Is Anesthesia Beneficial for the Ischemic Heart?

When coronary perfusion is decreased by anesthesia in healthy hearts, myocardial oxygen demand is diminished to approximately the same extent in dogs, pigs, and man. Apparently, autoregulation, which attempts to couple myocardial oxygen supply and demand, is not significantly altered by anesthesia. The demonstration of a similar effect in ischemic hearts is not so clear-cut. Studies in dogs with ligated coronary arteries have suggested that halothane anesthesia and epidural anesthesia improve myocardial perfusion and oxygenation. However, in both studies, the control state with which the anesthetic was compared was hyperdynamic. In particular, heart rates were greater than 150 beats/min. The anesthetics appreciably decreased those heart rates, as well as blood pressure. Perhaps, then, the major effect of anesthesia is the reduction of heart rate, blood pressure, and cardiac contractile performance, thereby lessening myocardial oxygen demand more than the concomitant decrease in coronary perfusion pressure decreases myocardial oxygen supply.

The readers of the Journal have been introduced to another aspect of myocardial perfusion, first in the study by Klassen et al., and now by the elegant article in the present issue by Verrier et al. The use of radio-labeled microspheres has allowed these investigators to measure regional myocardial perfusion. This may be important, for the intramyocardial tissue pressure causing cessation of the flow generated by coronary arterial pressure appears to be higher in the subendocardium than in the subepicardium, probably as a result of the greater influence of intercavitary (ventricular) pressure. Consequently, with reduced flow, the subendocardial vessels will dilate and lose their autoregulatory properties before the subepicardial vessels. In this situation, the ratio of subendocardial (inner) blood flow to subepicardial (outer) blood flow, known as the I/O ratio, may be an early indicator of myocardial ischemia. Kllassen et al. showed that epidural anesthesia could increase I/O ratios both when coronary perfusion pressure was reduced and when a coronary artery was ligated, although the changes were small. Verrier et al. did not document I/O ratios at the same coronary perfusion pressures with nitrous oxide-based and halothane-based anesthesia, but suggested that the "break point" where the I/O ratios decreased was lower with halothane than with nitrous oxide. In each study, epidural anesthesia and halothane anesthesia decreased heart rate (among other hemodynamic changes), and heart rate changes alone can influence I/O ratios.

In addition to I/O ratios, the current publication provides the first documentation of "coronary vascular reserve" during anesthesia, although the concept was introduced and discussed in scholarly detail in a previous editorial. Briefly, coronary vascular reserve is the coronary arterial blood flow differential at a given coronary arterial pressure between the flow measured during pharmacologically induced "maximal vasodilatation" and the flow during a given intervention. Theoretically, in the situation where there was a higher coronary vascular reserve, the coronary vessels could more effectively dilate in response to increasing myocardial oxygen needs. Whether coronary vascular reserve can be dissociated from the commonly accepted indices of myocardial oxygen supply—demand is questionable. In fact, Verrier et al. have preliminary data showing that the significantly higher coronary vascular reserve seen with halothane
compared with nitrous oxide is abolished when the heart rate differential between the two anesthetic states is eliminated by pacing.10

Another interesting aspect of coronary physiology and pharmacology (also discussed previously13) is the "zero-flow pressure intercept," or critical closing pressure of coronary arteries. By extrapolating the line relating coronary arterial pressure to coronary arterial blood flow during "maximal vasodilation" to zero flow, this figure is obtained. Although most earlier calculations of coronary vascular resistance had assumed that the pressure differential across the heart was arterial (diastolic or mean) pressure minus coronary sinus (right atrial) or left ventricular end-diastolic pressure, in fact, the zero-flow pressure intercept measurements from this and other studies have yielded pressures considerably higher than either right atrial or left ventricular end-diastolic pressure.10,13–15 However, the zero-flow pressure intercept is still greater in the subendocardium, and decreases progressively from subendocardium to subepicardium. It may be that the critical determinant of coronary vascular reserve is this zero-flow pressure intercept. It is tempting to speculate that this indicator of intramyocardial tissue pressure may be related to the tension developed by the contractile elements in the myocardium. Thus, a decrease in contractile force would not only decrease myocardial oxygen consumption, but would also decrease the zero-flow pressure intercept and improve coronary vascular reserve. The reduction in coronary vascular reserve produced by a heart rate increase could also be related to an increase in contractile force through the Bowditch or rate-treppe phenomenon.16

There can be little question that anesthetics are beneficial for the ischemic heart under certain circumstances. Tachycardia, sympathetic hyperactivity, and perhaps increased outputs of hormones such as angiotensin and vasopressin are deleterious to the ischemic heart. Anesthetics can improve myocardial perfusion by decreasing these effects. However, if myocardial perfusion and oxygenation are adequate without anesthesia, then the production of decreased coronary perfusion pressure by anesthesia in a heart where flow may be highly pressure-dependent is unlikely to benefit that heart, until proven otherwise. In addition, data from an artificial animal model must not be transferred too quickly to the clinical situation. (Verrier et al. have wisely refrained from making such a clinical correlation.) Although the rate-pressure product may be fallible as an index of myocardial oxygen consumption,17–19 there is still no solid evidence to challenge the clinical practice of trying to keep myocardial oxygen consumption as close to the unanesthetized angina-free value as possible while maintaining coronary perfusion pressure.

References


