ANAESTHESIA IN GENERAL PRACTICE

This is one of a series of articles, contributed by invitation

RISKS OF ANAESTHESIA—I

BY

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Difficulties of one sort or another are to be expected with certain types of patient or operative procedure. If the anaesthetist is on the lookout for them he should by intelligent anticipation be able to prevent their occurrence, or else by a suitable anaesthetic technique counteract their effects. During anaesthesia many situations arise which will develop into emergencies unless the anaesthetist recognizes the early signs of trouble and immediately takes the necessary steps to regain control. Sometimes an accident occurs with no warning at all, and a successful outcome may well depend on the right thing being done promptly and without hesitation. The difficulties encountered during anaesthesia, though small in number, crop up again and again in slightly different forms. Only the chief risks can be outlined here with the general principles of treatment; these principles, however, can be modified to apply to other emergencies which have in fact originated from similar faults.

Oxygen Want

The respiratory and circulatory systems are closely linked together, so that oxygen want in the tissues (anoxia) arising from failure or partial failure of one system will quickly embarrass the other. Anoxia being the underlying factor in so many anaesthetic troubles it will be well to emphasize some of its physical signs. These are: (1) increase or decrease in the rate of the pulse, which is slow, bounding, and often irregular in acute oxygen want; (2) rise or fall in systolic blood pressure; (3) altered breathing—depressed, quick, and shallow, or periodic respirations, or yawning; (4) nausea and vomiting; (5) muscular twitchings; (6) restlessness; (7) cyanosis. Cyanosis always indicates suboxygenation, but a high proportion of patients suffering from oxygen want show no cyanosis.

Anoxia is responsible for many of the troubles encountered during spinal anaesthesia, and is not infrequently present during or after general anaesthesia. Pyrexia, fear, pain, and hyperthyroidism all increase the patient's oxygen requirements.

Abuse of Carbon Dioxide

Nowadays carbon dioxide is commonly regarded as the "cure" for practically all anaesthetic difficulties. Actually its injudicious use has cost many patients their lives. It should be remembered that morphine and the basal hypnotics depress the breathing by decreasing the sensitivity of the respiratory centre to carbon dioxide, with the result that the alveolar content of carbon dioxide banks up to a higher level than usual. The patient's physiological processes are deranged by this excess, and will consequently not benefit from any addition to this burden. For the majority of patients who happen to be in this depressed state the lesser of two evils is probably the use of sufficient carbon dioxide or rebreathing during induction to secure normal pulmonary ventilation. Creating a higher concentration of carbon dioxide, with the objects of producing an exaggerated hyperpnoea and rapidly inducing anaesthesia with intolerable concentra-

tions of ether, or any piling up of carbon dioxide in an acidotic patient, is not to his advantage. Individual susceptibility to carbon dioxide is very variable, and if the normal immediate response to a low concentration—namely, an increased depth of respiration and a rise in blood pressure—does not appear overdose for that particular patient is a probable contingency, and the use of carbon dioxide must be immediately discontinued.

The signs of toxicity due to carbon dioxide are: a fall in blood pressure, depressed or gasping breathing, and muscular twitchings, which may pass on to generalized convulsions and death. Waters and his associates, who are responsible for most of the experimental work on carbon dioxide, have shown that the improvement in cases of circulatory depression and shock commonly attributed to carbon-dioxide-oxygen mixtures is in fact due to the good effects of the high oxygen concentration outweighing the ill effects of the carbon dioxide. The continued inhalation of a concentration of carbon dioxide above 10 per cent. will produce unconsciousness in normal unanaesthetized subjects. In this connexion it is of significance that asphyxiated patients and newborn babies recover more certainly and more quickly when pulmonary ventilation is carried out with 100 per cent. oxygen.

For success with the carbon-dioxide-absorption technique efficiency of the absorbent material and of the canister design and reduction of the dead-space to the minimum are essential. Towards the close of the operation the contents of the rebreathing bag must be repeatedly changed in order to wean the patient slowly from the warm, moist—and possibly high oxygen-containing—atmosphere which he has been breathing, and to restore him to normal atmospheric conditions.

It has been shown that carbon dioxide, oxygen, and all anaesthetic gases are rapidly and completely absorbed from the pulmonary alveoli. After operation the patient should be encouraged to take deep breaths. If he cannot or will not comply a gentle flow of pure carbon dioxide—a watch being kept for any signs of toxicity—may be blown at his face. This will make him fill his lungs with air, the presence of the *unabsorbed* nitrogen decreasing the risk of pulmonary collapse. The use of carbon-dioxide-oxygen mixtures, in contrast to this, may be followed by patchy areas of atelectasis or even complete collapse of lung.

Oxygen want and carbon dioxide overdosage, singly or combined, are liable to crop up on many occasions during anaesthesia, and are the underlying factors in many anaesthetic states which are difficult to explain on any other basis—at least I myself can call to mind situations which at the time I never attributed to carbon dioxide overdose, but in which I am now convinced excess carbon dioxide played an important part.

Overdose of Sedative Drugs

Among the effects of overdosage with sedatives are those of low alveolar oxygen and high alveolar carbon-dioxide concentrations. If it relieves the anoxia resulting from the low blood pressure and the depressed breathing an atmosphere enriched with oxygen will improve the respirations. The administration of carbon dioxide and oxygen may break the vicious circle, but on the other hand this additional carbon dioxide may be beyond the patient's tolerance and still further depress the breathing and the blood pressure. Artificial respiration with a high

percentage of oxygen and other treatments for shock are indicated in serious cases.

Picrotoxin is the specific antidote for barbiturate poisoning, but if injected too quickly or in excessive dosage it will produce convulsions, the treatment for which is an intravenous dose of a barbiturate. An unimpaired liver function is essential for the normal detoxication of a barbiturate. Apomorphine, 1/40 to 1/60 grain, aborts scopolamine reactions; coramine may be a useful temporary stimulant in cases of avertin depression. It is much safer, however, to place no reliance in analeptics and to be more careful in assessing the sedative dose for each individual patient.

Another serious risk which should be mentioned here is the existence of a partially obstructed airway. This can kill a patient from anoxia just as surely as can complete respiratory obstruction, and since it is so commonly overlooked it may be even more dangerous. When the breathing is depressed any further reduction of the alveolar exchange—such as occurs in the presence of an obstructed airway—immediately produces respiratory failure without any intervening period of respiratory stimulation.

Overdose of the Anaesthetic Agent

When the breathing is shallow or has stopped there is a high alveolar content of carbon dioxide as a result of insufficient pulmonary ventilation. The respiratory centre is, however, so depressed by the anaesthetic agent that its threshold has been raised above the point at which this high concentration of carbon dioxide can, so to speak, ring the bell. At the same time the insufficient respiratory exchange is failing to supply the body's requirements of oxygen, and anoxia itself will cause respiratory failure.

No more anaesthetic must be given until the situation is relieved, an atmosphere rich in oxygen being substituted after the patency of the airway has been verified. If respiratory movements are inadequate or absent a two-gallon bag should be filled with pure oxygen, an accurately fitting mask applied to the face, and the lungs rhythmically inflated by manual squeezing of the bag. A rapid recovery usually results. If this procedure has to be at all prolonged the bag must be emptied to remove the accumulation of exhaled anaesthetic and carbon dioxide, and then refilled with pure oxygen. When it appears that the circulation has also been brought to a standstill by the high concentration of the anaesthetic agent treatment for primary cardiac failure must be instituted unless there is a prompt response to artificial respiration.

If pre-anaesthetic sedatives have been given the failure is due to the combined depressant action of these and the inhalation agent. Recovery will naturally be more prompt when the major part of the depression has been evoked by the inhalation agent, since a volatile anaesthetic can—provided the circulation is reasonably efficient—be removed as rapidly as it has been introduced. When, however, the failure has been produced entirely with non-inhalation agents, such as intravenous pentothal or evipan, all that artificial respiration can do is to maintain life until such time as the body itself has detoxicated the drug. This unwelcome state is apt to arise when the operative stimulation has ceased, and again emphasizes the importance of not using these powerful drugs in a haphazard fashion. In these circumstances, as there is no inhalation anaesthetic to be eliminated, only carbon dioxide accumulates in the bag of oxygen; if a canister of soda-lime is in position between the mask and the rebreathing bag there will be no necessity to empty its contents during the period of artificial respiration. When oxygen has been administered freely throughout an operation its use must

be discontinued gradually, otherwise respiratory failure may occur before the patient has had time to readjust himself to atmospheric conditions.

Sometimes in cerebral cases and other poor operative risks patients cease to breathe as soon as they lose consciousness. Their general condition will be kept nearer normal if anaesthesia is induced and maintained by a manually produced respiratory exchange rather than if carbon dioxide is continually added with the idea of making these patients breathe.

A patient with chronic laryngeal obstruction has become accustomed to a high alveolar content of carbon dioxide, and his respiratory centre has adjusted its threshold accordingly. After the obstruction has been relieved the alveolar content of carbon dioxide at once returns to normal, and the patient may die of oxygen want before his respiratory centre is stimulated. The administration and gradual "tapering off" of carbon dioxide as his respiratory centre readjusts itself to the normal threshold will prevent such a tragedy. It is all a question of the difference between threshold and stimulus; a similar state of affairs may be seen following overventilation due to nervousness during induction (apnoea) or after the stopping of an anaesthetic in which carbon dioxide has been allowed to accumulate by rebreathing. The depressed and possibly irregular breathing of respiratory failure must be distinguished from the shallow or hesitant breathing of too light an anaesthesia.

(The conclusion of this article, which will appear in the next issue of the *Journal*, deals with respiratory obstruction, primary cardiac failure, circulatory depression, convulsions, and "other risks.")

SOME FACTORS IN NUTRITION

A special Chadwick Lecture on the study of nutrition was given in London on October 14 by Professor W. A. OSBORNE, dean of the faculty of medicine in the University of Melbourne. He attributed the increasing interest taken to-day in national and personal nutrition to seven causes: (1) the great war with its problems of food supply and the evidence found on examination of recruits; (2) public imagination caught by vitamin research; (3) economic success of scientific feeding of farm animals; (4) propaganda by food manufacturers, faddists and quacks; (5) increased resort to dietotherapy by the medical profession; (6) the widening democratic outlook; (7) prosecution of nutritional research.

Outlining the physiological aspect of dietetics the lecturer said that the sliding scales of calorie intake prepared by the League of Nations and by special committees were useful as working standards of minimal requirements; but the optimal was hard to fix owing to varying race, work, and climate, and the individual idiosyncrasies of human creatures. The importance of the rate of liberation of energy was often forgotten; nitrogenous fuel-food was more explosive, hence the killing power of the carnivora. Human instinct was a factor that played a part in nutrition, though it was inferior to the instinct in lower animals. The lecturer named as examples of instinct—salting of soup, distribution of calories over the three types of fuel foods, nausea when canned meat was taken for some time, and the instinctive liking for celery and salads. Speaking of drawbacks attending food enlightenment of the public, he suggested that people might become food-conscious, though this was contrary to Anglo-Saxon tradition. Exaggerated claims were made for improved nutrition—for example, that it would stop dental decay. It was forgotten that milk was not essential for adults, except mothers. Children in slums, even if fed correctly, would still be anaemic if they had inadequate sleep due to crowded and verminous beds. Neglect of social factors also delayed right education as to feeding. A man was not an isolated unit; the meal was a strong social bond. Disregard of aesthetic factors often prevented enjoyment of food; nutriment enjoyed was better digested.