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Do Let the Blood Pressure Drop and Do Use Myocardial Depressants!

IDEAS change with time just as ideals and fashions do. Presumably facts do not. We have to accept, preferably look for, change in those aspects of our practice based on the former and hopefully increase the portion based on the latter. The observations by Bland and Lowenstein in this issue of ANESTHESIOLOGY present an opportunity to evaluate a new idea, perhaps even a fact, and apply it to the practice of anesthesia, where time will decide its proper place.

For years I have been confused by the continuing admonitions of internists and many of my colleagues that patients afflicted with ischemic and other heart disease must be anesthetized with regimens that "don't let the blood pressure fall, and avoid myocardial depressants." These same internists, of course, continued to treat angina with the time-honored nitroglycerin, which lowered blood pressure—and effectively relieved the discomfort of myocardial ischemia. Consistency would have called for at least a trial of epinephrine! They also managed cardiac failure with rest and even sedation. During anesthesia, however, quite the contrary approach was urged. The natural course to follow when confronted by this sage advice was to utilize anesthetics that maintained or even elevated cardiac output and blood pressure. While a few argued that such an approach should actually be prohibited for sick hearts, support for the admonition grew and culminated in a popular swing to morphine anesthesia. The authors of the current study dwell in one of the highest temples of morphine anesthesia. They deserve much credit for subjecting a favorite tenet to study. While they do not compare halothane with any other type of anesthesia, they do demonstrate that the time-honored cliché about maintenance of blood pressure is subject to serious question.

The results of this study emphasize at least four concepts which, in importance, probably transcend specific pharmacologic observations here observed.

First, and perhaps most important, is the fallacy of accepting the "obvious." Intuitively, who could argue with a statement that arterial blood pressure must be maintained to perfuse the myocardium and that myocardial contractility must be preserved? One might as well argue that too much oxygen could be harmful.

Second, the common statement that anesthesia is a stress is challenged. Preservation of myocardial contractility in a stressful situation would certainly seem reasonable, but need anesthesia, as it is now practiced, be a stress? Or does this present study indicate that anesthesia is one of the least stressful of situations, actually a protection from other (surgical) stress.

Third, the long-standing debate as to whether there is a pharmacologically appropriate *choice of anesthesia* for each particular patient in each situation receives new input. Here are two rather diametrically opposed views as to which approach should be used for patients who have heart disease. Can we predict, on the basis of pharmacologic evidence, which will be best? Or does the knowledge, understanding and skill with which each is applied and modulated outweigh specific pharmacologic effects?

Fourth, improper use of experimental facts is pointed out in this controversy. Many have determined with certainty that halothane is a potent myocardial depressant. It has also been determined that pre-existing heart disease makes this depressive effect more pronounced. It does not follow, however, that halothane—in proper dosage—is any more harmful than other anesthetic agents for patients who have heart disease. In fact, from the pharmacologic observation that halothane is a myocardial depressant, one can equally well predict that halothane anesthesia has a favorable effect upon the myocardium. It is important to determine facts, but more important to use them wisely. To add to the authors' credit, there is no overextended suggestion found in their article. They realize that clinical application of their results must be based on further data.

Results of this study may oppose much

popular opinion, but should not be surprising. The "work-reduction" approach to treatment of myocardial infarction has received increasing support. Recently much information consistent with this study has been dispersed concerning the effects of blood pressure, heart rate, and anesthetic agents on myocardial oxygen demands. To be surprised is to overlook the tremendous clinical success halothane has enjoyed in clinical situations characterized by myocardial ischemia.

As with most good research, more questions are raised than answered by the work of Bland and Lowenstein, and the dangers of overinterpretation are real. Many other investigators are now engaged in pursuit of further information in this and closely related fields, and soon will provide further clarification. In the meantime, those of us who like the present results must recall that this study was done in dogs with acute localized disease imposed upon chloralose–urethane anesthesia. It may be a giant step to man with chronic diffuse coronary-artery disease without this baseline anesthetic. Other anesthetic approaches may produce similar or even better results. Attention must also be directed to emergence from anesthesia and to the early postoperative hours, when pain and discomfort may increase myocardial oxygen demands without the protective effect of the anesthetic. This perioid may be the real stress of an anesthetic experience and certainly needs early investigation. The matter of cardiac volume, and related effects on myocardial wall tension, is raised by the authors. Doses of halothane higher than those used by them have been shown to increase end-diastolic volume. The concentration of anesthetic will also be important in determining the severity of hypotension and the level of bradycardia. Non-critical interpretation of the results of this study might well lead to undesirable conclusions.

It is, of course, now *perfectly obvious* that the proper way to anesthetize patients with ischemic heart disease is to "reduce the determinants of myocardial oxygen demand." It is so obvious as to be true. This new admonition will undoubtedly achieve the same sanctity and respect as the warning to "avoid hypotension, give plenty of oxygen, and avoid myocardial depressants. . . ."

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Drug Abuse

HALOTHANE ABUSE IN HOSPITAL EMPLOYEES Access to centrally acting drugs is a significant problem in the hospital environment. Obvious areas of concern are narcotic-analgesics, barbiturates, and tranquilizers. Less obvious is the fact that the volatile inhalation anesthetics represent an easily concealed and transported substance with both major abuse liability and potential for inducing rapid death. The authors report deaths of three hospital employees (two operating room technicians and one emergency room technician) as a result of halothane inhalation. One breathed halothane vaporized through gauze. Two rebreathed halothane by the use of either a plastic bag over the head or a "tent-like arrangement of a sheet and pillows." A number of friends of these individuals reported attending parties where halothane "had been passed around for group inhalation." Access to such drugs should be controlled carefully, and education of those likely to misuse these substances is extremely important. (*Spencer JD, Rausch FO, Trefny FA: Halothane abuse in hospital personnel. JAMA 235: 1034–1035, 1976.*)