OXYGEN AND CARBON DIOXIDE TRANSPORT

Y.J. Salorinne and P. Haapalahti
Department of Clinical Physiology and Nuclear Medicine, Helsinki University Hospital and Helsinki University Medical Faculty, Helsinki, Finland.

Keywords: Hemoglobin, red cells, oxygen saturation, 2,3-diphosphoglycerate, mitochondria, acid base regulation, protein buffers.

Contents

1. Introduction
2. Oxygen Hemoglobin Dissociation Curve
3. Oxygen Delivery
4. Carbon Dioxide Transport
Glossary
Bibliography
Biographical Sketches

Summary

The binding and release of oxygen and carbon dioxide in blood must occur appropriately to fulfill the needs of tissues. The main characteristics of binding, transport and delivery have been clarified and described in sufficient detail to enable modern treatment of difficult respiratory and circulatory conditions.

1. Introduction

The partial pressure of oxygen in systemic venous blood entering the lungs is approximately 5 kPa. In the pulmonary capillary it increases to 13 kPa. At that pressure blood can only hold about 3 ml O₂ in physical solution.

The normal hemoglobin concentration of 150 g/l allows the red cells to carry about 193 ml oxygen per liter of blood, i.e. almost 65 times more than in physical solution.

Moreover, the dissociation curve of oxyhemoglobin (HbO₂) is a special sigmoid that describes how oxygen binding in the lungs and release in the target tissues are optimized. Figure 1 shows the relation of the blood oxygen saturation (SO₂) and partial pressure.

The arterial blood is almost fully saturated with oxygen and the O₂-saturation in the returning venous blood is still about 75%. In the normal situation 1 liter of arterial blood holds almost 200 ml of oxygen and if the cardiac output is 5 liters per minute, then the oxygen delivery is 1000 ml/min.

The resting consumption is approximately 250 ml/min, i.e. only 25% of the oxygen content is being used.
2. Oxygen Hemoglobin Dissociation Curve

The oxygen affinity of hemoglobin varies in many situations. Oxygen is released more readily i.e. the curve is displaced to the right (Figure 2) compared to the standard curve if the temperature rises, carbon dioxide pressure (PCO₂) or hydrogen ion concentration [H+] increases or there is more 2,3-diphosphoglycerate in the blood.

Various genetic forms of hemoglobin have a slightly different oxygen carrying capacity and they have been detected by measuring the 50% saturation pressure (P50). Fetal hemoglobin (HbF) is favorable to the fetus. In the newborn it is soon replaced by the adult form of hemoglobin (HbA).

Some forms of poisoning change the oxygen binding to hemoglobin profoundly. Carbon monoxide (CO) is especially dangerous, because it both changes the form of the dissociation curve and binds irreversibly to the oxygen binding site of hemoglobin (Figure 3). Nitrates and sulfates can oxidize the iron in hemoglobin and impair the oxygen carrying capacity.

Bibliography


Biographical Sketches

Yrjö Salorinne, MD, PhD, is presently chief physician in the department of clinical physiology and nuclear medicine at the Helsinki University Hospital and associate professor of clinical physiology in the medical faculty of the University of Helsinki, Finland. His scientific works are mainly on respiratory physiology and lung diseases, and he is a co-author on many textbooks and reviews. He has been active in medical organizations and a co-editor in the journal of Clinical Physiology.

Petri Haapalahti, MD, PhD, is at present a senior physician in the department of clinical physiology and nuclear medicine at the Helsinki University Hospital. His publications are mostly on surface ECG, but his clinical activity also includes a responsibility for respiratory function.