CYCLOPROPANE : A VINDICATION

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After administering approximately 10,000 cyclopropane anaesthetics, with and without pentothal and the muscle relaxants, to patients of all ages and for a great variety of operations, and including many already suffering from grave cardiac lesions, I am firmly convinced that cardiac arrhythmia occurring during the administration of this potent agent is not due, as universally believed, to the agent *per se*. I was fortunate not to have lost a single patient, although innumerable patients manifested varying degrees of collapse ('cyclo shock'), because of my initial inexperience of the closed circuit technique.

Time and again one has proved in practice that such arrhythmia is due to the ill-effects of hypoventilation—hypoxia and hypercapnia—resulting from defective technique and lack of appreciation of the condition. It has been repeatedly shown that the arrhythmia can be abolished with ease and safety by correcting these two factors, as will be described below. Of these two extraneous factors hypoxia is by far the more dangerous, and it is quite possible that those with low cardiac reserves may succumb from this cause long before the toxic effects of hypercapnia become grossly manifest. A slight rise in the blood pressure and a rather rapid pulse rate (the effects of hypoxia) are usually the early signs that all is not well. When the pulse becomes full and bounding, with a tendency to slowness, hypercapnia has joined forces with hypoxia.

Hypoventilation can produce practically every variety of abnormal cardiac irregularity. I have never encountered ventricular fibrillation. This complication, according to the pundits, is apparently bound up with chloroform, but I am strongly suspicious that it occurs only in the presence of too little oxygen.

Cyclopropane is a narcotic and *ipso facto* a central respiratory depressant—and a powerful one—but it is *not* a cardiac poison. Any poisonous effect on the heart is due to concomitant hypoventilation. For those who like formulae, the toxic set-up can be crudely expressed thus: $C_3H_6—O_2+(CO_2)n$

That is to say, the narcotic, in the presence of hypoxia and hypercapnia, can be shown to affect the heart. But the factor C_3H_6 (cyclopropane) can be removed from the closed-circuit situation and any other known anaesthetic agent substituted, excluding the halogen derivatives (because of the danger of phosgene formation), without affecting the issue. In other words, hypoxia combined with hypercapnia is liable to produce cardiac arrhythmia, *whatever the anaesthetic agent. In fact, I believe that hypoventilation* per se *can do so in the absence of narcosis*, but it is more prone to occur and with greater severity in the temporary pathological state of anaesthesia, in which the respiratory centre is always depressed, especially with powerful agents such as cyclopropane. The complicated method of administration of this agent is another contributing factor.

It should now be clear why adrenaline so dangerously aggravates the cardiac effects of hypoventilation. Its harmful effect has been ascribed (on the basis of a single death finding in dogs!) to its combination with cyclopropane,[1] but this is merely because hypoventilation is more commonly associated with the closed-circuit technique, which to many administrators implies cyclopropane. If cyclopropane is given with due regard to the prevention of hypoxia and hypercapnia, there is no reason why adrenaline may not be safely used. Indeed, it is the duty of the administrator to reassure the operator who wishes to use this useful vascular constrictor, that he may well do so without detriment to the patient. Obviously adrenaline should not be administered to any patient, anaesthetised or not, who is hypoventilating.

As regards the treatment of cardiac arrhythmia, all that is necessary is to empty the re-breathing bag of its accumulated CO_2 and to refill it repeatedly with liberal supplies of oxygen, while maintaining an increased tidal volume to ensure more rapid restoration of the normal blood levels of the respiratory gases. This procedure is carried out until the pulse returns to its pre-hypoventilating state. During the process of 'washing out' of excess CO_2 and 'filling up' with oxygen the cyclopropane can be turned off, as in practice the hypoxia and hypercapnia have themselves anaesthetized the patient sufficiently and there should be no fear of his waking up. Besides, cyclopropane is expensive and the gas will have practically no narcotic effect when so greatly diluted; also there is always the possibility of an explosion when this gas is forced out of the re-breathing bag.

If the anaesthetist is not inept and learns to prevent the onset of hypoventilation, there is no reason why cyclopropane should not be administered for hours on end, with benefit to patient and surgeon.

CONCLUSION

It appears that the extent of ventilation is too often over-estimated with the result that weird phenomena make their unwanted intrusion. It is high time, therefore, that there was a change in the teaching of anaesthesia in this respect. The normal tidal volume must be maintained with adequate oxygen intake, and excess CO_2 must be prevented at all times. This is a cardinal rule applicable not only to general anaesthesia but also to the treatment of and prevention of pulmonary conditions.

Most of the morbidity and mortality associated with narcosis can be prevented. Let us call a halt to making the *narcotic* the scapegoat of general anaesthesia.

1. Meak, W. J. (1940): Proc. Staff Meet., Mayo Clic., **15**, 237-240.