POSTOPERATIVE DISTURBANCES IN PULMONARY VENTILATION*

J. C. NICHOLSON, F.F.A.R.C.S., *Johannesburg*

Complications affecting pulmonary ventilation constitute one of the most important causes of morbidity and mortality associated with surgery and anaesthesia. The most dangerous time for the development of serious disturbances in pulmonary ventilation is the immediate postoperative period, especially before the return of consciousness.

While he is in the operating theatre, the patient's respiratory requirements are a first priority to the anaesthetist, but after his return to the ward there is often a deterioration in the standard of treatment and supervision that is available at the very time when it may be most needed. So well-recognized is this danger that the provision of special recovery wards for the postoperative management of surgical patients has come to be an accepted essential in all modern hospital planning. Unfortunately it is likely to be many years before the ideals embodied in this concept become a practical reality on a wide scale and, in the meantime, the hazards concerned must constantly be borne in mind.

THE IMMEDIATE POSTOPERATIVE PERIOD

Briefly summarized, the two main dangers at this stage are *obstruction to the respiratory passages,* which is predisposed to by the loss of reflex tone in the muscles guarding the upper respiratory tract, and *depression of pulmonary ventilation,* which may have a complex and variable aetiology, but in which drugs used in connection with the anaesthetic often play an important part. The two conditions are often present together and, when this is the case, each necessarily aggravates the effects of the other. Both obstruction and depression interfere with the two main functions of breathing, thus leading to *hypoxia* and *hypercapnia,* either of which, if not relieved, becomes a vicious circle with further deterioration of pulmonary ventilation (Fig. 1).

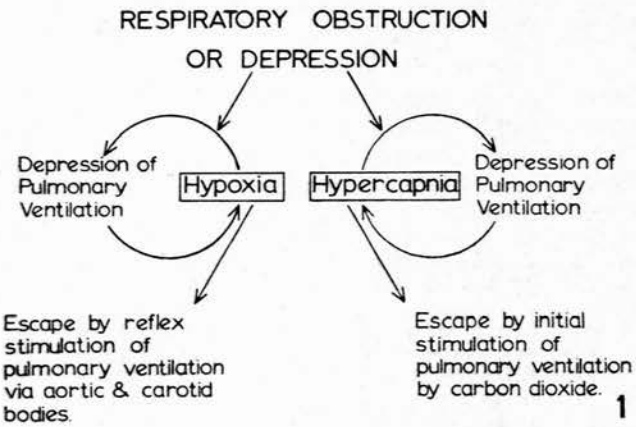

Fig. 1. Simplified diagram illustrating the vicious circles of hypoxia and hypercapnia with the possible escape mechanisms.

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There are natural defence mechanisms by which escape from these vicious circles is possible in favourable circumstances (Fig. 1). Hypoxia causes reflex stimulation of pulmonary ventilation through receptors in the aortic and carotid bodies before the respiratory centre succumbs to the effects of oxygen lack; and a raised carbon-dioxide tension in the blood is a powerful respiratory stimulant before depressant concentrations are reached. Unfortunately, if pulmonary ventilation is already seriously depressed by drugs or other agencies, these compensatory mechanisms may not be much in evidence and, with progressive hypoxia and hypercapnia, death from respiratory failure is inevitable in the absence of effective intervention.

The prevention and management of postoperative respiratory obstruction is largely a matter of competent supervision, but depressed pulmonary ventilation is a more complex and insidious factor from which, broadly speaking, serious complications may arise in two ways:

(*a*) From an error in the assessment of the adequacy and permanence of the re-establishment of normal breathing after anaesthesia.

(*b*) From the development of postoperative apnoea or grave respiratory depression requiring prolonged specialized treatment with artificial pulmonary ventilation.

Both these problems will be discussed.

POSTOPERATIVE PULMONARY VENTILATION

The transition from pulmonary ventilation under anaesthesia to normal breathing, after the discontinuation of the operation and anaesthetic, often involves considerable adjustment between the patient and his respiratory environment during which conditions may favour the development of hypoxia and hypercapnia and may contribute to a faulty assessment of his pulmonary function. Maximum readjustment is called for when the change is from passive pulmonary ventilation by intermittent positive pressure to normal breathing in the atmosphere but, even when spontaneous breathing has been allowed to continue throughout the operation, significant changes in the composition of the respiratory medium, the physiological dead space, and the tidal and minute volumes have to be considered.

Where rich oxygen mixtures have been used, it is obviously important to make sure that the patient does not become cyanosed when their administration is stopped. Further, the termination of the operation is frequently associated with a temporary functional decline in pulmonary ventilation, often accompanied by a fall in blood pressure. This is due mainly to cessation of surgical stimulation while some reduction in the carbon-dioxide content of the respiratory medium may be an additional factor. In a normal semi-closed anaesthetic, especially if an endo-

tracheal tube is used, the flow of gases has an insufflation effect which assists pulmonary ventilation and considerably reduces the physiological dead space. In these circumstances a very small tidal volume may be sufficient for adequate pulmonary ventilation, but may be insufficient when the patient is left to breathe in the surrounding air. The necessary adjustment to an increased tidal volume usually takes place within a few minutes, but if pulmonary ventilation has been unduly depressed, the change may be delayed or prevented leading to the rapid development of hypoxia.

The use of muscle relaxants has emphasized the necessity of ensuring the normal return of both diaphragmatic and intercostal action at the end of the operation and, when neostigmine is used for decurarization, sufficient time must be allowed to be certain that its effect is permanent. This precaution is particularly important where the overall dose of relaxant has been considerable, or when the last fraction has been given within half-an-hour of the termination of the anaesthetic.

Premature and excessive sedation, after return to the ward, may also be dangerous. Not only is pulmonary ventilation likely to be further depressed, but sometimes full anaesthesia may be re-established. This may easily lead to disaster if the patient is not carefully watched.

Briefly, the end of the operation and anaesthetic marks an important period of readjustment during which care and patience are essential to make sure that this readjustment has taken place effectively.

CAUSES OF POSTOPERATIVE APNOEA

In the foregoing section the complications discussed have been relatively minor degrees of respiratory depression which may become dangerous if not properly handled; but total apnoea (or something approaching it) may be apparent at the end of the operation and may require prolonged special treatment with artificial ventilation. Many factors may cause or contribute to a 'postoperative apnoea' but, in the majority of cases, the use of neuromuscular block plays an important part, and the term was certainly not widely used before the advent of the muscle relaxants. Since there is already an abundant literature on the subject,[1] a brief summary of currently accepted views will suffice. It is convenient to divide the main aetiological factors into the three groups:

1. *Drugs*

Drugs are usually the precipitating cause of a postoperative apnoea. They may be *centrally acting agents*, used either with premedication or as part of the anaesthetic, or *peripherally acting muscle relaxants*. A combined effect is sometimes seen where neuromuscular block is accompanied by injudiciously deep narcosis.

2. *Errors in Ventilation*

The two possible errors in the artificial ventilation of a patient during anaesthesia are *overventilation* leading to acapnia, and *underventilation* leading to hypercapnia.

Overventilation, by washing out CO_2 from the blood, may cause temporary depression of breathing, but is unlikely to be an important cause of a prolonged and intractable apnoea, since subnormal pulmonary ventilation will allow CO_2 to build up again so that the condition will tend to right itself. Acapnia, therefore, does not lead directly to a vicious circle, and its main significance is that the patient must be carefully watched so that hypoxia is not allowed to develop.

Underventilation is much more serious because high concentrations of CO_2 are depressant. It follows that, even if hypoxia is avoided, inadequate pulmonary ventilation from this cause will mean that the patient has already entered the vicious circle of hypercapnia.

It has been suggested that the stretch receptors in the lungs may be fatigued by the prolonged use of intermittent positive-pressure ventilation, especially if overventilation has been practised. Reflexes set up by rhythmically inflating the lungs certainly play an important part in helping to maintain respiratory quiescence during controlled breathing and it is possible that some sluggishness on the part of the patient to resume active breathing may result. To what extent this could be a major cause of serious apnoea is, however, problematical.

3. *Conditions in the Patient*

This, in some respects, is the most important group, since it comprises factors which may upset calculations relating to anaesthetic technique, especially in respect of the use of muscle relaxants. The condition from which the patient suffers which may contribute to postoperative respiratory failure may arise from acute or chronic pathological changes or may be an inherent constitutional factor. Further, such factors may either pre-exist or develop during the course of the operation, and therefore cannot always be anticipated. Without attempting to give a complete list of these factors, a few of the more important ones will be mentioned:

(*a*) *Intestinal obstruction.* Severe cases of intestinal obstruction are notorious for their liability to be followed by postoperative apnoea after the use of muscle relaxants. The features believed to be responsible are toxaemia, dehydration and electrolyte imbalance, especially potassium deficiency.[2]

(*b*) *Emphysema.* Although this condition has no direct influence on the duration or intensity of neuromuscular block, the increased dead space, the high threshold for CO_2, and the frequent dependence of these patients on some degree of hypoxia as a normal respiratory stimulus, may present problems during the period of changeover from intermittent positive-pressure ventilation to normal breathing.

(*c*) *Myopathies and neuropathies.* The presence of *myasthenia gravis* greatly exaggerates and prolongs the effect of *d*-tubocurarine or gallamine, which should never be used in these cases. *Carcinomatous neuropathy*[3] may also be associated with abnormal response to muscle relaxants, while the rare *dystrophia myotonica*,[4] on the other hand, seems to react normally with relaxants, but prolonged apnoea may follow the use of small doses of thiopentone in patients with this condition.

(*d*) *Deficiencies in the serum pseudocholinesterase.* This may be responsible for protracted apnoea following the use of depolarizing relaxants. Patients with pseudocholinesterase deficiency often suffer from cachexia, anaemia and impaired liver function, but may be, to all appearances, perfectly normal. It has been shown that

there are two different forms of pseudocholinesterase, typical and atypical, and that the atypical form lacks the normal ability to hydrolyse suxamethonium.[5] The two forms may be present in different proportions in different individuals, and investigation of a Canadian community showed 96·4% to have an adequate proportion of the active form; 3·6% had a slight to moderate deficiency, and about 1 in 3,000 were grossly deficient. Sensitivity to suxamethonium appears to have a familial basis.[6,7]

(*e*) *Circulatory failure.* Patients suffering from shock, haemorrhage and toxaemia, may go into respiratory failure from the effects of medullary hypoxia, and circulatory failure thus adds a central effect to the peripheral paralysis of the respiratory muscles by relaxants. The reduced blood supply to the muscles also intensifies the effect of the block itself.[8] In clinical practice circulatory failure, on account of the frequency with which it is encountered, is a very important factor in postoperative respiratory complications.

THE SIGNIFICANCE OF POSTOPERATIVE APNOEA IN THE GRAVELY ILL PATIENT

The wide general use of neuromuscular block with intermittent positive-pressure ventilation in anaesthesia, and even more the successful long-term employment of this technique in the treatment of tetanus and poliomyelitis, tend to encourage the outlook that postoperative apnoea is an inconvenient, rather than a dangerous, complication. This view seems justified when one is dealing with reasonably fit subjects and, in particular, the surprise apnoeas which sometimes follow the use of comparatively small doses of suxamethonium can be confidently expected to respond to patience and correct management, in these patients.

In gravely ill patients, on the other hand, apnoea may be a dangerous complication and, in this class, there are many postoperative deaths in which failure of pulmonary ventilation is a factor. Where muscle relaxants have been administered to such gravely ill patients, it is tempting to accept that respiratory failure is purely incidental and this is doubtless often correct. In other instances, however, it seems to have more significance and the extent to which the supervention of this complication may swing the balance is of considerable importance.

On theoretical grounds alone, it would be unwise to underestimate the effect of a protracted postoperative apnoea. Natural breathing is an important stimulus to the circulation which the most elaborate methods of artificial ventilation cannot entirely replace; the patient can ill afford to dispense with this stimulus at the end of a long and severe operation. Although ventilation by intermittent positive pressure is highly satisfactory in the presence of a well-maintained circulation, this is not the case where there is a tendency to circulatory failure, since such ventilation raises the intrapulmonary pressure, thereby obstructing the return of blood to the left heart and further decreasing the output. While its use during the operation is justified by the great advantages gained from the employment of neuromuscular block, any protraction of the period of enforced artificial ventilation is very undesirable.

In an attempt to throw further light on this problem, cases from the records of deaths in association with anaesthesia at the Johannesburg General Hospital, covering the past 12 years, have been studied. The criteria governing their selection were:

1. Muscle relaxants and intermittent positive-pressure ventilation were employed in accordance with generally accepted techniques.

2. Death was in the immediate postoperative period (up to 36 hours) without the return of consciousness.

3. Apnoea (or grossly inadequate pulmonary ventilation) was apparent at the end of the operation.

While not a condition of selection, all the subjects were in the poor-risk category, often to a marked extent relative to the operative procedures undertaken. In all, 15 cases were studied and were found to fall naturally into one or other of 3 groups: (*a*) cases where exposure to hypoxia was a possible factor, (*b*) cases showing primary respiratory failure, and (*c*) cases where progressive circulatory failure was the dominant feature.

(*a*) *Hypoxia a Possible Factor (4 Cases)*

In each case in this group, following the administration of prostigmine, the patient's pulmonary ventilation was judged to have been sufficiently restored for him to be returned to the ward but, thereafter, cardio-respiratory deterioration manifested itself, from which recovery did not take place.

The hiatus between the time of leaving the theatre and the summoning of assistance leaves an unfortunate element of doubt about the sequence of events, and the possibility that the patient was exposed to hypoxia cannot be dismissed. Whether he would have survived, even with full recovery-room facilities, is also a matter of pure speculation.

(*b*) *Primary Respiratory Failure (3 Cases)*

These cases presented a clear picture of primary respiratory failure, since apnoea was present from the beginning, but a fair circulation had been maintained for a considerable time by artificial ventilation. In view of the fact that failure of pulmonary ventilation undoubtedly preceded circulatory collapse, the anaesthetic technique must at least be regarded as a precipitating factor.

(*c*) *Circulatory Failure (8 Cases)*

This formed the largest group, and the subjects were obviously in very poor condition relative to the operations to which they were submitted; the dominant feature being progressive circulatory failure as the operation proceeded. If a patient to whom muscle relaxants have been administered becomes moribund during the course of the operation, it will naturally be impossible to restore normal breathing in the terminal stages and it is reasonable to accept the failure of pulmonary ventilation as a purely incidental factor. This view was further supported by several similar cases (not included in the series reviewed) in which secondary cardiac arrest occurred while the operation was still in progress.

Conclusions

The conclusions to be drawn from these observations are that, although respiratory failure in the gravely ill patient is often nothing more than part of the general picture of dissolution, postoperative apnoea, in this type of case,

is an extremely dangerous complication which *can* mean the difference between death and survival and which is something the anaesthetist must be at great pains to avoid. In this respect, the cases in group (*b*) are particularly significant.

PREVENTION OF POSTOPERATIVE APNOEA

While much is now known about the causation and treatment of postoperative apnoea, the subject of its prevention appears to have received little attention. This is not surprising, for prevention belongs more to the art, as opposed to the science, of anaesthesia, where each case has to be assessed and handled on its own merits and the individual judgement and experience of the anaesthetist are the vital factors. It is certain that different experts would have widely different approaches to the problem, with much the same degree of success. No hard-and-fast rules can be laid down and it is only possible to mention a few general principles:

1. Careful pre-operative observation is necessary to avoid missing any signs or symptoms which might have a bearing on the patients' response to muscle relaxants or other drugs.

2. Where factors such as dehydration or electrolyte imbalance are present, every effort should be made to correct them before operation is undertaken.

3. Great caution in the use of muscle relaxants is required when dealing with certain types of case. While this seldom amounts to an absolute contraindication, it may be desirable to utilize every available means to limit the total dose, and restraint is especially necessary in long and severe operations, particularly in the later stages. To this end, other methods of securing satisfactory relaxation may be used and intercostal block, spinal or extradural anaesthesia, may sometimes merit consideration.

4. With regard to general anaesthesia it is well to remember that, of the methods available, inhalation anaesthesia has the greatest measure of flexibility and freedom from cumulative depressant effects.

5. Finally, it is impossible to overemphasize the importance of carefully assessing the patient's respiratory condition at the end of the operation and keeping him under observation, with appropriate treatment, if there is any sign of inadequate pulmonary ventilation.

SUMMARY

Serious disturbances of pulmonary ventilation usually manifest themselves in the immediate postoperative period. Danger to the patient may arise from: (*a*) failure to recognize a slight or moderate degree of respiratory depression at the end of the operation, and the subsequent establishment of a vicious circle of hypoxia and hypercapnia; or (*b*) the presence of apnoea (or *grossly* inadequate pulmonary ventilation) which fails to respond to treatment.

In patients who are gravely ill, postoperative failure of pulmonary ventilation is, in itself, a dangerous complication which is likely to hasten the onset of circulatory failure.

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