Title: HALOTHANE NOT EFFECTIVE IN TREATMENT OF STRESS-INDUCED ELEVATION OF PULMONARY CAPILLARY WEDGE PRESSURE

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Introduction. Roizen et al[1] reported that increasing the dosage of volatile anesthetic can be used to treat increases in pulmonary capillary wedge pressure (PCWP) when such increases accompany increased surgical stimulation. It is illogical that use of a direct myocardial depressant to "treat" decreased ventricular function should always work. If the ventricular dysfunction is due to myocardial ischemia, then perhaps lowering myocardial oxygen demand might improve regional ventricular demand/supply relationships and result in improved performance. If, on the other hand, increases in PCWP are due to inherently decreased ventricular function rather than ischemia, then we questioned whether "treatment" with volatile agents would be effective. Accordingly, we administered halothane to 14 patients undergoing coronary artery surgery during the pre-bypass period, attempting to control arterial pressure at various stimulation levels with the volatile anesthetic, and observing resultant ventricular function.

Methods. Fourteen patients gave informed written consent to a protocol approved by the institutional Human Studies Committee. We selected the following periods as indicative of relatively "standard" degrees of stimulation: 1) controls at insertion of PA catheter; 2) prior to skin incision; 3) at skin incision; 4) at sternal split; 5) three minutes after sternal retraction; 6) six minutes after sternal retraction; 7) just before bypass. At each point, halothane was administered to attempt to control systolic arterial pressure at or near preoperative values. Hemodynamics and end-tidal halothane (Beckman LB-2) were recorded at each stimulus point. Groups 1 and 2 (see figure) were not treated differently until the point just before bypass. At that point, Group 2 was continued on halothane "treatment" of arterial pressure, whereas Group 1 was given IV nitroglycerin to reduce the halothane needed to maintain systolic blood pressure at or near control. Except for this last point, the groups were identically treated.

Results. Systolic, diastolic and mean arterial blood pressures were held within reasonable limits by the halothane "treatment". Maximal systolic pressures occurred with skin incision, averaging 137 mmHg for both groups, whereas minimal systolic pressures, just prior to skin incision, averaged 111 mmHg. Required halothane end-tidal concentrations (shown in Figure 1) increased at successive stimulus levels in Group 1, whereas Group 2 patients did not require as much halothane to control their arterial pressures (and consequently were not given nitroglycerin to replace halothane at the last stimulus period). Halothane "treatment" applied in this manner was not effective at any stimulus level in either group in reducing PCWP back near control (Figure 2). PCWP returned near control only at the last stimulus level in Group 1 when TNG was administered and halothane concentration reduced. Average cardiac indices remained near control levels throughout all stimulus periods in both groups.

Discussion. Ventricular function deteriorated at all stimulus levels in both groups when systolic arterial pressure was "treated" with halothane, as evidenced by increased PCWP despite relatively constant (slightly reduced) cardiac indices. Our halothane concentrations were not necessarily at steady state values because of the relatively short times between stimulus levels, but this does not detract from our finding that increasing the halothane concentrations resulted in deterioration of ventricular function in every patient. We doubt that any of our patients were actually ischemic, which possibly explains our different findings from that of Roizen et al. We conclude that, although volatile agent "treatment" of decreased ventricular function may be effective if it reduces ischemia, it is not effective, and indeed may be harmful if the elevation in wedge pressure is related to surgical stress (increased afterload) in patients with decreased ventricular dysfunction.

Figure 1

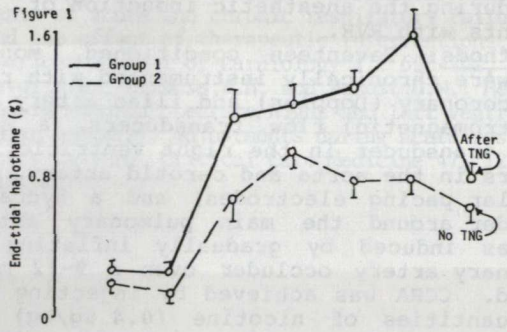
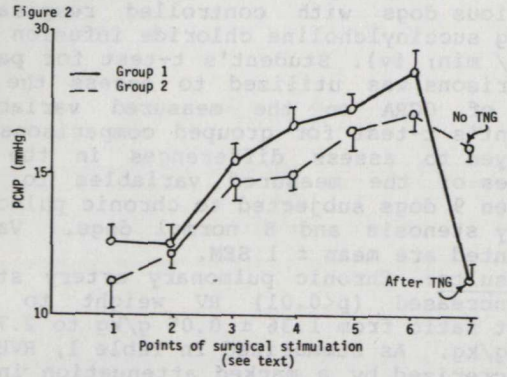


Figure 2



Reference.

1. Roizen MF, Hamilton WR, Sohn YJ: Treatment of stress-induced increases in pulmonary capillary wedge pressure using volatile anesthetics. Anesthesiology 55:446-450, 1981.