TITLE : ELECTROMECHANICAL MONITOR FOR ASSESSMENT OF HYPOTENSION: HALOTHANE vs BLOOD LOSS

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Introduction. An intraesophageal accelerometer responds to motions of the cardiovascular system and is affected both by myocardial depression and hypovolemia.[1] However, the accelerometer response differs in these two situations and this information can assist the clinician in deciding between halothane overdose or excessive blood loss.

Methods. A series of 12 mongrel dogs (15-25 kg) were anesthetized with sodium pentobarbital, intubated, paralyzed with pancuronium, and ventilated mechanically (100% O_2) with periodic blood gas monitoring. Several cardiovascular drugs were infused prior to inhalation of halothane. Hypovolemia was produced by bleeding. Sufficient time for recovery was allowed between the various maneuvers. Instrumentation included the electrocardiogram, catheter-tipped pressure transducers in the left ventricle and at the aortic root, and a Swan-Ganz catheter. The intraesophageal accelerometer was manufactured by Entran Devices, Inc. Model No. EGAL-125R-5D. With internal viscous damping the frequency response of the accelerometer is flat to 300 Hz. A conventional esophageal stethoscope encloses the accelerometer which was driven by conventional instrumentation for strain gauge bridges. The accelerometer was first positioned with respect to an external landmark (4th-5th rib interspace) and then adjusted slightly to maximize signal amplitude.

Results. Two major complexes are found in the accelerometer signal. The first wave (A1) occuring during isovolumic contraction and rapid ventricular outflow, corresponds to the "DE" complex of the precordial accelerogram[2]. Aortic valve closure produces a second wave (A2). The peak-to-peak A1 amplitude distinguishes between halothane and hypovolemia (Figs. 1 and 2). In these two figures the ordinate is the ratio of the A1 value during pharmacological or physiological intervention to the baseline value. The abscissa records the mean arterial pressure (MAP) in the same manner. Data on these two figures are almost completely separated by the (0,0)→(1,1) straight line. Although there is a region of overlap in the overall data, comparison of the data from an individual dog permits easy distinction between bleeding and halothane.

Discussion. In both figures all experimental data must pass through (1,1) and (0,0). Unfortunately, no statistical algorithms provide a polynominal regression with the final curve fitted through (0,0) and (1,1)[3]. However, two conventional regression curves were fit to the data and they have the following form:
Halothane $y = 0.0138 + 1.51\,x - 2.83\,x^2 + 2.27\,x^3$;
Bleeding $y = 0.0575 + 4.56\,x - 6.45\,x^2 + 3.09\,x^3$,
where $y \equiv A1/A1,b$ and $x \equiv MAP/MAP,b$. Halothane (<0.5%) produced simultaneous reduction in MAP and A1. This is anticipated as halothane causes some peripheral vasodilatation and direct myocardial depression. Bleeding should produce no intrinsic change in cardiac contractility until myocardial hypoxia results secondary to decreased myocardial perfusion. Thus, with bleeding a relatively large decrease in MAP would be expected before any appreciable decrease in A1 should occur. (Fig. 2).

References.
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2. Reuben SR, and Littler WA: Praecordial Accelerometry: An Indirect Assessment of Left Ventricular Performance, Europ J Clin Invest 3, 324-330 1973.
3. Rosenblatt J: Private communication Dept Biometry (CWRU).

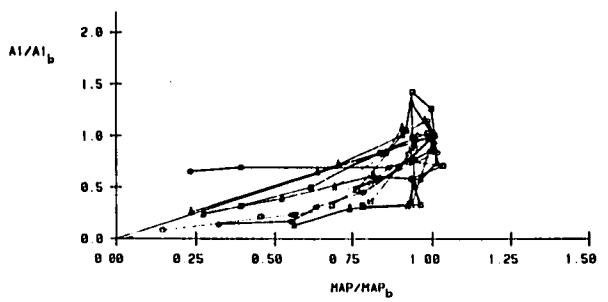

Fig. 1. Myocardial depression and hypotension caused by halothane inhalation in 12 dogs. Almost monotonic decrease of A1 as MAP falls.

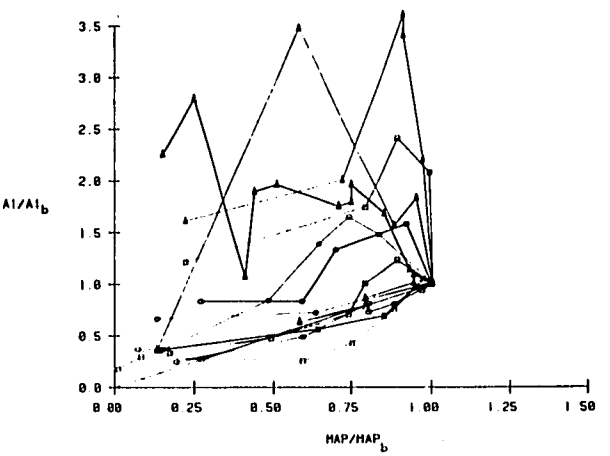

Fig. 2 Bleeding data from 12 dogs as in Fig. 1. A1 wave amplitude increased with initial blood loss.