Hypoxia: Its Causes and Symptoms

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> As exclusion of oxygen extinguishes a fire, so, too, in a man or animal a deficiency of oxygen in the blood and tissues induces death. There are many modes of dying, but there is only one final common cause of death. Whether the brain is destroyed, the lungs blocked, or the heart stopped, death finally occurs in but one way only. When breathing and the heart action come to a standstill, the supply of oxygen ceases. Unless a man is burned alive, the tissues of his body always die of anoxia. *(from "The Complications of General Anesthesia in Dentistry as Related to the Respiratory System", Douglas, B. I., Oral Surgery, Oral Medicine, Oral Pathology, 7:2, Feb. 1954).*

The failure of the tissues, for any reason, to receive an adequate supply of oxygen, is known as hypoxia or oxygen want.[1] Throughout the literature, the term "anoxia" has been used to designate a want of oxygen in the body. This term is a misnomer. Anoxia, literally translated, means "without oxygen." A patient "without oxygen" is dead. Hypoxia, therefore, is the more appropriate term.

Best and Taylor classify hypoxia into four main types.[2] These are:

(1) hypoxic hypoxia, (2) anemic hypoxia, (3) stagnant hypoxia, and (4) histotoxic hypoxia. To this listing a newer and fifth type of hypoxia may be added. It is known as fulminating hypoxia.

Hypoxemia is a term used to denote a low oxygen content in capillary blood.[3] In the first three types of hypoxia mentioned, hypoxemia is also present.

HYPOXIC HYPOXIA

In hypoxic hypoxia, there is a lack of oxygen in the arterial blood. The oxygen tension is lowered in both the lungs and the arterial blood, and the hemoglobin is not saturated with oxygen to its normal extent. This type of hypoxia affects the body as a whole and is one of the most serious forms of hypoxia.

Hypoxic hypoxia is often produced by low tensions of oxygen in the inspired air as is seen in high altitudes, breathing of inert gases, and the inhalation of anesthetic agents. Abnormal lung conditions may also produce hypoxic hypoxia. Emphysema, asthma, pneumonia, or pneumothorax encourage the formation of this type of hypoxia. Mechanical obstruction of the airway by foreign objects, laryngospasm, or bronchospasm inhibits the flow of oxygen from the atmosphere into the lungs, creating a state of oxygen want. Shallow respiratory movements from any cause, with either a decrease in rate or amplitude, may cause hypoxic hypoxia. A chronic state of hypoxic hypoxia may result from a patent foramen ovale and other embryo-

logical malformations of the heart and blood vessels.

ANEMIC HYPOXIA

The arterial blood contains oxygen at its normal tension in anemic hypoxia, but there is a shortage of functioning hemoglobin. Anemic hypoxia, on the whole, is less serious than hypoxic hypoxia. However, it does affect the whole body. Anemic hypoxia may be caused by acute or chronic hemorrhage, primary or secondary anemia, alterations in the hemoglobin of the blood (caused by nitrates, chlorates, or coal tar derivatives), and carbon monoxide poisoning.

STAGNANT HYPOXIA

Stagnant hypoxia is due to a decrease in the rate of flow of the circulating blood. Local regions of the body are usually involved, but it may affect the entire body.

The blood is saturated normally with oxygen, and the oxygen load, as well as the tension under which it is held, also may be normal. Hypoxia is produced because the amount of oxygen reaching the tissues is inadequate. Sluggishness in the rate of the circulating blood allows the blood to stagnate and give up a greater percentage of its oxygen. This slow circulation also permits the accumulation of a greater quantity of carbon dioxide in the tissues. Stagnant hypoxia is produced by failure of the circulation, impairment of venous return, and shock.

HISTOTOXIC HYPOXIA

As the term suggests, the tissue cells are poisoned and are unable to accept oxygen from the capillaries. In this type of hypoxia the cells are not able to utilize the oxygen, although the amount of oxygen in the blood may be normal and under normal tension. Histotoxic hypoxia is produced by cyanides. Theoretically, it may be produced by any agent which depresses cellular respiration.

FULMINATING HYPOXIA

Fulminating hypoxia is a newly recognized form of hypoxia. It is a very rapidly induced type of hypoxia caused by the inhalation of undiluted inert gases such as nitrogen, methane, or helium. In anesthesia, fulminating hypoxia may be produced by administering nitrous oxide anesthesia without the simultaneous use of oxygen.

EFFECTS OF HYPOXIA UPON THE BODY

Heart Rate. Acute hypoxia produces acceleration of the heart rate. As the barometric pressure is decreased, the heart rate is proportionately increased. The pulse rate will also vary according to the activity of the patient.

Heart and Circulation. In the early phases of hypoxia, a reduction in the effective venous pressure is produced, causing a shortening of the systolic phase of ejection of the heart. Hypoxia raises the initial tension in the left ventricle and also increases the ventricular ejection. These two opposing factors,

during the early phases of progressive hypoxia, remain evenly balanced and tend to maintain or slightly increase the normal systolic discharge. These beneficial effects of hypoxia, however, persist only until the concentration of oxygen drops to 9 per cent. Then, a circulatory crisis occurs. There is first a decrease in both the systolic and diastolic pressures and a reduction in pulse pressure. Beginning signs of circulatory failure appear because of a reduction in the minute output of the heart. The heart becomes slower, the ejection phase is still further shortened, and circulatory collapse becomes imminent, since the combination of these factors causes the cardiac output to diminish steadily. The blood pressure soon drops to a critical level incompatible with life.

Coronary Circulation. The volume of coronary flow increases greatly when the oxygen is reduced to 8 or 9 per cent. Hypoxia is a more powerful vasodilator of the coronary vessels than drugs such as amyl nitrate or histamine. Vasodilation at this time acts as a valuable compensatory mechanism while the patient is being subjected to severe hypoxia.

Conduction System of the Heart. Severe degrees of hypoxia produce a delayed action of the conduction system by directly affecting the conduction tissue.

Chemoreceptor Reflexes. Hypoxia stimulates the respiratory center reflexly by acting on the sensory nerve endings in both the aortic and carotid bodies. The threshold of stimulation of the chemoreceptors to hypoxemia is decidedly less than that of the respiratory center being stimulated by carbon dioxide.

The Difference Between Asphyxia and Hypoxia. During asphyxia there is an accumulation of carbon dioxide in the blood and tissues (hypercarbia). The oxygen content of the blood remains unchanged. Carbon dioxide accumulation causes a rise in blood pressure by direct action on the vasomotor center, and, to a much smaller degree, upon the carotid and aortic bodies. Hypercarbia therefore acts differently from hypoxia, since the main effects of hypoxia occur reflexly through aortic and carotid body response.

HYPOXIA IN ANESTHESIA AND SURGERY

Mechanical obstruction of the respiratory passages is the most common cause of hypoxia in anesthesia and surgery. Some of the more common mechanical obstructions are:

(1) Retrusion of the mandible following relaxation, permitting the base of the tongue to obstruct the epiglottis (this is epecially evident following the use of muscle relaxants in anesthesia).

(2) Mucus, blood, and other foreign bodies in the air passages.

(3) Spasm of the muscles of mastication (especially during the

induction or immediate postoperative periods of anesthesia).

(4) Laryngospasm.

Hypoxemia may result from inadequately controlled or assisted respirations in anesthesia. An overdosage of anesthetic agents may also cause the development of hypoxemia.

A patient lying in the supine position has less difficulty with respiration than one placed in a bizarre position. The ideal position on the operating table is the Fowler's or semi-sitting position. Positions such as the jacknife or Trendelenburg, decrease venous return and apply added pressure on the thorax and abdomen, thus increasing the incidence of hypoxic and stagnant hypoxia.

Spinal anesthesia, extending so high as to produce paralysis of the intercostal muscles, encourages the formation of hypoxic and stagnant hypoxia. Various drugs, such as morphine sulfate, the barbiturates and avertin, used preoperatively, depress metabolism and tend to produce histotoxic hypoxia.

CLINICAL SIGNS

The first clinical sign noted in the hypoxic patient is cyanosis. A bluish discoloration appears first in the capillary beds of the mucosa of the lips and lobes of the ears. Next, the conjunctiva and the fingernails take on a bluish tinge. Finally, a duskiness appears over the whole body.

A marked increase in rate with moderate increase in amplitude is the first respiratory indication of hypoxemia. A continued increase in rate with decreasing depth indicates approaching respiratory fatigue.

The pulse rate first increases and then may decrease in volume as well as rate. A temporary marked increase in blood pressure usually accompanies the rapid pulse.

Heart failure during anesthesia may occur from failure of the heart muscle, itself, due to acute hypoxia, or to failure of the myocardial conduction system.

The nervous system may show slight or severe effects. With the onset of hypoxia, the first symptoms are those of analgesia with exhilaration, warmth, and euphoria. The tongue feels thick and speech is slurred. Muscular rigidity is also an early sign in hypoxia.

Prolonged cases of hypoxemia lead to slower return to conciousness and the possible onset of postoperative headache and depression.

TREATMENT

Prevention is the best treatment. Proper evaluation of the patient is essential. Hypoxemia should be especially avoided in subnormal risk, toxic, and anemic patients. Patients with acute or chronic diseases often have some degree of hypoxia. Such patients especially cannot be further subjected to the dangers of hypoxia.

A patent airway is necessary at all times.

Administration of oxygen, sufficient to meet basal metabolic requirements, is vital.

In cases that have progressed to the point of respiratory depression and arrest, resuscitation is mandatory, with the administration of adequate amounts of oxygen under positive pressure.

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REFERENCES

1. BEST, C. and TAYLOR, N.—*The Physiological Basis of Medical Practice*, Third Edition, The Williams and Wilkins Company, Baltimore, 1943, p. 591.
2. *Ibid.*
3. *Ibid.*

The History of General Anesthesia* (Part II)

Published in The Boston *Atlas*, April 2, 1847

Paris, Feb. 17, 1847.

"Sir: — As you have recently published an extract from the Boston Medical and Surgical Journal, which recognizes me as the discoverer of the happy effects produced by the inhalation of exhilarating gas or vapor for the performance of surgical operations, I will now offer some suggestions in reference to this subject. Reasoning from analogy, I was led to believe that surgical operations might be performed without pain, by the fact that an individual, when much excited from ordinary causes, may receive severe wounds without manifesting the least pain; as, for instance, the man who is engaged in combat may have a limb severed from his body, after which he testifies that it was attended with no pain at the time; and so the man who is intoxicated with spirituous liquor may be treated severely without his manifesting pain, and his frame seems in this state to be more tenacious of life than under ordinary circumstances. By these facts I was led to inquire if the same result would not follow by the inhalation of some exhilarating gas, the effects of which would pass off immediately, leaving the system none the worse for its use. I accordingly procured some nitrous oxide gas, resolving to make the first experiment on myself, by having a tooth extracted, which was done without any painful sensations. I then performed the same operation for twelve or fifteen others, with the like results; this was in November, 1844. (Author's note: Dr. Wells was mistaken in his dates.) Being a resident of Hartford, Connecticut, (U.S.), I proceeded to

* From *Life and Letters of Horace Wells*, by W. Harry Archer.