Non-invasive determination of cardiac output by Doppler echocardiography and electrical bioimpedance

Sir,—Dr Northridge and colleagues (1990;63:93–7) have, inadvertently I am sure, incorrectly cited the proper name of the stroke volume equation currently implemented in NCCOM3. While it is true that the general form of the equation of Sramek et al., quoted under patients and methods, describes impedance derived stroke volume in a general, qualitative fashion, it is the quantitative definition of volume of electrically participating tissue (VEPT) in the equation that requires clarification. In the work cited by Northridge and associates, the VEPT defined by Sramek et al. is determined in practice uniquely as a function of body height alone. However, as correctly stated by Northridge et al., VEPT as currently used is a constant derived from both body height and weight. NCCOM3 computes stroke volume from the Sramek equation corrected for body habitus. This modified equation is now generally cited as the Sramek-Bernstein equation, which is the one, apparently, used in this study.

WILLIAM C SHOEMAKER
Editor, Critical Care Medicine, 251 E Imperial Highway, Suite 480, Fullerton, CA 92631 USA

1 Sramek BB, Rose DM, Miyamoto A. Stroke volume equation with a linear base impedance model and its accuracy, as compared to thermodilution and magnetic flow meter techni ques in humans and animals. Proceedings of the Sixth Conference on Electrical Bioimpedance, Zadar, Yugoslavia, 1983:38.


Sir,—We read with interest the validation of the transthoracic bioimpedance cardiac output device (BoMed NCCOM3) in acute myocardial infarction (1990;63:93–7). Northridge et al. concluded that in most patients the transthoracic electrical bioimpedance technique was accurate and reproducible. The validity of this technique has been questioned;1 we recently concluded that impedance cardiac output as implemented in the BoMed NCCOM3 was “not accurate compared with the thermodilution reference nor was it quantitatively.”

There are many pitfalls in the assessment of cardiac output and in undertaking validation studies in humans and assessment of devices likely to be applied in critical care should be rigorous rather than enthusiastic. Validation in vitro and in vivo should be attempted, the method should be accurate compared with the reference standard, and show similar reproducibility with ability to track physiological events and the effects of pharmacodynamic interventions.4 A simple rest assessment may have serious limitations and result in misleading conclusions.

Several crucial points in the Northridge paper may have contributed to the outcome. It is unclear whether investigators were blind to each other—this should be mandatory to avoid unconscious bias. Ten averaged estimates of impedance cardiac output were compared with three averaged thermodilution estimates: this is not reasonable. After the establishment of control haemodynamic stability, measurements should be paired, simultaneous, and consecutive; discarding of data (“outliers”) and averaging generates spurious accuracy. The reference thermodilution system is at best under conditions of physiological stability.5,6 Perusal of the data on control cardiac output (3–95 l/min) indicates that this lies outside the 95% confidence limits for acute heart failure (mean 4.31, 95% confidence interval +0–16) based on 32 studies of acute heart failure;7 this suggests haemodynamic compromise, with subjects unlikely to be haemodynamically stable. Validation studies should cover as wide a range of comparison points to ensure the method has no systematic bias; clearly this was not achieved because of the patient population selected. In three of the patients there were considerable discrepancies between the methods—were these with higher cardiac outputs?

Our assessment of the BoMed impedance device in 16 patients with stable coronary disease was as follows.1 Ten consecutive cardiac output measurements were compared with 10 impedance estimations over a 15 minute period. Subjects were studied fasting without premedication after a period of sub-maximal exercise to minimise haemodynamic variability. Observers were blind to each other; the thermodilution method had previously been validated in vitro and in vivo with accuracy and reproducibility determined.1 Data were not averaged and all data were included to obviate observer bias. The data (figure) clearly suggested large discrepancies between the impedance estimate of cardiac output and the thermodilution reference (average difference 1.33 l min−1 m−2; 95% limits of agreement 0.13 to 2.79 l min−1 m−2). During 4 minutes supine bicycle exercise, with techniques to minimise chest movement, these differences increased (average difference 2.36 min−1 l m−2; 95% limits of agreement −0.56 to 5.28 l min−1 m−2). We concluded that the BoMed impedance method as currently implemented in the NCCOM3 device produced uniformly low values compared with thermodilution estimates, suggesting that the incorporated algorithms were not appropriate to the ischaemic population. Our observations suggested that the method at present is unacceptable for scientific or clinical applications.

Devices which rely on empirically determined formulas (impedance changes in transthoracic impedance have been correlated with cardiac stroke output in volunteers) depend absolutely on the reliability of these relations; it may be simplistic to expect similar volume conduc-