Informed Consent

To the Editor:—Prospective studies such as the one described by Lankton et al. may be valuable to jurists when they are faced with deciding questions as to what and how much physicians must tell patients in order to obtain their informed consent. It is to be hoped that far larger numbers of patients will be included in future studies so that patient attitudes following complications more serious than a sore throat and hoarseness might be revealed. A statistically significant difference between the detailed and non-detailed groups might then become apparent. Although Lankton et al. were chiefly concerned with the emotional responses immediately prior to operation, one would like to know the responses after operation of those who suffered some significant injury.

What the anesthesiologist should tell his patient before operation remains an unanswered question. The suggested approach of Lankton et al. may indeed be "the most reasonable." Unfortunately, some jurisdictions require far more than what appears to be reasonable to the physician. Instead, certain basic minimum requirements have been laid down by the courts. It is incumbent upon every doctor to know what those requirements are in his own state.

I hope no-one is misled by Lankton's reference to Salgo v. Stanford (1957). It has been superseded by Calbex v. Grant (1972), which holds that "...a medical doctor has a duty to disclose to his patient the potential of death or serious harm...".

HAROLD L. ENGEL, M.D., J.D.
Los Angeles, California

REFERENCES


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Misapplication of the Warner Method for Computer Analysis of Cardiovascular Variables

To the Editor:—The clinical studies of the cardiovascular effects of scopolamine and nitrous oxide by Bennett and colleagues use, for measuring cardiac output and peripheral vascular resistance, a computerized technique that is not suitable for studies of this type. The imaginative efforts of Dr. Homer Warner and his collaborators have done much to enhance the application of computers to the solution of problems involved in care of hospitalized patients. The "Achilles heel" in their efforts has been the calculation of cardiac output and derived variables from the central arterial pulse waveform. The problem is that the method must be calibrated against an indicator dilution or Fick cardiac output. Whenever the peripheral vascular resistance changes, the pulse contour constants must be recalculated from a repeat control cardiac output.

To the best of my knowledge, no-one has yet devised a reliable means for recalibrating the constants without performing a control indicator dilution cardiac output. In Dr. Warner's original description of his method, this problem is evident in his titratable studies. Recently, Dr. Jurado and his collaborators reviewed the technique and concluded "Thus over periods of several hours between calibration, we are not able to show any advantage of pulse contour methods over the time-honored determination of simple blood pressure and pulse pressure."

Dr. Jurado's conclusions for patient monitoring must be even more applicable to studies of the cardiovascular effects of drug manipulation. If the effects on the peripheral vasculature of the drug in their study are known, the study should not be performed. If the effects are not certain, a validated method must also be used. The results reported by Bennett et al. may be valid, but one simply does not know.

If Dr. Bennett and collaborators have data that refute that already in the literature from several investigators, they should be published; however, the history of the method to date suggests such data are not obtainable.

MARK HILFERTMAN, M.D.
Assistant Professor of Anesthesia
Department of Anesthesia
Stanford University School of Medicine
Stanford, California 94305
Anesthesiology
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To the Editor:—Computation of cardiac output via computer analysis of the central arterial pulse waveform according to the “mean distending pressure method” of Warner1,2 is a simple, safe and accurate method for measuring cardiovascular dynamics in the clinical setting as well as in the experimental laboratory. While it is true that the pulse-wave analysis method has limitations, like all other methods for measuring cardiac output, it remains in our hands, as well as in those of others, a reliable means for determining cardiac output. Nevertheless, some investigators3,4 have presented strong evidence that at least under certain clinical situations, i.e., during hemodynamic instability and the use of drugs that produce profound changes in peripheral arterial resistance or cardiac output, and as duration increases after initial calibration, reliability of the Warner method, as well as all other pulse-wave analysis methods, decreases. The reasons for this are unclear, especially since Warner and co-workers found that in spite of threefold increases and decreases in heart rate, peripheral arterial resistance and cardiac output produced by a variety of vasopressor and vasodilator drugs, cardiac output computed from pulse waves correlated well (r = .90–.98) with output determined by Fick and dye-dilution techniques in man and dogs.5

In an attempt to define clearly the strengths and limitations of Warner’s method, as well as to explain the reasons why others have had so much difficulty validating the technique, we are comparing cardiac output determined by thermocination techniques with that measured by the Warner method in dogs anesthetized with a variety of concentrations of halothane before and during infusion of vasopressor and vasodilator drugs. Preliminary data document that cardiac output measured by the Warner method is highly correlated (r = .94) with that determined using thermocination with concentrations of halothane (0.5–2.5 per cent) in oxygen that produce large changes in cardiac output and significant alterations in peripheral arterial resistance. During infusion of vasopressor or vasodilator drugs correlation remains good so long as heart rate is between 50 and 150 beats/min, arrhythmias are not present, and a good dicrotic notch is obtainable on the arterial waveform. When the dicrotic notch is obscured or altered, as can happen during infusion of agents that change heart rate and peripheral arterial resistance, correlation of the two techniques is dramatically reduced, irrespective of absolute cardiac output or peripheral resistance values.

We believe that the data reported in our two recent manuscripts are valid because: good arterial waveform dicrotic notches were always present, the studies were complete within three hours of computation of the calibration coefficients (a period during which even those who question the Warner technique agree that correlation with other techniques is high), cardiovascular dynamics, although, changing were reasonably stable (no measured variable ever changed more than 50 per cent of control), arrhythmias were not present, and heart rate was always between 50 and 150 beats/min.

THEODORE H. STANLEY, M.D.
GEORGE M. BENNETT, M.D.
EDWARD A. LOESER, M.D.
RYOHEI KAWAMURA, M.D.
Department of Anesthesiology
University of Utah College of Medicine
50 North Medical Drive
Salt Lake City, Utah 84132

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