THE ANAESTHETIST AND THE EMPHYSEMATOUS PATIENT

BY

J. F. NUNN

Research Department of Anaesthetics, Royal College of Surgeons of England

It is surprising how few anaesthetists have ventured to publish their observations on the management of emphysematous patients. These patients are commonplace on operating lists but are notoriously difficult to anaesthetize. The anaesthetist is mainly concerned with emphysematous patients in four situations:

1. Anaesthesia for extra-thoracic operations.
2. Thoracic surgery which may result in a further reduction of pulmonary function.
3. Thoracic surgery intended to improve pulmonary function.

ANAESTHESIA FOR EXTRA-THORACIC OPERATIONS

The problems.

In this country the anaesthetist usually encounters emphysema as a complication of chronic bronchitis. In addition to the problems of chronic infection, such patients present difficulties due to disordered pulmonary function and an altered response to drugs. Further complications arise from the sequelae of chronic respiratory inadequacy such as cor pulmonale, cerebral hypoxia and raised C.S.F. pressure.

It is well known to the anaesthetist that patients with chronic bronchitis have unusually sensitive respiratory tracts. Ether inductions are prone to be interrupted by periods of breath holding and coughing, and mechanical irritation of the trachea—particularly intubation or inflation of the lungs—may result in bronchospasm. Anaesthesia is frequently followed by an exacerbation of bronchitis leading to bronchopneumonia, which may have fatal consequences in the patient whose reserve of pulmonary function is already reduced.

Various aspects of the disordered pulmonary physiology of the emphysematous patient become evident to the anaesthetist. Apart from bronchospasm there may be considerable resistance to the flow of gases in the respiratory tract. The small bronchi may be structurally narrowed, but the highest resistance is encountered during expiration from airway closure. A spirogram of a forced expiration in a conscious emphysematous subject will reveal a reduced flow rate and in addition the expiration is arrested at a higher lung volume than the patient's customary functional residual capacity (F.R.C.); this is due to air trapping. The level at which trapping occurs is related to the gas flow rate during expiration. During the early part of a normal expiration, the gas flow is retracted by a gradual "let-down" of the tone of the inspiratory muscles. During intermittent positive pressure ventilation of the paralyzed subject, however, the airway pressure is often suddenly reduced at the end of inspiration, and the expiratory flow rate therefore tends to be maximal at the commencement of expiration. Thus trapping will be very likely to occur and the patient's expiratory level will rise above the F.R.C. More vigorous attempts at inflation will merely raise the expiratory level further and a number of anaesthetists have reported that emphysematous patients have become hyperinflated during artificial ventilation (Johnstone, 1956; Jacques, 1957).

The rate of uptake of anaesthetic gas is reduced by the large F.R.C. and by the impaired distribution of inspired gas. This is of minor importance with agents which exert their effect at relatively low concentrations in the blood—such as halothane and cyclopropane. With ether, however, a much higher concentration is required to produce anaesthesia and the time taken for induction is considerably prolonged. Postoperatively a longer time will be required for elimination. Nitrous oxide occupies an intermediate position as regards solubility in blood. Even in severe emphysema the time required to reach a narcotic
concentration in the blood will be sufficiently short for the modern techniques of supplemented nitrous oxide anaesthesia for major surgery. During anaesthesia for short outpatient surgery, however, it is doubtful whether an adequate blood level would be reached in the time usually allowed for induction under such circumstances.

Many of the difficulties of the anaesthetist are related to the reduced response of the emphysematous patient to high alveolar concentrations of carbon dioxide (Scott, 1920). Much of the apparent tolerance is due to mechanical inability to respond. It is known that hypoxia plays an active part in the stimulation of respiration in these patients and saturation with oxygen will markedly reduce the ventilation in patients with severe emphysema (Donald, 1949). Many anaesthetists habitually use high concentrations of inhaled oxygen during anaesthesia and this practice will not only reduce the spontaneous ventilation of emphysematous patients, but will, to a large extent, mask the hypoventilation by preventing the appearance of cyanosis. The situation is analogous to the treatment of emphysematous patients in oxygen tents and dangerous carbon dioxide retention can occur without warning. If, on the other hand, the inspired gas mixture contains only 20 per cent oxygen, the patient with severe emphysema will probably be cyanosed during anaesthesia with unassisted spontaneous respiration. The anaesthetist may thus be confronted with the choice between a pink underventilated patient who is retaining carbon dioxide and a cyanosed patient with a reasonable minute volume. It is perhaps hardly necessary to say that the addition of carbon dioxide to the inhaled gas mixture is unlikely to increase the ventilation.

The emphysematous patient may differ from the normal subject in his response to drugs. In particular there may be considerable respiratory depression following opiates and barbiturates. The muscular pattern of inspiration is abnormal in emphysema and a partial myoneural block may result in a disproportionate reduction in ventilation. The use of relaxants is further complicated by the hazard of neostigmine increasing bronchomotor tone, which may be of critical importance during anaesthesia complicated by bronchospasm.

Cor pulmonale is a complication from which the patient may ultimately die. It is therefore important not to subject the right ventricle to an unnecessary strain, such as might arise from increased pulmonary vascular resistance during severe hypoxia or some forms of artificial ventilation. If the brain has been exposed to chronic hypoxia, it is probable that it will be less able to withstand an additional acute episode of hypoxia.

Assessment.

In this symposium Hugh-Jones (1958) and Dornhorst (1958) have discussed the assessment of the patient. For routine surgery, the anaesthetist often sees the patient only the night before operation when it is unusual to have available more than the history, physical examination and perhaps a chest X-ray. Fortunately the history yields some of the most valuable information.

The anaesthetist must first decide whether the risks of anaesthesia are justifiable and for this he must consider both the medical and surgical condition of the patient. Secondly, he must consider whether any advantage would be gained by postponing operation. Many patients in surgical wards may not have had effective medical treatment in the past, particularly if their condition has deteriorated while on the waiting list. It is not possible to improve all aspects of the emphysematous process, but it is wise to treat infection and bronchospasm. In patients with advanced emphysema it may be advisable to postpone operation until the warmer weather. The third problem for the anaesthetist is the choice of the anaesthetic technique and the drugs which should be used.

Management.

Most anaesthetists find the management of emphysematous patients difficult and there are a number of different schools of thought concerning how best they may be anaesthetized. Probably no method will be suitable for all patients and each should be independently reviewed.

Regional and local analgesia offer the advantage of minimal interference with the disordered physiology of the patient, and spinal analgesia was once popular for emphysematous patients, being particularly suitable for genito-urinary units where so many emphysematous patients are encountered. Recently, however, the neurological sequelae of spinal analgesia have greatly reduced the popularity of the technique in this country although epidural block may be a suitable alternative.
Unfortunately emphysematous patients are not always able to co-operate fully, particularly if their position on the table hampers respiration. A laparotomy under regional analgesia is at best an ordeal, but it will be far worse should the patient normally rely upon the assistance of his abdominal muscles during expiration. The role of the abdominal muscles is, however, open to considerable doubt (Campbell, 1958). Heavy sedation is dangerous in these patients as it may easily cause respiratory depression, possibly followed by a vicious circle of carbon dioxide retention. Nevertheless, for certain operative sites—particularly the upper limbs—regional techniques are very suitable.

In spite of the hazards of rendering emphysematous patients unconscious, surgery is usually carried out under general anaesthesia. Premedication is the first and not the easiest problem to be faced. Heavy sedation with opiates or barbiturates may be dangerous; pethidine is, however, a suitable alternative. Hyoscine may cause disorientation in the elderly and atropine should be substituted. Promethazine is a valuable drug combining sedation, antihistamine action and some anticholinergic effect.

If the patient is to be rendered unconscious the next decision is whether to permit spontaneous respiration or to use artificial ventilation. The proponents of spontaneous respiration can put forward a number of advantages for their method. The respiratory response of the patient to the inhaled gas mixture is under continuous review and some of the difficulties in the re-establishment of spontaneous respiration after artificial ventilation are thereby avoided. Auld (1956) described an emphysematous patient who, after a period of artificial ventilation, would not resume respiration until he became mildly hypoxic and thus restored the "anoxic drive" on which his respiration normally depended.

The author has generally found more difficulty in restoring the carbon dioxide tension to a satisfactory level. Spontaneous respiration is unlikely to commence if the arterial carbon dioxide tension is more than about 10 mm Hg below the tension to which they are accustomed, and in emphysematous patients that tension may closely approach the level at which respiration is depressed by the narcotic action of carbon dioxide. The anaesthetist may thus be reduced to raising and lowering the carbon dioxide tension alternately in the hope of striking a level which is compatible with spontaneous respiration. Those who are not practising anaesthetists may well criticize the empirical nature of this procedure, but it is virtually impossible to make a precise estimate of the patient's carbon dioxide tension after two or three hours of artificial ventilation. Analysis of the arterial blood is seldom practicable and unfortunately the alveolar gas of patients with severe emphysema is not sufficiently uniform in composition to warrant determination of its carbon dioxide composition. As it is rare for there to be any biochemical evidence of the cause of the apnoea in these circumstances, it is probable that many problems of this nature are dismissed as an abnormal response to relaxant drugs.

The second problem of artificial ventilation is mechanical. During inflation high pressures may be required to overcome the airway resistance, although still greater resistance is usually encountered during expiration. Trapping and diminished elastic recoil leading to progressive overinflation have already been mentioned. Fortunately it is uncommon to meet this complication which may reveal itself by a greatly reduced ventilation (Jacques, 1957) or by a displacement of the diaphragm into the abdomen (Johnstone, 1956). With an intact thoracic cage, hyperinflation will raise the intrathoracic pressure to a high level and it may not be possible to compensate for obstruction of the venous return by elevation of the peripheral venous pressure. The patient is thus simultaneously faced with respiratory and circulatory failure. Johnstone (1956) describes dramatic relief brought about by manual compression of the upper abdomen and chest while Jacques (1957) brought equally dramatic relief to her patient by the employment of a subatmospheric airway pressure during expiration.

Apart from assisting expiration and improving the venous return, a "negative" pressure phase may prevent excessive right ventricular strain, which is important if the patient has pulmonary hypertension. However, a sudden drop of airway...
pressure produces conditions similar to those during a forced expiration and may give rise to trapping—reducing rather than increasing the expiratory tidal volume. It is therefore advisable to reduce the expiratory airway pressure gradually while noting carefully any change in the tidal volume. The pressure could then be set a little higher than the pressure which resulted in the largest expiration. A pressure of the order of 5 cm water below atmospheric would probably be safe and should be applied gradually in the latter part of expiration. Siebecker and Curtis (1957) have described artificial ventilation in twelve emphysematous patients in whom they employed pressures 12 cm of water below atmospheric during expiration. They were able to maintain the mean arterial carbon dioxide tension at 45.5 mm Hg.

The third objection to artificial ventilation is the occasional development of severe broncho-spasm following intubation and inflation. This complication is not confined to artificial ventilation and there have been cases when spasm following intubation has presented an immediate threat to life. It has been shown by Campbell et al. (1958) that artificial ventilation of the normal subject causes some imbalance of ventilation-perfusion relationships compared with spontaneous respiration while conscious. The emphysematous patient, however, already has defective relative distribution of blood and gas. Whether artificial ventilation accentuates the existing imbalance is not known but there have been suggestions that it may actually cause an improvement.

There remains the difficult question of the possibility of alveolar rupture during artificial ventilation. Gas has been known to penetrate the alveolar wall and either enter the pulmonary blood vessels or dissect back along the perivascular sheaths to the hilum. Having reached the mediastinum, gas may enter the pleural cavity or track along various planes of the connective tissue (Marcotte et al., 1940; Whittenberger, 1955). Similar complications may follow the retention of gas in the lungs during escape from submarines by free ascent (Woolmer, 1958).

The danger of rupture is related, not to the absolute alveolar pressure, but to the gradient across the walls of the alveolus (transmural pressure). In his classical experiments on alveolar rupture, Macklin (1937) inflated only one lobe or a part of one lobe, so that it was possible for the transmural pressure of the inflated alveoli to approximate to the inflation pressure. This would not normally apply during inflation of the whole of both lungs, since the intrapleural pressure would follow the alveolar pressure—differing only by the elastic pressure of the lungs.

In the emphysematous patient, however, local differences in airway pressure affect the distribution of inspired gas according to the rate of inflation. Thus some alveoli might rapidly attain the airway pressure while adjacent alveoli were temporarily protected by the resistance offered by their ducts (Pask, 1958). In this manner it would be possible to develop considerable transmural pressure in parts of the lungs, particularly if high inflation pressures were required to overcome the airway resistance as a whole.

In summary, it may be said that artificial ventilation usually guarantees adequate gaseous exchange for the duration of the anaesthetic and many anaesthetists regard this as more important than the objections listed above. However, if little or no relaxation is required, spontaneous respiration will usually be satisfactory if moderate desaturation of the arterial blood is accepted. The addition of oxygen to the inspired mixture must be cautious, as respiratory depression is easily produced and may be masked.

Although spontaneous respiration may be satisfactory for operations which do not require relaxation, it is considerably more difficult to manage during laparotomy. Even in the normal patient relaxation suitable for abdominal surgery is frequently associated with a reduced ventilation and, in an emphysematous patient with disordered mechanics of breathing, there may be severe hypoventilation. It would appear that accessory muscles which are important to the emphysematous patient—such as the sternomastoids—do not contract effectively during deep anaesthesia. Thus, for laparotomy, there is a particularly strong case for the use of artificial ventilation as a routine for patients with severe emphysema. If it is intended to retain spontaneous respiration, the more powerful inhalational agents are easier to use than a combination of the relaxants and supplemented nitrous oxide. Ether is generally satisfactory once anaesthesia is established, but, for the reasons
given above, induction may not be easy. Much of the difficulty of the induction may be avoided by intubating the patient after thiopentone and suxamethonium, and subsequently building up the blood ether level during artificial ventilation before spontaneous respiration returns. Ether is an effective bronchodilator but this effect may be offset by increased secretion of mucus. Cyclopropane causes dangerous degrees of respiratory depression and certainly should not be administered with the customary high concentration of oxygen. It is possible that halothane will prove to be a satisfactory agent for emphysematous patients.

The choice between spontaneous and artificial ventilation rests not so much on the rival merits of the two methods, but rather on which has the fewer difficulties and dangers. It is impossible to be dogmatic on which technique is most suitable for all patients and the anaesthetist should be guided by the patient, the operation and also by his own experience and competence with the two techniques. It must be stressed that oxygenation is vastly more important than any other factor to do with respiration. This is particularly true in emphysematous patients in whom hypoxia will tend to cause pulmonary hypertension which may precipitate right ventricular failure.

In the emphysematous patient there is little doubt that topical anaesthesia should be the method of choice. If general anaesthesia is necessary, curarization will usually be required to control the cough reflex, and steps must then be taken to maintain oxygenation and relaxation while the bronchoscope is in place.

In the emphysematous patient there is little doubt that topical anaesthesia should be the method of choice. If general anaesthesia is necessary, curarization will usually be required to control the cough reflex, and steps must then be taken to maintain oxygenation and relaxation while the bronchoscope is in place. Preliminary inhalation of oxygen is valuable and oxygen may be insufflated down the side of the bronchoscope, although diffusion will not be wholly satisfactory. There is little danger of carbon dioxide retention in such a short procedure. A croup respirator used in conjunction with a short-acting relaxant is probably the safest form of general anaesthesia for this combination of a difficult procedure and a difficult patient.
The first steps should be taken to treat the causes of respiratory failure which are associated with the anaesthetic and are therefore temporary. Attention should be paid to residual curarization and to central depression from barbiturates or opiates which have been exhibited over the previous two or three hours. The dressing and position in bed should be examined and the pressure in the pleural cavity should be measured to exclude tension pneumothorax, since rupture of bullae may easily occur at thoracotomy. The presence of a drain will not necessarily rule out this possibility as drains can easily become blocked or kinked in the immediate postoperative period.

Chemical stimulation is worthy of trial. Bemegride or amiphenazole are the most suitable agents but the patient must be carefully watched and the drugs repeated as necessary. On occasion nikethamide will lighten the level of consciousness to the stage at which it is possible to obtain the cooperation of the patient and to exhort him to ventilate more vigorously. If, in spite of these measures, the patient is unable to accomplish satisfactory gas exchange, it will be necessary to ventilate him artificially until the temporary hindrances to respiration have subsided (Björk and Engström, 1955). This may take a few hours or a few days and, on rare occasions, it may eventually become apparent that the patient has become a permanent respiratory cripple.

After a thoracotomy it is seldom possible to carry out efficient artificial ventilation for any length of time without a tracheostomy. Not only is it essential for intermittent positive pressure respiration (which is the best method), but it is also valuable for clearing secretions. The reduction in deadspace amounts to about 70 ml and sometimes tracheostomy alone is sufficient to prevent the patient developing carbon dioxide retention. Certainly a tracheostomy will enable a patient to resume spontaneous respiration at the earliest possible time after artificial ventilation. Tracheostomies heal so quickly that some units perform them routinely if there is a likelihood of an emphysematous patient having respiratory inadequacy or troublesome secretions after pulmonary resection. It should be remembered that the efficiency of a cough is already reduced in emphysema and still further reduced in the postoperative period.

A mechanical ventilator is probably superior to manual ventilation in the treatment of carbon dioxide retention postoperatively because the ventilation is apt to be interrupted by periods of hypoventilation when an assessment of the patient's ability to breathe is made. It is more satisfactory to maintain artificial ventilation until the patient is fully conscious. Certainly little harm and even some good may be achieved by continuing artificial ventilation longer than it is required.

Postoperative carbon dioxide retention may not develop until after the patient has returned to the ward. Recognition of the condition is important and the nursing staff should be warned of the possible dangers of oxygen therapy which may precipitate and also mask hypventilation. A rising blood pressure is not invariably seen in carbon dioxide retention, but if the blood pressure after operation rises progressively above the pre-operative level it is sufficient evidence—particularly if it is associated with deepening of the plane of consciousness. A half-hourly blood pressure record is a valuable precaution and it is essential that all concerned should be aware of the significance of a rising pressure. Sweating associated with a flushed appearance is also suggestive of carbon dioxide retention and an irregular pulse has also been noticed at a late stage shortly before death. Postmortem examination reveals no pathognomonic features and the cause of death can seldom be established unless diagnosed during life.

Prediction of the ability of a patient to ventilate after pneumonectomy is difficult. Contralateral disease is of great importance and emphysema is probably the commonest cause of disability in this respect. Not only must the maximum breathing capacity be assessed but also what part of the total function is carried out by the lung which will be removed. Bronchospirometry would appear to be the most satisfactory method, but practical difficulties have retarded its use. Screening is often of value but considerable experience is required to relate the appearances to function. The respiratory quotients of gas expired from the two main bronchi will also indicate the relative function of the two lungs. Samples may be collected during bronchoscopy (Armitage and Taylor, 1956). The overall function should only be considered in relation to the differential function of the two sides,
but it is generally agreed that a maximum breathing capacity in an adult of 35 litres per minute or less should be regarded as a contra-indication to operative interference (Donald, 1953).

It is perhaps outside the scope of the anaesthetist to consider the rise in pulmonary artery pressure which may follow pneumonectomy in the emphysematous patient. During cardiac catheterization it is possible to occlude the pulmonary artery of the diseased side and so to predict the rise in pressure which may be expected to follow pneumonectomy (Hanson, 1954; Nemir et al., 1956). In this way it may be possible to avoid surgery in those patients in whom resection might be followed by right ventricular failure.

After pneumonectomy the remaining lung expands and this condition is known as compensatory emphysema. There is some doubt as to whether it constitutes distension or true emphysema. Certainly if the remaining lung is already emphysematous the distension will be harmful and this factor must be considered in the preoperative assessment. Thoracoplasty may be employed to prevent overdistension of the remaining lung but its use is not widespread in this country.

THORACIC SURGERY INTENDED TO IMPROVE PULMONARY FUNCTION

Denervation.
Patients who have considerable relief from bronchodilators may benefit from resection of the parasympathetic nerve supply to the tracheobronchial tree. There may also be some advantage in removing the pathway from the stretch receptors. Denervation procedures are, however, rare in this country.

Removal of air cysts.
Occasionally a patient’s disability may, in large measure, be due to air cysts or bullae. These may be entirely closed or in communication with the bronchus—the opening often being valvar and resulting in progressive distension. Improvement in function of the remaining lung may be obtained by obliteration or resection of the cyst (d’Abreu, 1953; Bromley, 1958).

Spontaneous pneumothorax.
Although this condition may occur in patients with no obvious pulmonary disease, it is frequently associated with either bullous or generalized emphysema (Brock, 1948). The recurrent condition may be treated by chemical pleurodesis, but some prefer to seal the leak at thoracotomy (Brewer et al., 1950). Anaesthesia presents no problems beyond those of thoracotomy in emphysematous patients.

ARTIFICIAL VENTILATION FOR CARBON DIOXIDE NARCOSIS

A small number of patients with severe emphysema pass into carbon dioxide narcosis which may be resistant to chemical stimulation. A tracheotomy alone may be sufficient to restore adequate alveolar ventilation but occasionally it is necessary to resort to artificial ventilation. A number of centres have used the technique and have managed to reduce the arterial carbon dioxide tension below narcotic levels. Many anaesthetists have now built up considerable experience in the long-term ventilation of patients in respiratory units, and their help may be sought in the treatment of carbon dioxide narcosis.

The principles of treatment have been outlined by Dornhorst (1958) in this symposium and it will be apparent that prolonged ventilation of unconscious emphysematous patients should not be lightly undertaken. To the problems of long-term artificial ventilation are added those of emphysema and specialized knowledge of each is required.

SUMMARY

Emphysema presents such a widespread disturbance of pulmonary function that anaesthesia may be complicated by a number of different factors. A knowledge of the disordered function due to the disease is essential to an understanding of the problems which confront the anaesthetist.

Associated bronchitis causes the tracheobronchial tree to be hypersensitive and anaesthesia may be complicated by coughing, breath holding or bronchospasm. Postoperative exacerbation of infection is a frequent occurrence and may have serious consequences.

Induction by inhalational anaesthesia is complicated by the delayed uptake of gases due to defective mixing. Respiration in emphysematous patients is particularly sensitive to depressant drugs and, in addition, they have a diminished
ventilatory response to a raised arterial carbon dioxide tension. Furthermore, reliance of these patients on the "anoxic drive" reduces the alveolar ventilation when the inspired oxygen concentration is raised unduly. These factors may precipitate a vicious circle of carbon dioxide retention leading to narcosis. After a long period of artificial ventilation, there may be some difficulty in restoring the blood gas tensions to levels which are compatible with spontaneous respiration.

Expiratory obstruction may complicate artificial ventilation and lead to hyperinflation with both respiratory and circulatory embarrassment. Cautious use of a reduced airway pressure during ventilation and lead to hyperinflation with both respiratory and circulatory embarrassment. Cautious use of a reduced airway pressure during expiration may be of value.

Neither spontaneous respiration nor artificial ventilation is free from difficulties and many prefer to use local or regional analgesia when such techniques are practicable.

After thoracotomy there is danger of postoperative hypoventilation, particularly if much functioning lung has been removed. Artificial ventilation may be the only effective treatment.

Mention is made of certain operations to improve function in emphysematous patients and attention is drawn to the possible role of the anaesthetist in the treatment of carbon dioxide narcosis by artificial ventilation.

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