Basic Pulmonary Mechanics

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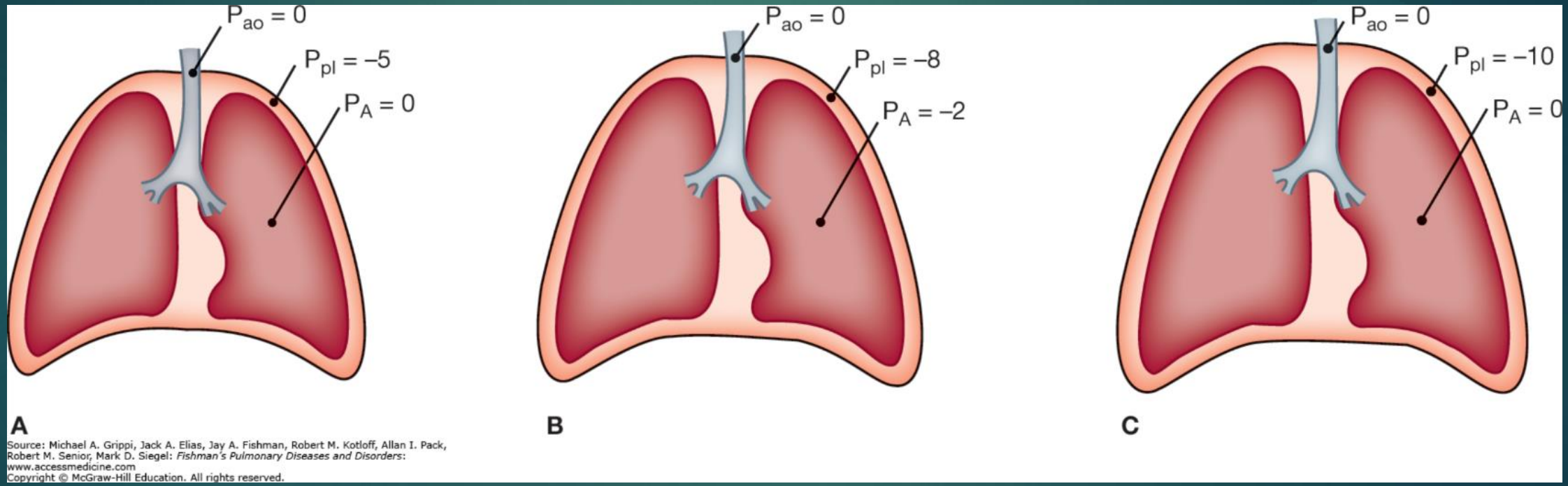

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Table 10-1

Lung Volumes and Subdivisions

The *functional residual capacity* (FRC) is the volume of air that remains in the lungs at the end of a normal expiration.
The *tidal volume* (TV) is the volume of air that is drawn into the lungs during inspiration from the end-expiratory position (and also leaves the lungs passively during expiration) in the course of quiet breathing.
The *expiratory reserve volume* (ERV) is the maximum volume of air that can be forcibly exhaled after a quiet expiration has been completed (i.e., from the end-expiratory position).
The *residual volume* (RV) is the volume of air that remains in the lungs after a maximal expiratory effort.
The *inspiratory capacity* (IC) is the maximum volume of air that can be inhaled from the end-expiratory position. It consists of two subdivisions: tidal volume and the *inspiratory reserve volume* (IRV).
The *total lung capacity* (TLC) is the total volume of air contained in the lungs at the end of a maximum inspiration.
The *vital capacity* (VC) is the volume of air that is exhaled by a maximum expiration after a maximum inspiration.

To assess the elastic properties of the ventilatory apparatus, it is expedient to evaluate the elastic properties of the lungs and chest separately. Elastic properties are conventionally assessed over a fixed range of volumes during periods of arrested airflow.

$$C = \frac{\Delta V_L}{\Delta(P_A - P_{pl})}$$

C = lung compliance
$\Delta(P_A - P_{pl})$ = change in transpulmonary pressure
P_A = alveolar pressure and P_{pl} = pleural pressure
ΔV_L = change in lung volume

The change in transpulmonary pressure required to effect a given change in the volume of air in the lungs is a measure of the distensibility, or compliance, of the lungs. Pulmonary compliance is calculated as the ratio of the change in lung volume to the change in transpulmonary pressure—that is,

Compliance

- Distensibility, ease of stretch
- Inverse = elastance (stiffness)
- Pressure volume characterstics are non linear
  - Volume increases, elastic elements approach limit of distensibility, more pressure increases produce smaller changes
- Elasticity favors relaxation of lung tissue and return to expiration

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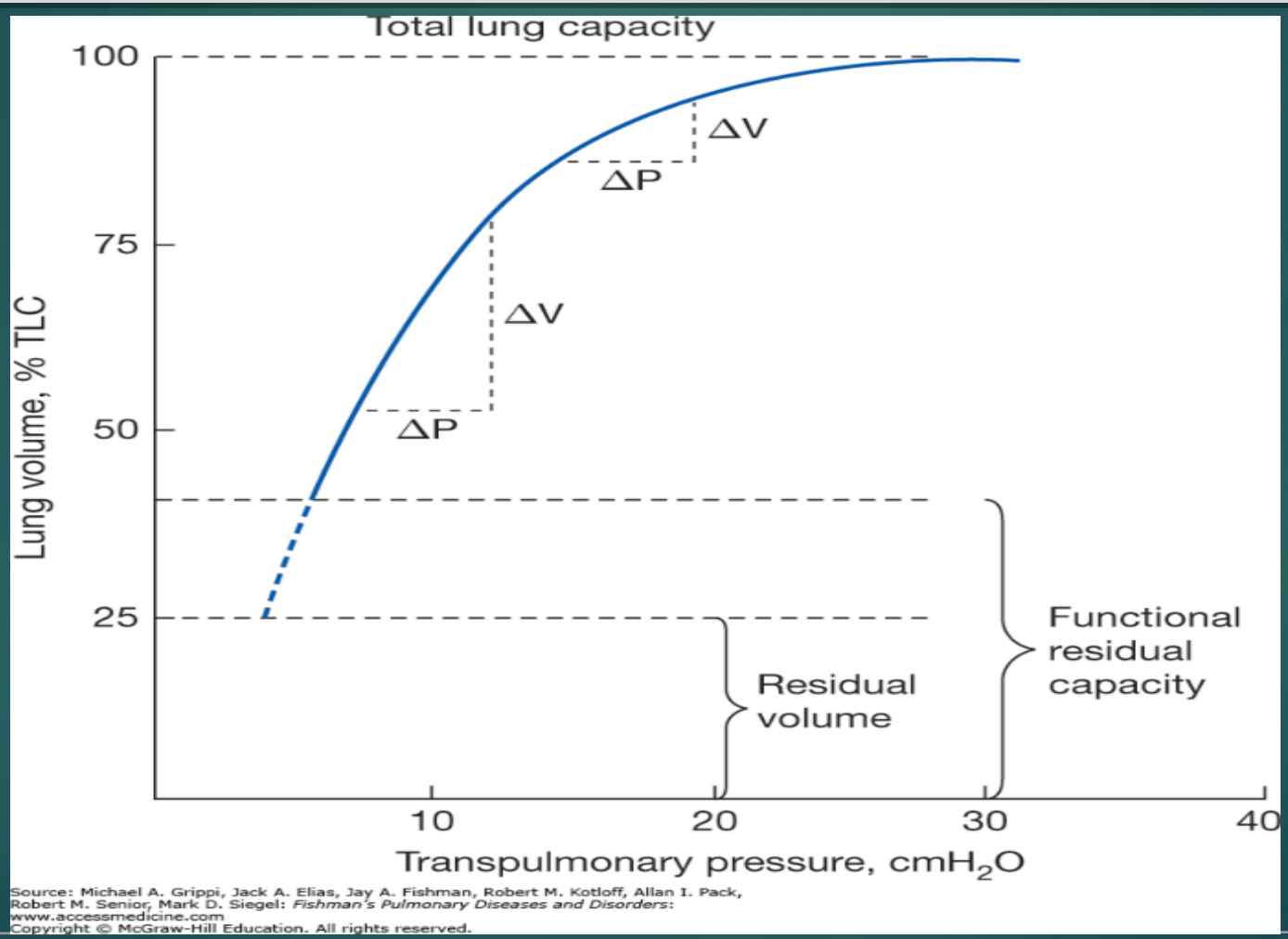

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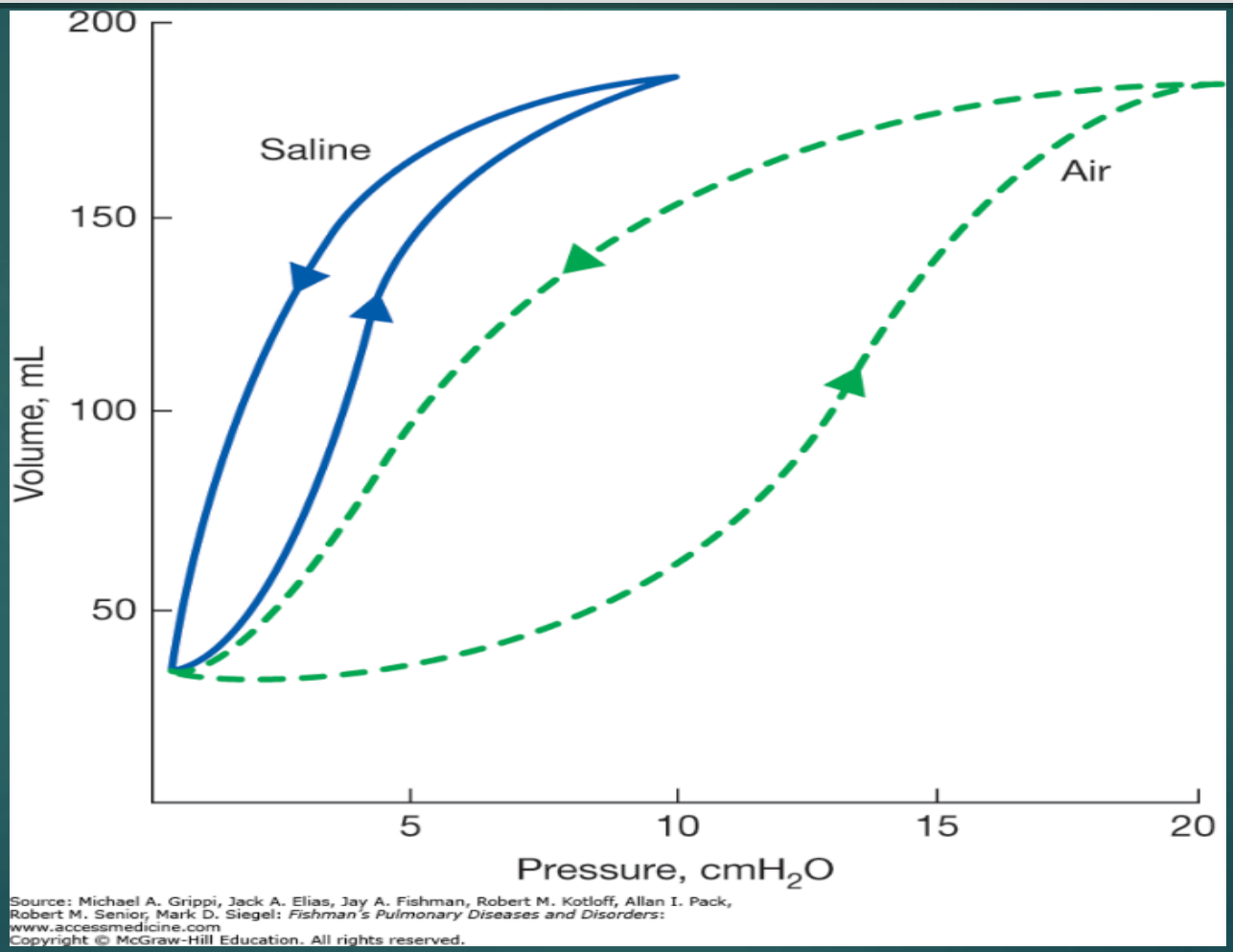

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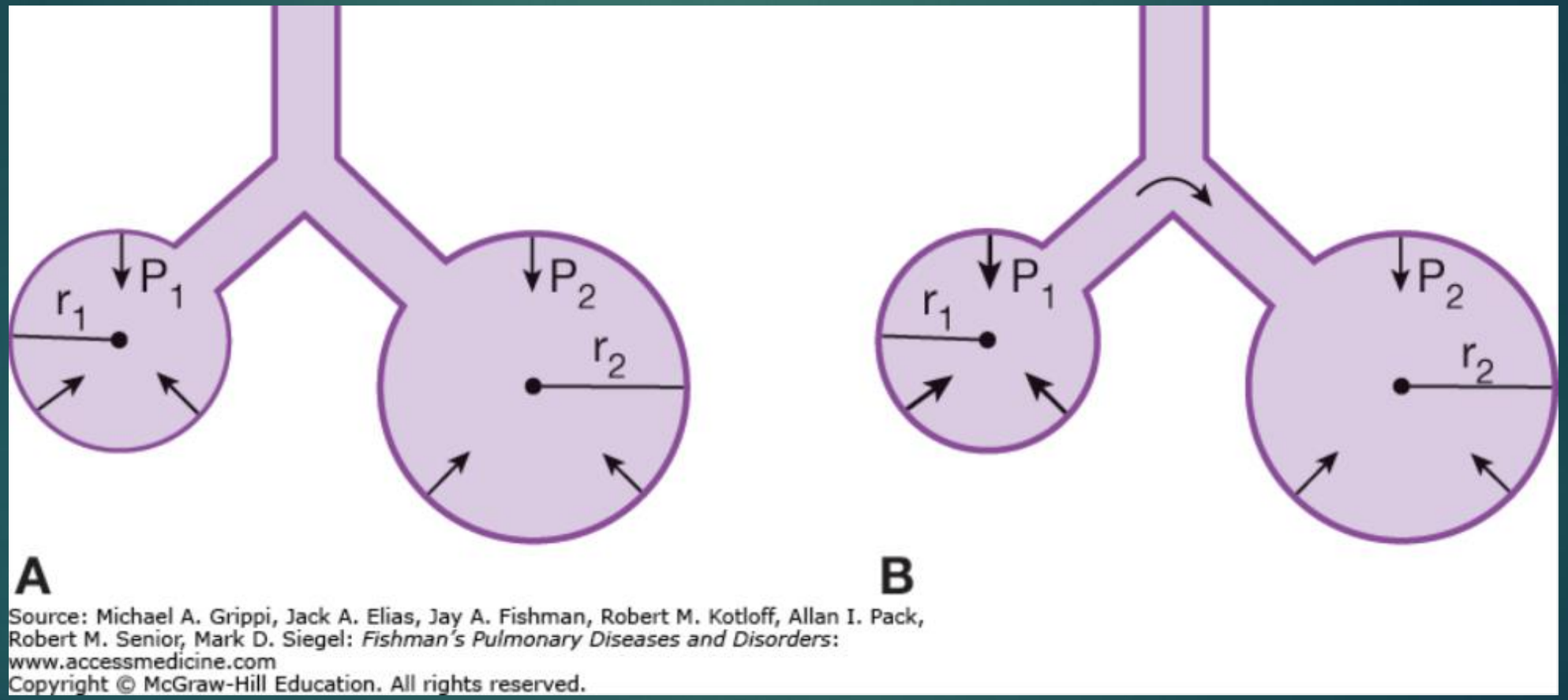

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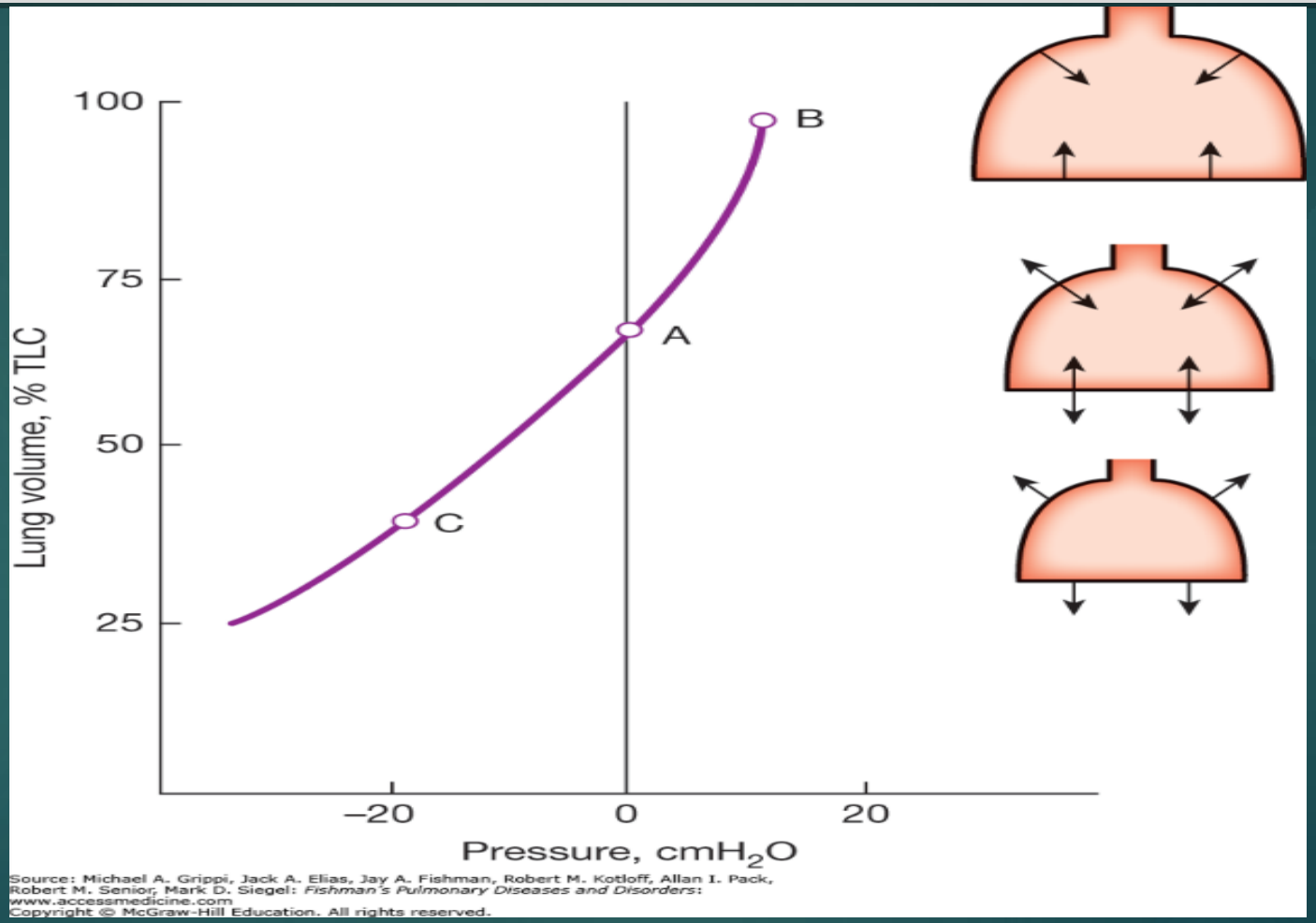

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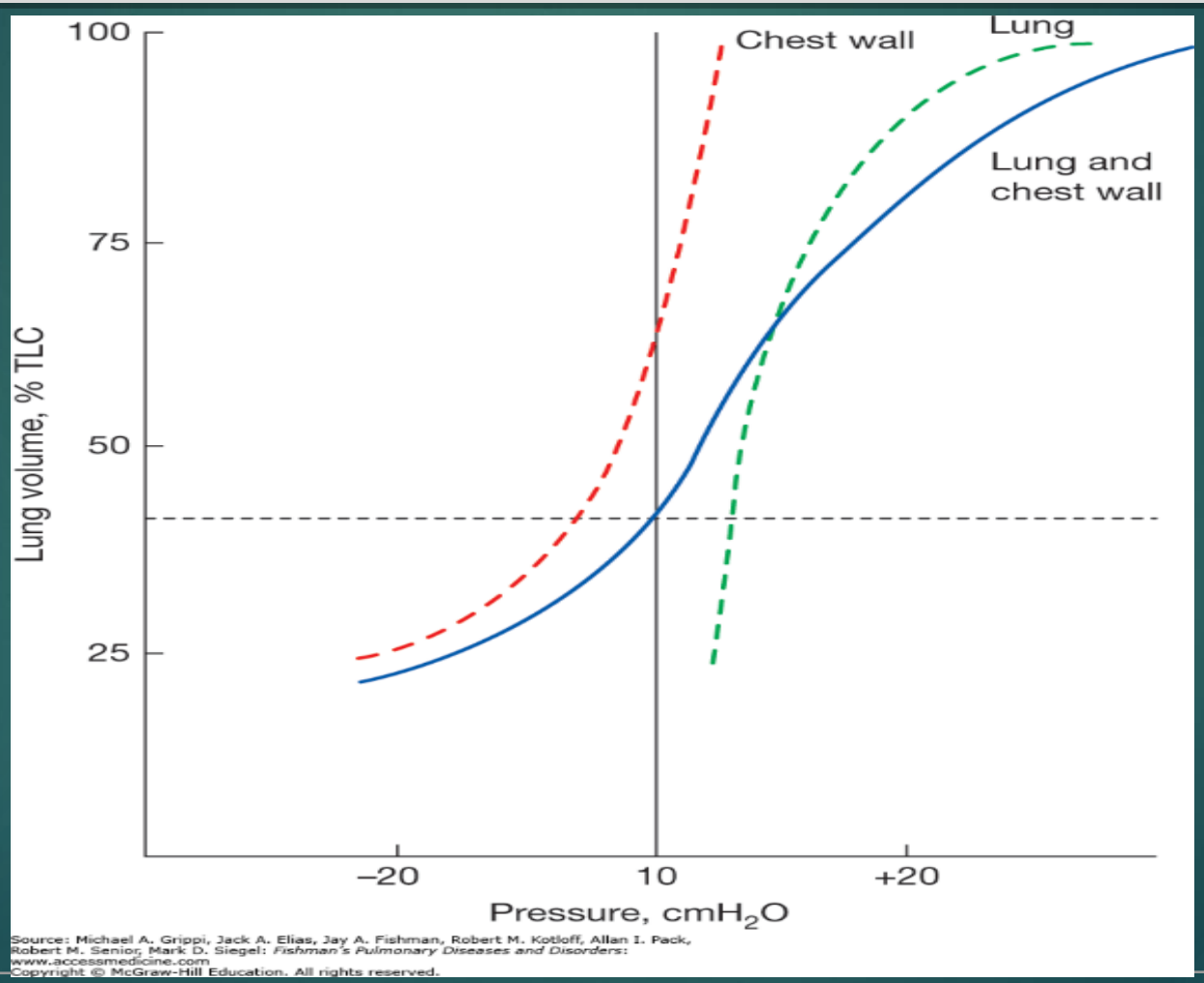

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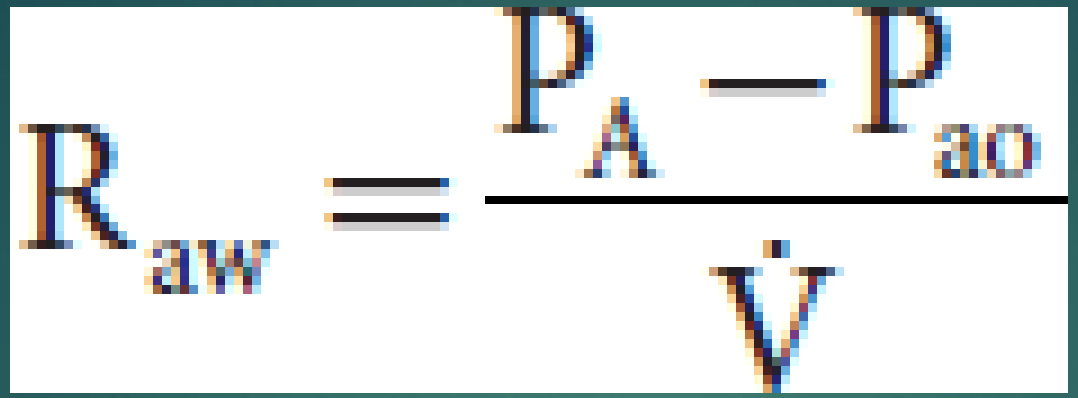
$R_{aw} = \frac{P_A - P_{ao}}{\dot{V}}$

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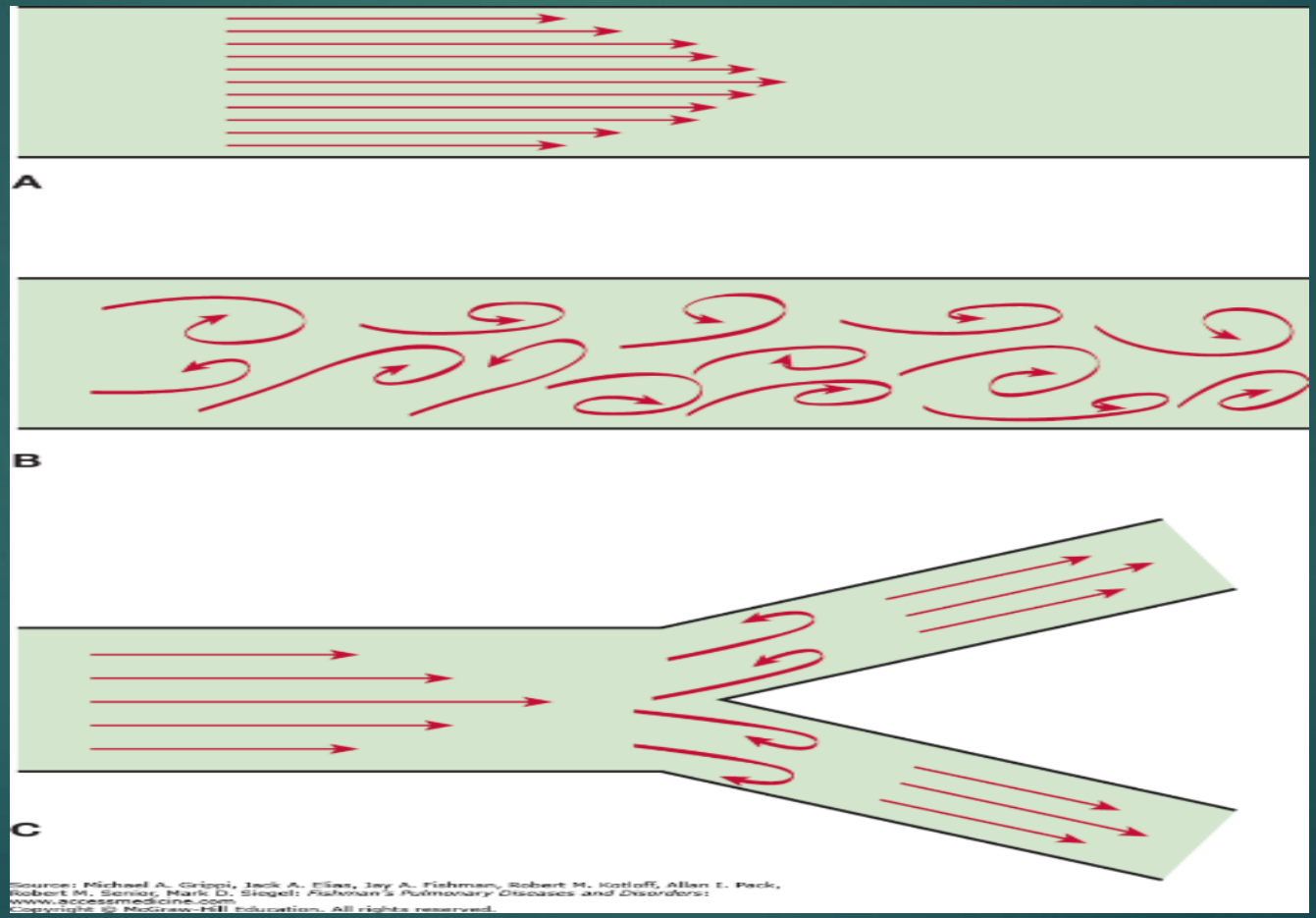

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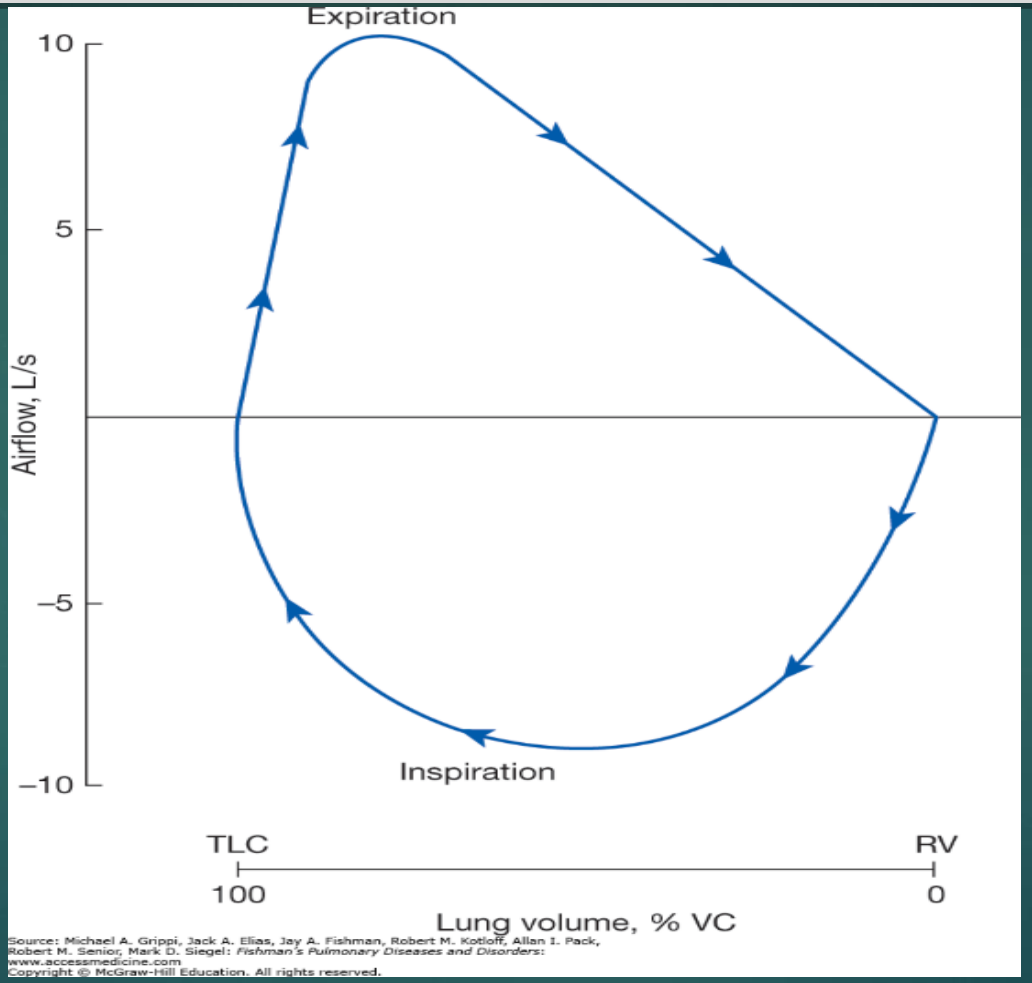

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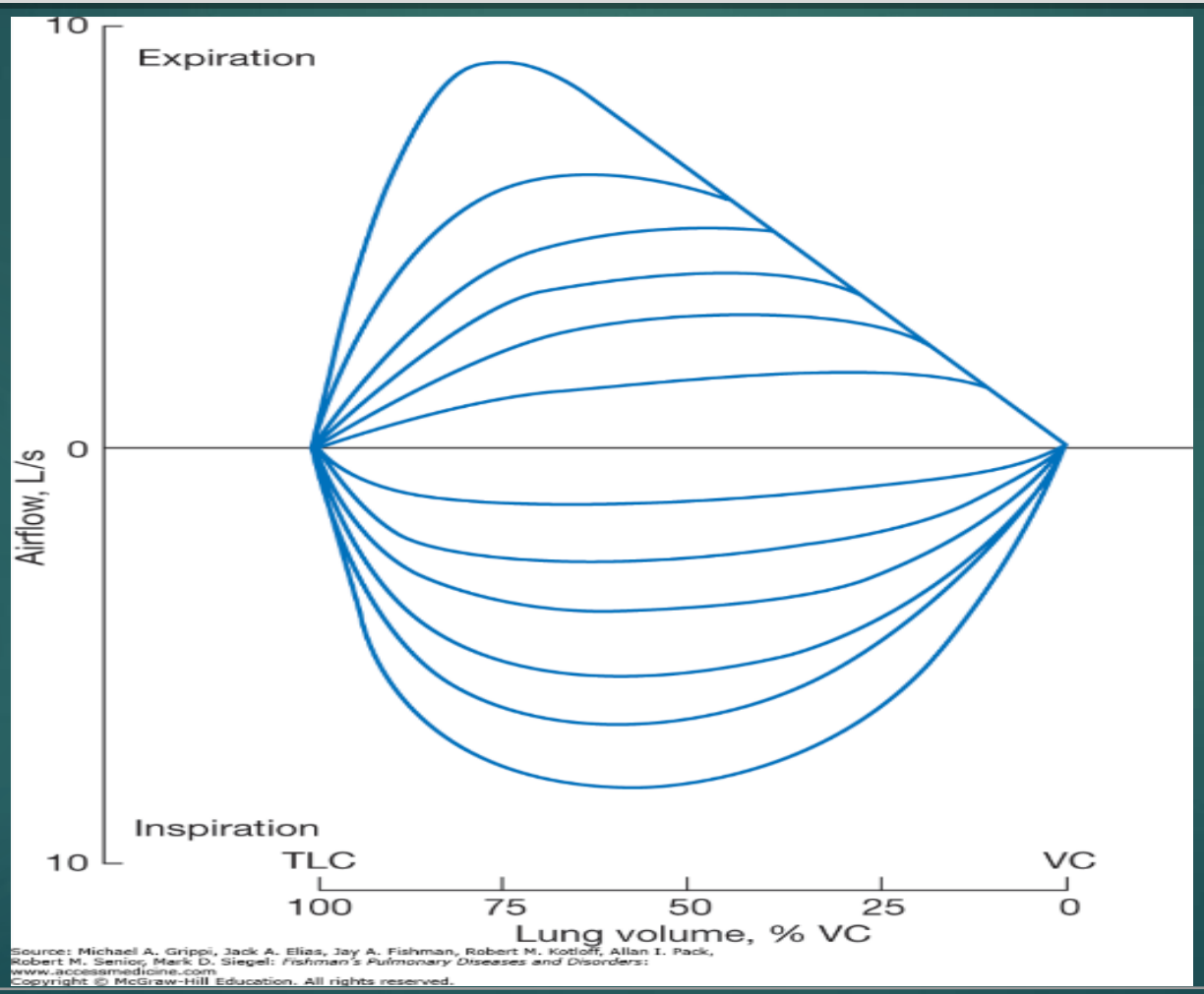

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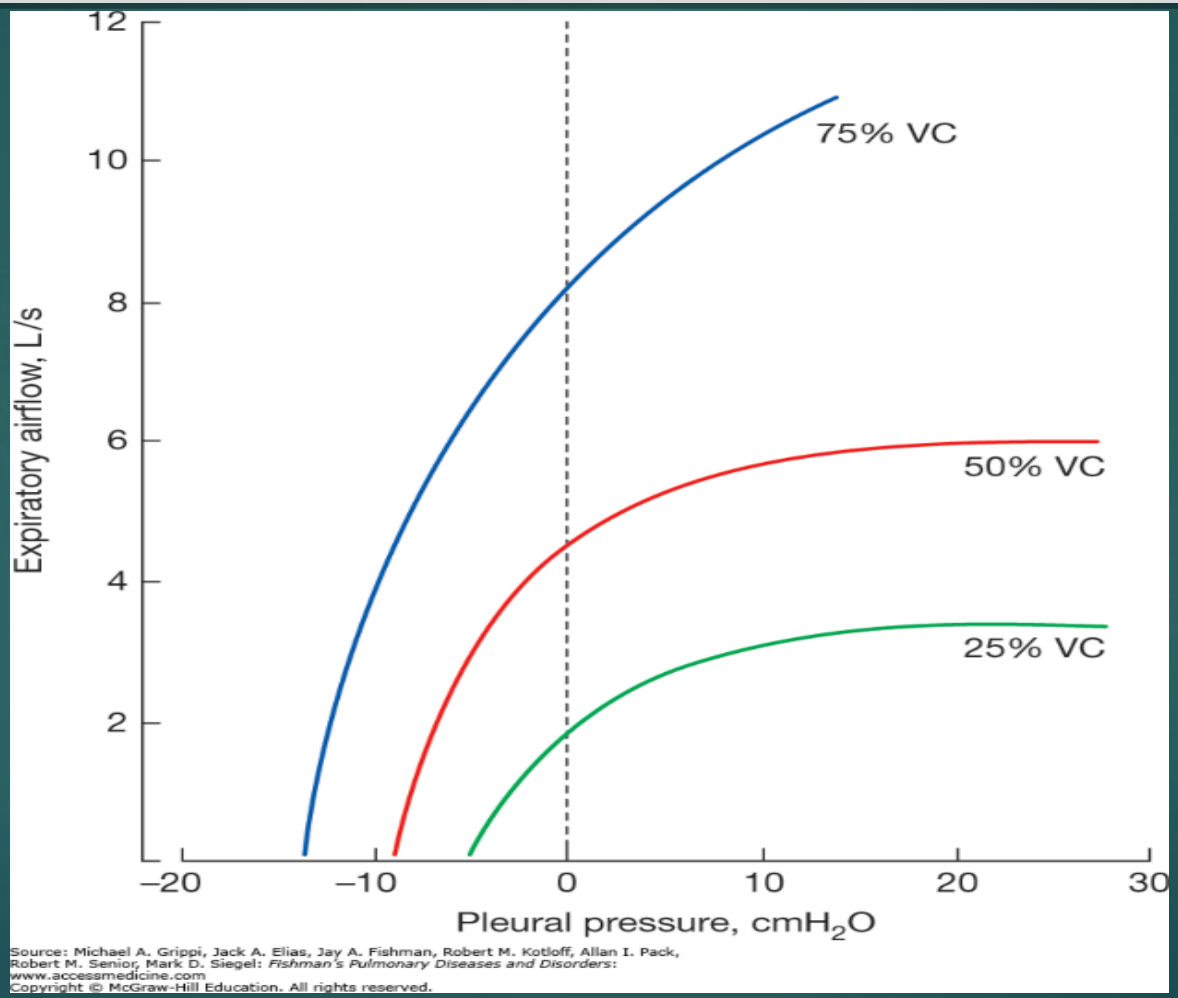

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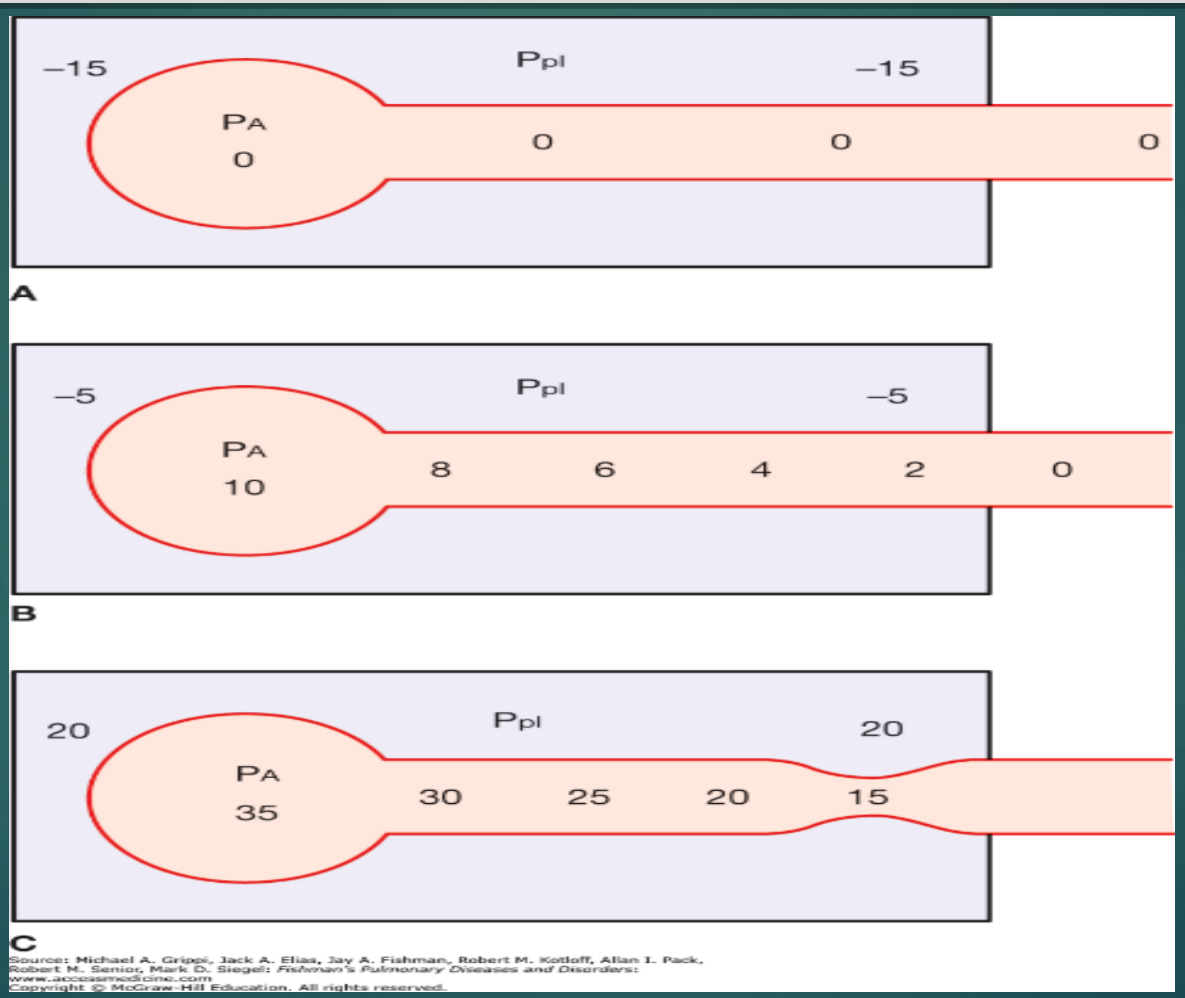

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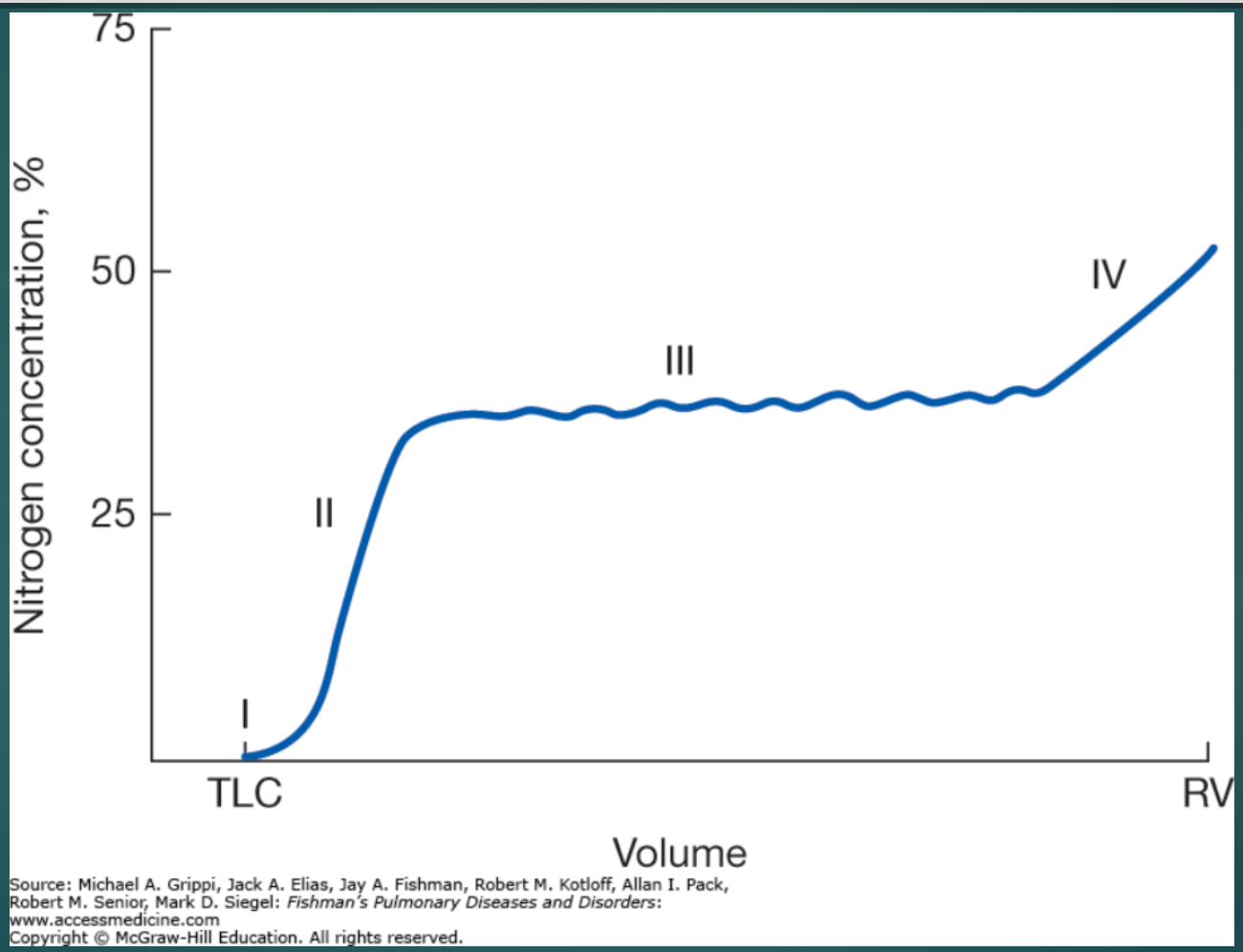

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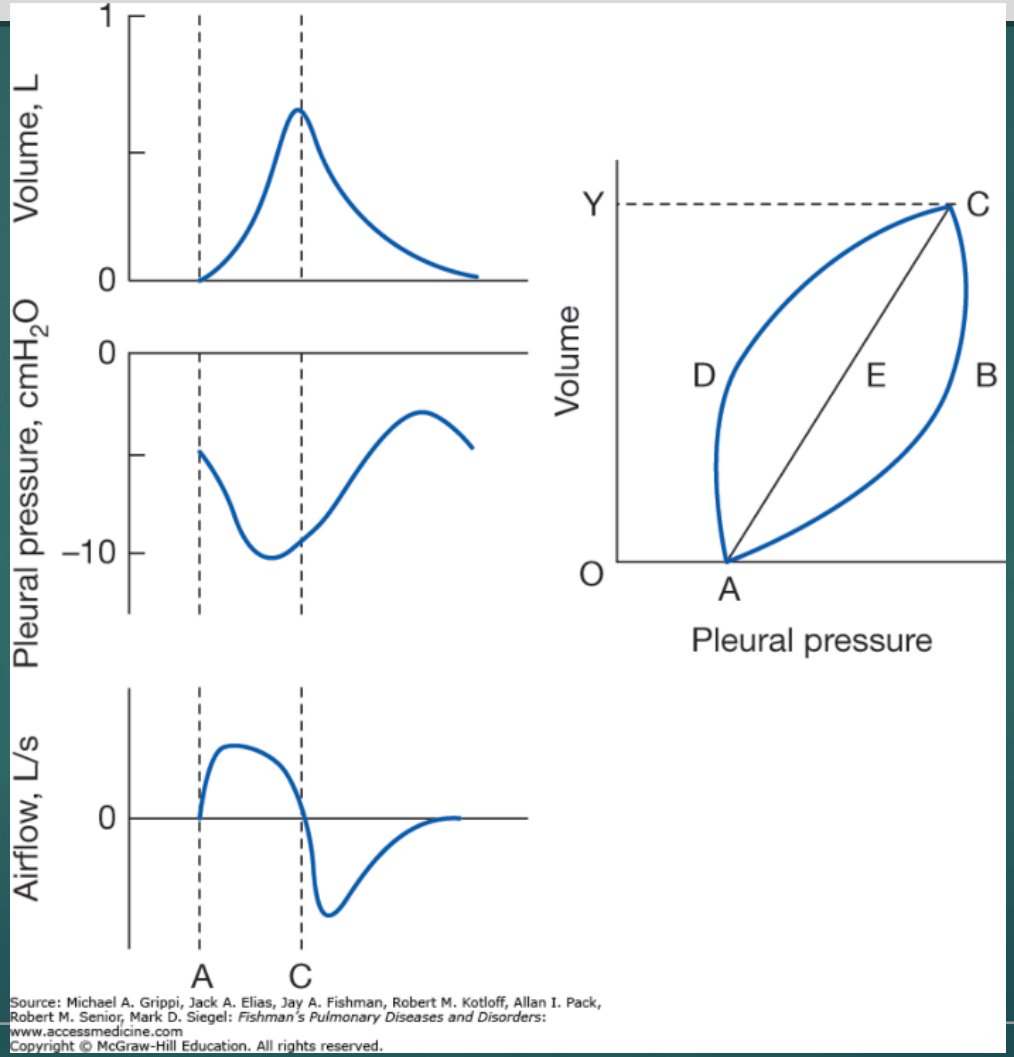

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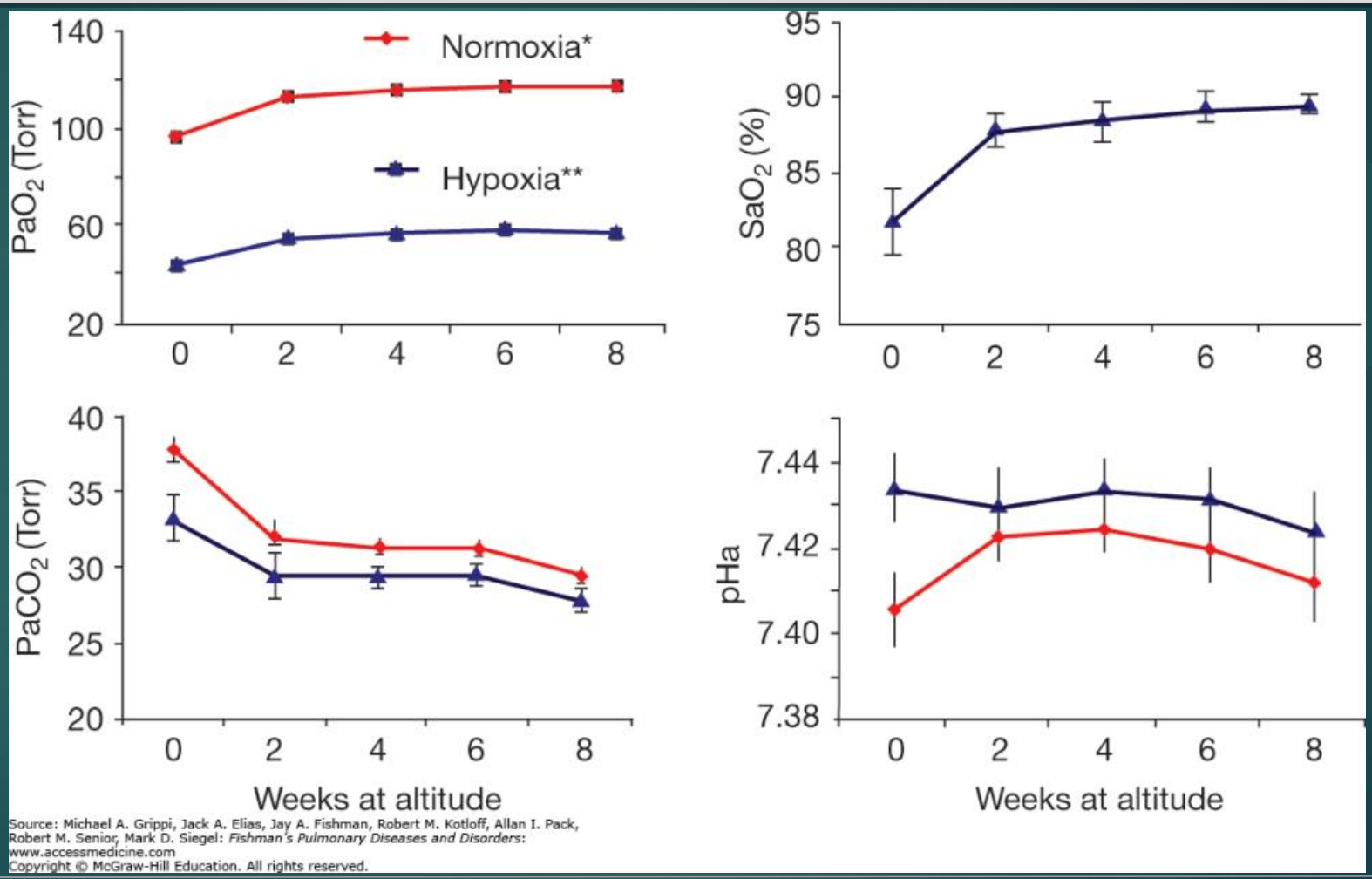

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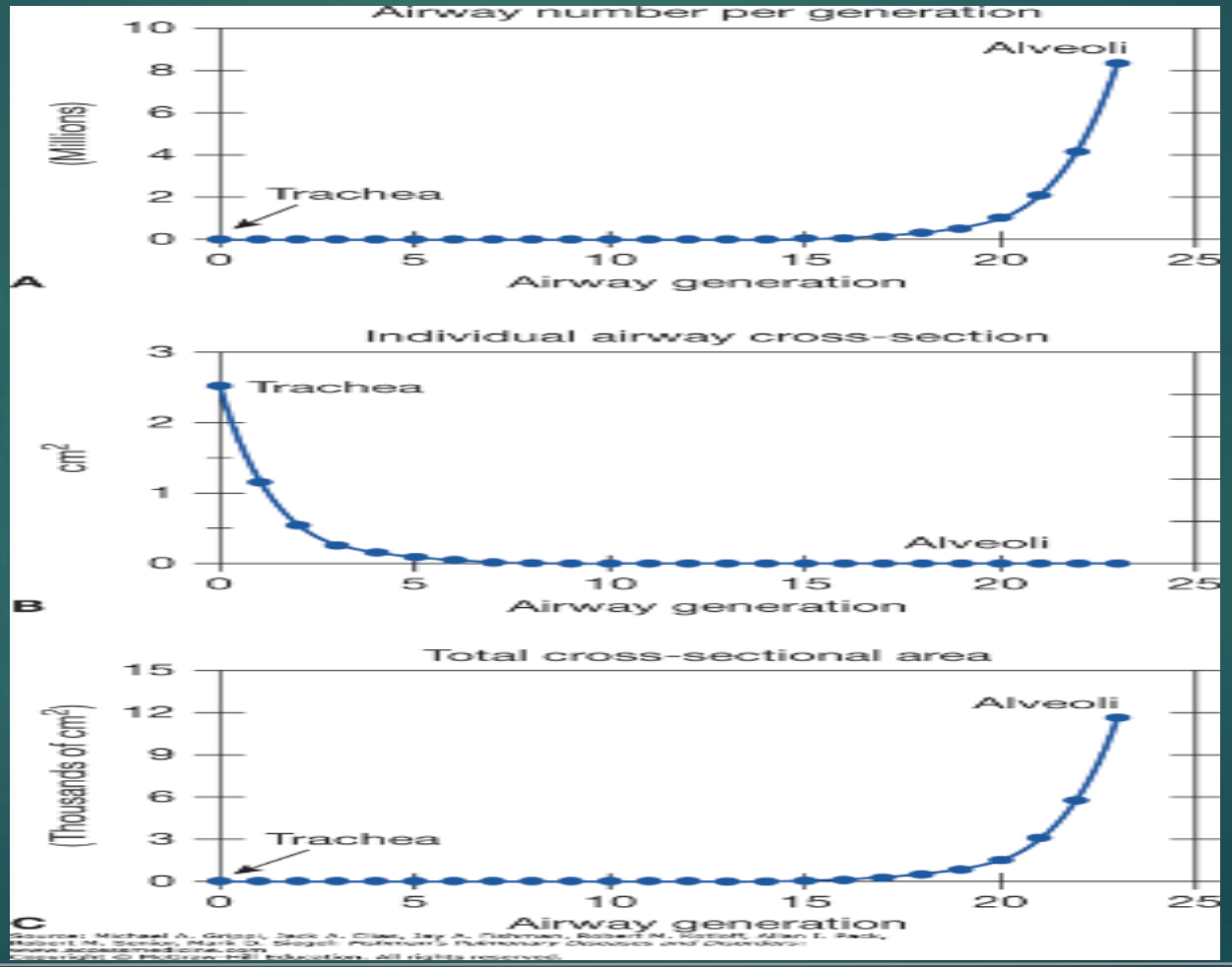

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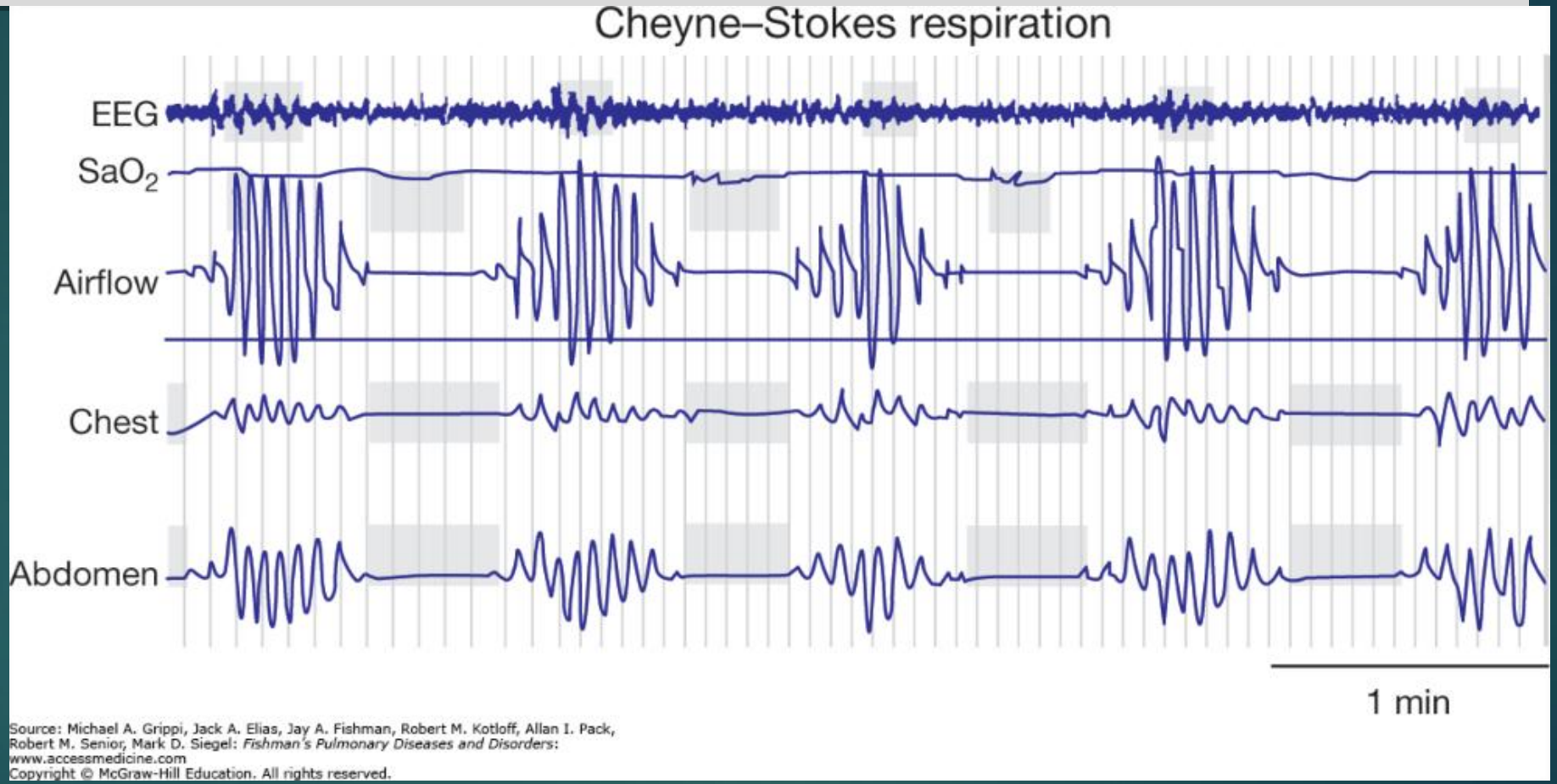

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Four principal potential mechanisms of failure of the O_2 transport pathway can lead to a reduced arterial P_{O_2} (i.e., to arterial hypoxemia):

1. Hypoventilation
2. Diffusion limitation
3. Shunt
4. Ventilation–perfusion ($\dot{V}A/\dot{Q}$) inequality.

Hypoventilation

Normal levels of ventilation produce a tightly regulated arterial P_{CO_2} at 40 ± 2 mm Hg in normal subjects with several control systems in place to ensure this (for details, see Chapter 11). However, if overall ventilation is reduced for any reason, alveolar P_{CO_2} (PA_{CO_2}), and therefore arterial P_{CO_2}, must rise to maintain constant the elimination of metabolically produced CO_2. Reciprocally, alveolar P_{O_2} (PA_{O_2}), and hence arterial P_{O_2}, will fall (and by relatively similar amounts as P_{CO_2} will rise). The alveolar gas equation[8] quantitatively relates PA_{O_2} and PA_{CO_2}, and is used to calculate how much PA_{O_2} will change for a change in PA_{CO_2}:

$$PA_{O_2} = PI_{O_2} - \frac{Pa_{CO_2}}{R} + PA_{CO_2} \cdot FI_{O_2} \cdot \frac{(1-R)}{R} \qquad (1)$$

PI_{O_2} and FI_{O_2} are inspired O_2 partial pressure and fractional concentration, respectively, and R is the respiratory exchange ratio, normally 0.8.

Hypoventilation represents a failure of step 1 of the gas transport pathway (see above) and can occur for several reasons: (1) the control centers in the nervous system that regulate ventilation could malfunction due to trauma, diseases, drugs, or anesthetics; (2) there could be neuronal or neuromuscular dysfunction of the nerves supplying the chest wall muscles of respiration; (3) the chest wall muscles could be fatigued, damaged, or paralyzed; or (4) the airways or chest wall could be disrupted from trauma or other mechanical derangement such as compression, or in the case of airways, obstruction.

Conceptually this type of problem is usually thought of as a whole-lung issue, usually with obvious causes, and can be reversed by recognizing the cause and taking appropriate reparative and/or ventilatory supportive steps.

Diffusion Limitation

Whereas diffusive transport plays a recognizable, if small, role within the airways and alveolar gas (see above), the concept of diffusive limitation affecting arterial P_{O_2} is more usually associated with transport step 6 – diffusion of O_2 from alveolar gas into the capillary and red cell.

This topic is specifically the focus of Chapter 16 and is not dealt with here. Indeed, the ensuing discussion of other factors sets aside diffusion limitation of O_2 transport for the sake of simplicity and assumes that the diffusive exchange of O_2 (and CO_2) between alveolar gas and capillary blood proceeds to completion within a single red cell's passage through the pulmonary microcirculation. This is reasonable under most conditions. Diffusion limitation in health is seen at sea level in some but not all athletes[9] but only at or near maximal exercise. It is universally seen in normal subjects exercising at altitude.[10,11]

Shunt

A shunt is a blood pathway that does not allow any contact between alveolar gas and red cells, so that no gas exchange occurs in the affected region. Consequently, blood passes through a shunt maintaining a mixed venous blood composition. When this blood reaches pulmonary veins, the left atrium and eventually arterial blood, it mixes with other blood that has undergone alveolar gas exchange. The result is a fall in arterial P_{O_2} and potentially an increase in arterial P_{CO_2} (arterial P_{CO_2} may not increase if the patient raises his or her level of ventilation, but hypoxemia will persist).

Classical pathophysiological scenarios giving rise to shunts are: (1) pulmonary edema, which fills alveoli with fluid, thereby abolishing their ventilation and any gas exchange; (2) alveolar filling with cellular and micro-organismal debris as in pneumonia, with the same result as in edema; (3) collapse of a region of lung due to pneumothorax, gas absorption distal to a fully obstructed airway, or to external compression; (4) rarely, the presence of abnormal arteriovenous vascular channels in the lungs, that can occur in, for example, hepatic cirrhosis; and (5) direct right-to-left vascular communications at the level of the heart or great (extrapulmonary) blood vessels.

Ventilation–Perfusion ($\dot{V}_A/\dot{Q}$) Inequality

The exquisite and complex branching architecture of the airways and of the blood vessels makes the lungs very susceptible to the potential problem of nonuniform distribution of alveolar ventilation and of pulmonary blood flow. Whenever alveoli are ventilated at less than average rates, for example if their feeding airways become partially obstructed for any reason, the ratio of ventilation to blood flow ($\dot{V}_A/\dot{Q}$ ratio) will fall (assuming their blood flow does not fall similarly). In certain other conditions, lung regions may suffer a reduction in local blood flow rather than ventilation, so that the $\dot{V}_A/\dot{Q}$ ratio rises above the average value in those areas.

Whenever there is a range of $\dot{V}_A/\dot{Q}$ ratios in a lung such that the $\dot{V}_A/\dot{Q}$ ratio is not identical everywhere, it is said that $\dot{V}_A/\dot{Q}$ inequality exists. The pathological cause of $\dot{V}_A/\dot{Q}$ inequality does not matter in this definition, nor whether the problem originates in the airways or blood vessels. The principal concept is that, compared to a lung having the same total alveolar ventilation and blood flow, a lung that has $\dot{V}_A/\dot{Q}$ inequality will exchange (all) gases in an inefficient manner.[12] The result is hypoxemia and, potentially, hypercapnia (raised arterial P_{CO_2}). A large section of this chapter presents the physiological reasons for this effect of $\dot{V}_A/\dot{Q}$ inequality.

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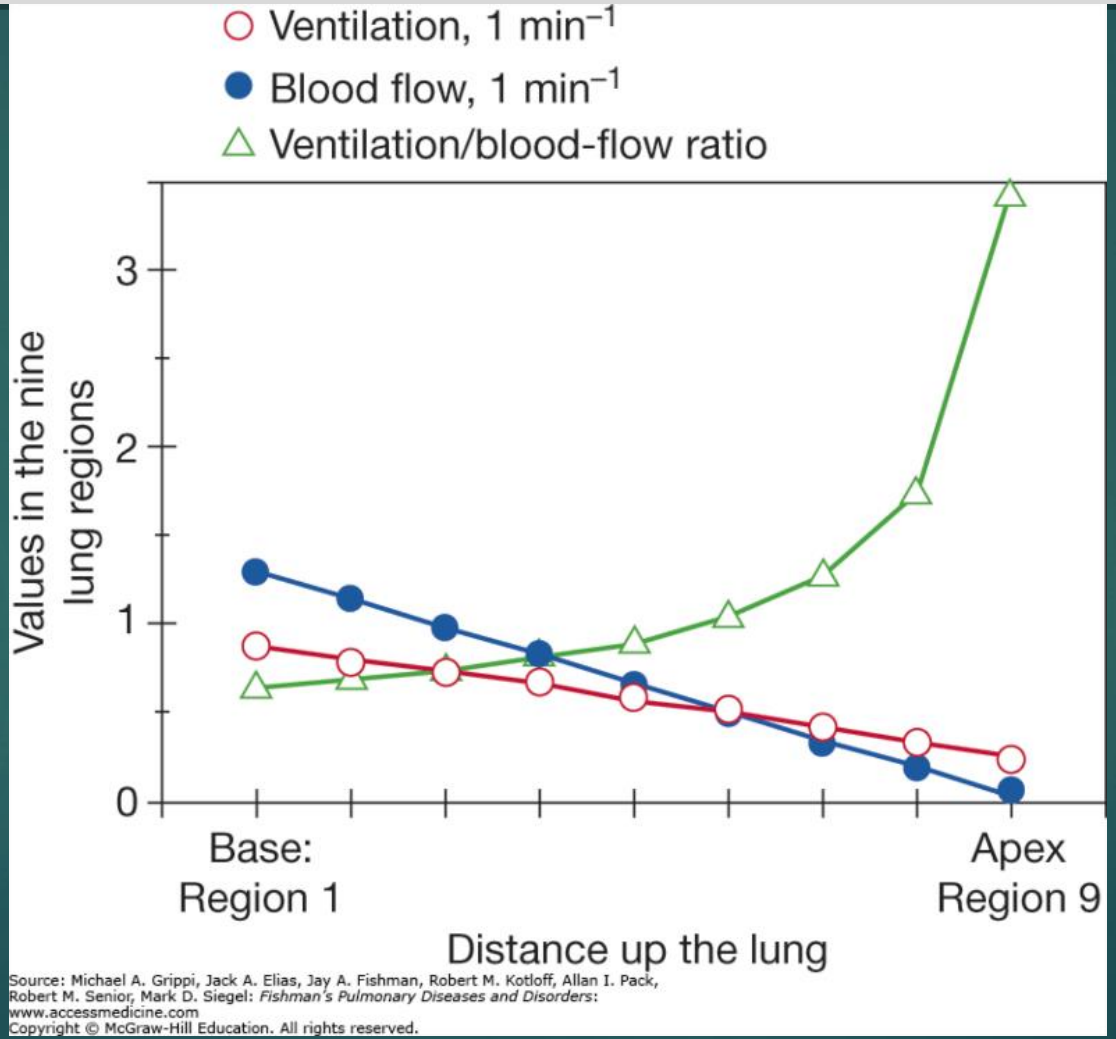

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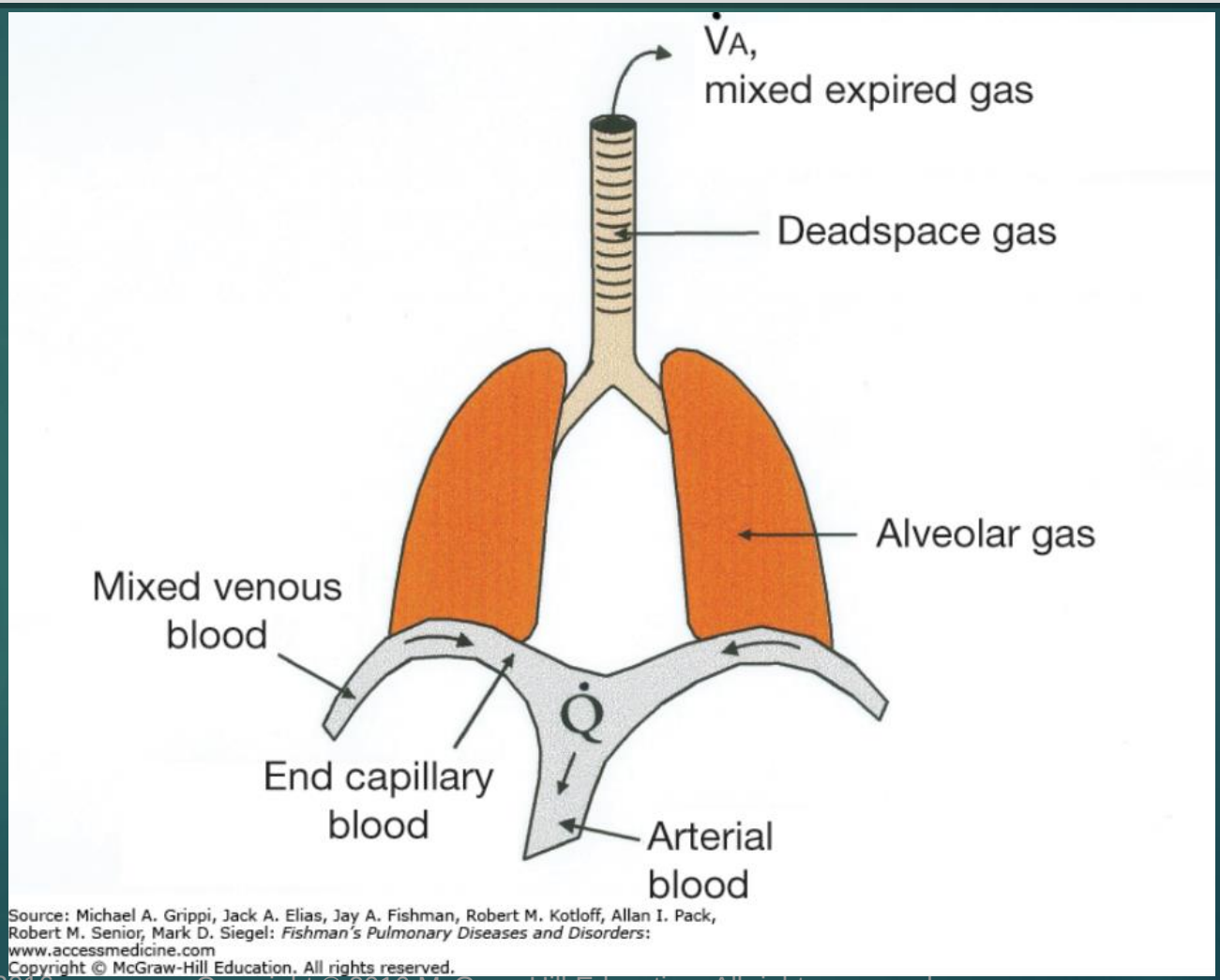

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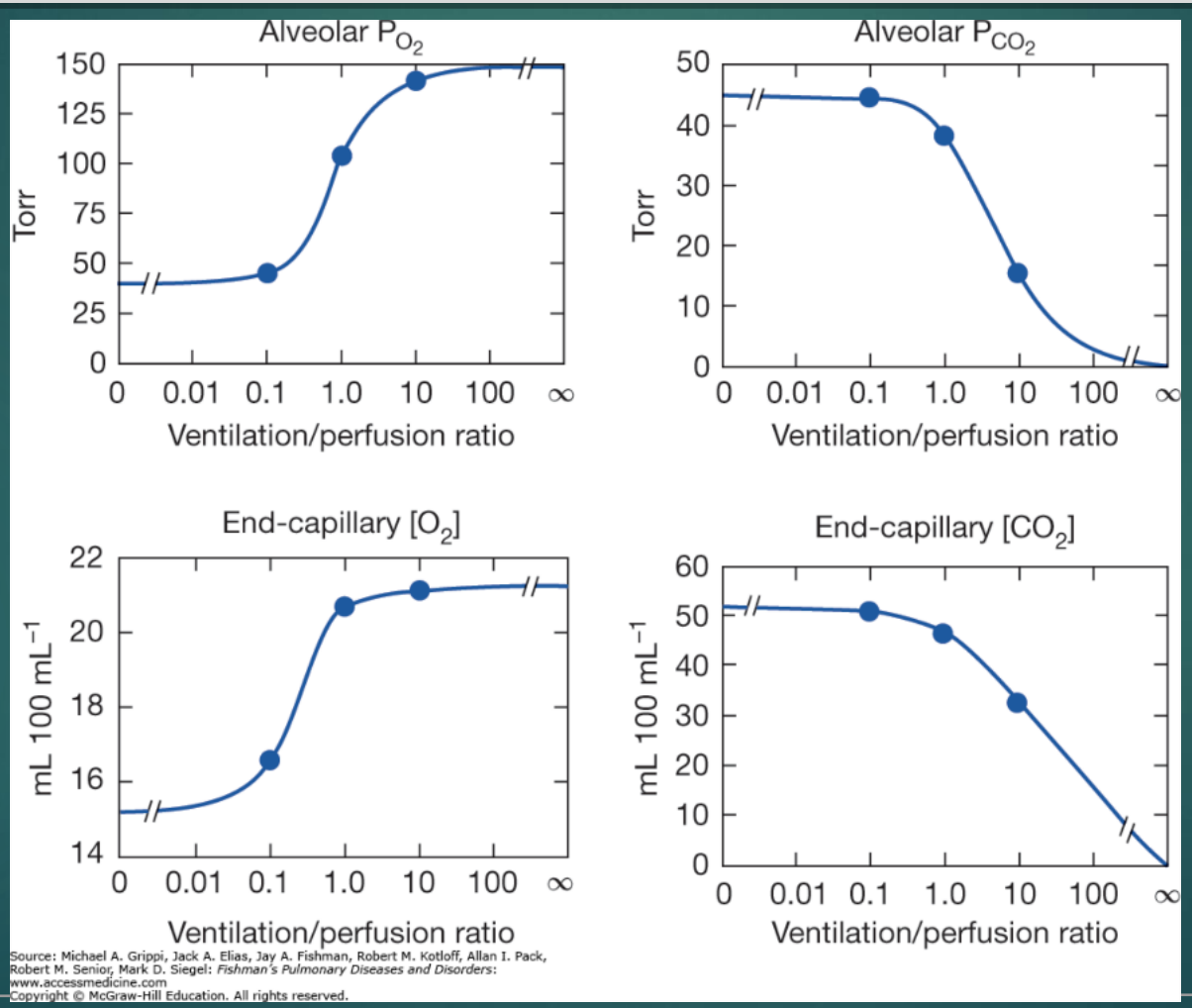

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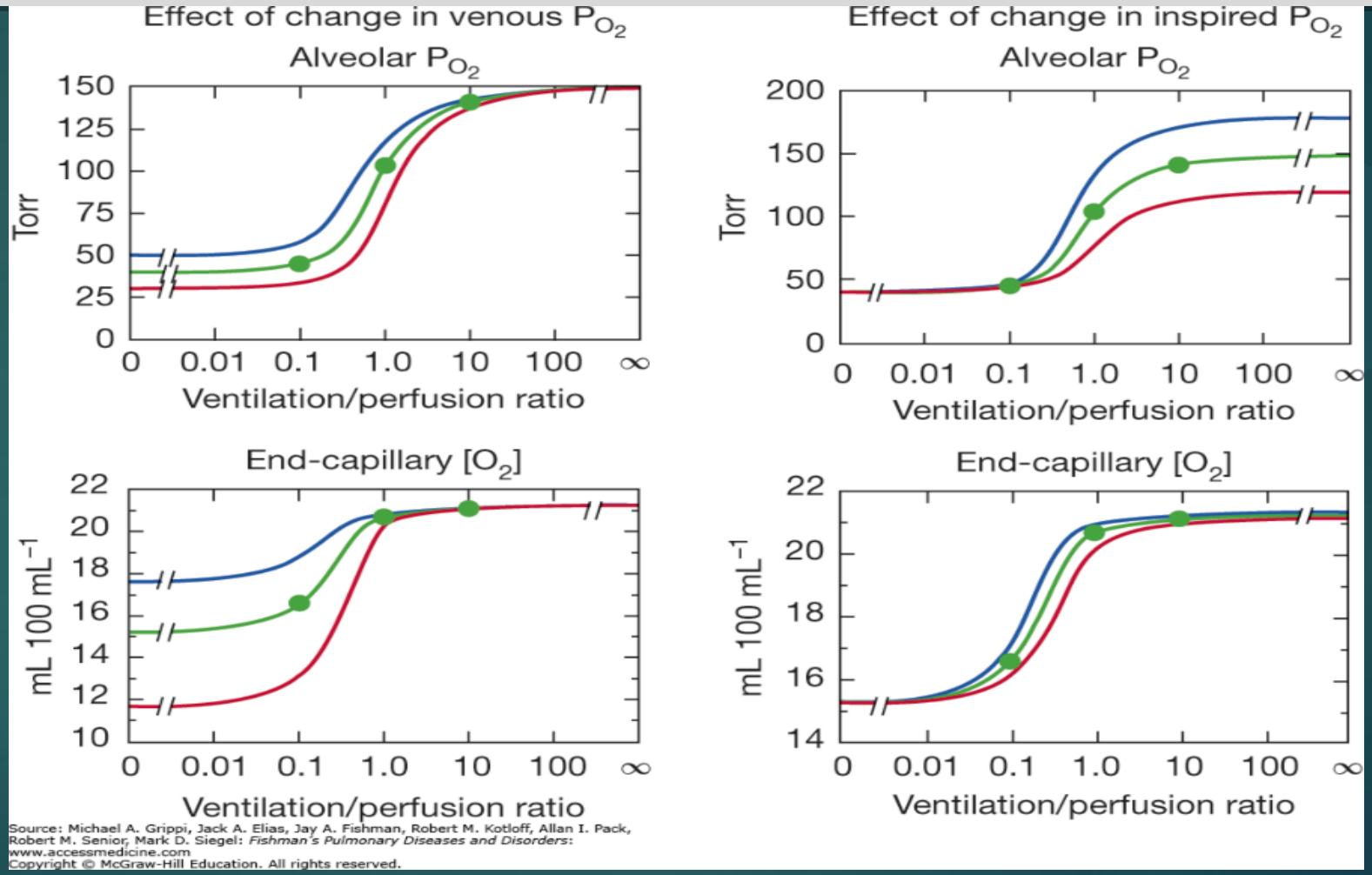

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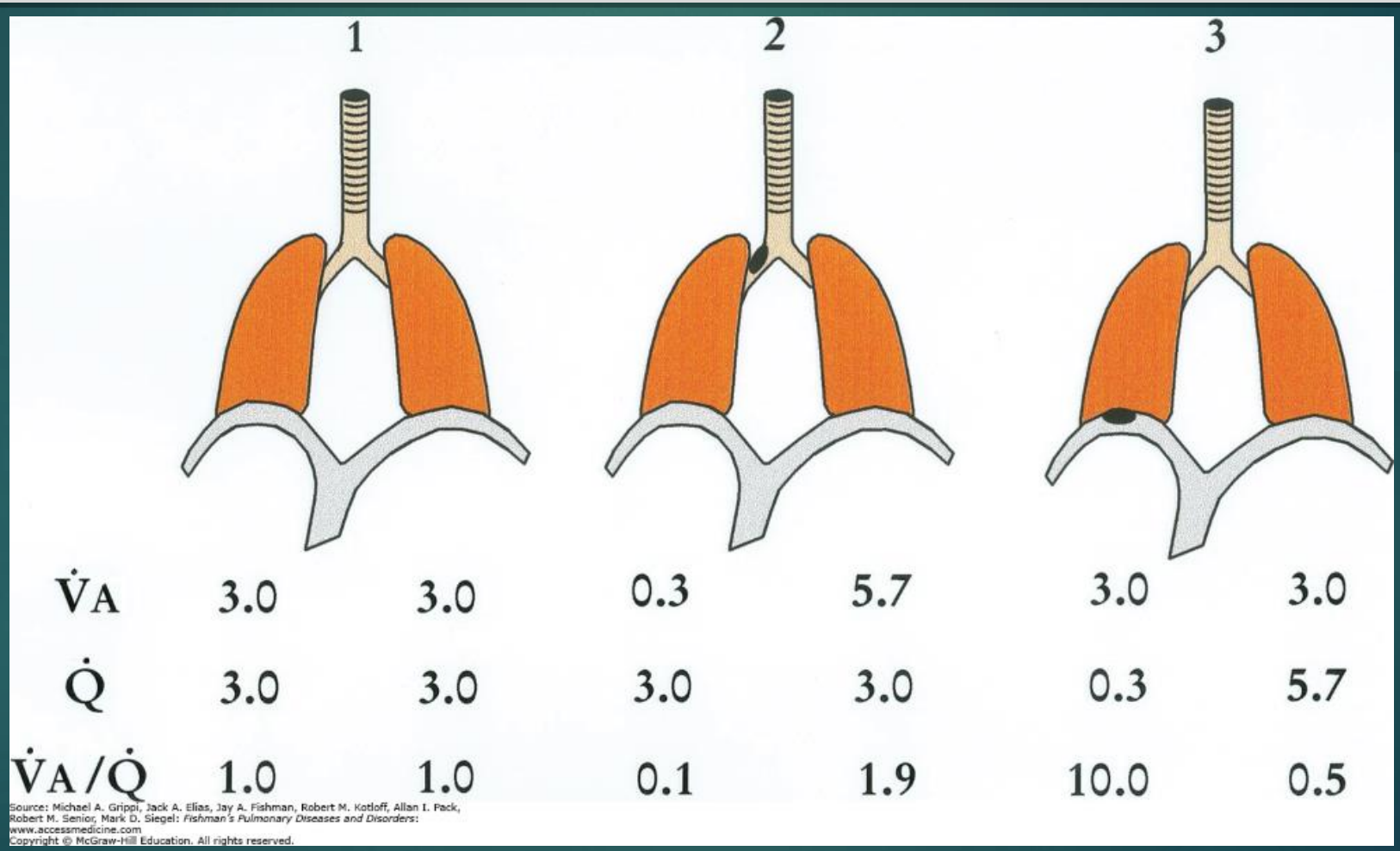

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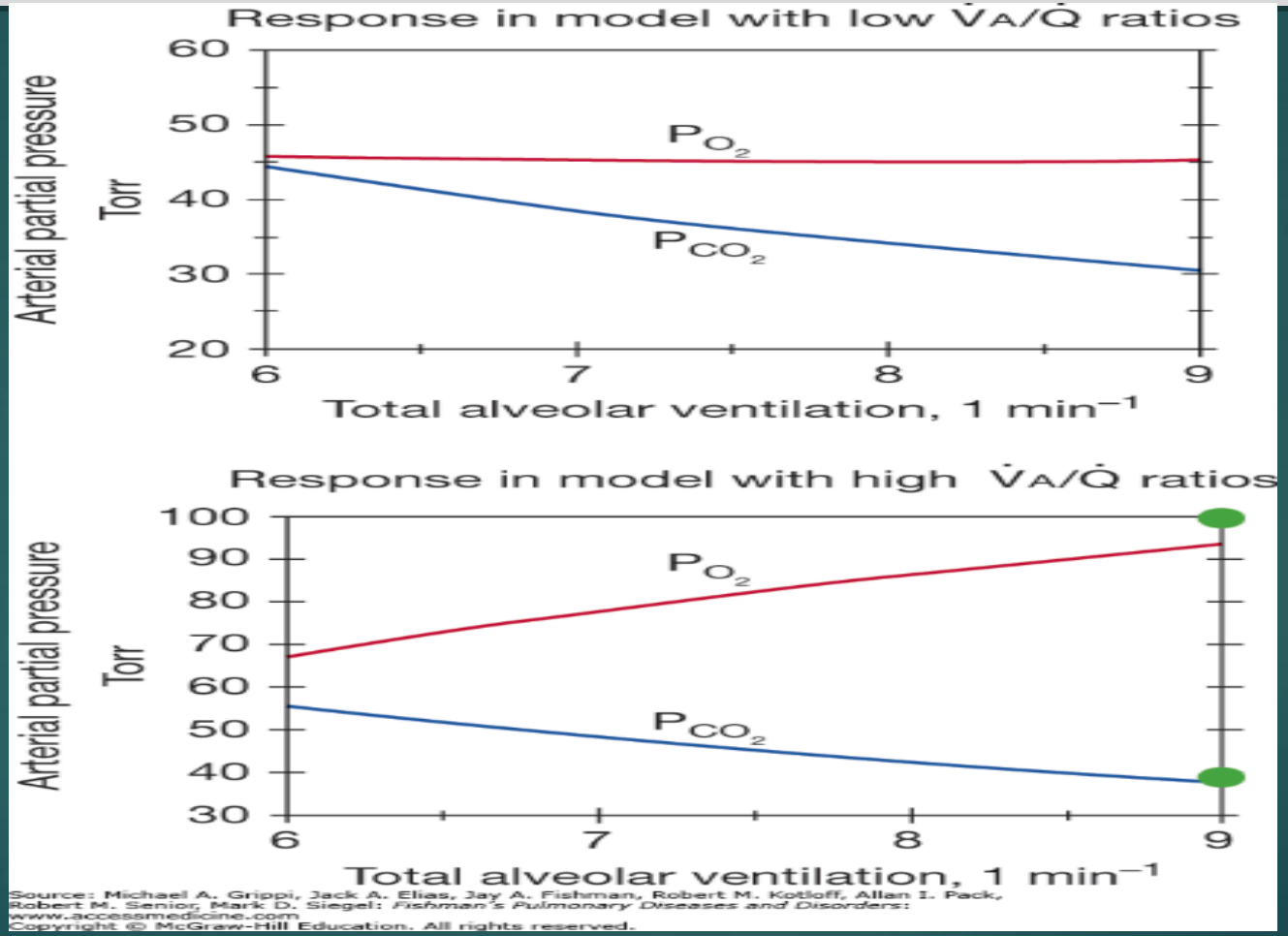

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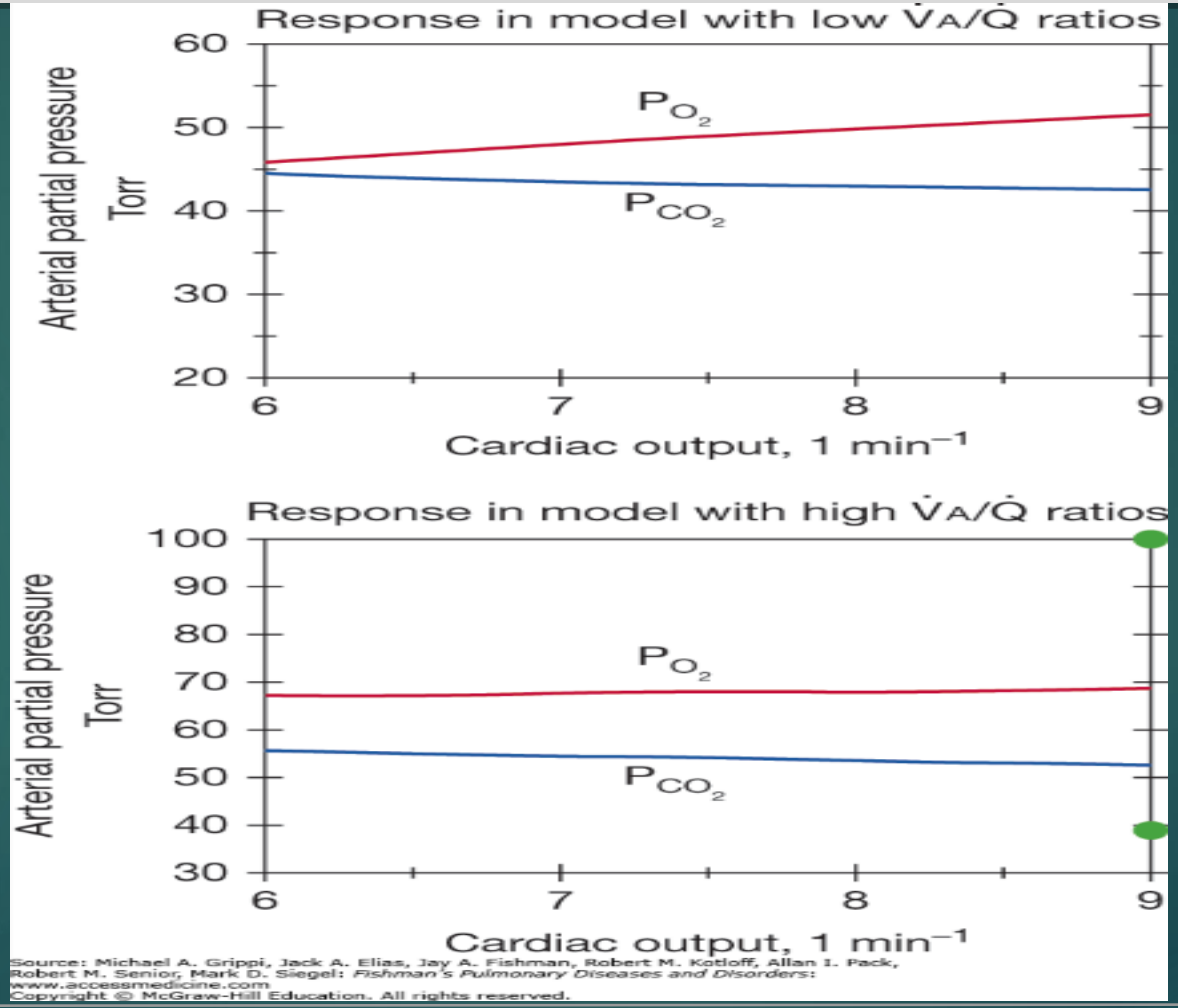

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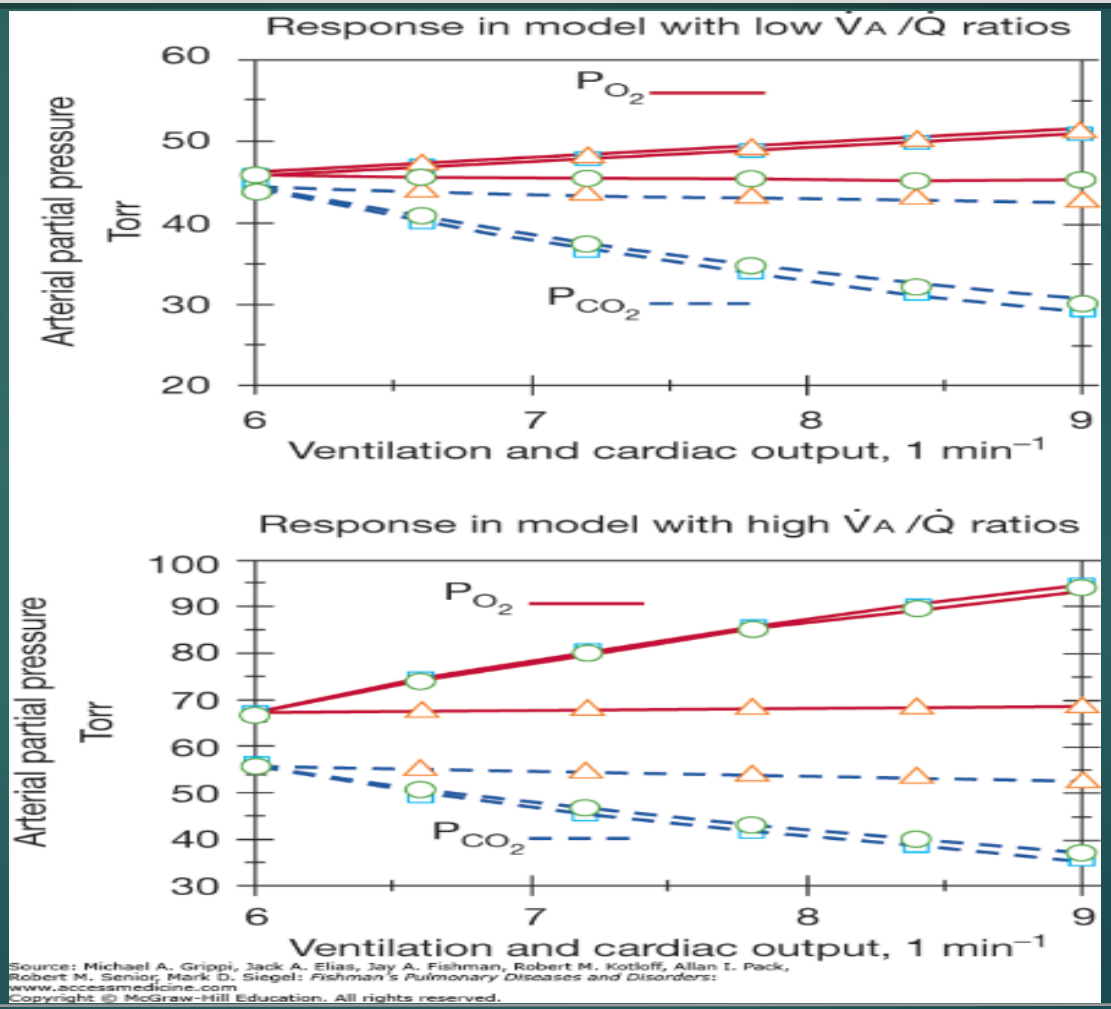

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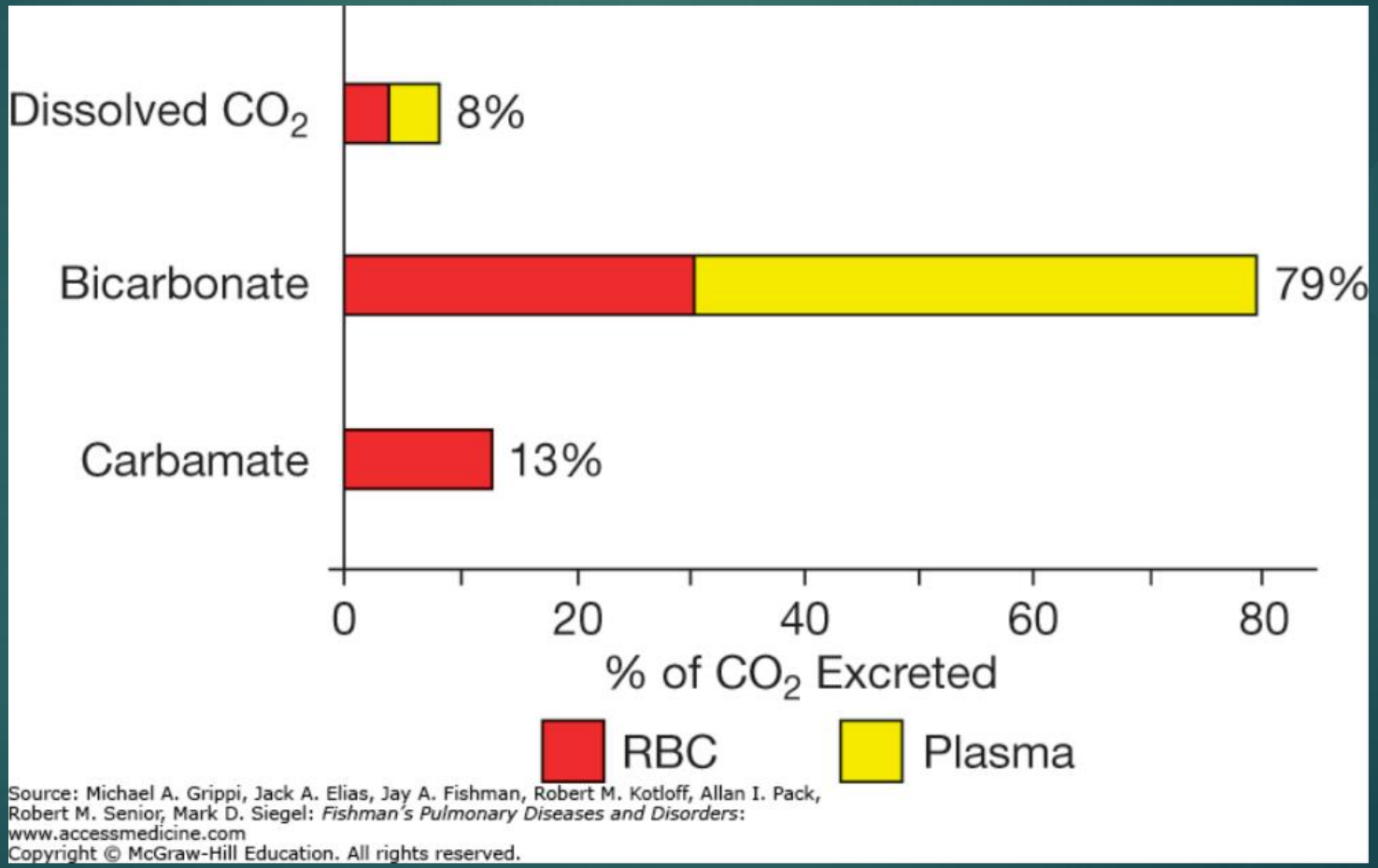

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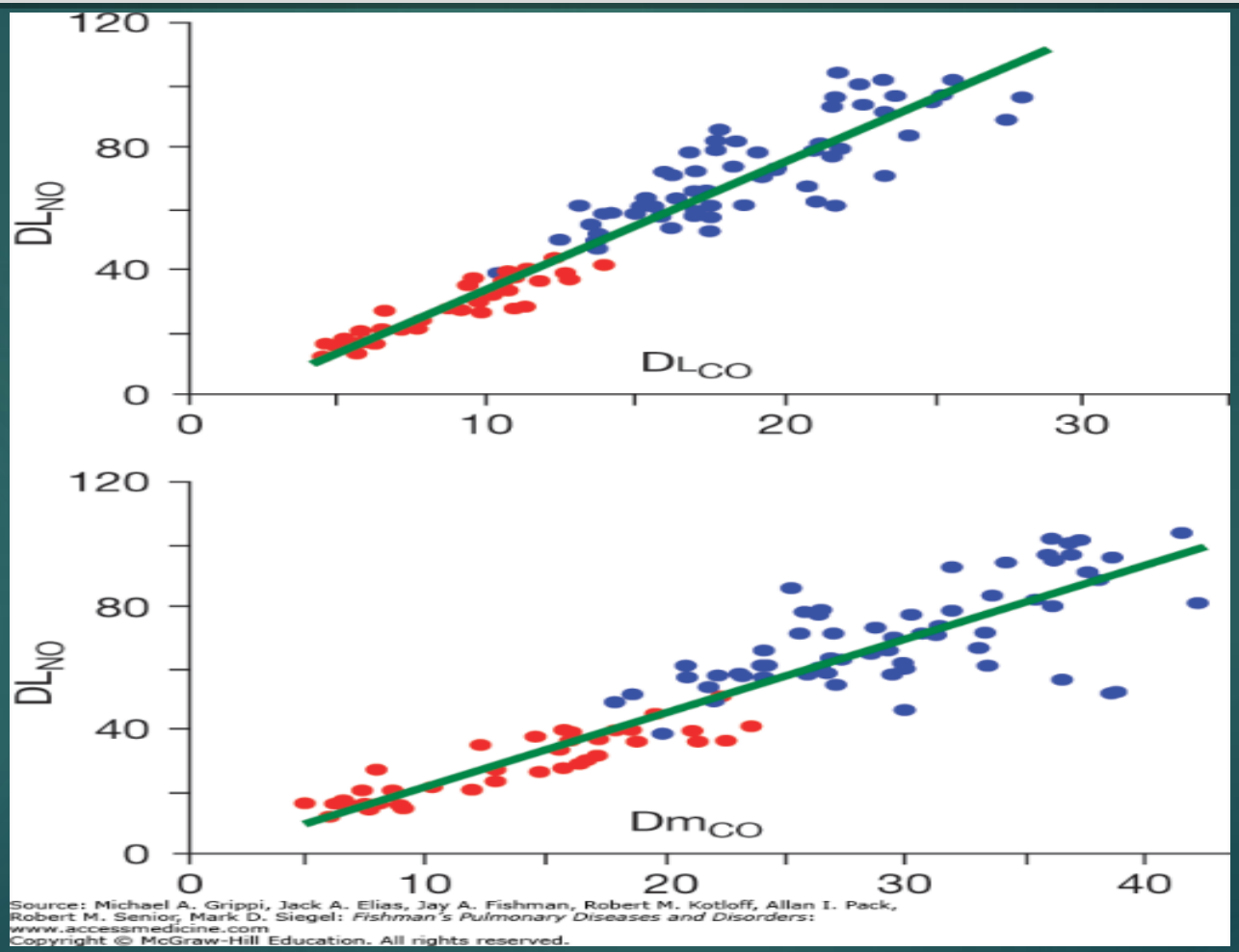

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