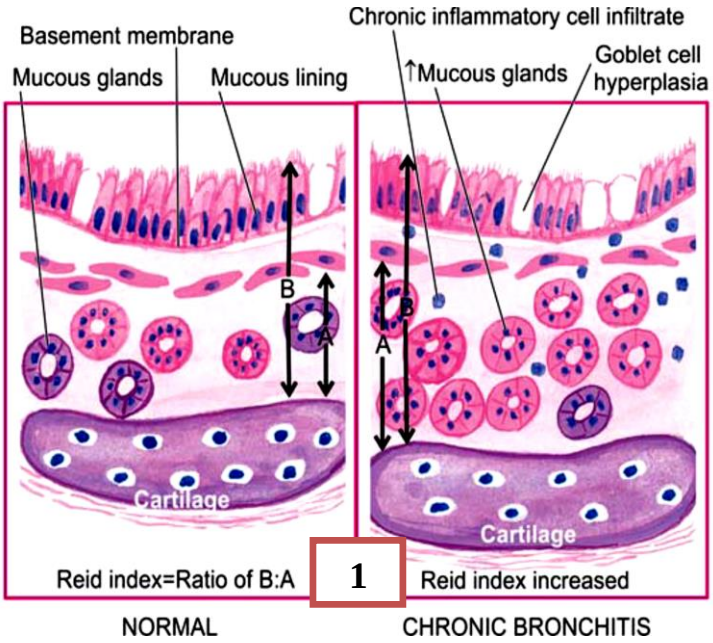
Chronic Bronchitic & Pulmonary Embolism

CHRONIC BRONCHITIS:

❖ **Pathogenesis:** The two most important factors responsible for the majority of cases of chronic bronchitis are: cigarette smoking and atmospheric pollution. Other contributory factors are occupation[16], infection[18,19], familial and genetic factors.

❖ **Morphologic Features:**

Gross section: The bronchial wall is thickened, hyperaemic and oedematous. Lumina of the bronchi and bronchioles may contain mucus plugs and purulent exudate.



Microscopic changes: Histologic define is an increased Reid index. *Reid index* is the ratio between the thickness of the submucosal mucous glands (i.e. hypertrophy and hyperplasia) in the cartilage-containing large airways for the total bronchial wall **(Fig. 1)**. The bronchial epithelium may show squamous metaplasia and dysplasia. There is little chronic inflammatory cell infiltrate. The non-cartilage containing small airways show goblet cell hyperplasia.

Three factors contribute to the increased thickness of the bronchial wall:

1. Infiltration of the submucosa by chronic inflammatory cells (**In**).
2. Marked hypertrophy of mucosal smooth muscle (**M**).
3. Marked hyperplasia of the mucous glands (**G**). with production mucus. **Slide 2**

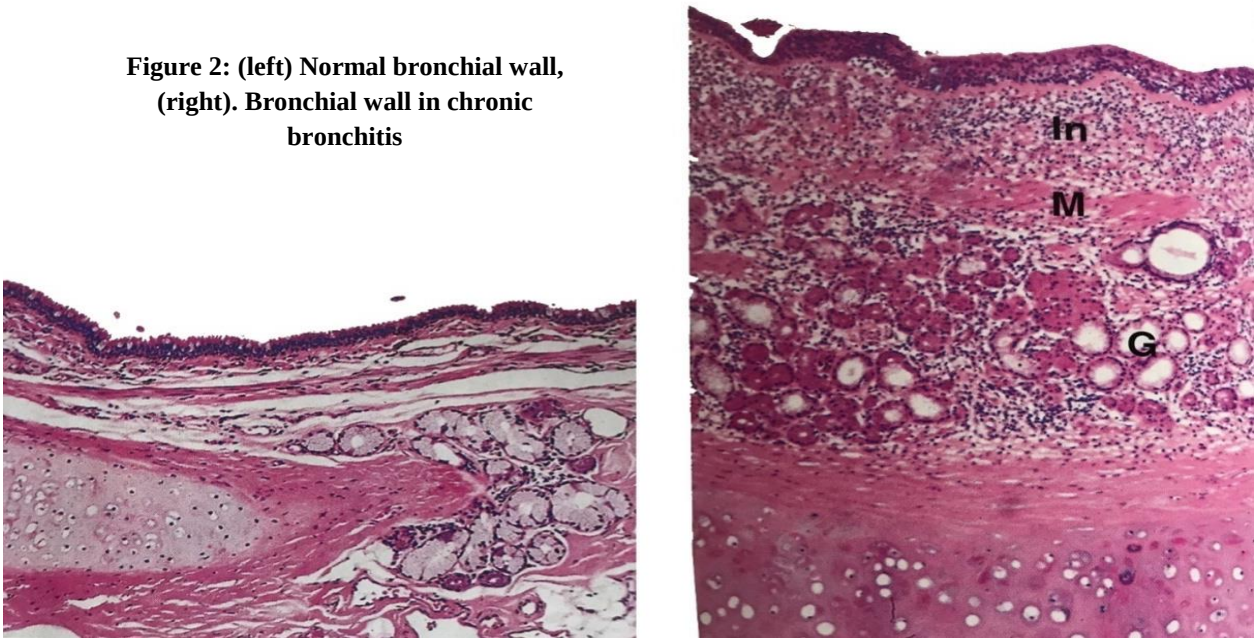
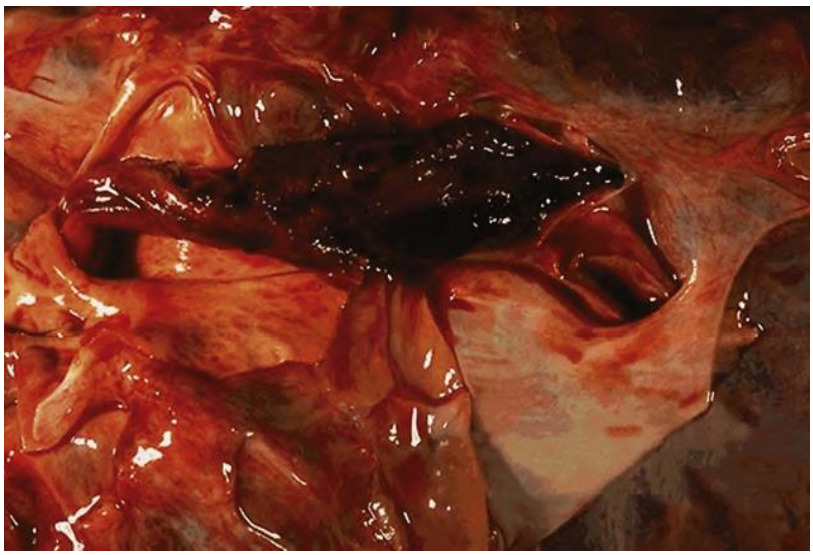


Figure 2: (left) Normal bronchial wall, (right). Bronchial wall in chronic bronchitis

Pulmonary Embolism

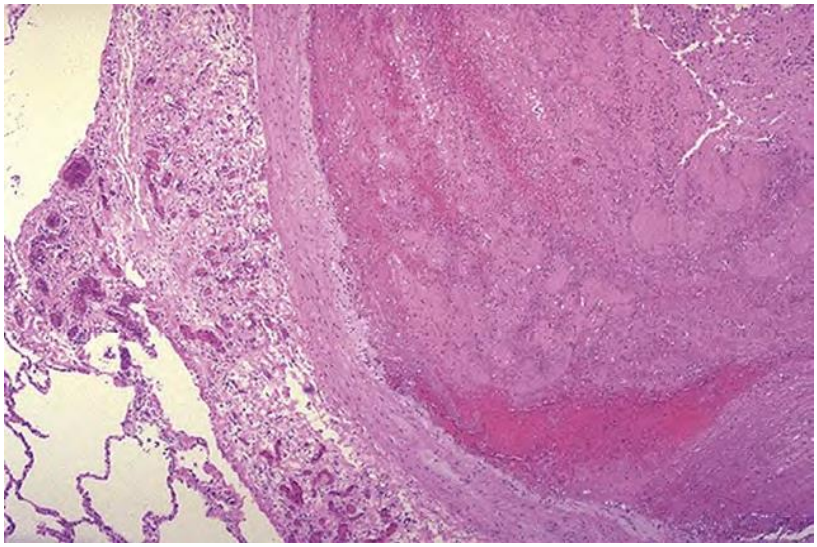
Embolism is the process of partial or complete obstruction of some part of the cardiovascular system by any mass carried in the circulation; the transported intravascular mass detached from its site of origin is called an *embolus*. Usually (90%) emboli are thromboembolic i.e. originating from thrombi or their parts from the vessel wall.

❖ **Pulmonary embolism**: is the most common and fatal form of venous thromboembolism. In contrast, pulmonary thrombosis is uncommon and may occur in pulmonary atherosclerosis and pulmonary hypertension. Risks for pulmonary thromboembolism are prolonged immobilization, advanced age, and hypercoagulable states.



- **Gross:** Here is a saddle embolus that bridges the pulmonary artery trunk as it divides into the right (➡) and left (*) main pulmonary arteries. There are pale areas mixed with dark-red areas.

Most large pulmonary thromboemboli originate within large deep veins of the lower extremities.



- **Microscopically:** Within this pulmonary artery are interdigitating areas of pale pink and red that form the lines of Zahn (⟵) characteristic of a thrombus. These lines act as layers of RBCs, platelets, and fibrin that are laid down as the thrombus forms within a vein. Here the thrombus has become a thromboembolus that has travelled up the inferior vena cava and the right side of the heart to become packed into a pulmonary artery branch. Over time, if the patient survives, the thromboembolus can undergo organization and dissolution.

In **slide 3** is a small peripheral pulmonary artery thromboembolus in the region of a hemorrhagic infarct. There is partial recanalization (♦) of this blocked artery. Such a small embolus probably would not cause dyspnea or pain, unless there were many emboli.