THE EARLY RECOGNITION OF RESPIRATORY FAILURE AND SIMPLE METHODS FOR ITS ASSESSMENT*

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Respiratory failure may occur in many different conditions, but they all have one factor in common — hypoventilation or underbreathing. It must be emphasized that there need not be primary pulmonary disease. The clinical picture of respiratory failure is the result of two factors: (1) the cause of the hypoventilation, and (2) the effect of the hypoventilation.

The causes may be classified as follows:

1. The obvious one of airway obstruction whether

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resulting from foreign body, trauma, oedema of the glottis or bulbar palsy.

2. Pulmonary conditions such as asthma, bronchitis and emphysema, pneumonia, or pulmonary oedema.

3. Musculoskeletal conditions such as kyphoscoliosis, stove-in-chest, ankylosing spondylitis and muscle dystrophies. Gross obesity may also be included here.

4. Neurological disorders such as head injuries, poliomyelitis, polyneuritis, myasthenia gravis and motor-neurone disease.

5. Iatrogenic conditions such as surgery on the abdomen or chest, anaesthetic drugs (especially muscle relaxants), and overdosage of narcotics.

This classification is an oversimplification, since many of the above conditions usually occur together. A patient with emphysema may be precipitated into respiratory failure by an acute chest infection, especially if he is heavily sedated. Reluctance to cough after thoracotomy or laparotomy, especially in obese subjects, may result in atelectasis and then respiratory failure.

CLINICAL PICTURE

The clinical picture of respiratory failure will vary according to the cause, and the examples mentioned above indicate that the clinical presentation cannot be uniform.

The underlying condition gives rise to symptoms and signs upon which are superimposed those of respiratory failure itself — namely hypoxia, hypercapnia and disturbances of heat regulation.[1] Awareness of the condition and its consideration in every seriously ill patient will prevent its diagnosis being missed.

Early on there is breathlessness or apparent hyperventilation, headache, somnolence, intermittent mental confusion, weakness, lassitude and irritability. The skin is warm, flushed and sweating. There is tachycardia and the blood pressure may be raised. Signs of cor pulmonale and right-sided heart failure may appear.

If the condition remains untreated and the hypoxia and hypercapnia increase, varying disturbances of consciousness occur, ranging from extreme restlessness to coma. Reflexes become depressed and muscle twitching and tremors of the extremities or face are common. There may be flaccid immobility; dilated, unequal or constricted pupils; or convulsions.[2] Obvious respiratory depression with deep cyanosis develops. Sweating ceases and the extremities become pale and cold, but if the trunk is felt, the high central-body temperature is easily appreciated.[1] If the patient is not energetically treated, coma deepens, blood pressure falls to unobtainable levels, and death ensues.

Perhaps worth mentioning as a physical sign of hypoventilation is the venous engorgement noted in the fundus of the eye. This can progress to papilloedema with retinal haemorrhages.[3,4] When this is detected in a patient with signs in the central nervous system, headache and somnolence, the resemblance to an intracranial lesion, especially a tumour, is very great.[5] If an intracranial lesion is diagnosed instead of respiratory failure, it may have the gravest consequences for the patient.

REGIME OF MANAGEMENT

Once a patient is suspected of having respiratory insufficiency, it is worth while adopting a regime of management and observation to assist in deciding what measures of treatment to carry out. Simple bedside observations are often all that are necessary.[1] The observations include: the mental state, the respiratory rate, the pulse rate, the blood pressure, the patient's colour (i.e. degree of cyanosis), and the rectal temperature.

A table of these observations is drawn up and from the record of these signs made at regular intervals it is usually easy to judge if the patient's respiratory function is unchanged, improving, or worsening. The frequency of observations will vary for each patient, from every 5 minutes to hourly. The patient's changing therapeutic requirements can be determined with objectivity as they arise and the effect of resuscitative procedures is manifest almost immediately from these simple observations. Fig. 1 illustrates the rapidity of response in a confused, stuporose

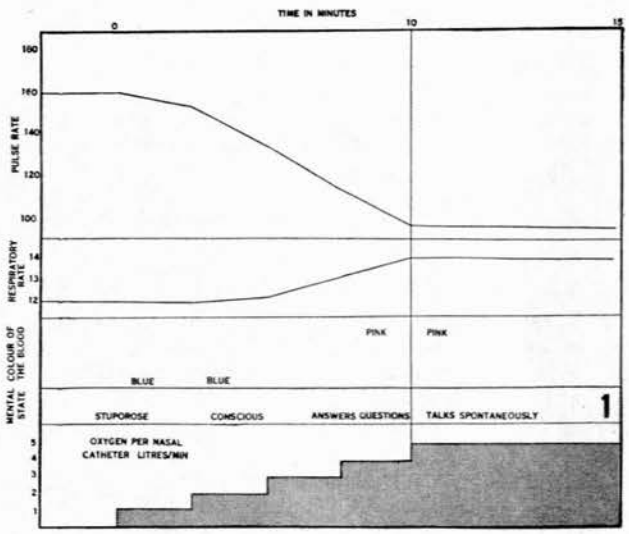

Fig. 1. Response to oxygen administration.

patient given oxygen by nasal catheters. After 10 minutes the patient's pulse rate had dropped from 160 to 100, the respiratory rate had increased, cyanosis had disappeared, and the patient was talking rationally.

SIGNS OF RESPIRATORY FAILURE

Hyperventilation

The importance of hyperventilation as a process of adaptation to either lung pathology or the increased oxygen requirements in other diseases is illustrated in Fig. 2. The resting minute volume (the amount of air breathed per minute) is plotted against the resting arterial oxygen saturation. Many of the patients with pulmonary or cardiac disease have virtually normal arterial oxygen saturations, but have notable increases in minute ventilation, that is, they require to hyperventilate.

The patients with arterial oxygen saturation of less than 80% have relatively small minute volumes compared to the others. It may be deduced that hyperventilation is the means by which a patient with pulmonary disease or increased oxygen requirements can maintain a normal supply of oxygen to the tissues. When hyperventilation can no longer be maintained and ventilation falls to normal levels

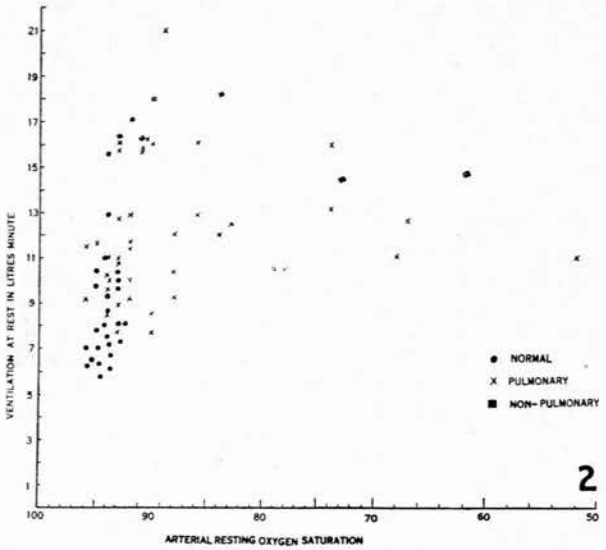

Fig. 2. Relationship between ventilation and arterial oxygen saturation at rest in normal subjects and in patients.

in such a patient, arterial oxygen desaturation or cyanosis occurs.

Cyanosis

The assessment of cyanosis clinically is generally regarded as unreliable, but we have found it valuable. This is probably due to the fact that the arterial oxygen tension in Johannesburg is considerably lower than at sea-level, making a fall in tension more easily detectable as

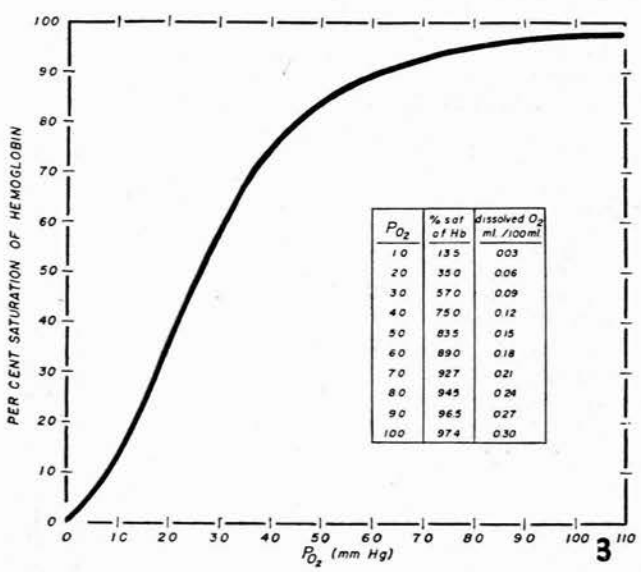

Fig. 3. Oxygen haemoglobin dissociation curve (data of Dill, pH 7·40, temperature 38° C.).

a fall in saturation. This is explained by the nature of the oxygen-dissociation curve (Fig. 3).

In respiratory failure one is concerned with central cyanosis, not peripheral cyanosis, therefore the colour of the tongue should be observed. If there is uncertainty, the ear lobe should be warmed to 37 - 40°C. and its colour judged. If still in doubt, the warmed skin should be pricked and the colour of the blood observed immediately it appears. Cyanosed blood rapidly becomes red on exposure to the atmosphere, whatever the cause of the cyanosis, with the exceptions of met- and sulphhaemoglobinaemia.

In general, the presence of cyanosis is an indication for oxygen therapy. If the patient is breathing room air and is not cyanosed, it can be inferred that ventilation is adequate (but not necessarily normal) for both oxygenation and removal of carbon dioxide, and there is little danger of carbon-dioxide narcosis. However, it must be remembered that, owing to the form of the oxygen-dissociation curve in blood, ventilation must be moderately depressed before a fall in oxygen saturation is detectable — oxygen tension may be reduced by almost 50%.

Hyperpyrexia

Hyperpyrexia is not generally appreciated to be part of the syndrome of respiratory failure.[1] Fig. 4 illustrates hyperpyrexia encountered in 4 patients going into respiratory failure. One had emphysema with an acute chest infection, one developed bilateral atelectasis after a cholecystectomy, one was being maintained on a respirator after a thoracotomy, and one had had a mitral valvotomy.

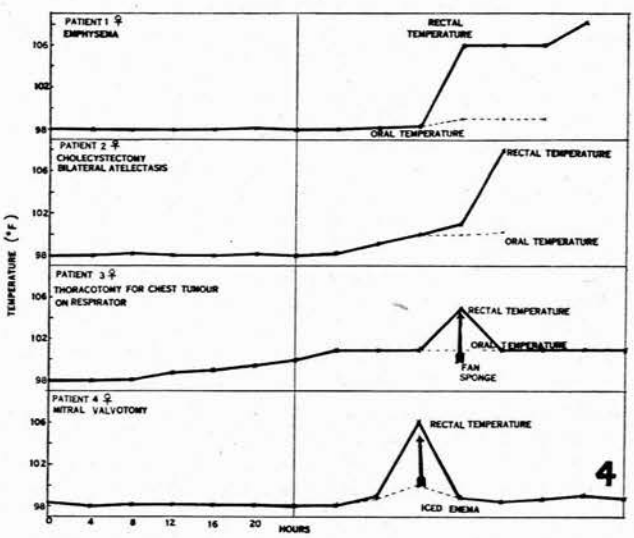

Fig. 4. Temperature curves in 4 patients who developed hyperpyrexia.

In each case, for approximately 24 hours before hyperpyrexia was detected, there was increasing tachycardia and tachypnoea and the skin was warm and moist. Oral temperatures were normal. Then, over the space of a few hours, marked deterioration in the patients' condition occurred with loss of consciousness and coldness of the extremities. The observation that the trunk felt hot to the touch led to the measurement of rectal temperature, which was 106°F. in the first case. In this case, oral temperature remained normal, even when the rectal temperature rose to 108°F., after which the patient died.

In the second patient the illness ran a similar course, but in the third and fourth the hyperpyrexia was energetically

treated by physical methods (fan, sponging, ice enema) and these patients survived.

The points that deserve emphasis are: (1) the unreliability of oral temperature measurements in dyspnoeic subjects; and (2) the importance of frequent estimates of rectal temperature, so that a rise can be detected early and treated before dangerous levels are reached.

In acute respiratory failure, body temperature may be raised, owing to infection, hypoventilation, increased work of breathing, or combinations of these factors. Normally the skin is the most important heat-regulating agent of the body since it provides a large surface area for radiation and evaporation.

Ventilation accounts for about 15% of heat loss by warming inspired air to body temperature and saturating it with water vapour. In ill patients, the major heat-losing mechanism, the skin, is ineffective. This is because the patients are usually covered with blankets, leaving only the face exposed to the atmosphere for heat loss. They are often dehydrated with reduced sweating, yet metabolic work and therefore heat production are often increased. The patient then has to rely on hyperventilation to lose heat. If he is unable to breathe adequately his temperature will rise; if this is not controlled, hyperpyrexia may occur and result in death. Physical methods must be used to cool the patient and ventilation may need to be assisted mechanically to save the patient's life. This situation of hyperpyrexia arises particularly in hot, humid weather, overheated wards, or overheated operating theatres.

During inspiration, by convection and evaporation, water and heat are added to the inspired air by the mucosa of the upper respiratory tract.[6] This cools the mucosa so that on expiration from the lung some heat and water is recovered by convection and condensation. There is a net loss of heat and water with each breath, however, since the expired air, though cooler than alveolar air, is warmer than inspired air and is fully saturated with water vapour.

The amount of heat and water exchanged between the body and the inspired air depends on the environmental conditions. For example, in a temperate environment (25°C. and 50% relative humidity) inspired air is 50% saturated with water vapour. During its passage to the alveoli of the lung its saturation increases to 100% and at the same time its temperature rises from 25°C. to 37°C., to conform with the alveolar environment. On expiration, the alveolar air is cooled from 37°C. to 32°C. with the recovery of some heat. As the temperature of the alveolar air drops, it is able to hold less water. Thus some water condenses and is recovered by the mucosa. With each breath, therefore, some heat and some water is lost from the body. In a cold, dry environment much heat and water is lost by ventilation, but in hot, humid environments little heat is lost in this manner. During surgical anaesthesia the ability of the respiratory tract to eliminate heat and water may be greatly decreased. CO_2-absorbing canisters both saturate inspired air and warm it to as high as 47°C. The respiratory tract may gain rather than lose heat under such conditions.[6]

TESTS FOR RESPIRATORY FAILURE

Carbon-dioxide Tension

Arterial carbon-dioxide tension ($_pCO_2$) shows a rapid response to any ventilatory change and is an excellent index of ventilation. However, carbon-dioxide tension cannot easily be estimated clinically. As proof of hypoventilation demands the detection of a raised carbon-dioxide tension in the blood, this will be discussed in some detail. Campbell has stated that knowledge of the $_pCO_2$ in respiratory failure is as valuable as measurement of blood sugar in diabetic coma, urea in renal failure, or sodium in adrenal failure.[7]

The arterial $_pCO_2$ equals the ratio between the volume of CO_2 produced in the body and the alveolar (or effective) ventilation. If alveolar ventilation is inadequate, the $_pCO_2$ rises; if alveolar ventilation is excessive, the $_pCO_2$ falls. There are various ways of measuring arterial $_pCO_2$, but most of them require expensive apparatus or are time-consuming and difficult.

Only Collier's method,[8] as modified by Campbell and Howell,[9] will be discussed. These workers showed[10] that by rebreathing from a bag containing 2 litres of 100% O_2 for 90 seconds, a CO_2 mixture could be prepared which had a higher tension (4 - 10 mm.Hg greater) than that of mixed venous blood, i.e. the blood entering the pulmonary capillaries. If after a 2-minute interval the patient rebreathes from this simply prepared CO_2 mixture, the gas in the bag mixes with the alveolar air in the lung after a few breaths and the venous blood equilibrates with the CO_2 in the bag. At this stage, blood enters and leaves the lung without changing its $_pCO_2$. This equilibration must occur in less time than it takes the blood passing through the lungs to recirculate, because blood which has passed through the lungs without being able to offload any CO_2 picks up more CO_2 from the tissues and returns to the lungs with a higher $_pCO_2$ than before. It takes approximately 20 seconds before this recirculation causes the alveolar and bag CO_2 to start rising.

If at the end of the period of equilibration, the CO_2 in the bag is measured, it equals the CO_2 of mixed venous blood. To derive the arterial $_pCO_2$, 6 mm.Hg is subtracted from the mixed venous $_pCO_2$. The whole procedure takes 5 - 10 minutes and yields results sufficiently accurate for clinical purposes. The apparatus required to measure CO_2 is either a rapid infra-red analyser or a modified Haldane apparatus.[11] This method has been used for detecting respiratory failure in emphysema, carbon-monoxide poisoning, cerebrovascular accidents, hypothyroidism, polycythaemia and other conditions.[9]

Ventilatory Volume

While measurement of the CO_2 tension is the best method of estimating the extent and progress of respiratory failure, the measurement of ventilatory volumes is both a simple and a valuable alternative method.[1] There are various ways of doing this: (1) by spirometry; (2) by collection of expired air in a Douglas bag; (3) by dry-gas meter or gasometer — an instrument which resembles and works like the domestic gas meter, the amount of air breathed per minute being read directly off a dial; and (4) by placing ventilation meters such as the Wright anemometer in continuity with the airway.

With any of these instruments, the minute volume (MV) of a patient can be measured and compared with standard tables to determine if ventilation is subnormal or not, but it must be remembered that most ill patients require to

ventilate more than normal subjects. Also, it is alveolar ventilation (minute volume minus dead-space ventilation) that is the important index. An arbitrary, but clinically satisfactory, estimate of the respiratory reserve may be obtained from the ratio MBC : MV,[1] where MBC is the maximum breathing capacity, i.e. the maximum amount of air that can be breathed per minute. It is measured on the same apparatus that is used for the minute volume The ratio MBC : MV is normally about 15 : 1. In respiratory insufficiency this ratio may be reduced because of a decrease in MBC and/or an increase in MV. When this ratio is greater than 4 : 1 there is no immediate danger. As this ratio decreases to 2 : 1, carbon-dioxide narcosis is imminent.

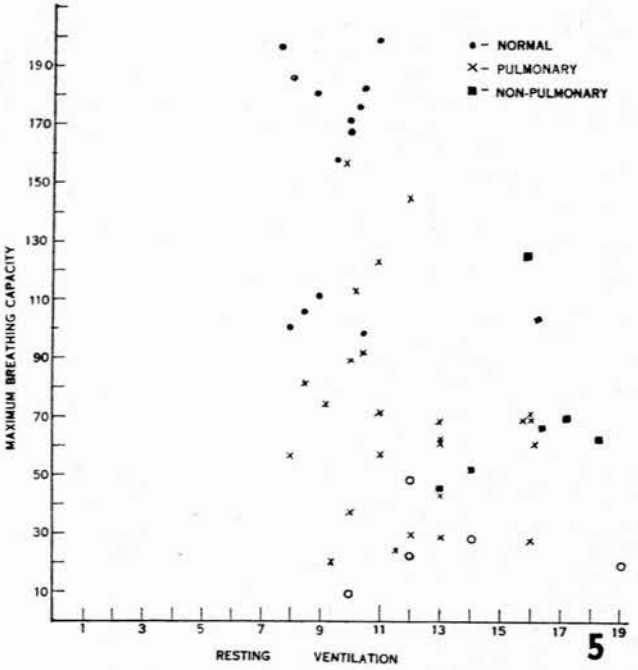

Fig. 5. Respiratory reserve (ratio of maximum breathing capacity to minute volume at rest) in normal subjects and in patients.

Fig. 5 compares the maximum breathing capacity with the resting minute ventilation in a group of patients and normal subjects. The respiratory reserve of the patients is decreased. Five patients who required tracheotomy in the treatment of their respiratory failure are indicated by open circles and they all had a respiratory reserve (ratio MBC: MV) in the region of 2 : 1.

SUMMARY

Respiratory failure should be considered in the differential diagnosis of every seriously ill patient. The cause of the respiratory failure and its effects (hypoxia, hypercapnia and hyperpyrexia) must be detected and treated. The measurement of blood carbon-dioxide tension is the single most useful test for the condition, but measurement of ventilation is simple and valuable.

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