Lecture 8

Tutorial #2:

Thursday February 2nd, 5h30-6h30, McIntyre building room 522

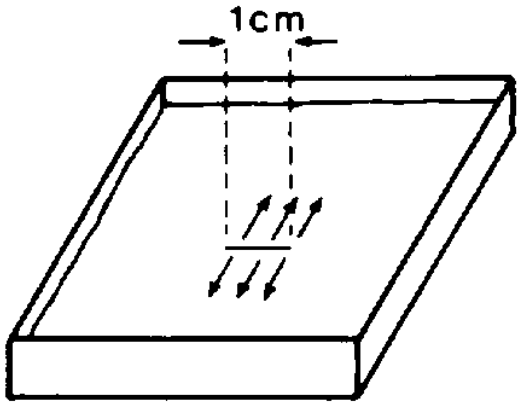
F. Surface Tension

•The surface tension of the liquid film lining the lungs is an important contributor to the mechanical properties of the respiratory system.

•This tension arises because the molecules in the surface of the film tend to arrange themselves in the configuration involving the lowest energy.

•Being more attracted to themselves than to air, they like to “hold hands” rather than freely associate with air molecules.

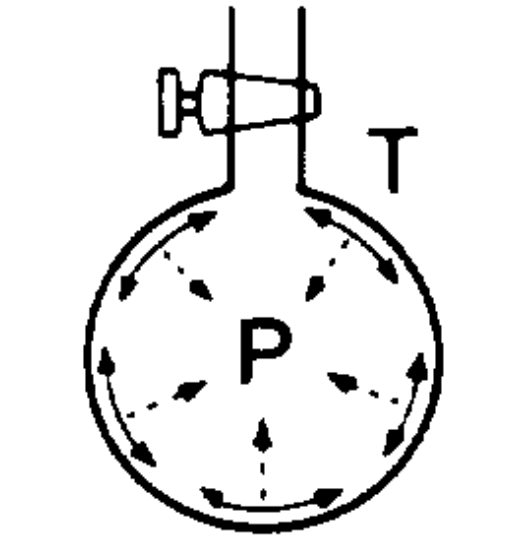
•This causes a tension to be generated across the film surface.



A

F. Surface Tension

•If the surface is curved, such as on the inside of an alveolus or airway, this tension can produce a pressure.



SOAP BUBBLE

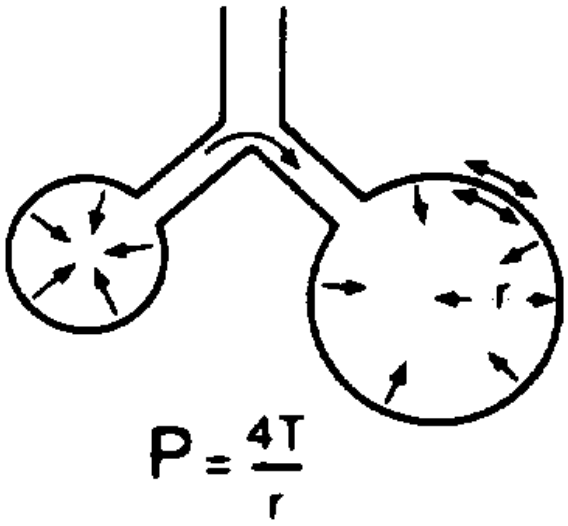
B

F. Surface Tension

•Alveoli can be modeled to some degree of approximation as being like a collection of soap bubbles. The pressure, P, inside the soap bubble of radius R, resulting from a surface tension T, is given by LaPlace's Law:

$$P=4T/r$$

• This equation shows that the pressure inside a small bubble is greater than that inside a large bubble.



C

G. Pulmonary Surfactant

- Therefore, small alveoli should collapse into large ones. This is **prevented** from happening **by** secretion of pulmonary **surfactant** by alveolar type II cells.

Pulmonary surfactant has 2 principal roles:

1) Making the surface tension inside the alveoli change with the lung volume in a way that prevents the pressure inside the small alveoli from exceeding that of the large alveoli. (i.e. biophysical properties that decrease surface tension to a greater extent in the smaller than in the larger alveoli).

2) Reducing overall surface tension so that we are able to breathe. If the surface tension in the liquid lining layer was equal to that of water, we simply would not be able to inflate our lungs.

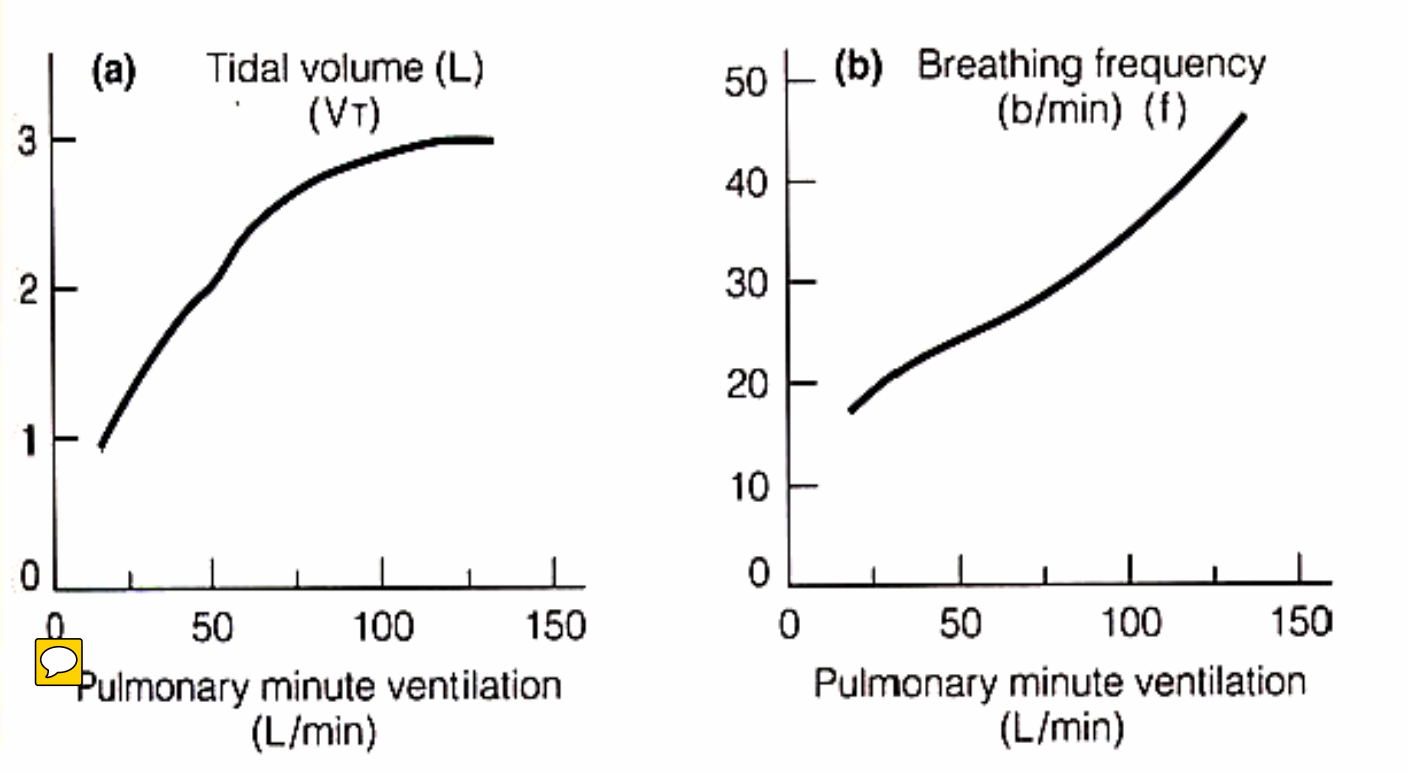
VIII VENTILATION DURING EXERCISE

A. Tidal Volume and Breathing Frequency during Exercise

Minute ventilation ($\dot{V}E$) during progressive exercise up to maximum:

- When exercise starts, both tidal volume (V_T) and breathing frequency (f) increase proportionally.

- However, at some point VT plateaus.



-Remember that lung compliance decreases at very high lung volumes.

-Thus, high ventilatory rates during hard exercise are due to incremental increases in f.

Brooks, G.A., et al., Exercise Physiology,Human Bioenergetics and its applications, 3rd edition, California: Mayfield, 1999.

A. Tidal Volume and Breathing Frequency during Exercise

- Because of the increased breathing frequency, inspiratory and, expiratory times decrease during progressive exercise but expiratory times fall relatively more than inspiratory time.

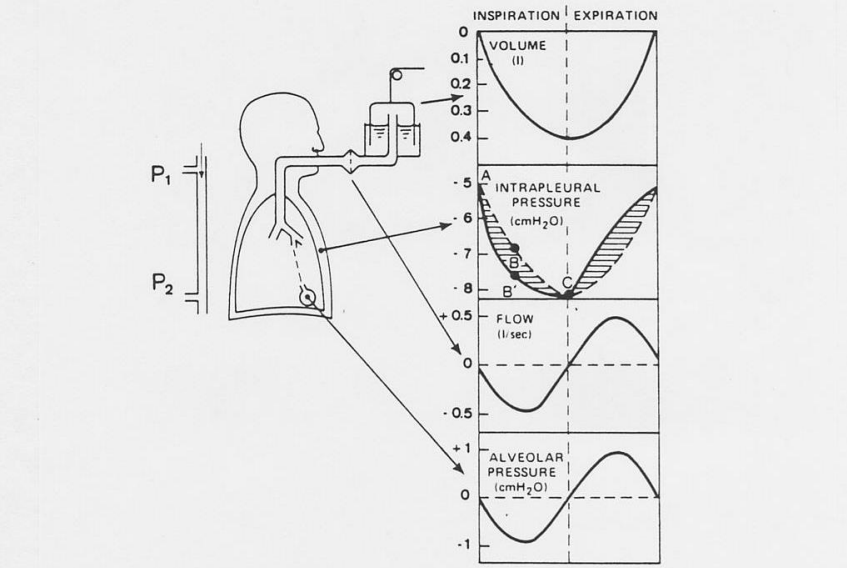
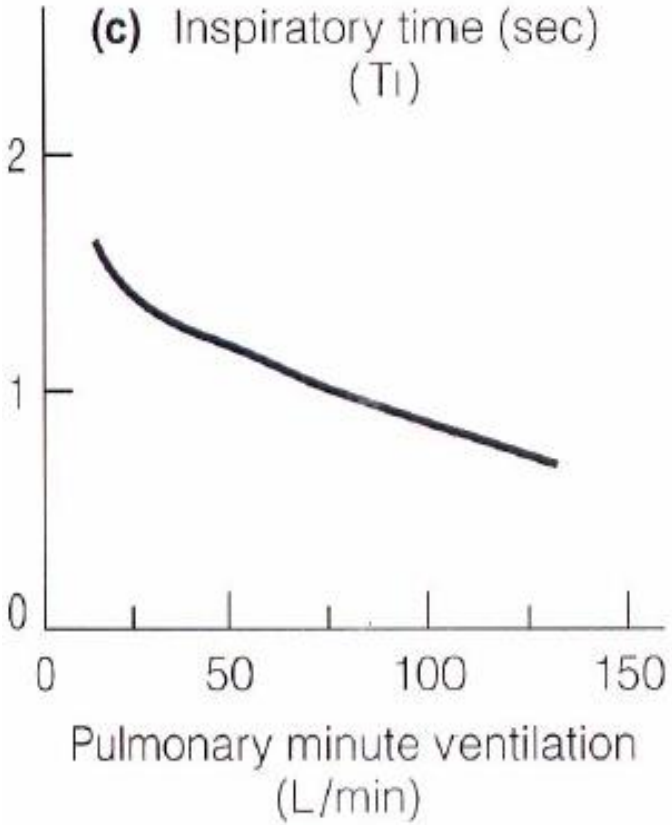
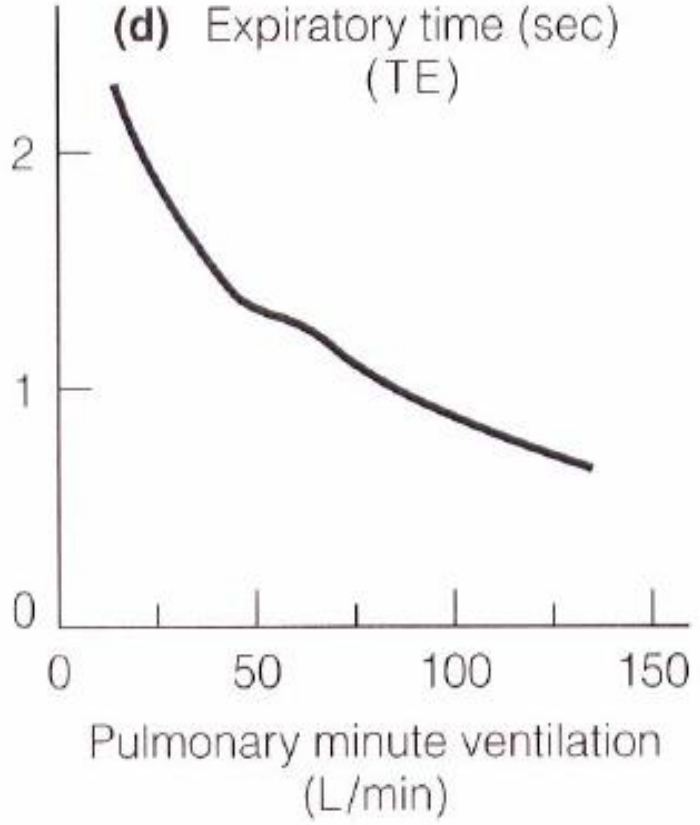


Figure 7.13. Pressures during the breathing cycle. If there were no airway resistance, alveolar pressure would remain at zero, and intrapleural pressure would follow the *broken line ABC*, which is determined by the elastic recoil of the lung. Airway (and tissue) resistance contributes the *hatched portion* of intrapleural pressure (see text).





Consequently, peak expiratory flow rate increases more than peak inspiratory flow rate.

Brooks, G.A., et al., Exercise Physiology,Human Bioenergetics and its applications, 3rd edition, California: Mayfield, 1999.

Is Ventilation a limiting factor in aerobic performance at sea level? ($\dot{V}E/\dot{Q}$)

-Resting values of $\dot{V}E$ can increase 35 fold during exercise (from 5L/min to 190 L/min, in a fit individual).

-Resting values of cardiac output (CO) can increase 5-6 fold during exercise (from 5L/min to 25-30 L/min, in a fit individual).

-The $\dot{V}E/Q \cong 1$ at rest. Because $\dot{V}E$ can increase more than $\dot{Q}$ during exercise, there is an increase in $\dot{V}E/\dot{Q}$. The increase in this ratio is one reason why ventilation is not believed to limit aerobic performance.

-In a less fit individual, the absolute values of $\dot{V}E$ and $\dot{Q}$ will be less but the ratio will increase to a similar extent.

Is Ventilation a limiting factor in aerobic performance at sea level? (alveolar surface area)

-The alveolar surface area is 50m^2 (1/2 of a single tennis court).

-The average blood volume is 5L.

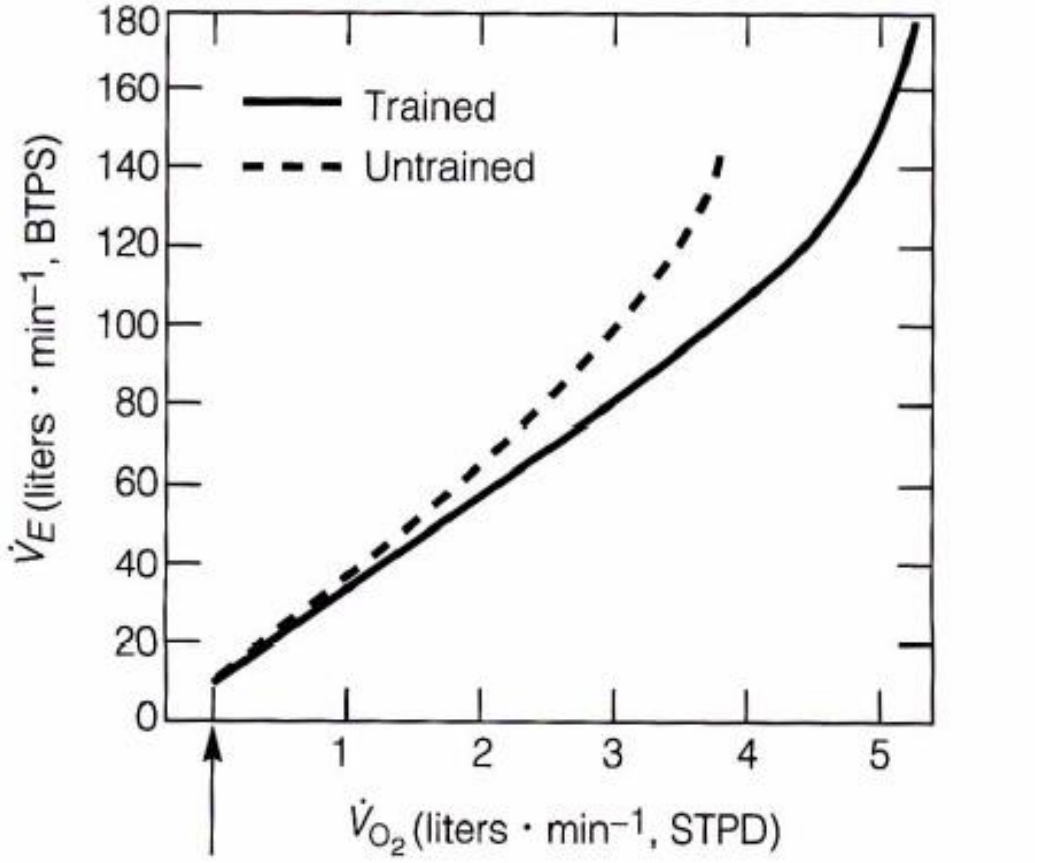
-4% of this 5L is in the pulmonary system at any one time during maximal exercise.

-Therefore, there is a large capacity for gas exchange which is another reason why ventilation is not believed to limit aerobic performance.

B. Minute ventilation and metabolic rate during exercise

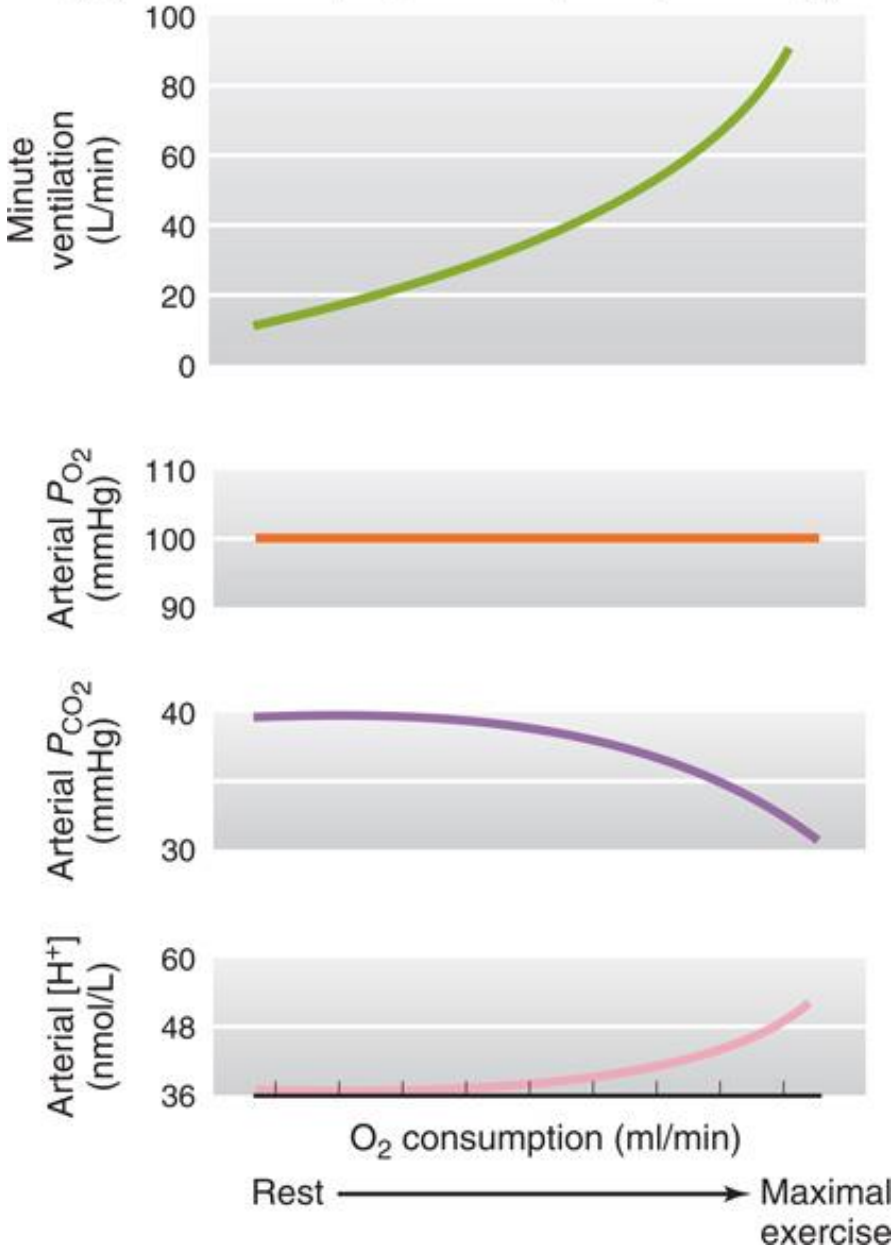
- In both untrained and trained subjects, minute ventilation ($\dot{V}E$) increases linearly with metabolic rate ($\dot{V}O_2$) up to about 50% to 65% of $\dot{V}O_2$ max.

- Thereafter, $\dot{V}E$ increases at a rate disproportionately greater than the change in $\dot{V}O_2$. Note that an effect of endurance training is to delay the ventilatory inflection point (*T*vent).


180
160
140
120
100
80
60
40
20
0
Trained
Untrained
$\dot{V}_E$ (liters · min^{-1}, BTPS)
1
2
3
4
5
$\dot{V}_{O_2}$ (liters · min^{-1}, STPD)

C. Control of Ventilation during Exercise

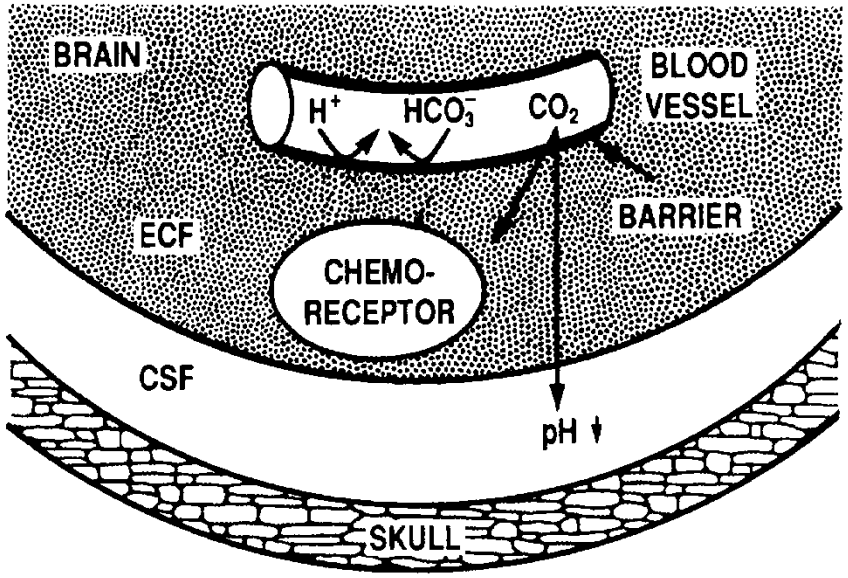
What controls ventilation during exercise?



C. Control of Ventilation during Exercise

The Central Chemoreceptors During Exercise:

•During exercise, there is an alkalotic (↑pH) response in the medullary ECF. This decreases the ventilatory response. Therefore, the role of the central chemoreceptors is important at rest but not so much during exercise.



Control of Ventilation during Exercise (Cont'ed)

Peripheral Chemoreceptors:

•Peripheral chemoreceptors are mainly sensitive to changes in PO_2, but are also stimulated by increased PCO_2 and decreased pH.

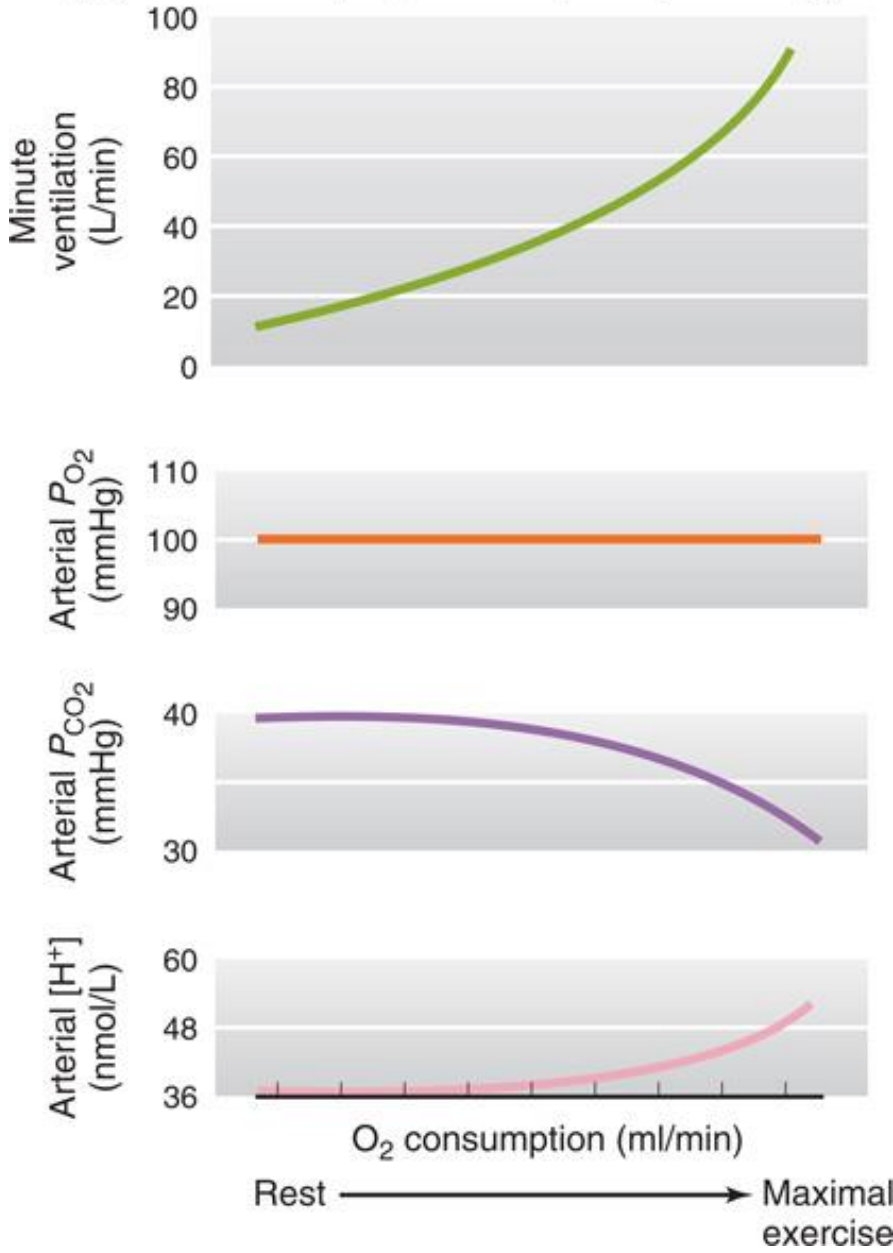
Control of Ventilation during Exercise (Cont'ed)

Peripheral Chemoreceptors (Cont'ed):

-PaO_2 remains rather constant during exercise.

-$PaCO_2$ is often seen to decrease during exercise.

-However, during exercise, arterial pH does decrease (**lactic acid**) and PaO_2 fluctuates subtly with arterial pulse waves. Therefore, it is possible that during exercise, these fluctuations in PaO_2 increase the sensitivity of the peripheral chemoreceptors to CO_2 and H^+.



Control of Ventilation during Exercise (Cont'ed)

Peripheral Mechanoreceptors during Exercise

-The pulmonary mechanoreceptors, the muscle spindles, the Golgi tendons, and the skeletal joint receptors were thought to play a role in the increase in $\dot{V}E$ during exercise.

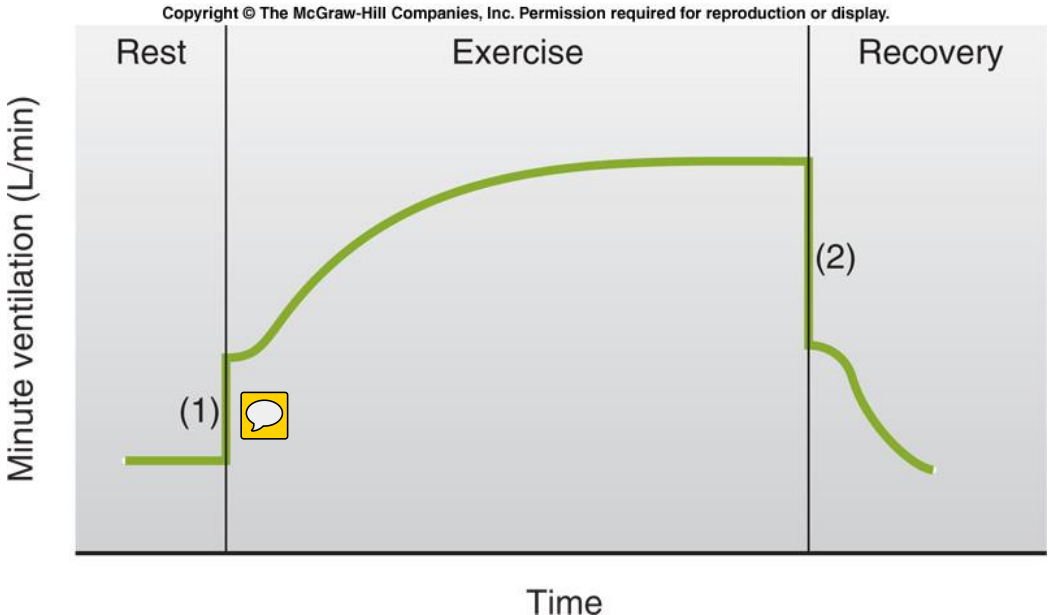
-Stimulation of these mechanoreceptors does produce an increase in $\dot{V}E$, but it is small compared to the large and abrupt increases observed during exercise.

Control of Breathing during Exercise Cont'ed)

Onset and Recovery from Exercise

- $\dot{V}_E$ is known to start increasing even before the exercise has started. This control is thought to be neural. A similar control is thought to operate at the end of exercise because a very rapid decrease in $\dot{V}_E$ is observed.

- Humoral control is believed to be responsible for the ventilatory response during the exercise event.



The end