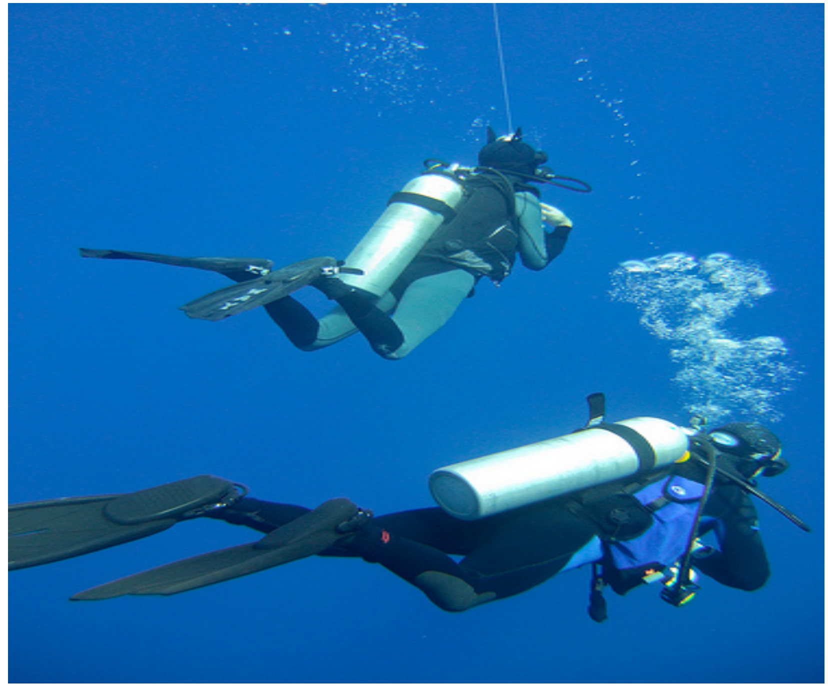
Effects of low and high gas pressure on the body



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Objectives

By the end of this lecture you should be able to:

1-Describe the effects of exposure to low and high barometric pressures on the body.

2- Describe the body acclimatization to low barometric pressure.

3-Define decompression sickness and explain how it can be avoided.

4-Understand the effects of high nitrogen pressure, and nitrogen narcosis.

Effect of increased barometric pressure (Deep sea diving)

- When human descends below the sea, the pressure around them increased.
- To prevent the lungs from collapse air must be supplied also under high pressure.
- This exposes the blood in the lungs to extremely high alveolar gas pressure (hyperbarism).
- Under certain limits these high pressures cause tremendous alterations in the physiology of the body.

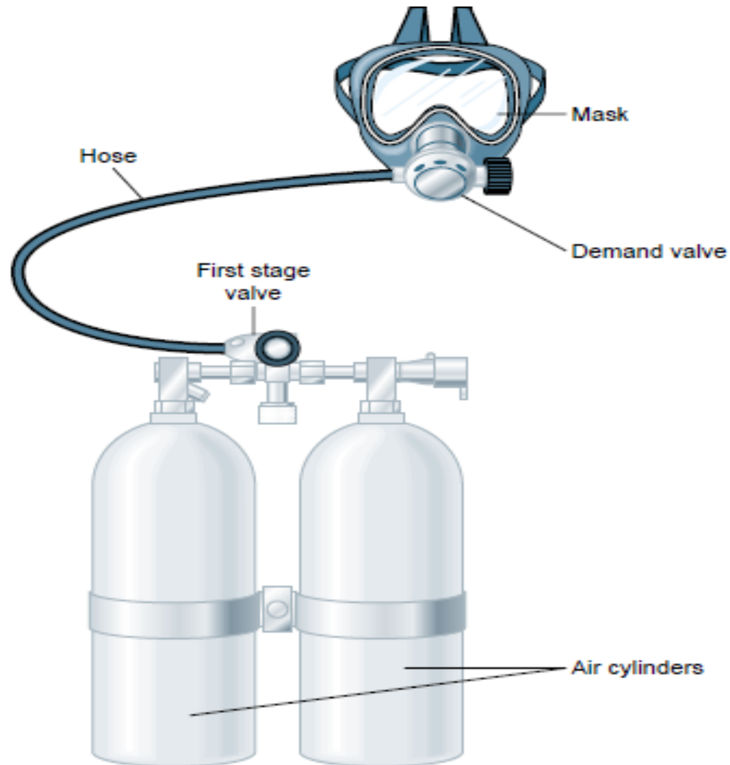


Figure 44–4

Open-circuit demand type of SCUBA apparatus.

Cont..

The surrounding pressure increases by 1 atmosphere for every 10 meters (33 feet) of depth in sea water. So the diver is exposed to 2 atmospheric pressures.

Therefore, at a depth of 31 meters (100 feet) in the ocean the diver is exposed to a pressure of 4 atmospheres.

These problems confront SCUBA (self contained under water breathing apparatus..)

Effect of depth on the volume of the gases:

At depth, there is compression of gases to smaller and smaller volumes. i.e 1L (sea level)→1/2 L at 33 feet and so on.

Effect of depth on density of gases:

There is increase in the density of gas and hence increased work of breathing.

Nitrogen effect at high nitrogen pressure:

Nitrogen will have 2 principle effects:

* Nitrogen narcosis (anesthetic effect)

* Decompression sickness.

Depth (feet)	Atmosphere(s)
Sea level	1
33	2
66	3
100	4
133	5
166	6
200	7
300	10
400	13
500	16

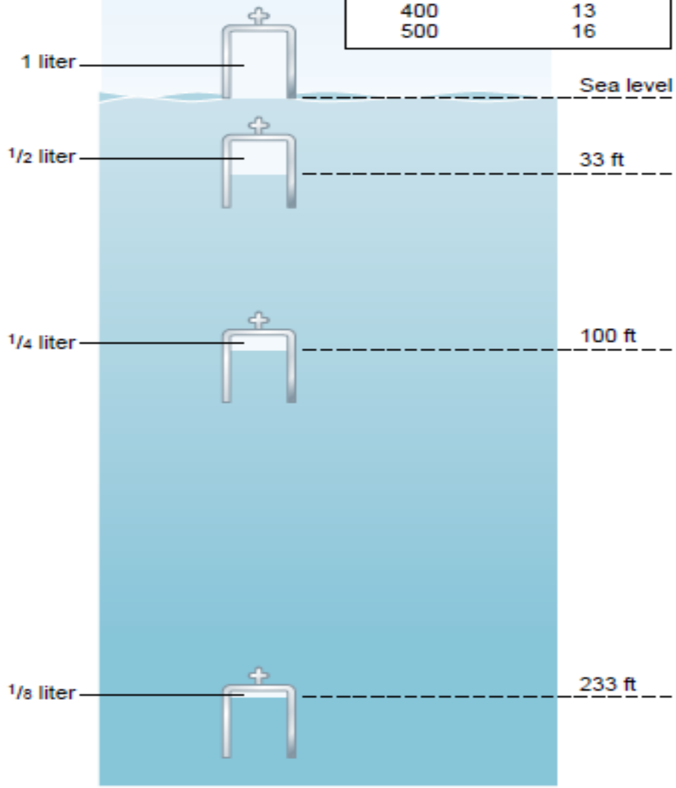


Figure 44–1

Effect of sea depth on pressure (*top table*) and on gas volume (*bottom*).

Oxygen toxicity when breathing hyperbaric air (Effect of Very High PO2 on Blood Oxygen Transport)

When the Po2 in the blood rises above 100 mm Hg, the amount of oxygen dissolved in the water of the blood increases markedly.

Acute Oxygen Poisoning:

The extremely high tissue Po2 that occurs when oxygen is breathed at very high alveolar oxygen pressure can be detrimental to many of the body's tissues.

At 4 atmospheres pressure of oxygen (Po2 = 3040 mm Hg) will cause brain *seizures followed by coma* in most people within 30 to 60 minutes.

Other symptoms include nausea, muscle twitchings, dizziness, disturbances of vision, irritability, and disorientation.

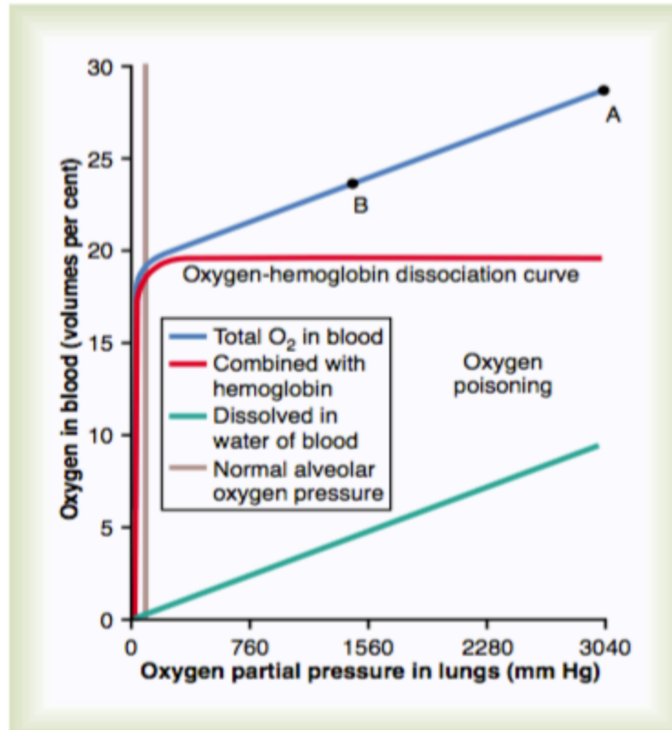


Figure 44–2

Quantity of oxygen dissolved in the fluid of the blood and in combination with hemoglobin at very high Po_2s.

Cont. Oxygen toxicity at a high PO2.

- Molecular oxygen (O2) has little capability of oxidizing other chemical compounds. Instead, it will first be converted into an "active" form of oxygen called: oxygen free radicals.

For example: superoxide and hydrogen peroxide.

- At high levels, these oxygen free radicals can have serious destructive and even lethal effects on the cells.

Nitrogen narcosis

Nitrogen, like most other anesthetic gases, dissolves freely in the fats of the body including the membranes and other lipid structures of the neurons(after one hour).

This leads to alteration of the electrical conductance of the membranes, reduces their excitability and subsequent narcosis develops.

At 120 feet: the diver loses many of his cares.

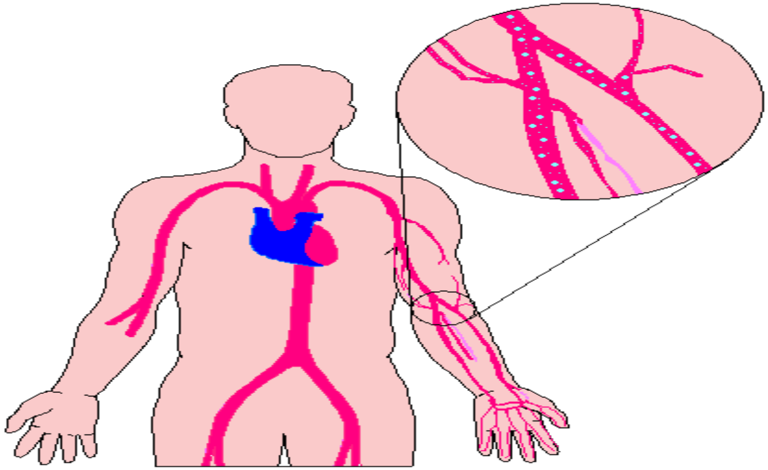
At 150 feet: there is a feeling of euphoria and drowsiness and impaired performance.

At higher pressure: loss of coordination

Feet	Liters
0	1
33	2
100	4
200	7
300	10

Decompression Sickness (Bends, Compressed Air Sickness, Caisson Disease, Diver's Paralysis, Dysbarism).

- During descending, the high partial pressure of nitrogen (encountered when breathing compressed air at depth) forces this gas into solution in body tissue particularly in fat (it has a high N2 solubility).
- On ascending, this inert gas comes out of physical solution forming a gaseous phase (bubbles), leading to symptoms and signs.

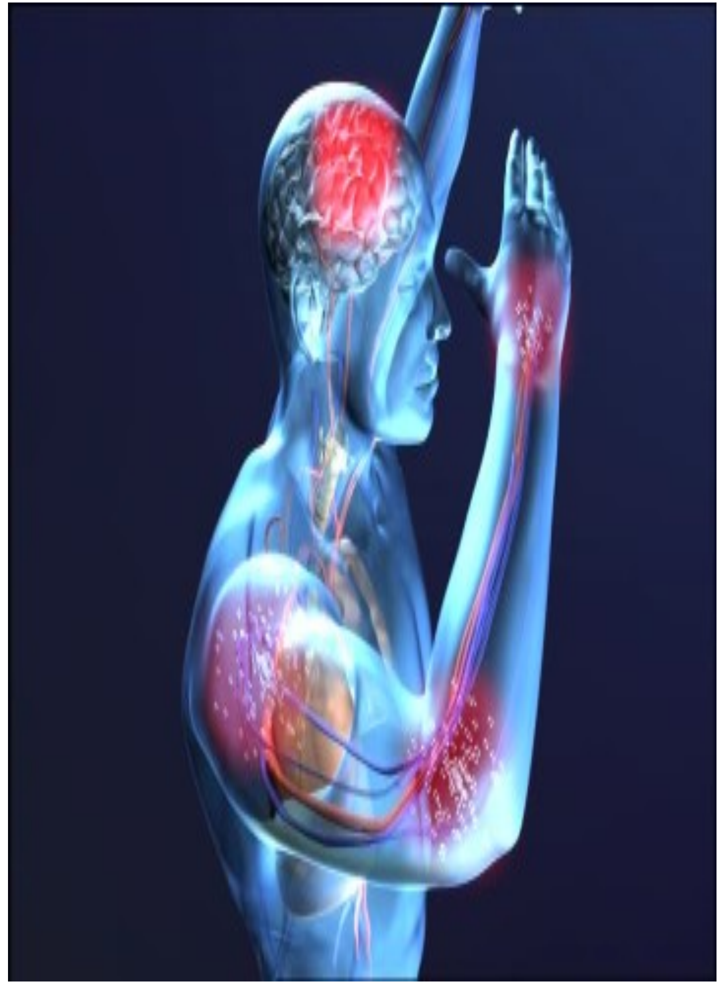


Cont.. Decompression sickness

- During slow ascending:

N2 is slowly removed from the tissues since the partial pressure is higher than that in the arterial blood and alveolar gas.

- If decompression is rapid: bubbles of gaseous nitrogen are released, in tissues and blood, causing the symptoms of decompression sickness (the bends or caisson disease).



Symptoms & signs of decompression sickness

The mildest form of DS is fatigue or drowsiness after decompression.

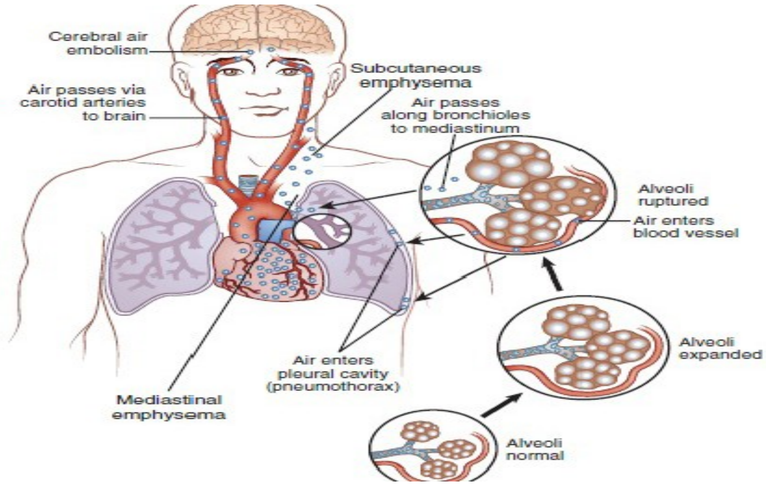
Locally there is skin itch.

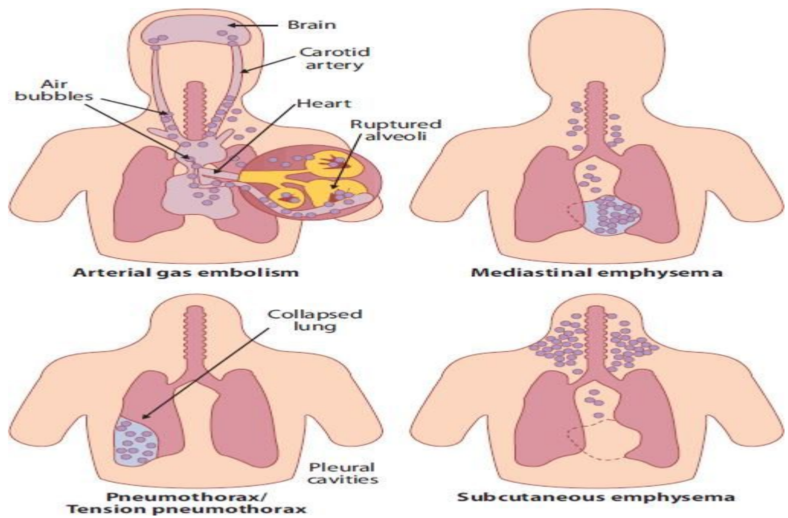
other sever symptoms may occur e.g.:bubbles in the tissues cause severe pains particularly around the joints.

Neurological symptoms including paresthesia, itching, paralysis, and inner ear disturbances.

Cont.. Decompression Sickness (S&S)

- Thoracic pains: dyspnea, substernal pain, cyanosis, and cough.
- Bubbles in the coronary arteries may cause myocardial damage.
- Decompression sickness shock, capillaries become permeable to plasma and hypovolemia rapidly develop.
- Edema may be prominent and shock is also usually complicated by pulmonary edema.





Treatment of decompression symptoms

Rapid recompression in a pressure chamber followed by slower decompression.

This reduces the volume of the bubbles and forces them back into solution.

In a very deep dive, the risk of decompression sickness can be reduced if a helium-O2 mixture is breathed during the dive.

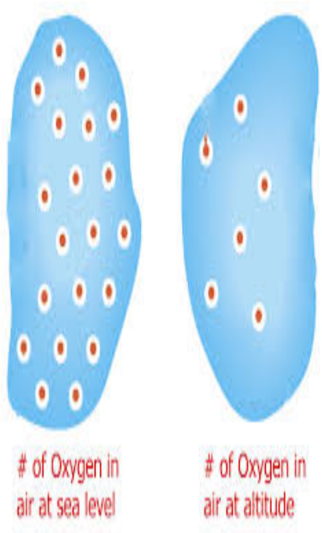
Also it is important to reduce the oxygen concentration in the gaseous mixture to avoid oxygen toxicity that would

Cont.. Treatment of Decompression sickness

- Helium is more desirable than nitrogen in deep dives because it has:
- ¼-1/5 the narcotic effect of nitrogen on CNS.
- 1/7 the molecular weight of nitrogen.
- low density leading to decreased air way resistance of diver.
- Helium is about 1/2 as soluble as nitrogen in body fluids. This reduces the quantity of bubbles that can form in tissues when the diver is decompressed after diving.
- Diffuses out of the tissues during decompression several times as rapidly as does nitrogen, thus reducing the problem of decompression sickness.

Effects of low oxygen pressure on the body
(Aviation-ascend to high altitude)

- At the sea level the barometric pressure is 760 mmHg.
- At 10,000 feet is 523 mmHg
- At 50,000 feet 87 mmHg.
- This decrease in barometric pressure is the basic cause of all the hypoxia problems in high altitude.



Alveolar PO2 at different altitudes

- As the barometric pressure decreases, the oxygen partial pressure decreases proportionally, remaining less than 21 % of the total barometric pressure:
- At sea level PO2= 159 mmHg.
- At 20,000 feet PO2= 40 mmHg.
- At 50,000 feet PO2= only 18 mmHg.

Effects of acute hypoxia

- Some of the important acute effects of hypoxia beginning at an altitude of approximately 12,000 feet, are: Drowsiness, lassitude, mental and muscle fatigue, sometimes headache, occasionally nausea and sometimes euphoria.
- All these progress to a stage of twitching or convulsions above 18,000.
- Above 23,000 feet the un acclimatized person can enter into coma.

Chronic Breathing of Low O2 Stimulates Respiration — Phenomenon of "Acclimatization"

- Mountain climbers have found that when they ascend a mountain slowly, over a period of days rather than a period of hours, they breath much more deeply and therefore can withstand far lower atmospheric O2 concentrations than when they ascend rapidly. This phenomenon is called *acclimatization*.
- The reason for acclimatization is that, within 2 to 3 days, the respiratory center in the brain stem loses about four fifths of its sensitivity to changes in PCO2 and hydrogen ions.
- It causes fewer deleterious effects on the body and it becomes possible for the person to work harder without hypoxic effects or to ascend to still higher altitude.

Principles of acclimatization

1- Increase in pulmonary ventilation.

2- Increased red blood cells.

3-Increased diffusing capacity of the lungs.

4- Increased vascularity of the tissues.

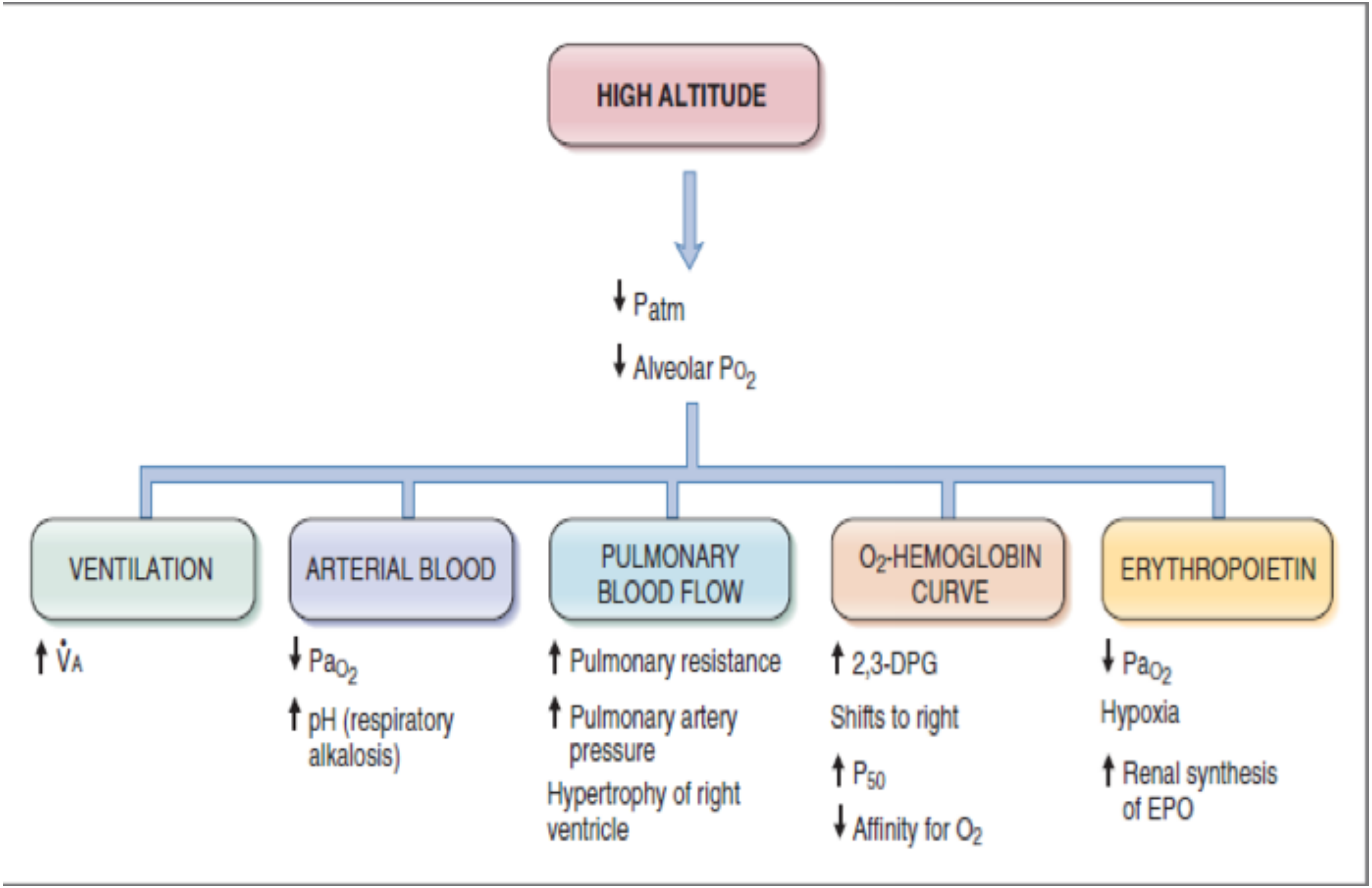
5-Increased ability of the cells to utilize oxygen despite the low PO2 through increased number of mitochondria and oxidative enzymes activity.

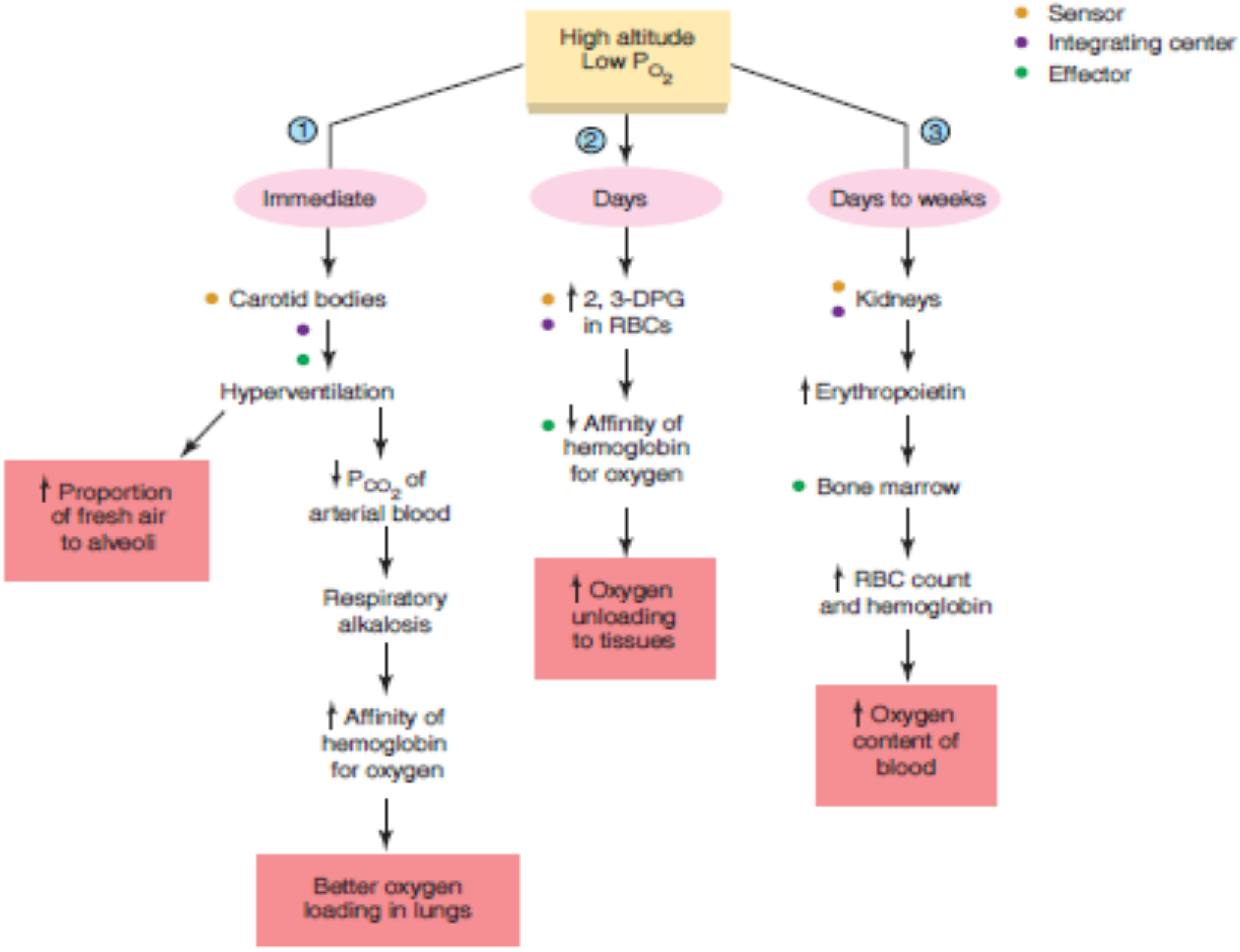
TABLE 5.4 Summary of Adaptive Respiratory Responses to High Altitude

Parameter	Response to High Altitude
Alveolar P_{O_2}	↓ (due to decreased barometric pressure)
Arterial P_{O_2}	↓ (hypoxemia)
Ventilation rate	↑ (hyperventilation due to hypoxemia)
Arterial pH	↑ (respiratory alkalosis due to hyperventilation)
Hemoglobin concentration	↑ (increased red blood cell concentration)
2,3-DPG concentration	↑
O_2-hemoglobin dissociation curve	Shifts to right; increased P_{50}; decreased affinity
Pulmonary vascular resistance	↑ (due to hypoxic vasoconstriction)
Pulmonary arterial pressure	↑ (secondary to increased pulmonary resistance)

2,3-DPG, 2,3-diphosphoglycerate.

Acclimatization to low PO2





Sensor
Integrating center
Effector
High altitude
Low P_{O_2}
1
2
3
Immediate
Days
Days to weeks
Carotid bodies
Hyperventilation
↑ Proportion of fresh air to alveoli
↓ P_{CO_2} of arterial blood
Respiratory alkalosis
↑ Affinity of hemoglobin for oxygen
Better oxygen loading in lungs
↑ 2, 3-DPG in RBCs
↓ Affinity of hemoglobin for oxygen
↑ Oxygen unloading to tissues
Kidneys
↑ Erythropoietin
Bone marrow
↑ RBC count and hemoglobin
↑ Oxygen content of blood