Today's Treatment

Diseases of the respiratory system

Respiratory failure

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The term respiratory failure is used as a shorthand means of indicating an abnormally low arterial oxygen tension (P_{aO_2}), an abnormally high arterial carbon dioxide tension (P_{aCO_2}), or both. Abnormal blood gas values may arise in a wide variety of clinical circumstances. There is some virtue in oversimplifying a little so as to distinguish among three types of respiratory failure: (a) hypoxic respiratory failure, in which the P_{aO_2} is low but the P_{aCO_2} is not raised (and the defect is inefficient gas exchange in the lungs); (b) ventilatory failure, in which the P_{aCO_2} is raised and the P_{aO_2} is abnormally low in proportion (and the defect is underbreathing); and (c) a mixed form, in which the hypoxia is more severe than can be accounted for by underbreathing alone (and in which defective gas exchange in the lungs and underbreathing are both present). A common example of this third type of respiratory failure arises during a severe exacerbation of chronic bronchitis with severe airways obstruction, the management of which I want to discuss.

Recognition

Respiratory failure is confirmed by blood gas measurement, but its presence can usually be inferred from clinical observation. The patient is usually known to have had longstanding cough and dyspnoea, usually amounting to severe disability. Respiratory failure sometimes starts abruptly during a severe respiratory tract infection but more often it develops over days or weeks. Some patients present with acute worsening of chronic compensated respiratory failure of long standing. Respiratory failure may be signalled by increasing lethargy and somnolence, headache, and the onset of oedema. Examination shows central cyanosis and signs of airways obstruction (characteristic muffled cough, signs of overinflation, and paradoxical indrawing of the anterior costal margin being more reliable than the presence or loudness of wheezing or rhonchi). When the neurogenic drive to breathe is well preserved the patient will appear very breathless and the P_{aCO_2} may not be very high. When the drive to breathe is poor the P_{aCO_2} is high and the most obvious features may be confusion, stupor, or even coma rather than breathlessness. The periphery is generally

warm and the pulse strong. Muscular twitches and myoclonic jerking may be apparent. The jugular venous pressure is usually difficult to assess but may be clearly raised, and there may be tender enlargement of the liver and oedema.

Management

Any illness in which respiratory failure develops acutely is always serious and the patient should be admitted to hospital. In practice, assessment and treatment proceed hand in hand without delay. Management of the respiratory failure takes priority but as soon as possible information must be sought about the patient's history. Firstly, the patient's previous respiratory status and in particular what may be termed his "range"—that is, the distance that he has ventured unaided during the three months or so before the illness. The prospects for recovery are good when the patient has done shopping or worked and successively poorer if the range has extended only to the front door, to one floor, or no further than the bedside. The physician should ask the patient's closest contacts and the family doctor about the extent to which the patient's life has been acceptable or intolerable and whether he has had appropriate medical treatment and, where relevant, the necessary social support. Next the doctor should investigate the possibility of underlying asthma. The features that might suggest this are: childhood onset, nocturnal attacks, persisting morning symptoms, surprising variability, a previous response to steroid treatment, a family history, and also allergies, eczema, nasal operations, and lifelong abstinence from smoking.

An early chest radiograph is desirable in case of unrecognised pneumothorax or pneumonia.

OXYGEN TREATMENT

Because individuals with established respiratory failure tend to be insensitive to carbon dioxide and dependent on hypoxia for their drive to breathe, oxygen treatment should be undertaken only after careful thought. Severe hypoxia is dangerous and leads to confusion, worsening heart failure, acidosis, and ultimately depression of vasomotor reflexes and of ventilation itself; but in practice it is difficult to recognise severe hypoxia that is less than overwhelming. Cyanosis itself is not an indication for oxygen treatment. It is generally encountered below P_{aO_2} of about 6.5 kPa (49 mm Hg) but many patients are perfectly stable with a P_{aO_2} well below this level.

Sweating, tachycardia, restlessness, and moaning may indicate appreciable hypoxia at brain-stem level, but these are unreliable clues. Measuring the P_{aO_2} is the best means of assessing the severity of hypoxia, but it must be remembered

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that the arterial oxygen tension or content is only one aspect of the delivery of oxygen to the body and that cardiac output and haemoglobin concentration are two other factors to be considered. Most agree that a P_{aO_2} of less than 4.7 kPa (35 mm Hg) represents serious hypoxia however satisfactory the cardiac output, and that this is an indication for oxygen treatment. Above a P_{aO_2} of 6.5 kPa (49 mm Hg) oxygen is generally unnecessary. Between 4.7 and 6.5 kPa (35-49 mm Hg) the indications for oxygen treatment are debatable but strongest when there is severe heart failure, coexistent ischaemic heart disease, or severe breathlessness (when underbreathing on oxygen is less likely to develop).

Unfortunately those with the lowest P_{aO_2} tend to be those with the highest P_{aCO_2} and hence they are least able to tolerate further underbreathing when the hypoxic drive to breathe is assuaged by oxygen. In practice, when hypoxia is severe enough to merit oxygen treatment intensive measures to stimulate breathing are generally also needed. Oxygen should be administered in low concentration when the P_{CO_2} is raised and a Ventimask delivering 24% oxygen is suitable. Controlled low concentrations of oxygen do set limit on the underventilation that can occur, but it is incorrect to regard 24% oxygen as "safe." Some patients will underventilate sufficiently to allow the P_{aCO_2} to rise by a further 2 kPa (15 mm Hg) or more when breathing this mixture. Higher concentrations of oxygen (28 or 35%) may be useful in patients with a relatively low P_{aCO_2} but severe reduction in P_{aO_2} . Attempts to avoid the adverse effects of oxygen by giving it intermittently are illogical and potentially dangerous. If oxygen is necessary it is needed continuously until there has been substantial improvement, and close supervision is required. Whereas underventilation on oxygen may be evident within minutes from clinical observation or the results of blood gas measurements it may also develop insidiously over hours (commonly overnight) or days.

ENCOURAGEMENT TO COUGH

It is vital to ensure that the patient coughs adequately and produces sputum—deterioration is inevitable otherwise. This requires regular efforts from physiotherapists and other available staff. The patient should be exhorted to cough at least hourly until sputum is actually produced. The severely ill can be helped by the injection of nikethamide, 2 ml, together with aminophylline, 250 mg, over two minutes. This generally produces arousal, coughing, and commonly retching. In somnolent patients coughing can be induced by passing a nasal suction catheter far enough to irritate the larynx—the sputum being aspirated as proof of effect. If the patient does not respond to these measures then artificial ventilation will almost certainly be needed.

ENSURING ADEQUATE VENTILATION

Adequacy of ventilation is reflected by P_{aCO_2} measurements, which serve as a useful guide to severity and trend, but it tends to be judged ultimately by whether or not the patient remains lucid and co-operative. Respiratory stimulants may help, and intermittent intravenous injections of nikethamide and aminophylline before coughing sessions are quite effective and can be repeated up to every two hours. Alternatively, doxapram may be given by continuous intravenous infusion at a rate of 2 mg/min, increased if necessary to 4 mg/min. This largely protects against underventilation produced by controlled oxygen treatment.

A separate infusion of aminophylline may be given, with a loading dose of 500 mg for an average man by slow intravenous injection followed by 500 mg over four hours. This may produce useful ventilatory stimulation as well as bronchodilatation. Almost always breathing is more effective if the patient sits

upright, and skilled nursing to ensure this can transform the progress of a critically ill patient.

BRONCHODILATATION

Response to bronchodilator drugs is limited in a severe exacerbation of chronic bronchitis but even a small effect is worth while in respiratory failure. Aminophylline by intravenous injection or infusion is still the most widely used treatment. Clinical impression suggests that results are improved by combining it with inhalation of salbutamol. Patients in severe respiratory failure do not use pressurised aerosols well and the drug can be given more effectively by a mask incorporating a nebuliser (Bard "Minineb" or equivalent). Salbutamol 0.5% solution, 1 ml diluted to 5 ml with water for injection, may be given for two minutes every two hours and may be repeated after one hour over short periods. If oxygen is inappropriate the mask should be driven by compressed air. If the patient needs oxygen supervised stimulation of breathing and coughing will be necessary during the inhalation, the mask being exchanged for a Ventimask or equivalent immediately afterwards.

TREATMENT OF BRONCHIAL INFECTION

Acute bronchial bacterial infection can be assumed in a severe exacerbation. Treatment is directed towards *Haemophilus influenzae* and the pneumococcus unless there is high fever or other indication of pneumonia. Amoxycillin or talampicillin (500 mg eight-hourly for three doses then 250 mg eight-hourly) is satisfactory, and co-trimoxazole is generally equally effective. Patients too ill to take treatment by mouth may be given ampicillin by intravenous or intramuscular injection or co-trimoxazole by infusion. In a desperately ill aging patient with particularly foul purulent sputum chloramphenicol is a reasonable first choice and can produce surprising results.

OTHER MEASURES

When there is well-established pre-existing chronic ventilatory failure a powerful diuretic such as frusemide helps and may be needed in the long term after recovery from the acute episode. Patients with raised mean jugular venous pressure are commonly given digitalis, but it is doubtful if this has an important effect when heart rate is modest. When there are indications of coexisting asthma corticosteroid treatment should not be withheld from an ill patient. Whether or not steroids are relevant to the particular case is rarely answerable during the acute episode, and the need for such treatment must usually be assessed after recovery by withdrawal and reintroduction of the drug.

Noisy delirium may develop during severe respiratory failure, usually some time after the blood gases have improved. There is always a temptation to manage the delirium with sedatives (for the sake of other patients) but this must be resisted. The patient must simply have expert nursing, with the best available sound-proofing. Sedative drugs have no place in managing respiratory failure, and the patient's prescription sheet should be annotated accordingly. An occasional exception to this rule may arise in the care of the dying. Some patients with intense breathlessness who have had terrible immobilising disability for years despite optimal treatment may reach an end stage where control of unbearable dyspnoea and anguish has a higher priority than actual length of survival.

ARTIFICIAL VENTILATION

When the above measures fail artificial ventilation by intermittent positive-pressure ventilation (IPPV) must be con-

sidered. The indications for this are failure to achieve adequate oxygenation or persisting inability to cough up secretions because of stupor or exhaustion. When reliable information indicates that during the months before the episode the patient had already reached an intolerable stage of disability despite all available treatment it may not be kind to embark on IPPV but it should be started whenever doubt exists. Oxygen in whatever concentration is necessary should not be withheld in the short period between the decision and intubation and it must be continuous. When the patient is restless intubation should be undertaken only by the skilled, but when deep coma has supervened intubation will be easier and should be carried out without delay.

The aims of IPPV are to secure adequate oxygenation, to remove secretions by tracheal and bronchial suction, and sometimes to rest an exhausted patient. Vigorous ventilation is undesirable, and there is no need to lower the Paco_2 below about 8 kPa (60 mm Hg) initially as this may induce circulatory collapse and at a later stage lead to difficulty in withdrawing IPPV. Most patients will need IPPV until at least the morning after intubation, allowing the whole day for assessment after withdrawal. Further attempts at withdrawal are made daily. Tracheostomy is seldom needed. IPPV is usually appropriate when some reversible event such as pneumonia, abdominal surgery, or inappropriate sedation is a contributory factor, and is sometimes needed when the patient is admitted in coma or there is inadequate information about the earlier history. When oxygen is used sparingly and thoughtfully, however, and vigorous measures to stimulate breathing and coughing have been

performed, relatively few patients with a prospect of recovery will need IPPV.

ARRESTED IMPROVEMENT

Some patients relapse after initial improvement and remain somnolent and ill with a high Paco_2 of 10.5 kPa (80 mm Hg) or so, apparently needing oxygen from time to time. In these circumstances it may help (a) to have the patient sitting out of bed; (b) to give a large dose of frusemide; (c) to add dichlorophenamide 50 mg 12-hourly; (d) to stop oxygen treatment altogether and remove the mask; and (e) to consider venesection if the packed cell volume is over 53%.

Aftercare

After the patient has recovered from the illness several steps should be taken to improve his respiratory condition. He should be persuaded to stop smoking and, if obese, to lose weight, and sedatives should be avoided. Exacerbations should be treated promptly with antibiotics, and the patient and his family should be taught to recognise features of recurring respiratory failure promptly. The possibility of an asthmatic component must be investigated and treatment given if necessary. Finally, the patient should be encouraged to exercise regularly within the limits imposed by dyspnoea.

Medical History

Schumann's hand injury

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The tragic story of the life and death of Robert Schumann has fascinated both musicologists and medical men. The history of the mental illness that afflicted him from the age of 18 until his death at 46 has been amply recorded. For some 24 years this illness was characterised by fluctuations of mood, with many episodes of depression and identifiable, though less frequent, spells of elation. The composer recovered between these bouts of affective disorder. Around 1852, when Schumann was 42, symptoms of organic brain disease appeared, and in 1854 he entered a mental hospital after attempted suicide in the Rhine. There he died with terminal convulsions in 1856.

Slater and Meyer¹ looked at the history of these illnesses and reviewed previous diagnoses. They concluded that the true psychiatric diagnosis was manic-depressive psychosis with

ultimate dementia paralytica, thus siding with Gruhle² against Möbius,³ who believed that the diagnosis was dementia praecox, or schizophrenia.

Schumann complained of many neurological and psychiatric symptoms over the years, most of which appear to have been due to the affective disorder or later organic nervous disease. Slater and Meyer¹ attributed his giddiness and tinnitus (apparently without notable deafness) to a peripheral process, probably syphilitic. In fact, the known evidence does not indicate whether the causal lesion was central or peripheral. In February 1854 auditory hallucinations occurred, which can be ascribed to organic brain disease.

Exploration of neurological complaints in historical figures, before the introduction of systematic clinical examination and diagnostic laboratory investigations, is commonly unsatisfactory. The conclusions are generally tentative or speculative. Nevertheless, we were moved to review the evidence on Schumann's hand injury by reading Sams's two articles in *The Musical Times*.^{4,5} The second article contained new material from the Leipzig City Archives.⁶ By courtesy of Dr Sams we have been able to examine a copy of Rothe's report and discussion of the correspondence found at Leipzig. While further information may emerge in the future, the time seems ripe for a neurological study of this one aspect of Schumann's medical history. Our main sources have been Sams's two communications, Rothe's work, and Walker's biographical chapter in *Schumann, The Man*

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