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Respiratory Control, Ventilation, and Regulation of P_aCO_2

M1 – Cardiovascular/Respiratory Sequence

Louis D'Alecy, Ph.D.

Fall 2008

Tuesday 11/18/08, 10:00

Respiratory Control

vs.

Control of Breathing (Ventilation)

vs.

Regulation of P_aCO_2

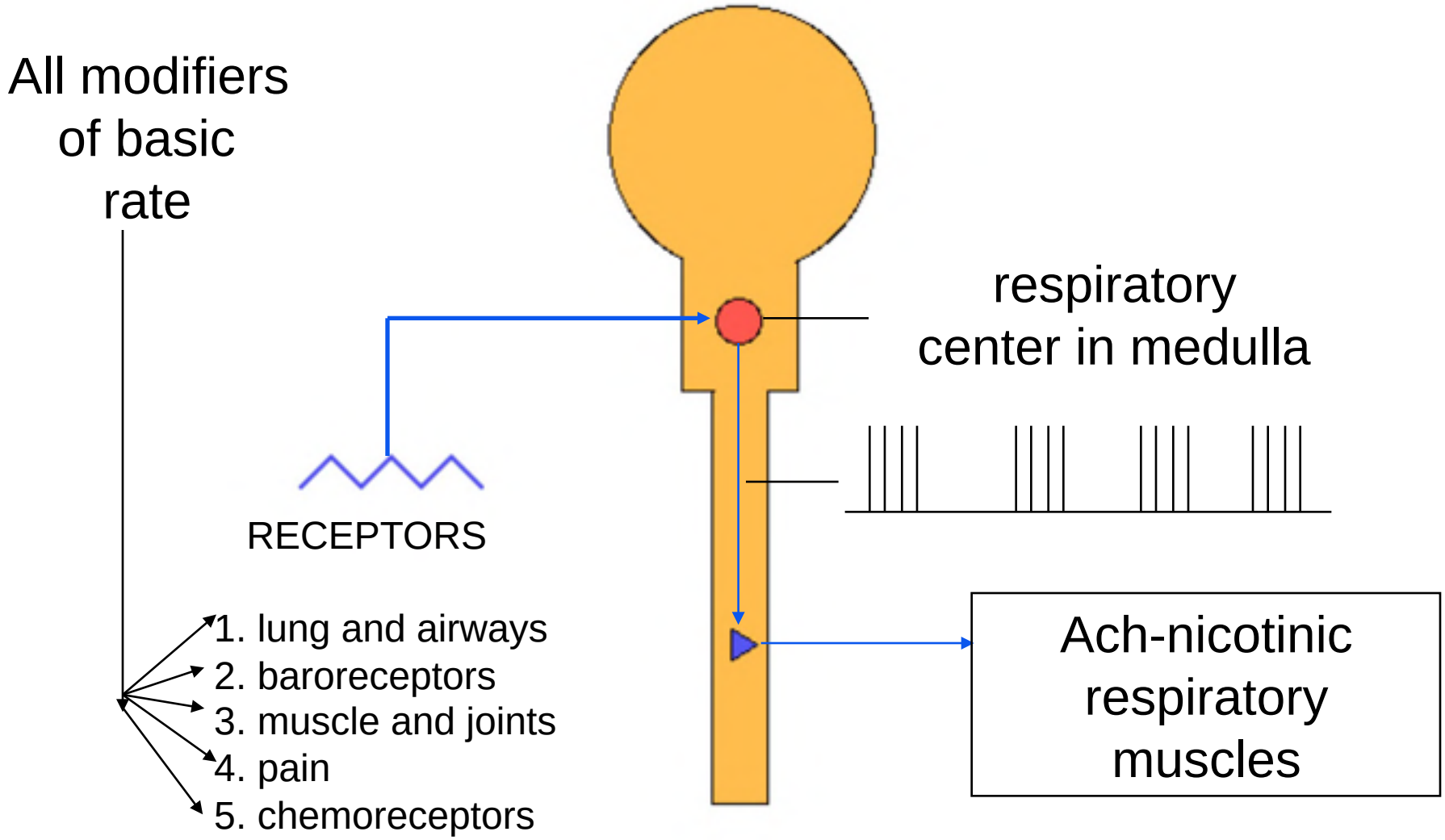
(22 slides, 50 minutes)

Apnea, Eupnea, Tachypnea, Hyperpnea

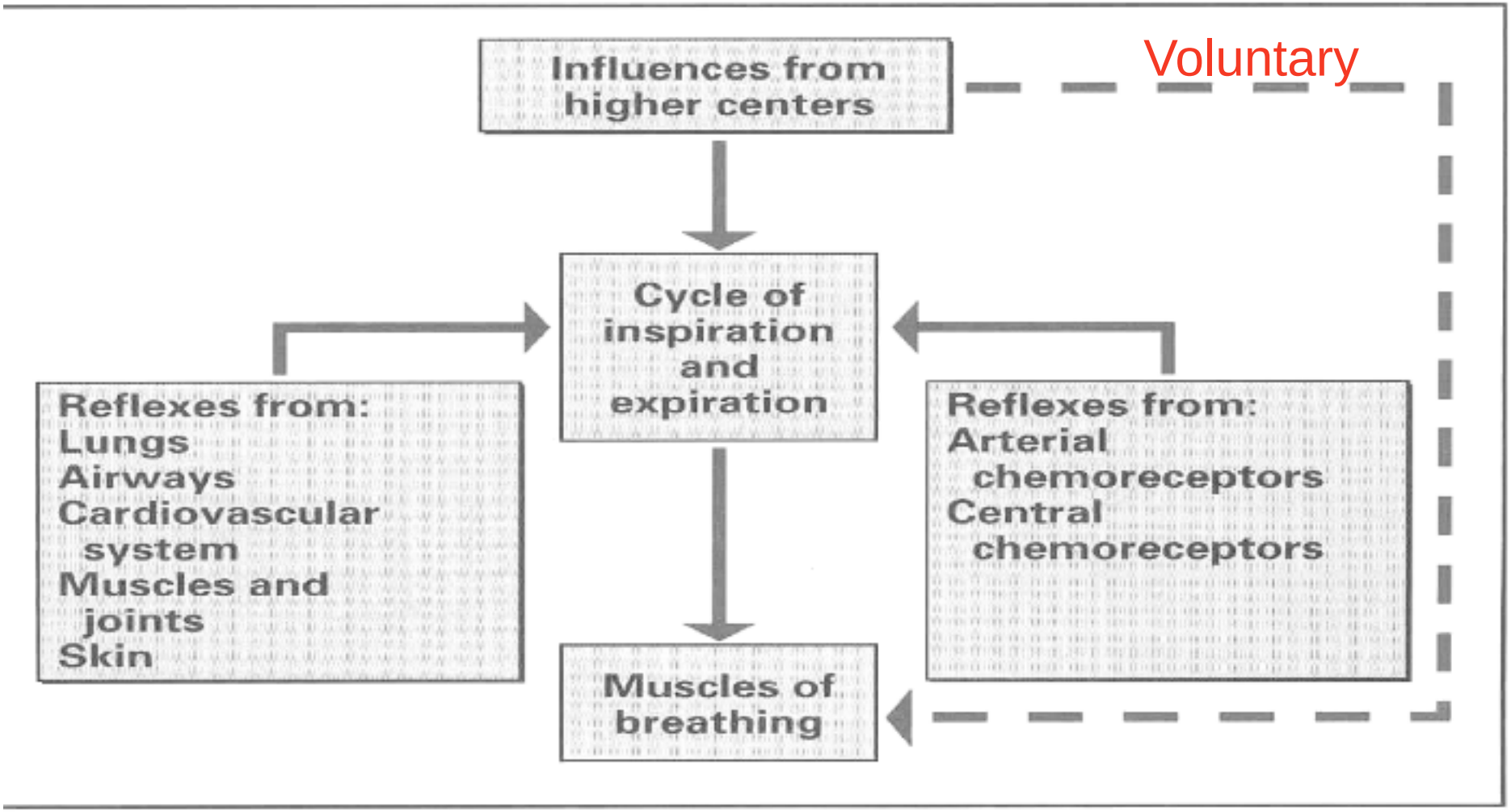
Hyperventilation & Hypoventilation

Eupnea: normal quiet ventilation

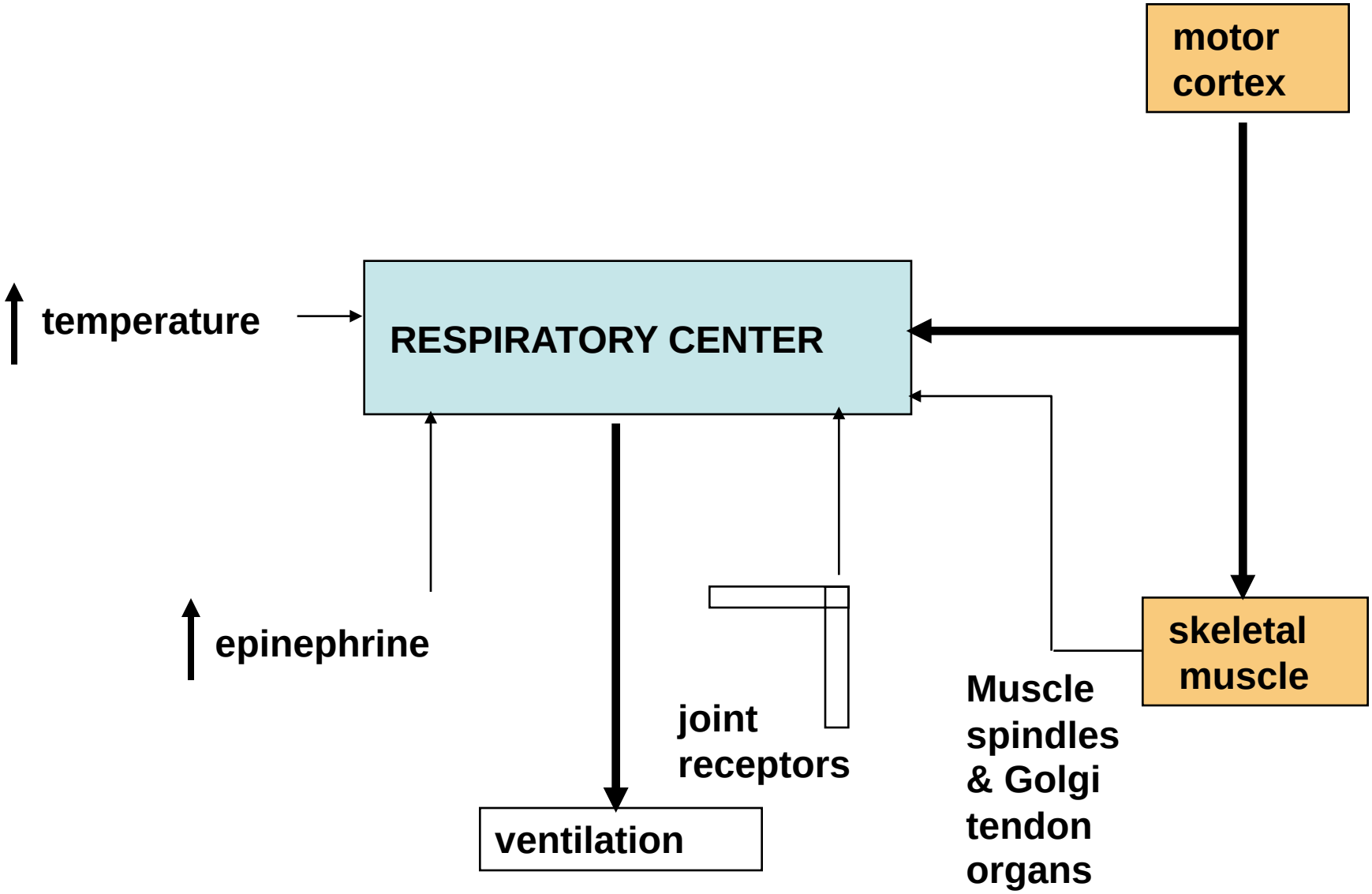
- Automatic & cyclic
- Originate in CNS - medulla
- “final common pathway” via phrenic nerves
- Bursts of nerve firing cause inspiration
- Interval between groups determines rate
- Frequency and # of motor units - depth (TV)
- Diaphragm (2/3 supine)+external intercostals


All modifiers of basic rate
respiratory center in medulla
RECEPTORS
1. lung and airways
2. baroreceptors
3. muscle and joints
4. pain
5. chemoreceptors
Ach-nicotinic respiratory muscles

Control of Respiration



CONTROL OF RESPIRATION DURING EXERCISE



Receptor Modulation of Ventilation

1. Lung and airway receptors

    A. stretch receptors - inflation inhibits inspiration
        Hering-Breuer reflex

    B. irritant receptors - sneeze and cough

    C. juxtapulmonary receptors - stimulated by
        pulmonary edema - sensation of dyspnea

2. Baroreceptors -decreased MAP increases ventilation

3. Muscles and joints - movement increases ventilation

4. Pain - gasp reflex

Altered Breathing

- Apnea: absence of breathing
- Eupnea: normal quiet breathing
- Tachypnea: rapid breathing
- Hyperpnea: increase $\dot{V}_T$ + or - increased $\dot{V}_A$
- Hyperventilation: excess ventilation that
  - ******* Lowers PaCO2 *******
- Hypoventilation: inadequate ventilation
  - ******** Increases PaCO2 *******

$$\dot{V}_T \equiv \dot{V}_E$$

Minute Ventilation

Alveolar Ventilation

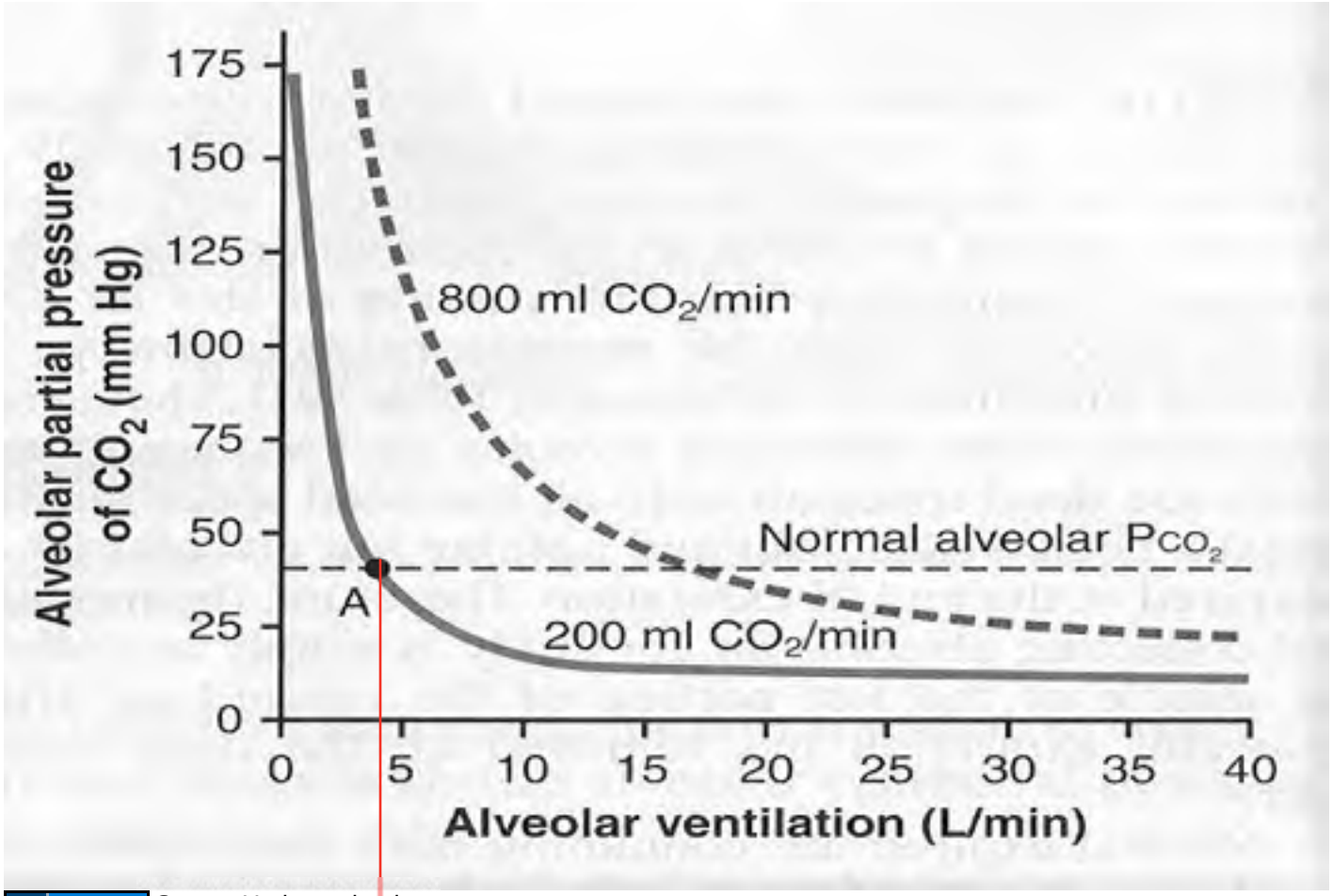
Alveolar Ventilation

Dead Space Ventilation

$$\dot{V}_A = \dot{V}_T - \dot{V}_D \quad \text{or}$$

$$\dot{V}_A = \dot{V}_E - \dot{V}_D$$

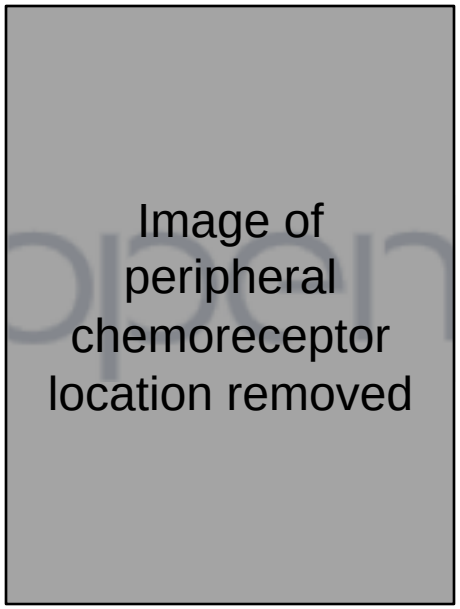
Ventilation on PA_{CO2}



PD-INEL Source Undetermined

Hypoventilation ← Normal → Hyperventilation

PERIPHERAL CHEMORECEPTORS



Please see: http://
www.medicine.mcgill.ca/physio/
resp-web/Figures/Figtt20.jpg

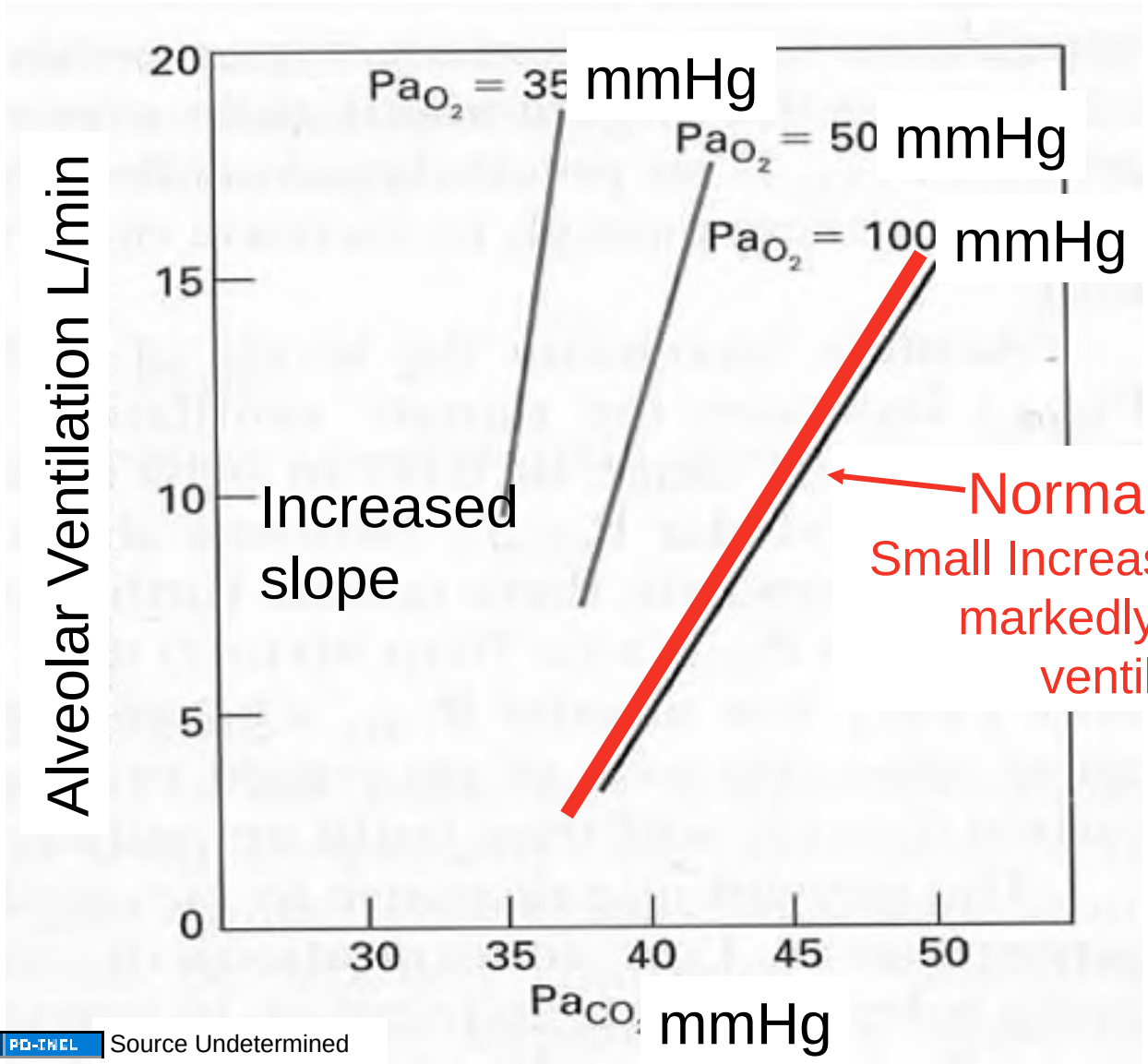
carotid sinus

common carotid artery

Stimulated by

- ↓ arterial P_{O2}
- ↓ arterial pH (= ↑ H^+)
- ↑ arterial P_{CO2}

↓PaO_2 leads to ↑ CO_2 Response



Hypoxia increases ventilatory response to CO_2.

Normal slope.

Small Increases in PaCO2 markedly increase ventilation.

ACIDOSIS = an increase in the [H^+] concentration of blood

Respiratory Acidosis - accumulation of CO_2 (hence H^+) due to **hypoventilation**.

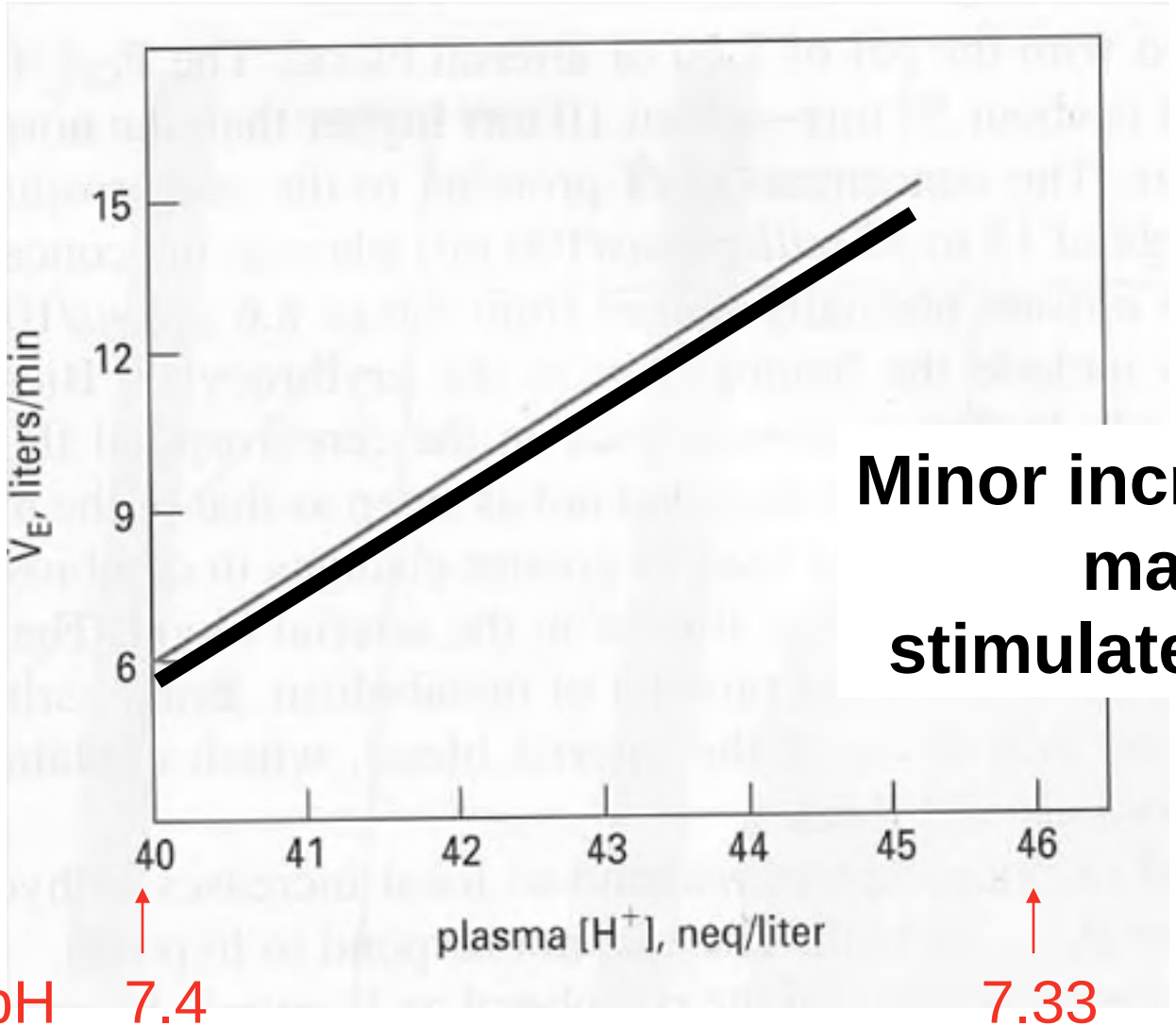
Metabolic Acidosis - Any accumulation of acid (H+) that does not come from retained CO_2.

From: a) metabolic production of organic acids such as lactic acid or ketone bodies,

b) ingestion of acidic substances or

c) failure of kidneys to excrete acid.

Plasma Acidosis ↑ Ventilation



Minor increases in [H$^+$] markedly stimulate ventilation.

PERIPHERAL CHEMORECEPTORS

Response to Acid & CO_2

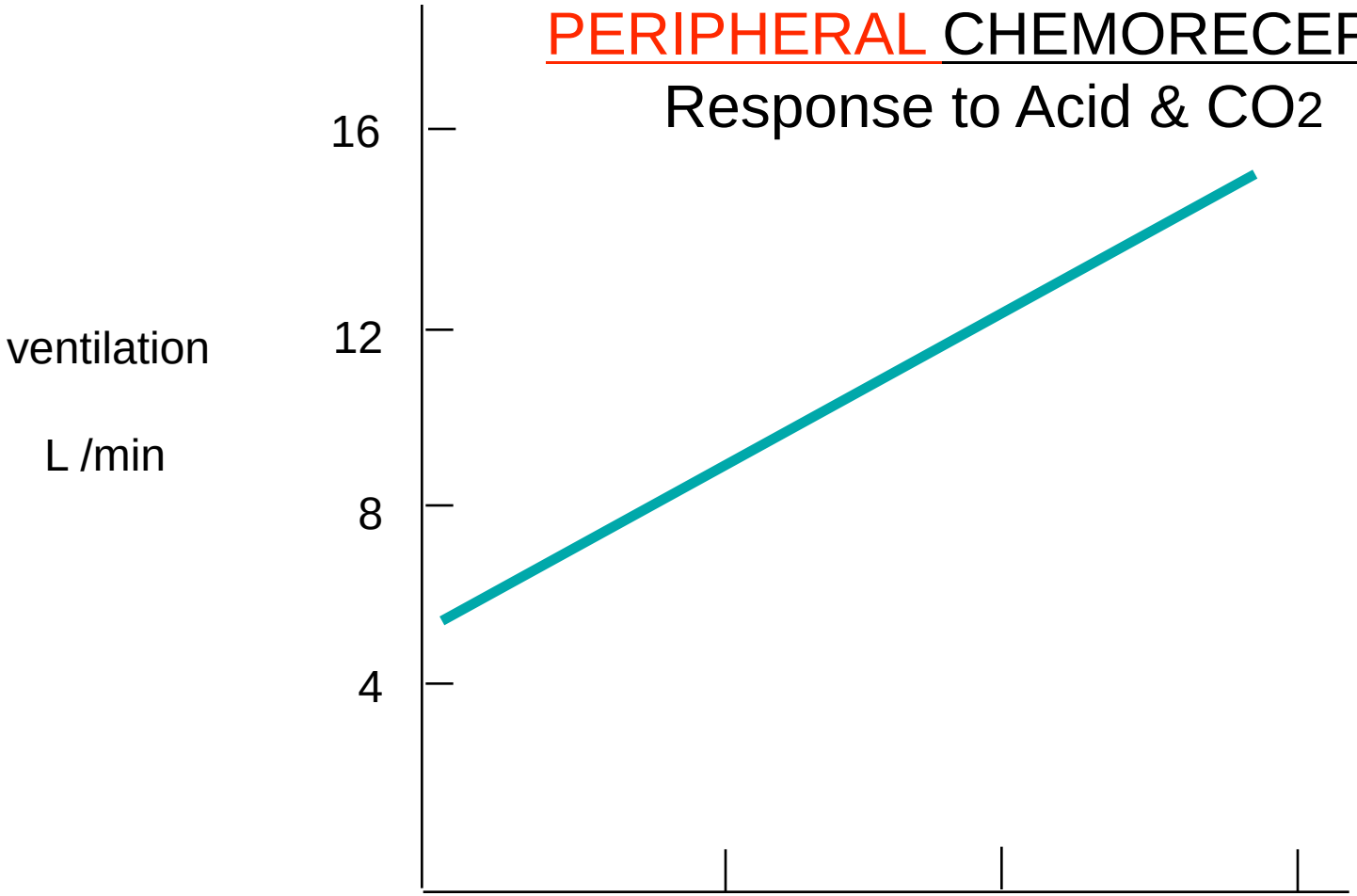
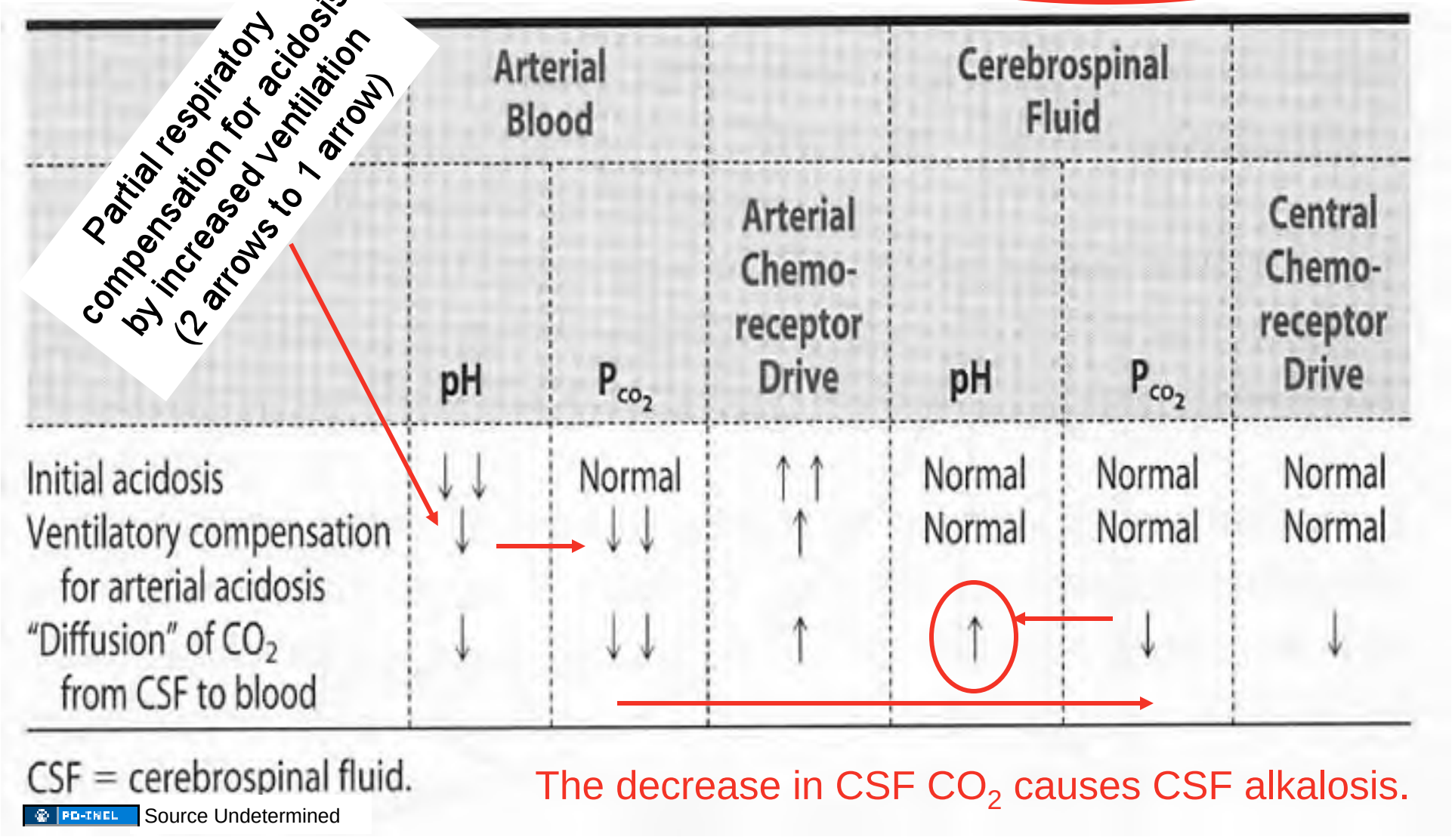


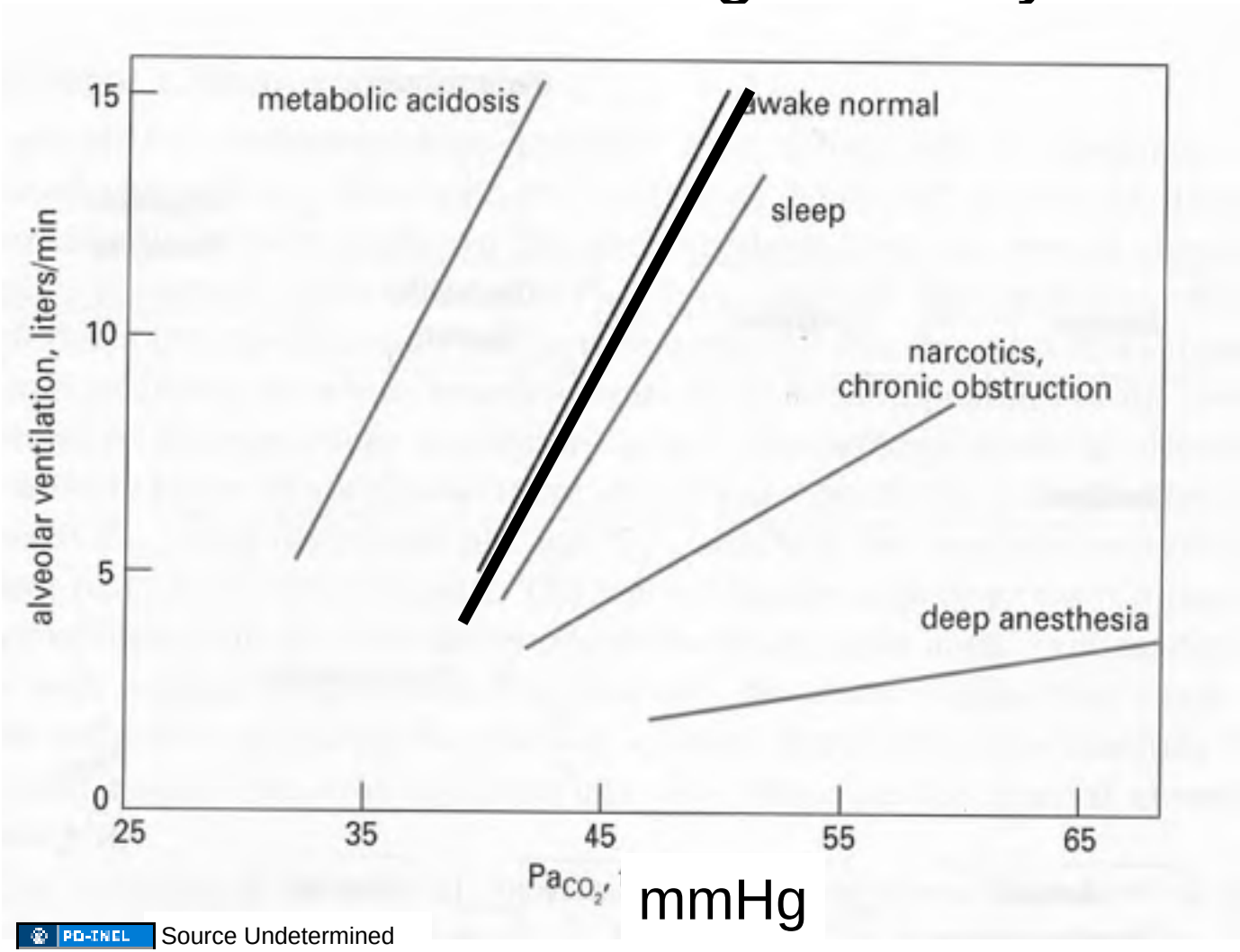
Fig 9-3

Arterial Acidosis → CSF Alkalosis



Arterial CO_2 is major determinant of ventilation BUT this CO_2 response can be altered significantly.

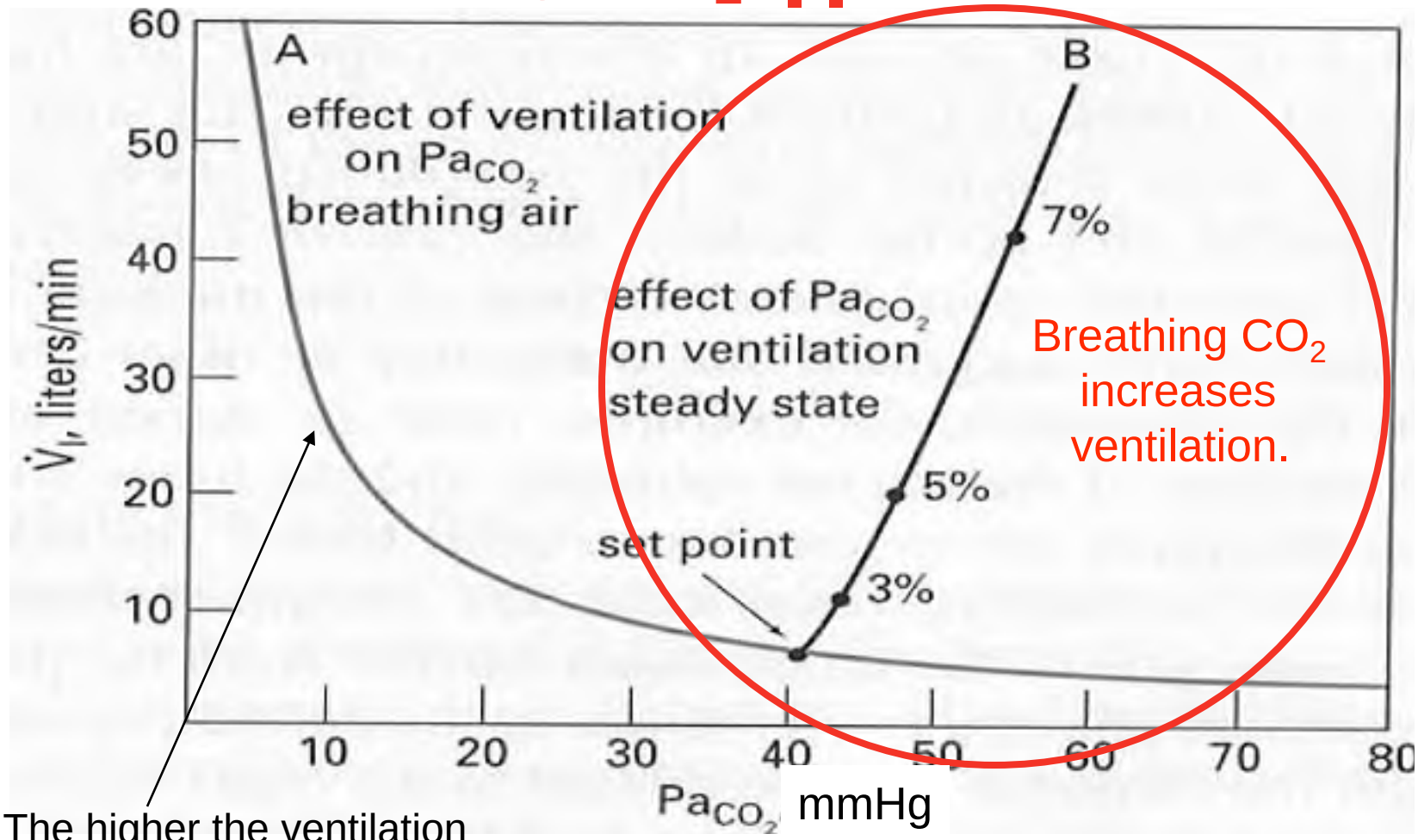
$\dot{V}_A$



PD-INEL Source Undetermined

Hyperventilation: ↓↓ $PaCO_2$

breathing CO_2 ↑↑ ventilation



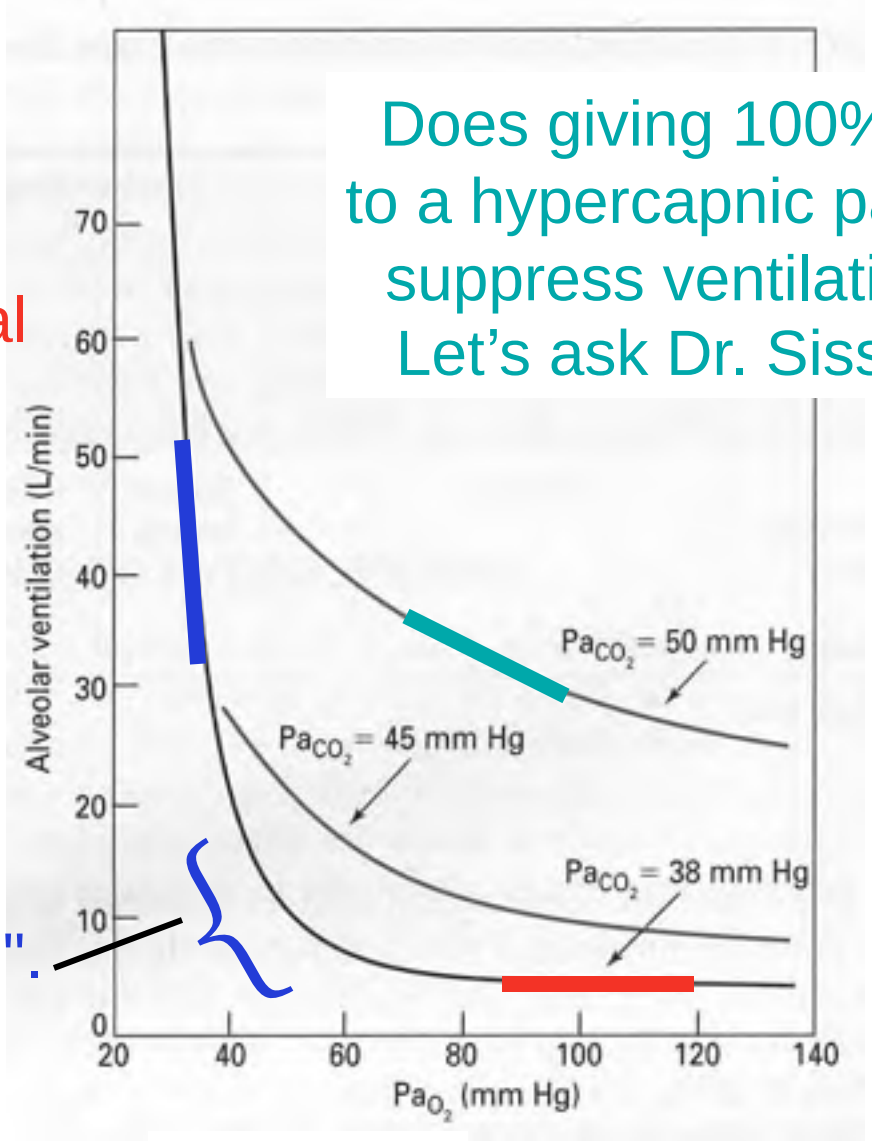
The higher the ventilation the lower the $PaCO_2$.

 Levitzky. Pulmonary Physiology. McGraw-Hill, 2003. 6th ed.

O_2 Response* Interacts with CO_2

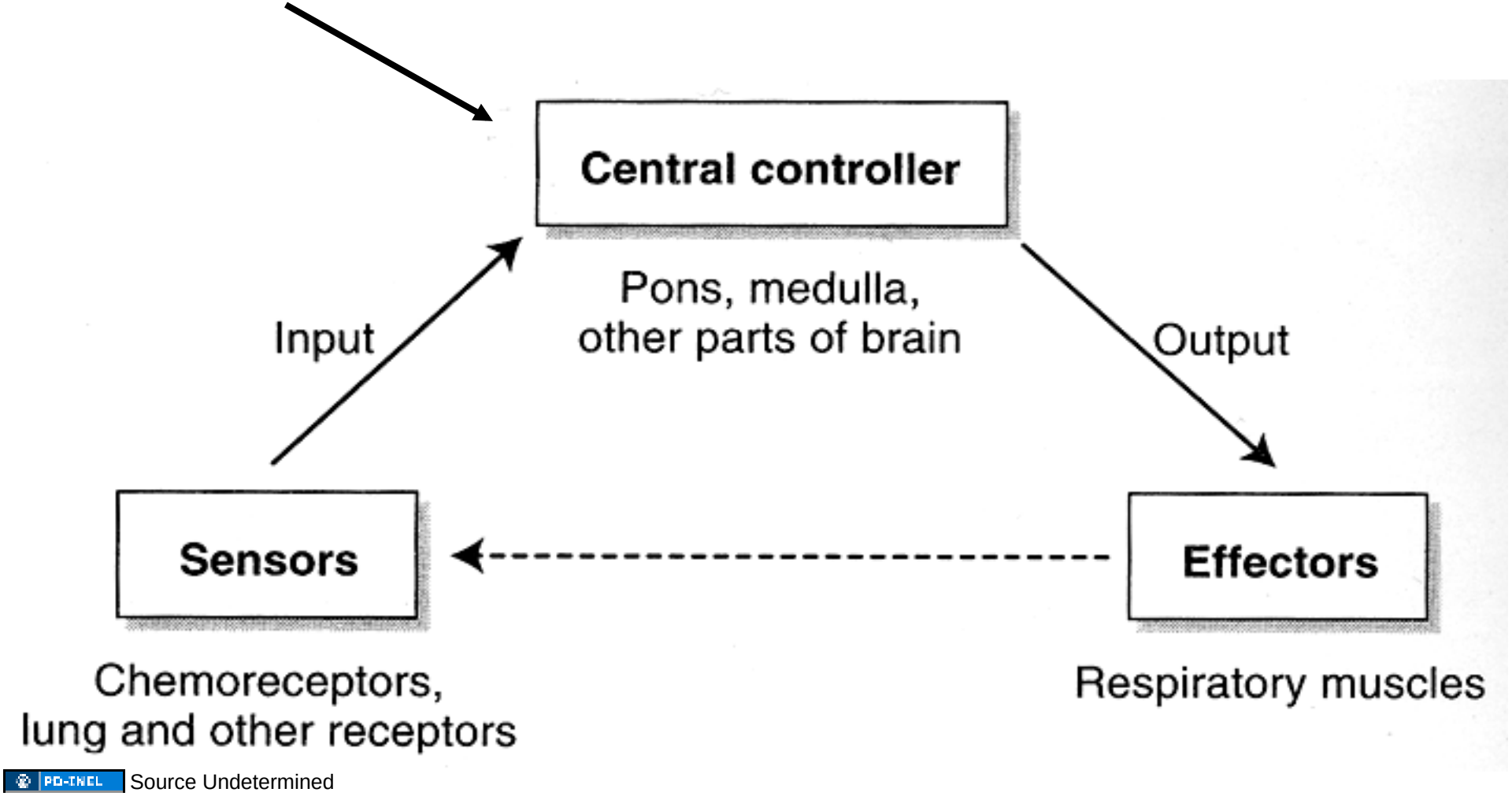
- Insensitive at normal PO_2
- O_2 *response all peripheral
- P_aO_2 <u>not content</u>

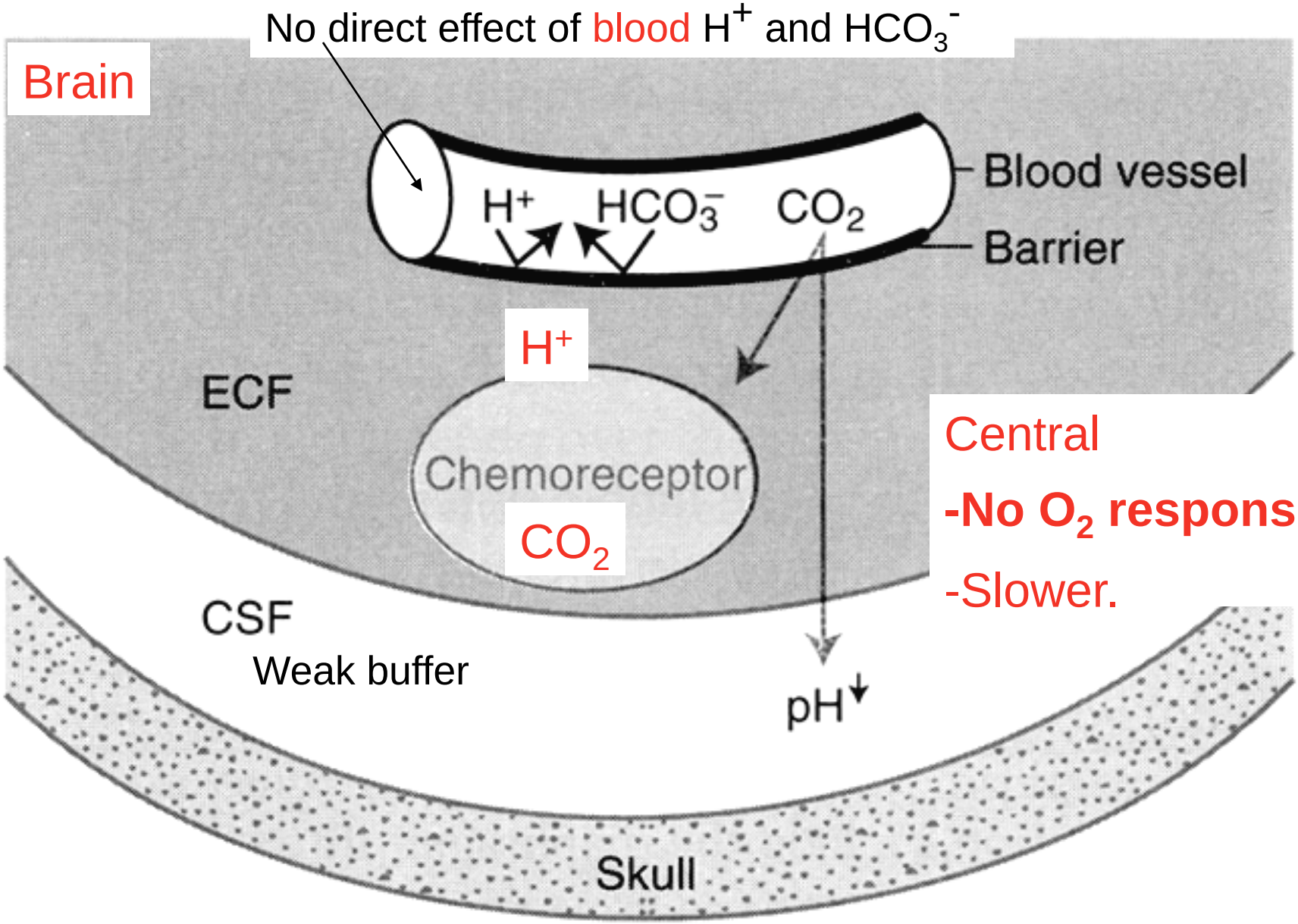
Does giving 100% O_2 to a hypercapnic patient suppress ventilation? Let's ask Dr. Sisson.

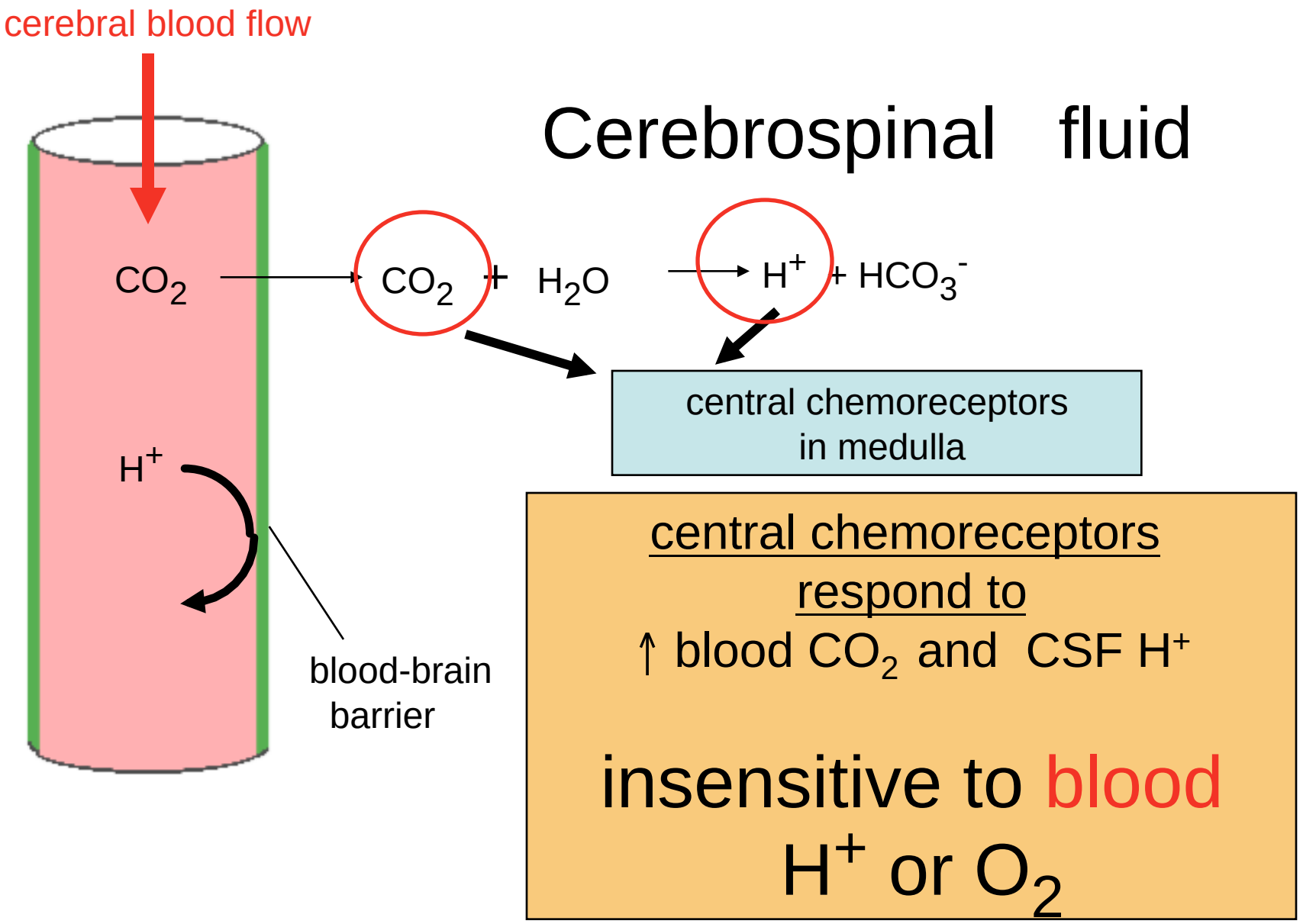


Low PO_2 response "fail safe".

What about central chemoreceptors ?






cerebral blood flow
Cerebrospinal fluid
CO_2
CO_2 + H_2O
H^+ + HCO_3^-
central chemoreceptors in medulla
H^+
blood-brain barrier
central chemoreceptors respond to
↑ blood CO_2 and CSF H^+
insensitive to blood H^+ or O_2

Summary:

Control of the respiratory system is based on responses to peripheral chemoreceptor stimulation resulting in tight regulation of arterial CO_2 **(P_aCO_2).**

Additional Source Information

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Slide 7: Source Undetermined
Slide 8: D'Alecy
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Slide 19: Source Undetermined
Slide 20: Levitzky. Pulmonary Physiology. McGraw-Hill, 2003. 6th ed.
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