Narcosis

There are some theories, explaining the mechanism of narcosis.

Lipid Theory. The nerve cells and membranes contain lipids, the anesthetic is thought to gain access to nerve tissue by virtue of its lipid solubility.

Adsorption Theory. The adsorption of anesthetic agents may change the effective dielectric constant and permeability and may also alter structural relationships between those enzymes supporting oxidative phosphorylation and electron transport.

Cell Permeability Theory. The anesthetics cause a change in permeability of the cells of the CNS. Certain agents may physically stabilize the cell membrane by preventing the increase in ion permeability. Some anesthetics partially prevent the decrease in membrane resistance that normally occurs during excitation. They depress the selective permeability of the cell to **sodium ions**.

Biochemical Theory. Recent investigations have demonstrated that clinically used concentrations of inhalational anesthetics markedly and reversibly depress the oxidation of **glutamate and NADH**. They inhibit the oxygen consumption. **Calcium uptake** brain mitochondria is inhibited.

Neurophysiological theory. Anesthetics **inhibit the ascending reticular formation,** which is very important for the maintenance of wakefulness.

Physical theory. Anesthetic agents within the CNS are able to orient water molecules around them in an order manner. This interaction with water (rather than with lipid) results in the formation of **hydrated microcrystals**, which **can interfere with neuronal excitability**.

Physicochemical theory. Anesthetics can expand **the lipid phase of the membrane**, thus increasing fluidity or disorder or, perhaps, altering the shape of pores within the membrane. Narcosis may arise either as a direct result of these changes or be produced by changes in enzyme activity.

Uptake and distribution

One of the most important factors influencing the transfer of an anesthetic from the lungs to the arterial blood is **its solubility**. When an anesthetic with low blood solubility diffuses from the lung into the arterial blood, relatively few molecules are required to raise its partial pressure and the **arterial tension rises** quickly. Conversely, for anesthetics with moderate to high solubility, more molecules dissolve before partial pressure changes and arterial tension of the gas increases less rapidly. Changes in the rates of blood flow to and from the lungs influence transfer processes of the anesthetic gases. **An increase in pulmonary blood flow** (increased cardiac output) **slows the rate of rise in arterial tension**, particularly for those anesthetics with moderate to high blood solubility. This is because **increased pulmonary blood flow exposes a larger volume of blood** to the anesthetic; thus, blood "capacity" increases and tension rises slowly. **A decrease in pulmonary blood** has the opposite effect and **increases the rate of rise of arterial tension** of inhaled anesthetics.

Organ System Effects of Inhaled Anesthetics
Effects on Cardiovascular System:

- **all decrease arterial pressure** in direct proportion to their alveolar concentration;
- the reduced arterial pressure appears to be caused by a **reduction in cardiac output**, because there is little change in systemic vascular resistance despite marked changes in individual vascular beds (e.g. increase in cerebral blood flow);
- other anesthetics have a **depressant effect on arterial pressure** as a result of an endothelium – mediated decrease in systemic vascular resistance; they have **little effect** on cardiac output;
- inhaled anesthetics change heart rate either by directly altering the rate of sinus node, depolarization or balance of autonomic nervous system activity;
- **bradycardia** is often seen;
- all inhaled anesthetics (IA) tend to **increase right atrial pressure** which reflects **depression of myocardial function**;
- IA **reduce myocardial oxygen consumption.**

Effect on Respiratory System

All inhaled anesthetics cause a **decrease in tidal volume and an increase in respiratory rate**. They are **respiratory depressants.** Inhaled anesthetics also depress **mucociliary function** in the airway. Thus, prolonged anesthesia may lead to pooling of mucus and then result in **atelectasis and respiratory infections.** However, IA tend to be **bronchodilators.** This effect has been used in treatment of status asthmaticus.

Effects on Brain

IA decrease the metabolic rate of the brain. Nevertheless, most of them **increase cerebral blood flow** because they **decrease cerebral vascular resistance**.