ON THE THEORY OF BLOOD-TISSUE EXCHANGES:
III. CIRCULATION AND INERT-GAS EXCHANGES AT THE
LUNG WITH SPECIAL REFERENCE TO SATURATION

ENS. M. F. MORALES AND LIEUT. R. E. SMITH, H-V(S), USNR*
NAVAL MEDICAL RESEARCH INSTITUTE, NATIONAL NAVAL MEDICAL CENTER,
BETHESDA, MARYLAND

The results of a simple analysis of the situation in the lungs with
regard to the uptake of inert gases is presented. It is shown that the gas
transfer at the lung governs the early stages of gas uptake by a body
region so that the latter conceivably could be used as an index of lung
function. It is shown that the so-called Fick Principle, which serves as
the basis of most indirect cardiac output methods, is an approximation to
a more general equation. When the latter equation is applied to a slowly
permeating gas, it offers the possibility of determining functional lung
surface.

Being the spatial intermediates between the atmosphere and all
body tissues, the lung membranes potentially govern all inert gas ex-
changes. It is, therefore, unavoidable that their role be included in
such exchange theories as have been attempted (Smith and Morales,
1944a, hereinafter referred to as SM). Furthermore, the kinetics of
inert gas exchange across the lung membranes has been made the
basis for certain indirect studies of circulatory function, and so, from
this point of view, a study of these kinetics has immediate practical
application. In appreciation of these two incentives, the following
note attempts to formulate certain transfer problems at the lung, and
then to incorporate the solutions into more comprehensive theories
and applications.

As must forever be the case in biology, the physical situation in
the organism must be considerably simplified and schematized before
the problem involving it can be formulated. For our present discus-
sion the theoretical model is pictured in Figure 1. We imagine the
entire pulmonary circulation to pass through a lung blood chamber of
volume, \( V \) cm\(^3\). The walls of this chamber are the lung membranes

* The material in this article should be construed only as the personal opinion
of the writers and not as representing the opinion of the Navy Department
officially.

\(^1\) This is not to imply that only lung membranes may pass an inert gas.
Behnke (1941) has shown that \( N_2 \) can be exchanged by the skin in significant
amounts. The same author (1938) has also demonstrated that He is passed by
the bladder wall in very slight amounts.

141
THEORY OF BLOOD-TISSUE EXCHANGES

The time course of saturation may be pictured as follows: At the instant that the subject takes the first breath of the gas, he builds up the alveolar pressure of the gas to the value $p$. We designate that instant as zero time. The blood at that moment in the lungs will then begin to flow out with a progressively increasing concentration of gas. Finally, the last part of that blood will flow out with a concentration of gas to be sustained for as long as $p$ is constant. This initial stage, equal to the length of time which blood spends in the lung, we may designate as stage (a). There will now follow a stage in which the arterial blood is carrying and delivering blood to the various absorbing regions, and during which the venous return does not yet carry gas. This stage (b) ends when the veins first begin to return charged blood to the lung, and it is, therefore,

---

142

142

THEORY OF BLOOD-TISSUE EXCHANGES

LUNG PERMEABILITY+$h$

LUNG VASCULAR SURFACE+$S$

SYSTEMIC CIRCULATION

FIGURE 1

themselves. On the external side of the membranes the gas in question is assumed to exist at a mean constant pressure of $p$ dynes cm$^{-2}$. The remaining symbolism is either obvious from Figure 1 or is consistent with that previously introduced (SM).

Gas exchanges at the lung can be divided conveniently into those processes which result in a net gain of gas by the body as a whole (saturation), and those processes and phases which result in a net loss (desaturation). Not only has the chief experimental concern of the authors been with the former process, but also the analysis of lung desaturation, even at the level presented here, is beset with a great many complications which at this point we have found it impossible to simplify. For these reasons the attention of this paper is focused on saturation phenomena, and mention of the converse process will be slight. Some measure of simplicity is attained by dividing saturation itself into certain temporal stages, and this accordingly has been done. We now turn to the processes themselves.

**Saturation.** The time course of saturation may be pictured as follows: At the instant that the subject takes the first breath of the gas, he builds up the alveolar pressure of the gas to the value $p$. We designate that instant as zero time. The blood at that moment in the lungs will then begin to flow out with a progressively increasing concentration of gas. Finally, the last part of that blood will flow out with a concentration of gas to be sustained for as long as $p = $ constant. This initial stage, equal to the length of time which blood spends in the lung, we may designate as stage (a). There will now follow a stage in which the arterial blood is carrying and delivering blood to the various absorbing regions, and during which the venous return does not yet carry gas. This stage (b) ends when the veins first begin to return charged blood to the lung, and it is, therefore,

---

2 Actually we will neglect the coronary return (about 5% of the total) which is the first such return.