Effects of Anesthetics and Vasodilators on Aortic Input Impedance

It is noteworthy that Gersh, in his thesis, 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.1 Gersh et al.1 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,1 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 arterial coupling, which rely both on the mechanical properties of the left ventricle and on the arterial circulation.2 In contrast, aortic input impedance depends only on the mechanical properties of the arterial vasculature. Gersh et al.1 also suggested that lack of oscillations in the magnitude of 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 authors1 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, reduce pulse pressure, and diminish oscillatory power loss.

Our study2 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 power spectral analysis to enable 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 compliance on wave reflection timing and magnitude. It is this ability to quantitatively describe volatile anesthetic-induced alterations in left ventricular afterload that separates our investigation3 from the study of Gersh et al.1 Nevertheless, despite the differences in experimental preparation and the techniques used to generate aortic input impedance spectra, the findings of Gersh et al.1 with 1.5 MAC halothane were similar to our observations. The results of our investigation also form the basis for the comparison of other anesthetics,4,5 including examining the influences of desflurane, sevoflurane, and propofol in a similar chronically instrumented canine model.

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