Effects of Anesthetics and Vasodilators on Aortic Input Impedance

To the Editor.—The article by Hettrick 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." Gersh,' working with me in the Nuffield Department of Anaesthetics at Oxford, studied the subject in depth between 1968 and 1970 and published our experimental findings on halothane and its effects on the relations 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 et al.6 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 Hettrick et al. Our interpretations were based on both the Windkessel and the transmission line models. 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.6 8 We also used an arteriolar dilator, trimethaphan, in the first study6 to test the concept that the effects of halothane were different from those of a potent arteriolar dilator. Nonetheless, our main conclusion, that "neither the inductive nor the capacitative characteristics of the aorta and peripheral vascular beds could play a significant role in the haemodynamic responses to halothane anesthesia" reads remarkably similar to that of Hettrick et al. We subsequently studied the effects of sodium nitroprusside,7 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 dispensability 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 endoxanemia, and with profound anemia from natural causes or from isovolumic hemodilution.

It is noteworthy that Gersh, in his thesis,7 also defined precisely the requirements for both pressure and velocity (flow) measurements for accurate evaluation of hemodynamics.

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In Reply—We thank Prys-Roberts and Gersh for their interest in our investigation. Clearly, Prys-Roberts and his colleagues performed early pioneering work examining the influence of halothane on left ventricular afterload. However, there are important differences between their work and ours. Gersh et al. studied the effects of a single concentration of halothane (1.5 MAC) on the aortic input impedance in open-chest, barbiturate-anesthetized dogs in the absence of autonomic nervous system activity. Halothane-induced alterations in discrete, harmonic Fourier series spectra were qualitatively described in this experimental model. Halothane-induced alterations in aortic input impedance were not examined using Windkessel parameters, and no quantitative measurement of aortic compliance was made. In this study, conclusions about the effects of halothane on aortic capacitive properties were inferred from measurements of the ratio of pulsatile to mean power and oscillations in the magnitude of the frequency spectra harmonics. However, pulsatile and mean power are indexes of left ventricular contractility, which rely both on the mechanical properties of the left ventricle and on the arterial circulations. In contrast, aortic input impedance depends only on the mechanical properties of the arterial vasculature. Gersh et al. also suggested that lack of oscillations in the impedance spectrum indicated that reflected waves from distal sites in the arterial circulation exerted a minimal influence over the arterial circulation as a resistive force opposing left ventricular ejection. However, the authors' inference that the absence of large reflected waves helps to minimize pulsatile energy loss may be incorrect, because reflected waves reaching the aortic root during diastole augment diastolic pressure and shift pressure and diminish oscillatory power loss.

Our study examined the effects of several concentrations of halothane and isoflurane on aortic input impedance in chronically instrumented dogs. This model allows direct comparison between the conscious and anesthetized states in the same dog, avoids the potential confounding influence of a baseline anesthetic (such as a barbiturate with profound hemodynamic actions) and acute surgical instrumentation, and maintains the functional integrity of the autonomic nervous system. In contrast to the methods of Gersh et al., we used a power spectral analysis to determine the determination of complete, and not discrete, aortic input impedance spectra. Importantly, alterations in the aortic input impedance spectrum produced by volatile anesthetics were quantified using parameters of a three-element Windkessel model of the arterial circulation. Each of the Windkessel parameters, including total arterial resistance, total arterial compliance, and characteristic aortic impedance, represents a physically meaningful mechanical property of the afterload system. In addition, by quantifying the relationship between mean arterial pressure and total arterial compliance, we were able to demonstrate a sharp contrast between the effects of volatile anesthetics and sodium nitroprusside on this relationship. In addition, we were able to ascertain the effects of aortic input impedance and total mean arterial pressure and its first derivative. Cardiovasc Res 1978; 12:49–55.


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