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Anesthesiology
1996; 84:478-9
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Lippincott-Raven Publishers

Effects of Anesthetics and Vasodilators on Aortic Input Impedance

To the Editor.—The article by Hetrick et al.1 claims that "the effects of volatile anesthetics, including isoflurane and halothane, on quantitative indexes of left ventricular afterload have not been described." In Gersh, working with me in the Nuffield Department of Anaesthetics at Oxford, we studied the subject in depth between 1968 and 1970 and published our experimental findings on halothane and its effects on the interactions between myocardial contractility, aortic impedance, and left ventricular performance in a series of four articles.2-5 These were supplemented by the studies of Fox,6 also at that time working with me in Oxford, on the effects of carbon dioxide on the systemic and pulmonary vasculature during anesthesia.

The aortic input impedance spectra during halothane anesthesia and sodium nitroprusside infusion, which we obtained then, were essentially similar to those described now by Hetrick et al. Our interpretations were based on both the Windkessel and the transmission line model. However, for our studies of both systemic and pulmonary vasculature, we preferred to use the ratio of pulsatile left (or right) ventricular work (and power) to the total work (pulsatile + steady work) to define the efficiency with which the relevant arteriolar bed was decoupled from the heart. We also used an arteriolar dilator, trimetaphan, in the first study to test the concept that the effects of halothane were different from those of a potent arteriolar dilator. Nevertheless, our main conclusion, that "neither the inductive nor the capacitative characteristics of the aorta and peripheral vascular bed could play a significant role in the hemodynamic responses to halothane anesthesia" reads remarkably similar to that of Hetrick et al. We subsequently studied the effects of sodium nitroprusside,10 and our conclusion that "when hypotension is induced by widespread arteriolar dilatation, it is achieved at some loss of efficiency in the coupling of the left ventricle and its load" differs little from theirs. Our other conclusion, that "the increased resistivity of the arterial bed accounts for the marked changes in the profile of the arterial pressure pulse," is not only consistent with their finding of an increased C in their model but has relevance in the interpretation of clinically observable changes in the arterial pressure wave during the hypotension associated with drugs such as sodium nitroprusside, with endotoxemia and with profound anemia from natural causes or from isovolumic hemodilution.11

It is noteworthy that Gersh, in his thesis,2 also defined precisely the requirements for both pressure and velocity (flow) measurements for accurate evaluation of hemodynamics.

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Anesthesiology, V 84, No 2, Feb 1996
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(Accepted for publication November 6, 1995.)

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(Accepted for publication November 6, 1995.)