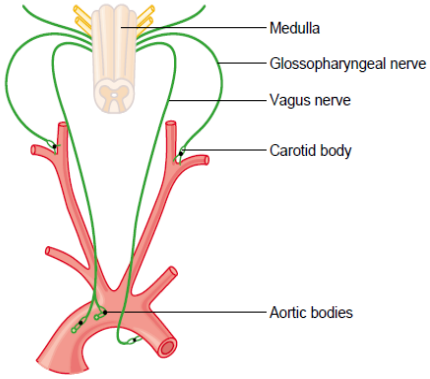
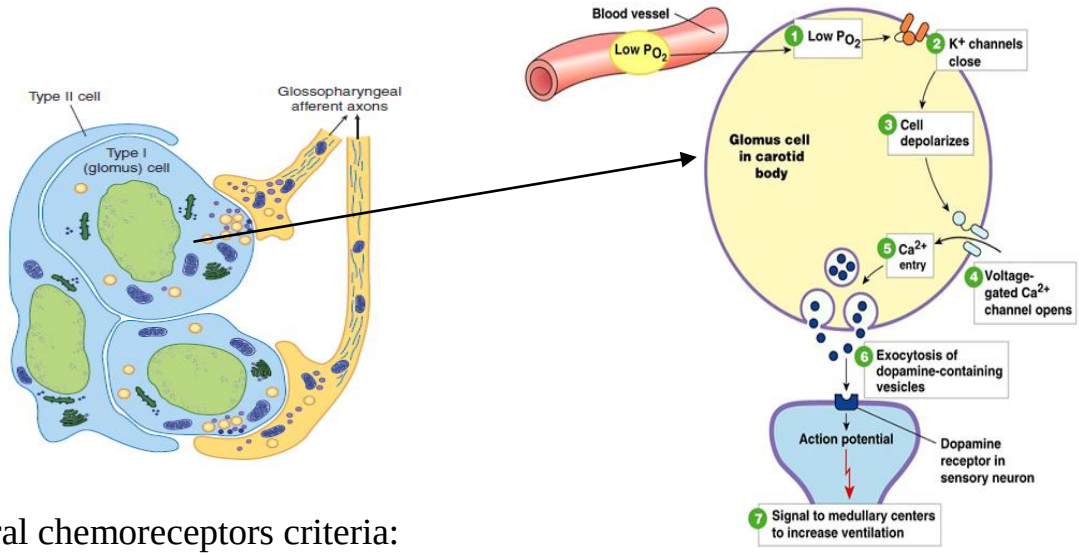
Peripheral chemoreceptors :

- **present in two locations**
  - **1-** Carotid bodies : located bilaterally at the bifurcation of common carotid artery. transmit impulses through glosopharyngeal nerve
  - **2-** Aortic bodies : located along the aortic arch and send impulses through vagus nerve.



➢ **Structure of carotid and aortic bodies** :

1. Type I or Glomus cells :
   They have O 2 -sensitive K + channels, their conductance ↓in hypoxia →reduces the K + efflux, →depolarizing the cell and causing Ca 2+ influx by voltage gated Ca_2^+ channels→ action potential and release of transmitter **(dopamine)** → excitation of the afferent nerve endings through **D2 receptors** .
2. The type II cells are glia-like, and each surrounds 4-6 type I cells.



Peripheral chemoreceptors criteria:

- They have huge blood supply→ O2 needs of the cell can be maintained by **dissolved O2** also makes the cells always exposed to the arterial PO2 .
- The response of carotid bodies in human > aortic bodies

- Removal of carotid bodies only → little change in ventilation at rest but the ventilator response to hypoxia is lost and ventilator response to CO2 ↓30 %

Note : The smooth muscle of pulmonary arteries contains similar O 2 - sensitive K + channels, which mediate the vasoconstriction caused by hypoxia.

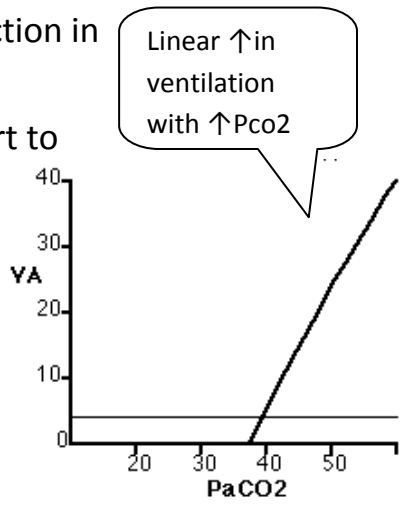
Factors stimulating peripheral chemoreceptors:

1. Po_2 (hypoxia is the main stimulant) and to a lesser extent to CO2 and H ion concentration .
2. Vascular stasis →↓ The amount of O_2 delivered to the receptors
3. Cyanide (prevent O_2 utilization at the tissue level)
4. ↑ plasma K^+ as in exercise (one reason for exercise induced hyperpnea).

- they respond only to a reduction in the dissolved O_2 in blood (PO2)
- There is No stimulation of respiration in anaemia and CO poisining ? Dissolved O2 is not affected in these conditions ,which is the main stimulant of Peripheral chemorecptors

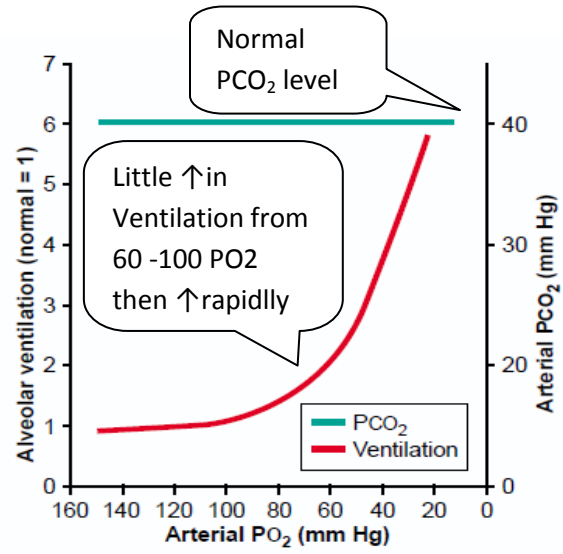
<u>Ventilatory response :</u> the respiratory center is designed to **optimize PaCO2** ,but changes in PaO and pH influence respiratory sensitivity to change in PaCO2 .

1) **Ventilator Response to changing in CO2 level** :
   - ↑tissue metabolism →↑PaCO2(very potent ventilator stimulant) →stimulate respiratory center through chemoreceptors→ ↑alveolar ventilation→ keep the excretion and production in balance
   - If the inspired gas is more than 7% CO2→the PCO2 start to ↑ in the blood inspite of the hyperventilation → (Hypercapnia)→depresses the central nervous system, including the respiratory center ,produce headache , confusion ,and coma (CO2 narcosis).



2) Ventilatory response to hypoxia :

- The ventilation is slightly ↑ when the PO_2 of the inspired air is **> 60 mm Hg** at normal PCO2(40 mmHg),why ?



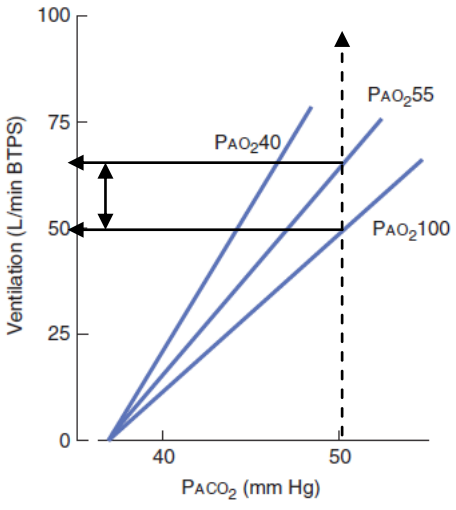
1. counter balance inhibitory effect by:
   - H^+ ion :↓PaO2→↑deoxy hemoglobin which is strong acid base buffer →binds H ion →plasma PH ↑→↓periphral chemoreceptor stimulation .
   - ↓PCO2 :hypoxia → ↑ ventilation → ↓PCO2 →inhibits respiratory center (because respiratory center main goal is to optimize CO2)
2. The of level of PaO_2 from 60 -100 mmHg represents the flat upper part of the O2-Hb dissociation curve in which there is a little change in Hb saturation when there is large change in PO_2

<u>interaction of chemical factors in regullation of respiration :</u>

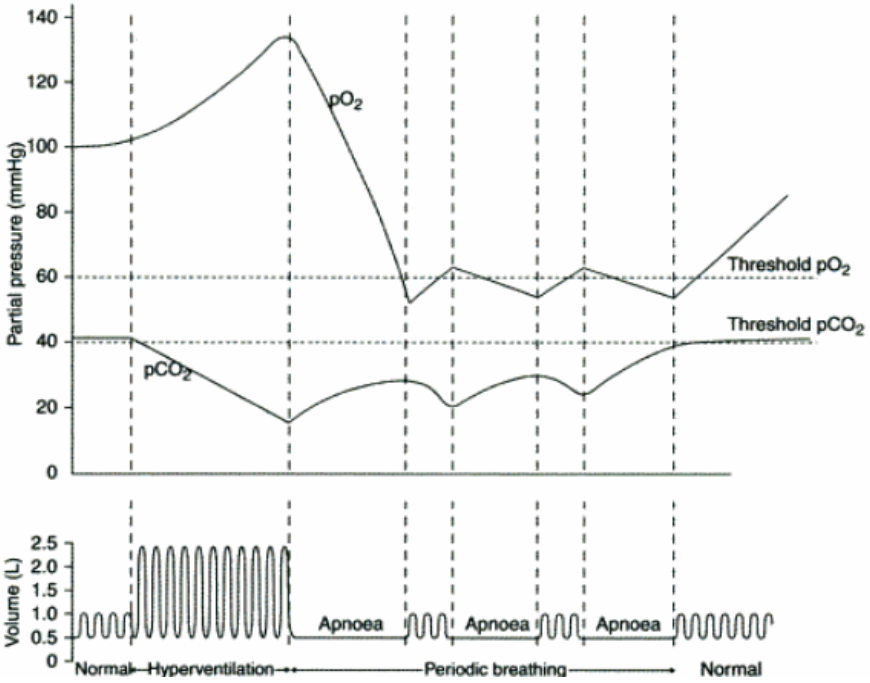
1. at PCO2 = 40 →ventilation ↑ **slightly** with hypoxia until PO2 <60 mmHg
2. at PCO2 > 40→ventilation **↑rapidly** as the PO2 ↓(potentiate hypoxia effect)
3. The ventilation response to PCO_2 increases when PO_2 ↓
   Hypoxia(↓PO2) increases chemoreceptors sensitivity to hyperacpnia (↑PCO2).
4. ↓pH →↑respiratory response to ↓ pCO2 : (additive effect)

Increase Ventilatory response to hypercapnia :

At PO2 =40mmHg >55mmHg > 100 mmHg



- **The acute effects of voluntary hyperventilation demonstrate the interaction of chemical mechanism regulating respiration , how ?**
  - If a person hyperventilates for 2‑3 min and then stops and allow respiration to continue without voluntary control →a period of apnea occurs followed by a few shallow breaths and then by another period of apnea, followed by a few breaths **(periodic breathing).** The cycles may last for some time before normal breathing is resumed .



Explanation :

- The apnea is due to a ↓ of CO 2 (main regulator of ventilation)
- During the apnea, the alveolar PO_2 ↓ and the PCO_2 ↑.
- Breathing resumes because of hypoxic stimulation of the carotid and aortic chemoreceptors before the CO 2 level has returned to normal.
- A few breaths eliminate the hypoxic stimulus, and breathing stops until the alveolar PO_2 ↓again. Gradually PCO_2 returns to normal, and normal breathing resumes.
- periodic breathing may present in some diseases. ex: congestive heart failure and brain disease