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Respiratory Inadequacy – its study, diagnosis, and treatment

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Abstract

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Woolmer (1956) states that respiratory inadequacy exists "when the gas exchange between the lungs and blood falls below that between the tissues and the blood : when external respiration cannot keep place with internal respiration " . (17) Melville Arnott (1960) is more direct, stating that respiratory inadequacy is the preliminary stage of respiratory failure, namely, "that condition in which the amount of oxygen and carbon dioxide in the blood stream is altered by an abnormality of the respiratory system" . (23) Comroe et al. state specifically as the basic facts of respiratory inadequacy, hypoxaemia, CO2 retention, and respiratory acidosis.

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RESPIRATORY INADEQUACY

- its study, diagnosis, and treatment

By CHARLES HOPE

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PULMONARY FUNCTION STUDIES

Pulmonary function studies will not tell where the lesion is, what the lesion is, or even that a lesion exists, if it does not interfere with the function of the lungs. They supplement but do not replace a good history and physical examination, and radiological, bacteriological, bronchoscopic, and pathological investigations. How can we use these studies to our advantage? The most useful method is to divide respiratory function into three zones—[11, 23]

1. VENTILATORY ZONE — comprising volume and distribution.
2. DIFFUSION ZONE — discussion of O_2 and CO_2 relationships and the effect of the alveolar capillary membrane.
3. PERFUSION ZONE — comprising the volume and the distribution of the pulmonary capillary blood flow.

Ventilatory Zone

Ventilation normally maintains the alveolar gas tensions at levels which ensure that the pulmonary capillary blood is saturated with oxygen before it leaves the respiratory part of the capillary and that it has rid itself of the necessary amount of CO_2 over the tension range of 46 - 40 mm. Hg. The requirements for this are therefore the appropriate function of the bellows mechanism and airway configuration to ensure a uniform distribution of the inspired air. Remembering that gas exchange occurs rapidly only in the alveoli, it is therefore obvious that the measurement of **alveolar ventilation** is the most important single measurement of this zone of function. It must be appreciated that measurements of lung volumes do not evaluate function. These are essentially anatomical and are unchanged by altered physiology.

Alveolar ventilation is the amount of gas which reaches the pulmonary alveoli, and will depend on the frequency of respiration, the tidal volume, and the respiratory dead space. Frequency normally lies between 11 - 14 breaths per minute in healthy basal conditions, but taken alone, cannot be very useful as an index of alveolar ventilation. The tidal volume is seldom measured clinically although this can be done easily even in a sick person by the use of various open and closed methods, by the use of a Douglas bag, or by the use of an ingenious plastic bag which can be carried easily in the pocket.[12] The normal average is from 500 - 600 mls. Again, this is not a useful value if considered alone, but if associated with the frequency, it may be useful in detecting gross hypoventilation.

The respiratory or physiological dead space is equivalent to the anatomical dead space, plus two additional volumes, namely, the volume of inspired gas ventilating alveoli which have no pulmonary capillary blood flow, and the volume of inspired gas ventilating some alveoli in excess of that required to arterialise the pulmonary capillary blood flowing around them. These two factors operate significantly only in patients with pulmonary disease, taking this in its broadest sense. In the normal person, the anatomical dead space is very nearly equal to the physiological dead space except in exercise. From this it is seen that the respiratory dead space is easily calculated from the basic equation—

ALVEOLAR VENTILATION/MINUTE = (T.V. - R.D.S.) × frequency.

For patients in respiratory inadequacy it is easier to measure ventilation by their effectiveness in washing out alveolar CO_2.

$$\text{ALVEOLAR VENTILATION (mls.)} = \frac{\text{Vol. of exp. } CO_2 \text{(mls.)}}{\% \ CO_2 \text{ in alv. gas}} \times 100$$

This should demonstrate the importance of thinking along the lines of alveolar ventilation. It is not sufficient merely to count the respiratory frequency. Much more attention must also be paid to tidal volume.

Diffusion Zone

The rate of transfer of oxygen and carbon dioxide depends on the tension differences, length of the path, the specific solubility and diffusibility of the gases and the area of the membrane. CO_2 is much more diffusible that O_2 to the extent of 20 : 1 and the tension difference of 6mm. Hg. between the pCO_2 of the mixed venous blood and the alveolar air is sufficient to eliminate an adequate amount. Oxygen requires a greater diffusion gradient of the order of 64 mm. Hg., from 104 mm. Hg. in the alveoli to 40 mm. Hg. in the venous blood. The Hb is barely saturated when it leaves the alveoli, and in health a small tension deficit of 9 mm. Hg. exists, partly due to shunting and partly due to the admixture factor existing as a result of mechanisms in the ventilatory zone (previously discussed) whereby an increase in the dead space may occur.

Reduction in the diffusion capacity may be due to an increase in the diffusion pathway or a decrease in the area of membrane. Arterial unsaturation can be due to either these factors or to ventilatory insufficiency, when present in the absence of gross shunting. The arterial pCO_2 however, allows a differentiation. In ventilatory insufficiency there will be a decreased alveolar pO_2 and an increased alveolar pCO_2, and, as CO_2 has to accumulate in the plasma until it obtains the 6 mm. Hg. ascendancy over alveolar pCO_2 necessary for it to escape, there will be both hypoxia and hypercarbia.

In decreased diffusion capacity, or alveolar-capillary block, CO_2, being highly diffusible, still diffuses out at normal tensions. In fact, hyperventilation may actually wash-out CO_2 to produce an abnormally low arterial pCO_2. As arterial pCO_2 is 6 mm. Hg. less than venous pCO_2, it can be calculated from the value of the alveolar pCO_2 which is equivalent to that of mixed venous blood when a subject has rebreathed to equilibrium with a bag of air. Alveolar pCO_2 is calculated either by a rapid infra-red analyser, or by the Haldane method or one of its modifications. This promises to be of great value in clinical practice and will show unsuspected elements of hypercarbia in early stages.

Perfusion Zone

In respiratory inadequacy the usual defect shown by studies of this zone is pulmonary hypertension. The perfusion zone can be studied by cardiac and pulmonary catheterisation, and by the use of tracer isotopes and dyes.

Forced Expiratory Volume (F.E.V.) [21, 4]

This is a practical measure of the maximum ventilatory capacity (M.V.C.) and is easily measured in health and disease. The inspiratory and expiratory phases of the vital capacity as measured and recorded on a fast drum produce a record known as the vital spirogram. In the initial linear fraction of the expiratory phase, the volume and rate of air flow are related to those obtained in the expiratory phase of maximum voluntary ventilation (M.V.V.) The F.E.V. is half of the mean of six readings of the first 0.75 second periods of the expiratory curve. (0.75 is equivalent to the theoretical breathing rate of 40/minute.)

F.E.V. gives a better measure of the maximum ventilatory capacity than the M.V.V. It is easier and quicker to perform and has only a small standard error. F.E.V. is thus a measure of the vital capacity which is useful to an individual for hyperventilation (i.e. it is a valid measure of M.V.C. for expiration at a uniform rate proportional to the vital capacity).

AETIOLOGY OF RESPIRATORY INADEQUACY

Respiratory inadequacy occurs as a result of hypoventilation, that is, decreased alveolar ventilation, and is due to any process interfering with the normal function of the bellows mechanism, either in its moving parts or in its central and/or peripheral control mechanisms. These processes may be classified under several headings (Table 1.)

These aetiological factors may act either singly or in combination. At the time of commencement of therapy there may be little or no indication of the exact cause of the respiratory failure.[11, 23, 24]

CLINICAL PICTURE OF RESPIRATORY FAILURE

The clinical signs and symptoms in respiratory inadequacy can be many and varied. The distressed patient, dyspnoeic, cold, clammy, apprehensive, exhibiting purposeless movements; the paradoxical respiration, cyanosis, and perhaps coma of the major road casualty; the surgical risk, grossly obese, postoperatively in poor condition, with slightly lowered blood pressure and average, thready or full pulse: these and many others are the clinical syndromes which the house physician or surgeon, or the general practitioner may be required to assess and treat.

Dyspnoea can be the result of an obstructed airway caused by inflammation or secretion, the result of emphysema or of gross obesity with its concomitant mechanical embarrassment. It is the result of CO_2 retention and hypoxia. Cyanosis may be present in severe cases, provided there is enough circulating haemoglobin. This is due to arterial desaturation, and if this has been of long standing, there may be a compensatory polycythaemia. The symptoms of hypoxia are many and varied :— increased ventilation, increased pulse rate, incoordinated movements and decreased muscular efficiency ; progressing to further increase in pulse and respiration, Cheyne-Stokes breathing, cyanosis,

TABLE I.
Aetiology of Respiratory Inadequacy

Depression of Respiratory Centre:

General anaesthesia	Increased intracranial pressure
Excess morphine or barbiturates	Electrocution
High concentrations CO_2	Prolonged hypoxia or cerebral
Cerebral trauma	ischaemia

Interference with neural conduction or with neuromuscular transmission to the respiratory muscles :

Traumatic spinal cord lesions	Neuromuscular block due to:
Poliomyelitis	curare; decamethonium; succinyl
Peripheral neuritis	choline; nerve gases; myasthenia
	gravis; botulinus or nicotine poisoning.

Diseases of the Respiratory Muscles — Acute myositis

Limitation of Thoracic Movement :

Arthritis	Scleroderma
Kyphoscoliosis	Gross obesity
Emphysema	Chest wall injury
Ankylosing spondylitis	(traumatic and surgical)

Limitation of Pulmonary Movement :

Pleural effusion	Thickened pleura
Pneumothorax	

Pulmonary Diseases :

Decrease in functioning lung tissue:	Emphysema
Atelectasis	Pneumonia
Tumour	Pneumoconiosis
Decreased distensibility of lung tissue:	
Fibrosis	Congestion
Obstructive lesions:	
Tracheal compression	Asthma
Inhaled foreign material	Bronchospasm
Obstruction due to faulty endotracheal tube.	

Primary Hypoventilation Syndrome

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vomiting, asthenia, and fatigue. Further hypoxia leads to severe cyanosis, excitement, fall in blood pressure, syncope, and coma, with respirations initially deep, becoming shallow and frequent with inspiratory spasm, and ultimate death.[20]

Carbon dioxide retention is not separated clearly from hypoxia with regard to its clinical effects. There is mental confusion, drowsiness, coarse irregular muscular twitching, warm extremities, raised blood pressure and full pulse. There may be tachycardia, arrhythmias, accentuation of vagal stimulation, ventricular fibrillation, cardiac arrest and bronchoconstriction. Acute CO_2 excess produces a peripheral vasodilatation with a full bounding pulse. Central effects produce vasoconstriction with a raised blood pressure which is maintained until further excessive retention depresses the vasomotor centre to produce a sudden and dramatic fall in the blood pressure. The effects are similar to an excess of circulating adrenaline, and there is such a release in this condition.[17]

CO_2 retention produces a respiratory acidosis and decreased blood pH. The kidneys compensate by increasing the reabsorption of Na^+ and HCO_3^-, and increasing the excretion of H^+ and NH_4^+.[32] There may be oliguria with all its grave complications. Experiment in animals can produce oliguria in diffusion respiration, but it does not occur until CO_2 accumulates. Perhaps this is the cause—it has not been proven.[17]

Clinically, when the onset is relatively gradual and observation is possible,

there may be evidence of CO_2 retention in that there is a slow rise in the pulse and blood pressure, followed later by a fall.[24, 11, 23]

TREATMENT OF RESPIRATORY INADEQUACY

The general principles of therapy are early energetic treatment, with a clear airway, and an adequate effective ventilation. To some extent the exact method of treatment will be indicated by the factors predisposing to inadequacy, and in certain cases the method of treatment will be instrumental in treating the cause. Treatment will be discussed under the headings—oxygen therapy, artificial respiration, drug therapy and tracheostomy.

OXYGEN THERAPY

It is necessary to administer oxygen to patients in respiratory failure. It is traditional to do this by the use of intermittent oxygen, and various schedules have been recommended—[23]

(a) oxygen given for 20 minutes per half-hour.
(b) oxygen given for half an hour in every hour.
(c) oxygen given for 40 minutes in every hour.

By these means one is said to prevent progressive CO_2 retention as the removal of oxygen will produce a hypoxic stimulus to breathing and will allow " blowing-off " of retained CO_2. This is achieved however only at the risk of producing periods of extremely serious hypoxia, mainly because the oxygen and carbon dioxide stores in the body are very different. [Farhi & Rahn 1955[7]] Any change in the ventilation or in the inspired air will produce a change in the oxygen concentration within 1 - 2 minutes. whereas change in the concentration of carbon dioxide takes 10 - 20 minutes. Within 2 - 10 minutes after stopping the administration of oxygen the persistent high pCO_2 from the blood reduces the alveolar pO_2 to produce an arterial anoxaemia or rather hypoxaemia of a greater degree temporarily than if oxygen had never been given at all. It may be argued that patients overventilate to " blow-off " CO_2, but in many cases the mechanical function of the chests are much too poor to permit this. It is also argued that the poorly ventilated parts of the lungs retain sufficient oxygen to prevent hypoxaemia for several minutes. That this latter argument is completely spurious is seen if one recalls the previous pulmonary physiological evidence. It has also been found that within 5 - 6 minutes after stopping oxygen the arterial pO_2 has dropped to 15 mm. Hg. on the average. This is equivalent to a 20% saturation.[7]

It should be remembered that in some cases the only stimulus or mechanism maintaining respiration is the decreased pO_2 stimulus to the chemoreceptors, the respiratory centre being completely depressed by the high pCO_2. Oxygen therapy may then produce apnoea which may prove fatal, e.g. Cor Pulmonale.

By these and many other arguments the requirements for giving oxygen in respiratory failure can be deduced.[7] In summary they are that oxygen must be given continuously, that the oxygen concentration should be controlled to prevent it rising high enough to produce respiratory depression, or falling low and permitting recurrence or increase of the hypoxia. The results of various exhaustive trials suggest that these requirements demand control of the inspired oxygen concentration with an accuracy of $\pm$ 1% (7 mm. Hg. pO_2.) in the range 24 - 35%. The dead space must be reduced as much as possible to prevent CO_2 retention.[8]

To achieve these requirements, conventional methods have proved to be rather inadequate. In oxygen tents the concentration of oxygen cannot be controlled with sufficient accuracy and at the low flow rates required there is CO_2 retention. The nursing and physiotherapy problems are also intensified.

With nasal and pharyngeal catheters the dead space is greatly reduced, but the oxygen concentration produced is extremely variable as a result of the degree of patency of the nasal passages, whether or not the mouth is open or closed, and whether inspiration is deep or shallow. Masks are the best method to use when high concentrations are required, the high flow rate flushing the dead space and preventing reinspiration of expired air, but at low flow rates, the mean inspired oxygen concentration is extremely variable. The required control of inspired oxygen is thus difficult and the difficulty is increased with lack of knowledge of the minute volume and with leaks in the system. The dead space of masks is too large to be acceptable when the gas flow rate is below 5 - 6 litres per minute. One should also note that if respiratory depression occurs when a patient is given oxygen by conventional methods at low flow rates, the concentration of inspired oxygen is thus automatically increased. Hypoxia may therefore not return to *stimulate* ventilation.

As a result of these difficulties attempts were made to devise systems using air at high flow rate with controlled oxygen enrichment. This was obtained by a loose-fitting mask, with air supply from a cylinder or pump at a flow rate of 30 - 50 litres per minute, and with additional oxygen at a controlled rate to produce an accurate control of the inspired oxygen concentration. With this method, rebreathing is negligible because of the flushing ; there is no dead space, and the flow rate is very much greater than the inspiratory peak flow rate. The many considerable practical disadvantages were a contraindication to its use until the Venturi principle was adopted. The present design has a jet set to deliver 1.5 - 2 litres of oxygen per minute. This produces sufficient negative pressure to entrain 50 litres of air per minute, and give a basic flow rate of 50 litres of air containing 24% oxygen per minute. Additional oxygen is then added at low pressure through a second tube to produce any desired oxygen concentration. Air sampling has shown that the concentrations produced are $\pm$ 0.5% of the predicted concentration, and that the concentration of CO_2 is less than 0.5%. This is simple to use, economical of oxygen, and requires no humidification as atmospheric air is used. However additional further humidification is possible. Campbell (1960) suggests a very sensible routine for the treatment of respiratory inadequacy or failure.[8]

1. Decide if ventilatory failure is present or not by clinical or clinico-physiological methods and biochemistry. In the absence of failure one should give oxygen in the conventional way with high flow rates.

2. If ventilatory failure is present or suspected, 24% oxygen is given by the Venturi principle. The oxygen concentration is then increased by 2% steps (14 mm. Hg.) at 3 - 4 hourly intervals *provided there is no CO_2 retention.* When 35% oxygen is being tolerated, the conventional methods may be brought into use.

3. If there is CO_2 retention, continue at the highest rate which can be tolerated (approx. 25 - 26% O_2). This will relieve the hypoxia without further CO_2 retention.

4. Unsatisfactory progress will require Intermittent Positive Pressure Respiration with possible tracheostomy.

ARTIFICIAL RESPIRATION

Methods of artificial respiration have been in use for many years. There are three main groups.[17, 18]

1. Methods utilising the pressure changes on the chest well, abdomen, or diaphragm.

    (a) Manual methods.
    (b) Eve rocking bed or stretcher.
    (c) Cuirass or jacket respirator.
    (d) Iron lung or cabinet respirator.

All of these may be of use in the treatment of inadequacy as an emergency measure, but they have several obvious limitations. They can be used with safety and effectiveness only in the normally healthy patient who has no thoracic cage fixity or osseous fragility, or any decrease in pulmonary movement or decreased distensibility of lung tissue. They require a clear airway.

2. Methods utilising pressure changes through the airway.[13]

    (a) Mouth to mouth respiration.
    (b) Manual compression of the reservoir bag or other device ; i.e. the anaesthetic machine.
    (c) Intermittent Positive Pressure Respiration.

(a) According to Cox, Woolmer, and Thomas[13], mouth to mouth, or mouth to airway EXPIRED AIR RESUSCITATION offers an effective method of respiration, and should replace the standard methods of emergency artificial respiration. In expired air resuscitation, tidal volumes in excess of one litre can be moved with each breath. Certain investigators have shown that standard manual methods without an artificial airway do not move a tidal volume greater than the victim's dead space in 25 - 80% of cases. Aesthetic objections are overcome by the use of an airway such as the Brook airway. Gastric distension is of apparently little importance except in the infant, and lung rupture is unlikely unless there is gross disease or if the victim is an infant. Infection is possible, but can be prevented by the use of a bacterial filter. Such risk is unimportant, however, in a case of emergency. CO_2 levels are found to be satisfactory in subjects and tolerable in donors, and the possible circulatory effects will occur in most other methods of artificial respiration also. However, this may be a difficult method for use with the conscious patient. A clear airway is also necessary and can be maintained by the usual manual methods.[15, 22, 29]

(b) THE ANAESTHETIC MACHINE may be used effectively in the treatment of respiratory inadequacy. The most effective combination is one including a soda lime cannister for the absorption of CO_2. The principles of the mechanism are similar to I.P.P.R. machines, with the exception that the pumping is done by hand.

(c) INTERMITTENT POSITIVE PRESSURE VENTILATORS are of three types—

    (a) Pressure cycled ventilators.
    (b) Volume cycled ventilators (pressure limited).
    (c) Time cycled ventilators (pressure limited).

Pressure cycled ventilators have as a cycling mechanism a pressure sensitive valve which is actuated by changes in the patient's lungs at a pressure set by the anaesthetist. Volume cycled machines deliver a pre-set volume to the lungs irrespective of the pressure produced. They must be fitted with a pressure limiting device. Time cycled ventilators have the length of the inspiratory and expiratory phases determined by the anaesthetist. The valves open and close under electronic control, the absolute cycling times determining the number of cycles per minute. The pressure or air-flow is then varied to produce the required tidal volume.

It should be noted that triggers and humidifiers are available for employment in the ventilator circuit.[17, 18, 25]

Controlled respiration has a definite effect on the circulation. The low pressure pulmonary vascular system has an average pressure of 10 mm. Hg. with a range of 7 - 15 mm. Hg. I.P.P.R. transfers a similar pressure to the alveolar wall, and if the alveolar pressure is high, there is ' milking out ' of blood from the lungs and a transient slight increase in cardiac output from the left heart, a damming back of blood to the right heart, and a decreased filling gradient of the right heart from the great veins thus leading to decreased cardiac output. A significant fall in cardiac output will not occur if the positive pressure is limited to a short portion of the respiratory cycle, if the expiratory phase at ambient air pressure is made as long as possible, and if no severe blood loss or other cause of hypovolaemia exists. Therefore a high instantaneous air flow, the positive pressure inspiratory phase, will produce an adequate tidal air. This is followed by a rapid passive deflation to zero pressure. Addition of a negative phase (not greater than – 10 cms. H_2O) when the airflow has stopped at the end of the expiratory phase will reduce the mean pressure and improve the cardiac output.[17]

3. Methods using electrical stimulation.
  (a) Electrophrenic respirators.

These have practical disadvantages which limit their use considerably. In the past they have been used almost exclusively in the resuscitation of the newborn infant.

Drug Therapy

Some investigators and physicians advocate the use of respiratory stimulant drugs. Either 2.5% aminophylline or 25% nikethamide[23] may be given intermittently or continuously by a slow intravenous infusion. Close medical supervision is required as large doses are necessary, the aim being to increase the ventilation without producing the side effects of nausea and vomiting (aminophylline) or of convulsions (nikethamide). These investigators advocate the use of these drugs in conjunction with the conventional face mask methods. However, it is stressed that should these measures prove ineffective, due to the amount or stickiness of secretions, or due to dangerous ventilatory depression, tracheostomy and I.P.P.R. is indicated and should be resorted to without delay. Nevertheless because of the insecurity of these measures the patient may require such therapy for many days, and because the aetiological factors of the inadequacy may be contraindications to the effectiveness and desirability of drug therapy, I feel that although these measures may play an important part in the treatment of certain selected and mild cases, they have, as a whole, little indication for their use. Further it is not generally appreciated that the administration of intravenous respiratory stimulant preparations can produce an immediate respiratory and/or cardiac arrest in cases of inadequacy. This may possibly be due to the fact that these drugs are stimulating already exhausted and overworked vital centres.

TRACHEOSTOMY

Tracheostomy is indicated in all cases of respiratory inadequacy in which the dead space must be reduced either as a preventive or ancillary measure in the treatment. It is necessary for adequate bronchial toilet, in cases in which assisted respiration will be required for a considerable time, and in cases in which there is relatively acute or acute on chronic upper respiratory tract obstruction. With tracheostomy, strict aseptic technique is essential and humidification may be necessary to prevent tracheal crusting with its complications. Adequate toilet must be carried out to remove secretions and daily bronchoscopy may be necessary. Prevention of tracheal pressure necrosis must be ensured, and naso-gastric feeding will be essential as feeding difficulties arise with cuffed tubes and I.P.P.R.[17, 16]

GENERAL MANAGEMENT AND GENERAL NURSING CARE

It is possible to mention but the headings of the many duties which must be performed routinely while the patient is in the dangerous state of inadequacy or failure. The management is described for the patient on I.P.P.R. with tracheostomy.

1. PULSE and BLOOD PRESSURE must be taken and recorded every 10 - 15 minutes. This is essential to ensure satisfactory progress and must be enforced. It is preferable to have this done by a doctor initially.
2. There must be a special senior nurse and doctor (preferably an anaesthetist) in the presence of the patient 24 hours per day.
3. Proper TRACHEOSTOMY management must be carried out. This includes daily bronchoscopy.
4. DAILY CHEST X-RAY and clinical examination must be carried out to ensure against pneumothorax or some such complication. Equipment should be easily available to institute pleural drainage if necessary—e.g. water-seal drainage for spontaneous pneumothorax.
5. SEDATION must be adequate, and intravenous opiates or bariturates are indicated. Curarisation may be performed—e.g. in the tetanus case.
6. RENAL FUNCTION must be continually observed. 24-hour specimens of urine must be collected. if necessary by an indwelling Gibbon catheter. Fluid balance charts must be kept accurately.
7. BIOCHEMICAL INVESTIGATIONS must include B.U.N., serum electrolytes, Na^+, K^+, and Cl^-, and CO_2 combining power, as a minimum. Arterial pO_2 and pCO_2 readings would be of much greater value but present administrative difficulties make this impossible on a nation-wide scale.
8. NASO-GASTRIC TUBE FEEDING is essential to maintain the general nutrition of the patient. Overloading of the stomach should not be allowed to occur. This is a possible complication as a paralytic ileus occasionally follows tracheostomy.
9. PARENTERAL FLUIDS are required to maintain hydration, renal function, and correct the acidosis. Glucose, M/6 lactate, saline and added KCl are usually required.
10. HAEMATOLOGY should be done routinely as a precautionary measure. This should include a P.C.V. and white cell count. The haemoglobin level is of secondary importance to these above estimations.
11. ANTIBIOTICS must be given if indicated.
12. PHYSIOTHERAPY must be enlisted in attempt to prevent deep venous thrombosis, and to prevent joints from stiffening.
13. Aseptic dressing technique of the tracheostomy wound.
14. General nursing duties—prevention of pressure sores, bed-baths, change of clothes, etc. This is good for the patient's morale.

This formidable list is essential in every detail for the treatment of such cases. As the position improves, the intensity of management can be reduced, but only under the direction of the specialist in charge, and the patient can be weaned from the ventilator, and ultimately weaned from the tracheostomy, to commence a period of rehabilitation.

CONCLUSION

In conclusion I would emphasise that some knowledge of the condition of respiratory inadequacy should be essential for all medical attendants. I trust too that I have emphasised sufficiently what I consider the only permissible and rational approach to oxygen therapy. May I ask once more—How frequently do such patients die, and how frequently do we claim their death as being inevitable ?

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