SOME PHYSIOLOGICAL ASPECTS OF RESPIRATORY EMERGENCIES

J. B. L. HOWELL, B.Sc., Ph.D., M.B., M.R.C.P.
Lecturer in Medicine, University of Manchester; Department of Medicine, Manchester Royal Infirmary, Manchester, 13

The term 'respiratory emergency' denotes a state of interference with respiratory exchange to such an extent that life is endangered. While the term is commonly limited to acute disturbances of ventilation in dramatic circumstances (drowning, electrocution, poliomyelitis), in fact a much wider range of conditions can properly be regarded as respiratory emergencies and should be treated as such.

This paper seeks first to classify the various causes of such emergencies on the basis of the physiological processes which have been disturbed. Then follows a description of some of the more important changes in the respiratory gases which result from these disturbances.

Classification of Respiratory Emergencies

(i) Ventilation

Of the four processes involved in pulmonary function, ventilation, distribution, perfusion and diffusion, the one most commonly involved directly in the production of a respiratory emergency is ventilation. The chain of events producing inspiration is initiated by nervous drive originating in the brainstem. This drive is conducted over nervous pathways to the inspiratory muscles, contraction of which generates inspiratory force which is transmitted to the thorax and lungs. This force overcomes the combined elastic and frictional resistances of these structures and inspiration results.Expiration follows passively when the nervous drive wanes. This sequence can be broken at any one or more of these points, and examples of the situations in which this may occur are shown in Table 1.

(ii) Ventilation-Perfusion Relationships

In normal lungs the inspired air is distributed relatively evenly to all alveoli so that the alveolar gas is equally diluted. Similarly, the blood flow around the alveoli is evenly distributed in relation to their ventilation. When the ratio of ventilation to perfusion is the same throughout the lung, respiratory gas exchange is achieved with maximum efficiency. When these relationships are disturbed so that some regions of the lung are over-ventilated in relation to their blood flow, and others are over-perfused in relation to their ventilation, the efficiency of operation of the lungs is impaired. The former disturbance causes a smaller proportion of the tidal ventilation to be effective in achieving gas exchange (increased physiological dead space), and the latter disturbance inevitably results in some inadequately oxygenated blood reaching the systemic circulation (venous admixture).

The importance of disturbed ventilation-perfusion relationships in this context is twofold: (a) They may in themselves cause respiratory emergencies in conditions such as bronchial asthma and pulmonary oedema; and

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<th>Table 1 Classification of Causes of Respiratory Emergencies</th>
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<tr>
<td>Disturbance of</td>
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<td>(i) Ventilation:</td>
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<td>Nervous drive</td>
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<td>Transmission of muscular force</td>
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<td>Resistances of lungs:</td>
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<td>(a) Frictional</td>
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<td>(b) Elastic</td>
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<tr>
<td>(ii) Ventilation-perfusion relationships</td>
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<tr>
<td>Atelectasis</td>
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<td>Pulmonary oedema</td>
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<td>Bronchial asthma</td>
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<td>(Emphysema)</td>
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<tr>
<td>(iii) Diffusion</td>
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<td>Pulmonary oedema</td>
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*Paper presented to the Meeting of the Harveian Society, October 19, 1960.*
(b) they may so aggravate the effects of a
reduction in total ventilation that emer-
gencies may arise out of conditions which
otherwise would not be so serious.

The commonest circumstance in which (b)
occurs is when obstructive emphysema compli-
cates other causes of hypoventilation. By emphy-
sema is meant an established increase in functional
residual capacity (FRC) of the thorax associated
with predominantly expiratory obstruction. When
patients with this condition develop acute venti-
latory failure, they have disturbances of most of
the processes of pulmonary function and it is
often difficult to apportion the relative importance
of each. This is the reason why emphysema
appears in several places in the table.

(iii) Diffusion

The development of pulmonary œdema is the
commonest way in which a disturbance of the
diffusion of gases across the alveolo-capillary mem-
brane causes an emergency. In addition to the
increased thickness of the membranes due to the
exuded fluid, bubbles form in the airspaces and
occlude or partially occlude the airways. This
superimposes a severe disturbance of distribution
upon that of diffusion. Despite this there is no
overall hypoventilation, the arterial PCO₂ remain-
ing normal or even being reduced.

Consequences of Hypoventilation

The behaviour of the carbon dioxide is the key
to the understanding of the events which follow
ventilatory arrest.

Initially, the mixed venous blood is returning
to the lungs with a carbon dioxide tension (PCO₂)
of about 46 mm.Hg. The alveolar gas is some
6 mm. lower since it is being ventilated with air.
Assuming an FRC of 2.5 l., the addition of about
25 ml. carbon dioxide to the FRC would abolish
this gradient completely. This should occur
within a few seconds, after which the blood flow-
ning through the pulmonary capillaries will not
lose carbon dioxide, though oxygen will still be
taken up. Consequently, the arterial blood return-
ing to the tissues will be loaded with carbon
dioxide and will be unable to take up the usual
amount from them. The fate of the carbon
dioxide accumulating in the tissues is important.
Because of the relatively large volume of the
body fluids and because of the solubility of carbon
dioxide in them, the tissue capacity for carbon
dioxide is very large. This results in the tissue
PCO₂, and therefore that of the mixed venous
blood, rising relatively slowly. This rise has
been found to be less than 6 mm./minute
(Campbell and Howell, 1960), and corresponds to
the discharge of about 25 ml./minute of carbon
dioxide into the alveolar gas. The remainder
of the carbon dioxide produced by the tissues during
this time must therefore have been accommodated
by the tissue fluids, and in the normal resting
state would amount to about 175 ml. Over the
same period the resting subject would continue to
absorb oxygen from the lungs at the normal rate,
about 250 ml./minute, causing a volume deficit in
the lungs of about 225 ml./minute. Whether the
chest would actually decrease in volume in this
way would depend upon whether the airways were
patent or not.

If ventilatory arrest occurs with the airways
patent there is a mass movement of air into the
lungs from the atmosphere enriching the alveolar
air and it can be calculated that the oxygen
pressure (PO₂) of the alveolar gas after apnoea of
one minute’s duration would be 10-11 mm.
higher than if this mechanism were not operative.
This process has been variously called diffusion
respiration (Draper and Whitehead, 1944), apnoeic
oxygenation (Nahas and L’Allemont, 1956), but
the term ‘aventilatory mass flow’ (AMF) (Bart-
lett, Brubach and Specht, 1959), though cumber-
some, is more accurately descriptive.

If at the onset of the apnoea the subject’s airways
were connected with a supply of oxygen, the
AMF would consist entirely of oxygen (Fig. 1),
and the PO₂ of the alveolar gas would be largely
maintained, decreasing only at the rate at which
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![Diagram of gas stores](image)

**Fig. 2.**—Diagrammatic illustration of the body gas stores for oxygen and carbon dioxide. The large capacity of the carbon dioxide store is in contrast to that of the oxygen store.

the Pco₂ was rising. This would ensure the delay of lethal levels of anoxia for at least ten minutes. If the subject had been ventilated with oxygen to remove the body’s nitrogen stores before the onset of the apnea, oxygenation could be maintained for longer periods. This is not put forward as a desirable method of treatment of ventilatory failure because it allows an increasing respiratory aci-

dosis. However, the mechanism of AMF may well be encountered more frequently in the future in the treatment of ventilatory failure if the use of the hydrogen-ion buffer ‘THAM’, which can prevent the elevation of the Pco₂ (Nahas, Jordan and Ligou, 1959), proves to be capable of therapeutic application.

The rate at which hypoventilation develops affects the changes which occur in the two respiratory gases. When ventilation ceases abruptly, because the store of oxygen in the body is only about 1 litre (Fahri and Rahn, 1955), the P0₂ falls rapidly. Lethal levels of anoxia will develop within a few minutes, but the Pco₂ will have risen only slightly. In contrast, when hypoventilation develops more slowly, the meagre oxygen stores are supplemented by the ventilation so that the hypoventilation can persist for a longer period, giving the carbon dioxide an opportunity to accumulate in the body tissues and elevate the Pco₂ to a greater degree.

These relationships are shown in Fig. 2, where the body gas stores for oxygen and carbon dioxide are shown diagrammatically. The size of the carbon dioxide stores results in a buffering of the tissue Pco₂ against changes in ventilation. This becomes of practical importance in the assessment of the ventilatory status of a subject if ventilation is altering rapidly (as, for example, in Cheyne-Stokes respiration or in the apprehensive subject), when the arterial Pco₂ might be misleading. In contrast estimation of tissue Pco₂ by measurement of the Pco₂ in the mixed venous blood would be little affected by such transient changes and would reflect the average alveolar ventilation. Simple methods of estimating mixed venous Pco₂ are now available (Collier, 1956; Campbell and Howell, 1960).

This review has been confined to respiratory aspects of the physiological events occurring during respiratory emergencies. There are important cardiovascular aspects which have not been discussed, which are largely concerned with the consequences of the altered intrathoracic pressure relationships produced by artificial ventilation.

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