

Differences between M-mode and Two-dimensional Echocardiography

To the Editor:—When studies of similar design yield conflicting results, the reasons for the discrepancies are always worth exploring. Recently, two groups of investigators utilized echocardiography to assess the effects of halothane and isoflurane on cardiac function in infants and children. Using M-mode echocardiography, Wolf *et al.*¹ observed that halothane produced significant myocardial depression, while cardiac function was well preserved with isoflurane. Conversely, Murray *et al.*² using pulsed-wave Doppler and two-dimensional echocardiography (2D-echo), demonstrated that halothane and isoflurane had a similar depressant effect on the myocardium. Murray *et al.*² attributed the differences between their results and those reported by Wolf *et al.*¹ to basic differences between the echocardiographic techniques that were used to assess ventricular function. Their discussion, however, contained a number of controversial statements.

They stated, for instance, that Wolf *et al.*, by using the pre-ejection period (PEP), the left ventricular ejection time (LVET), and the systolic time interval (STI), were limited to a unidimensional evaluation of the myocardium. PEP, LVET, and STI are, indeed, unidimensional, since they are temporal and describe the duration of various cardiac events. M-mode echocardiography is, however, ideally suited to assess temporal events because, by design, it provides a time-history of cardiac events. While it is often true that the determination of PEP, LVET, and STI does not allow conclusions regarding cardiac output, the reasons for this are not to be found in the unidimensional nature of the M-mode *per se*, but, rather, in the limited ability of these indices to assess myocardial performance.

Murray *et al.*² also stated that M-mode measurements of contractility were only valid if it could be assumed that the effects of halothane and isoflurane on heart rate, cardiac conduction, and afterload were similar. The M-mode derived shortening fraction, used by Wolf *et al.*,¹ and the 2D-echo derived ejection fraction, used by Murray *et al.*,² are both ejection phase indices of contractility. All ejection phase indices are influenced, to some degree, by the ventricular loading conditions, because ventricular ejection itself is influenced by them. It is, therefore, totally unjustified to imply that changes in ventricular loading conditions affect the M-mode ejection phase indices to a greater extent than the 2D-echo indices, or vice versa.

Finally, their claim that a more accurate measure of

ventricular volumes and ejection fractions is possible with 2D-echo than with single dimension M-mode deserves further scrutiny. In one study, referenced by Murray *et al.*,² end-diastolic volumes (EDV), obtained by 2D-echo, correlated significantly better with cine-angiographic volumes than the M-mode EDV.³ However, no statistically significant differences were observed for the correlations between M-mode or 2D-echo end-systolic volumes (ESV) and cine-angiographic ESV. Opposite findings were obtained by Schiller *et al.*⁴ They observed that 2D-echo ESV, rather than M-mode ESV, correlated significantly better with cine-angiographic ESV, but they observed no differences for EDV. In a third study, the correlations between 2D-echo EDV or ESV and the same cine-angiographic volumes were not significantly better than those between the M-mode EDV or ESV and the corresponding cine-angiographic volumes.⁵ It is further noteworthy that the differences between the various correlation coefficients were not statistically evaluated in any of these reports.³⁻⁵ The statistical significances, mentioned in this letter, are based on our own calculations.⁶ The superiority of 2D-echo over M-mode for the measurement of volumes is, therefore, often based on vague impressions, rather than on solid, statistically significant evidence. Comparisons between correlations of 2D-echo or M-mode ejection fractions and cine-angiographic or radionuclide ejection fractions are similarly inconclusive.³⁻⁵

One could even wonder whether the studies by Wolf *et al.*¹ and Murray *et al.*² really resulted in totally different findings. Although Murray *et al.* concluded that the changes in hemodynamics produced by halothane and isoflurane were similar, their study also showed evidence for "increased cardiovascular reserve" in patients receiving isoflurane. This observation can be interpreted as a suggestion that isoflurane produced less myocardial depression than halothane, which is the exact conclusion reached by Wolf *et al.* If one believes that the results of the two studies were truly dissimilar, one should not attribute these differences to differing echocardiographic techniques. Indeed, there is little evidence in the literature to confirm this hypothesis. A better explanation might be that dissimilar populations were studied. The substantial difference between the mean ages of the two populations supports this argument, and it should have been considered in the discussion.

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In Reply:—Thys and Hillel correctly point out that our study, using pulsed Doppler and two-dimensional echocardiography, of the cardiovascular effects of halothane and isoflurane was in infants and small children (mean age = 12 months),¹ while the M-mode echocardiographic study by Wolf *et al.* compared cardiovascular effects of similar end-expired halothane and isoflurane concentrations in older children (mean age = 5.7 yr).² This important difference was, indeed, one of the reasons for conducting the study. While it is tempting to state that the cardiovascular differences between the two studies are related to age, we believe that the information derived from the measurement techniques are different and difficult to compare for a number of reasons.

In our study, we used two-dimensional echocardiography to assess ventricular volumes, and pulsed Doppler echocardiography to assess cardiac output and stroke volume. These measurements of myocardial performance were used to determine differences between halothane and isoflurane. Because no direct measures of afterload and only an indirect measure of contractility (ejection fraction) was available in our study, we neither intended nor reported in our paper that pulsed Doppler or two-dimensional echocardiography provided better contractility assessment than M-mode echocardiography. As Thys and Hillel point out, both techniques, M-mode and two-dimensional echocardiography, when used to assess contractility, are affected by loading conditions. Specific conclusions about contractility are difficult to support without measurements of loading. In fact, all *in vivo* attempts at measurement of contractility are limited, to varying degrees, by this problem.

Because we used two-dimensional and pulsed Doppler echocardiography, rather than M-mode, to measure global performance, our paper does not include a full discussion of the reasons why M-mode measurements are limited in the evaluation of left ventricular volumes. Nonetheless, prior clinical studies in older children used M-mode, so our discussion did address some of the methodology differences between these echocardiographic techniques.

The peer-reviewed studies referenced in our paper and those referred by Thys and Hillel *all* conclude that two-dimensional measures are superior to M-mode measurements in assessment of ventricular volumes.³⁻⁵ These comparative clinical studies were in subjects with normal left ventricular function, because it is recognized that M-mode volumes and ejection fractions, unlike those with two-dimensional echo, correlate poorly with angiographic determinations of left ventricular volume in situations of dyskinesia or akinesia.^{6,7} The two-dimensional echo technique underestimates ventricular volumes, as we discuss in our paper. For this reason, we added pulsed Doppler echocardiography to measure cardiac outputs and stroke volumes. Ejection fractions were derived by combining the stroke volumes determined by pulsed Doppler echocardiography and left ventricular end-diastolic volumes determined by two-dimensional echocardiography. By this, we believe that we minimized the underestimation of stroke volume. In an editorial that accompanied early M-mode studies during anesthesia, Meyers discussed the limitation of using the cube of a single diameter dimension to assess left ventricular volumes.⁶ While M-mode measurements can be used to derive ventricular volume, cardiac output, stroke volume, and ejection fraction by