POSTOPERATIVE RESPIRATORY FAILURE:
AN ANAESTHETIC HAZARD?

J. C. STODDART

Since the state of anaesthesia possesses no intrinsic merit and as far as the patient is concerned merely comes between him and the real business of surgery, the ideal anaesthetic technique would be one of which he was totally unaware. It would have no unpleasant preliminaries or sequelae and certainly would be devoid of all hazards, irrespective of the state of health of the patient, or of the planned operation. For the surgeon, perhaps the ideal anaesthetist would be a modest assistant who provides sound but not time-consuming or delaying advice before operation, who transforms the patient into a perfect medium for the exercise of his talents without in any way obtruding upon his activities, and then dissolves into the aether, leaving behind a patient who is both none the worse for the anaesthesia and cured by the surgical procedure.

Since both of these ideals are as yet unattainable it may be salutary to examine the present state of the art and science of anaesthesia with particular reference to its undesirable effects upon the respiratory system. Many of these have recently been reviewed in this journal (Hewlett and Branthwaite, 1975) and it would be superfluous to reiterate them. It is therefore the purpose of this presentation to describe some of the ways in which the modern anaesthetist may, if he is not careful, fail short of the ideals expressed above.

It is based upon the sweeping assumption that, since most of the complications and hazards listed below are well recognized and can be either prevented or minimized by correct treatment, they should rarely lead to disaster.

The list is by no means exhaustive, but is merely an outline of the many ways by which a patient may find himself in respiratory difficulty after a sojourn in the operating theatre.

The ultimate causes of respiratory failure following surgery and anaesthesia may be grouped as shown in tables I, II and III.

The part played by bad or ill-considered anaesthesia in the development of respiratory failure will be considered in each of the three phases of the anaesthetist's management of the patient, namely the preoperative assessment and preparation, the induction and maintenance of anaesthesia and the recovery and postoperative phases.

PREOPERATIVE ASSESSMENT AND PREPARATION

Although the conscientious anaesthetist always sees his patient in advance of the planned procedure, some patients arrive in the anaesthetic room never having been seen by the anaesthetist. He may know about the patient in the sense that an announcement of arrival has been made, and the patient may have been "clerked" and premedicated by a medically qualified person. The anaesthetist who is to perform the act will have read the notes made by a house surgeon and have come to a conclusion regarding the patient's

---

Table I. Causes of hypoventilation

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bronchitis, emphysema, asthma, pulmonary fibrosis</td>
</tr>
<tr>
<td>Pleural effusion</td>
</tr>
<tr>
<td>Pneumothorax, pneumoperitoneum</td>
</tr>
<tr>
<td>Thoracic trauma, thoracic surgery</td>
</tr>
<tr>
<td>Kyphoscoliosis, obesity</td>
</tr>
<tr>
<td>Neuromuscular blocking agents</td>
</tr>
<tr>
<td>Analgesics, anaesthetics, etc.</td>
</tr>
<tr>
<td>C.n.s. disorders</td>
</tr>
<tr>
<td>Hypoxia, hypercapnia</td>
</tr>
</tbody>
</table>

Table II. Causes of impaired diffusion and gas exchange

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atelectasis</td>
</tr>
<tr>
<td>Pulmonary fibrosis</td>
</tr>
<tr>
<td>Pulmonary oedema, pneumonia</td>
</tr>
<tr>
<td>Septicaemia</td>
</tr>
<tr>
<td>Pulmonary thromboembolism</td>
</tr>
<tr>
<td>Pneumonectomy</td>
</tr>
</tbody>
</table>

Table III. Causes of ventilation/perfusion inequality

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic bronchitis, emphysema</td>
</tr>
<tr>
<td>Asthma</td>
</tr>
<tr>
<td>Atelectasis, endobronchial intubation</td>
</tr>
<tr>
<td>Pneumonia—aspiration, bacterial or chemical</td>
</tr>
<tr>
<td>Thromboembolism</td>
</tr>
<tr>
<td>Shock lung</td>
</tr>
<tr>
<td>Septicaemia</td>
</tr>
</tbody>
</table>

© Macmillan Journals Ltd 1978
fitness to undergo the planned procedure. He may then, however, simply reproduce his standard anaesthetic technique which he knows "does" for patients who are to have the planned operation and which allows him to get away to his next list on time.

It may be confidently affirmed that the assessment of the patient's state before operation is the second most demanding part of the anaesthetist's task. The development of respiratory failure after operation begins with the patient's condition before operation. The anaesthetist's most important contribution to the patient's well-being may lie in persuading the surgeon that he is not yet (nor perhaps ever will be) ready for the proposed operation. Some anaesthetists take pride in never having refused to anaesthetize a patient, possibly ignoring the sequel which may be either to reduce his expectation of life, or else to add postoperative pain to terminal illness.

The preoperative conditions which increase the hazard of respiratory failure after operation can be summarized as follows:

- Pre-existing respiratory disease
- Peritonitis; ileus; septicaemia
- Dehydration
- Pain
- Obesity, kyphoscoliosis
- Vomiting, intestinal obstruction, electrolyte disturbance

The factors listed are also postoperative hazards for a variety of reasons. The pre-existence of respiratory disease is often cited as a contraindication to elective surgery. A patient with severe chronic obstructive airways disease can almost always be expected to develop some embarrassment after operation, so that with foresight the necessary arrangements can be made to cope with this. A patient with respiratory disease who is also dehydrated runs the additional risk of being unable to expectorate his thickened mucus, and so deserves an even more careful preparation before surgery.

Regurgitation and vomiting with or without intestinal obstruction and paralytic ileus have been the anaesthetist's nightmare for many years (Crockett, 1857; Edwards et al., 1956), and various techniques have been advised to reduce their dangers (Hall, 1940; Snow and Nunn, 1959; Inkster, 1963). The fact that so many methods are recommended emphasizes the dangers and difficulties which accompany anaesthesia in this situation; but again, to be forewarned is to be forearmed. The anaesthetist should never be the only sucker in the anaesthetic room.

The site of the planned operation is also of major significance. It has long been believed that abdominal and thoracic operations are more frequently followed by respiratory difficulty than are operations performed upon the extremities (Minnitt and Gillies, 1945). It may be in part a reflection upon careful selection before operation that Jordan (1923) could report anaesthesia for 11 200 orthopaedic operations with only one episode of bronchopneumonia. Recently the distinction between the effects of central and peripheral operations has been blurred, for Thompson and Eason (1970) and Scheidegger and his colleagues (1976) have stated that hypoxia occurred just as frequently after operations upon the extremities as after central operations. Nevertheless, the association between thoracic and abdominal procedures and major respiratory difficulties is well recognized (Beecher, 1933; Dripps and Denning, 1946; Diament and Palmer, 1966). The reasons for this association are multiple, and some are discussed later. That there is an association between obesity and respiratory difficulty has been known since the inception of anaesthesia (Minnitt and Gillies, 1945; Catenacci, Anderson and Boersma, 1961; Gould, 1962). The extreme example of this problem, the so-called "Pickwickian Syndrome" (Burwell et al., 1956), is not often encountered. Whenever possible, operations should be postponed to allow patients to achieve a satisfactory weight loss.

There is some evidence that the severity of pain after operation and therefore the incidence of postoperative pulmonary complications is influenced by the patient's neuroticism index (Parkhouse, Lambrechts and Simpson, 1961; Parbrook, Steel and Dalrymple, 1973). This appears to suggest that the anaesthetist requires an even greater understanding and skill than is normally exercised in handling surgeons, to reassure the patient before operation and to recognize pathological anxiety.

**PEROPERATIVE FACTORS**

The induction of anaesthesia is accompanied by many hazards, some of which have already been described, including asphyxia and aspiration of regurgitated gastric material. The process of endotracheal intubation carries multiple dangers which include mechanical obstruction and kinking, but that which is most commonly associated with respiratory complications after surgery is an unrecognized endobronchial intubation, followed by lung collapse. Endobronchial intubation can be avoided by the simplest possible manoeuvre (Stoddart, 1975) and is really inexcusable. When endotracheal intubation is performed casually
it may be more hazardous for the patient than not to attempt intubation at all. The same dangers exist for the patient with a tracheostomy.

During the maintenance of anaesthesia many other potential problems arise. Inadequate ventilation may cause progressive hypercapnia and hypoxia which may make the re-establishment of normal ventilation at the end of the procedure difficult. This may also be the result of a relative overdose of anaesthetic or of adjuvant drugs or of the too casual use of an automatic ventilator.

Much has been written about the importance of the bronchial cilia and their ceaseless activity in scavenging mucus and particulate matter from the depths of the lung. It has been shown that, in addition to many opiate and anaesthetic drugs, the inhalation of dry gases can depress their activity, but there is disagreement as to the ill-effects of short-term ciliary stasis. Some observers consider it to be potentially harmful (Dalhamm, 1956; Burton, 1962), predisposing to bronchiolar plugging and atelectasis. Other workers, although recognizing that ciliary stasis exists, doubt its significance (Knudsen, Lomholt and Wisburg, 1973). However, a more recent paper showed that the ill-effects of dry gases on mucus velocity appeared within 3 h (Hirsch et al., 1975). This must be considered an undesirable accompaniment of long operative procedures. The same authors showed that mucus movement began again when the inspired air was adequately humidified. Many anaesthetists routinely humidify the anaesthetic gases in order to maintain ciliary activity, to discourage the formation of mucus plugs, to conserve body heat (Lewis and McKenzie, 1972) and to increase the patient’s comfort after operation.

Although a great deal of work has been done to clarify the problem of postoperative atelectasis, it may be of interest to note that the first description of airless alveoli was given by Lichtheim (1879). Beecher (1933) was probably the first to relate it to anaesthesia, when he described a syndrome of hypoxia without underventilation and considered that atelectasis was its cause. The syndrome of absorption atelectasis after cyclopropane anaesthesia was proposed by Jones and Burbford in 1938 and the practical aspects of absorption atelectasis were investigated by Robertson and Farhi (1965) and by Dery and colleagues (1965).

It was hoped that the addition of insoluble nitrogen to the anaesthetic gas mixture either during or at the end of anaesthesia would reduce the incidence of atelectasis and hypoxia by “splinting” the alveoli, but this did not prove to be the case (Stevens et al., 1966; Webb and Nunn, 1967). Moreover, Colgan and Whang (1968) failed to show any difference in the occurrence of atelectasis and hypoxia following either inhalation or i.v. anaesthesia. It is now believed that, to reduce this hazard, efforts must be made to keep the alveoli fully distended (Cooper, 1972); but whether this is best achieved by delivering large tidal volumes to the patient, as suggested by Hedley-White, Laver and Bendixen (1964) and by Vissick, Fairley and Hickey (1973), or by the maintenance of positive end expiratory pressures, as advocated by Ashbaugh and Petty (1973) is still open to question.

The problems created by obesity have been mentioned already, but abdominal distension from other causes such as ileus or peritonitis may equally impede respiratory movements. It has also been known for some years that if air is allowed to collect beneath the diaphragm during upper abdominal surgery it can interfere with diaphragmatic descent and therefore with ventilation of the lower parts of the lungs (Bevan 1961a, b). Given thought, this is easily preventable by suction just before peritoneal closure.

It is frequently necessary to place a patient in the lateral position for surgical procedures. Langton Hewer (1943) stated that this interfered with breathing both during and after surgery and it has been confirmed that this position can give rise to the “dependent lung syndrome” with hypoxia before and after operation which may lead to persistent postoperative pulmonary oedema and atelectasis (Craig, Bromley and Williams, 1962).

There is no doubt that many patients are given excessive amounts of anaesthetic agents and adjuvants and are returned to the surgical ward without having fully regained their protective reflexes. Although most such patients survive, the combination of this state with the hazards of bleeding into the upper airway or regurgitation following abdominal surgery increases the probability of aspiration pneumonia (Bosomworth and Hamelberg, 1962; Alexander et al., 1973). Less obvious examples exist. Some valuable modern drugs, such as fentanyl, which are widely used to supplement anaesthesia are said to have a short duration of action and to be completely antagonized by other drugs, such as naloxone. Experience has shown that neither of these statements is always true and patients may disconcertingly lapse into sleep and respiratory depression after leaving the operating theatre, when this combination has been relied upon. This has been explained by pharmacokinetic studies (Bower, Holland and Hull, 1976). All depressant analgesic drugs share this potential for disaster.
Other examples can be found without difficulty. The epidemic of "neostigmine-resistant curarization" which was described in 1956 was in most cases explained by a relative overdose of relaxant drug followed by a relative overdose of neostigmine followed by further polypharmacy, respiratory stimulation by carbon dioxide and underventilation (see Gray, 1956). This was sometimes combined with an unwillingness to ventilate the paralysed patient for a sufficient period to allow all the drugs which had been given to disperse. Feldman (1963) has since shown that electrolyte disturbances and, in particular, hydrogen dispersion can influence the duration of action of muscle relaxants. More recently, the large number of operative procedures which are performed upon patients suffering from renal failure has shown that the duration of action of many drugs, including muscle relaxants such as tubocurarine and pancuronium, can be greatly increased (Gibaldi, Levy and Haytor, 1972; McLeod, Watson and Rawlins, 1975; Miller and Cullen, 1976; Rouse, Galley and Bevan, 1977). Nevertheless, their duration of action is still dose-related, which suggests that recognition of the problem and reduced dosage is all that is required (Matteo, Spector and Horowitz, 1974). The variation in the duration of other relaxant drugs such as suxamethonium is also well understood (Hodges and Harkness, 1954) and should never lead to disaster. In any situation in which doubt exists as to a patient's ability to breathe adequately, continued controlled ventilation and blood-gas analysis is all that is required. No matter how inconvenient this may be for the surgeon, the anaesthetist, the theatre staff or the other patients upon the list, polypharmacy and other means of stimulating a patient to breathe must never be adopted.

Attention has recently been drawn to the formation of pulmonary microemboli from fragments in stored blood (Olcott, Barber and Blaisdell, 1971; McNamara et al., 1972). This may be a cause of respiratory dysfunction after operation if large volumes of old blood are rapidly transfused. It is possible that the use of special filters will reduce this hazard (Cullen and Ferrara, 1974), although developments in the collection, separation and storage of blood for transfusion will probably eliminate this problem in the near future (Wallace, 1977).

THE RECOVERY PERIOD AND THE POSTOPERATIVE PHASE

It has been stated that respiratory failure after surgery may be initiated in the earlier phases of the proceedings. However, pain and terminal airway closure during quiet ventilation in the recovery period can also play an important part in its development. Pain from the operation site or from peritonitis will directly interfere with coughing. Langton Hewer (1943) stated that the pain of upper abdominal surgery causes shallow breathing and bronchiolar occlusion, followed by "apneumatosis", which presumably meant atelectasis. This opinion is shared by most other authors and Bromage (1955) used spirometry to assess the efficacy of analgesia after operation. In a later publication he stated that abdominal pain increased the transpulmonary pressure, reduced the functional residual capacity and encouraged airway closure; and he showed how this could be corrected by prolonged extradural analgesia (Bromage, 1967). Spence and his colleagues (Spence, Smith and Harris, 1968) confirmed these findings and also remarked upon the fact that patients in the control group who did not have extradural anaesthesia but were without obvious respiratory difficulty had still not achieved their preoperative arterial oxygen tension by the 5th day after operation. Later work from the same group and others more accurately defined the problem of terminal airway closure associated with pain and restricted diaphragmatic movement (Alexander et al., 1973; Hedenstierna, McCarthy and Bergström, 1976). It is now generally accepted that this is one of the most important causes of hypoxaemia after surgery, and that its prevention and treatment are difficult. It has been shown that physiotherapy carried out while the patient is in bed is of little value, but that ambulation immediately after operation may be more effective (Finley, 1968; Scheidegger et al., 1976). This is not always practicable, but it does emphasize that the condition is potentially remediable. Whilst on the subject of pain relief, it should not be necessary to reiterate that too much analgesia is as bad as too little; and that analgesic drugs should be prescribed for the individual patient, rather than simply to conform with ward custom.

Less important but still measurable factors may be implicated. Hypothermia resulting from exposure and reduced metabolic rate may cause shivering and increased oxygen consumption after operation (Roe et al., 1966; Bay, Nunn and Prys-Roberts, 1968). However, this is only of significance if the patient has a limited cardiac reserve and a low or fixed cardiac output. The adoption of heated humidification of the inspired air and other warming devices should reduce the frequency of this complication. Hypothermia per
and surgery have emphasized the importance of a satisfactory outcome is to be expected. Critical state for one or more of the reasons outlined measurement. Provided the patient is not already in a proper clinical observation aided, if necessary, by operating theatre can readily be recognized by the care of patients after operation has emphasized most of these problems have been known for many years. The increased involvement of anaesthetists in the references selected for this paper indicate that following operations are the result of simple mechanisms for the elderly patient in the periods both during and after operation. This simply confirms the opinions of other workers already quoted. It should be a stimulant to anaesthetists to provide what is now certainly the optimum method of anaesthesia for such patients, particularly when the alternative methods are so frequently unsatisfactory.

CONCLUSION
This review is intended to remind the anaesthetist of some of the respiratory hazards of his craft. Most of the complications mentioned are at least theoretically preventable by the clinician who is aware of their existence. Many of the problems are the results of omissions rather than of commission. Failure to visit and assess the patient before and after operation means that both the patient and the surgeon are deprived of both the potential benefit of good advice and the early recognition of specific problems which lie within the anaesthetist’s province. The use of polypharmacy when simple anaesthesia may suffice, or failure to recognize that some techniques really are better for specific situations, and that a standard method, although tried and trusted, is not always the best, may also be contributory factors. The anaesthetist who, by design or by force of circumstance, hands over his patient at the end of the operation before he is in a safe condition is simply tempting providence.

Most of the respiratory problems which are seen following operations are the result of simple mechanical or pharmacological causes. The dates of some of the references selected for this paper indicate that most of these problems have been known for many years. The increased involvement of anaesthetists in the care of patients after operation has emphasized one fact: the hazards which may attend a visit to the operating theatre can readily be recognized by proper clinical observation aided, if necessary, by measurement. Provided the patient is not already in a critical state for one or more of the reasons outlined above, a satisfactory outcome is to be expected.

Increasingly sophisticated methods of anaesthesia and surgery have emphasized the importance of attention to first principles. With recognition of this fact, the incidence of postoperative respiratory failure attributable to anaesthesia should decline.

REFERENCES