NITROUS OXIDE AND THE COMPROMISED HEART

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Since its introduction in the middle of the last century, nitrous oxide has been used almost universally in anaesthesia and for analgesia and its effects on the circulation have often been considered negligible. However, because of the increase in the number of anaesthetics given to patients with a severely compromised cardiovascular system, the effects of nitrous oxide on the heart and the circulation, deserve re-examination.

Nitrous oxide and the normal heart.

As late as 1955, Fieldman and his colleagues (1) explained the decreases in cardiac output, blood pressure and stroke volume observed under thiopental sodium and nitrous oxide anaesthesia entirely in terms of the effects of thiopental sodium, tacitly assuming that nitrous oxide had no effect on the circulation. Similarly, Heller and co-workers, (2) on the basis of their observations of "excellent cardiovascular homeostasis" under anaesthesia with nitrous oxide, oxygen and curare, concluded that "nitrous oxide does not produce myocardial depression ... and interferes minimally with the integrity of the peripheral vascular bed". A number of recent reports, however, have demonstrated that nitrous oxide has significant cardiovascular effects particularly in patients suffering from coronary artery disease.

Information concerning the effect of nitrous oxide on the normal heart has been gained from studies of isolated heart muscle preparations, heart-lung preparations, intact animals, and man.

Studies of isolated heart muscle preparations have the advantage that the effect of drugs, including anaesthetic agents, can be observed without interference from neurogenic or hormonal responses. However, in the case of nitrous oxide, such experiments have their limitations. As the concentration of nitrous oxide increases, the effects of hypoxia may be superimposed to those of nitrous oxide because oxygenation of the preparation is solely by diffusion. This problem is particularly important when preparations are tested at 37°C, as opposed to room temperature, because of the higher oxygen consumption. Using papillary heart muscle preparations tested at 37°C, Goldberg and his colleagues (3) found that cardiac performance was reduced to almost the same extent by the addition of nitrous oxide and that of nitrogen; these authors concluded that nitrous oxide did not by itself depress myocardial contractility. In more controlled studies, carried out at room temperature and after eliminating heart muscle preparations that could not contract normally in a mixture of 50% nitrogen in oxygen, Price (4) demonstrated that the replacement of nitrogen by nitrous oxide caused a significant reduction in contractile performance. The severity of myocardial depression was greatest when the ionised calcium concentration was lowest, (Fig.1).

![Diagram](image-url)  
**Figure 1.** Depression of cardiac performance induced by nitrous oxide is a function of calcium ion concentration (after Price, 1976).