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Is Anesthesia Beneficial for the Ischemic Heart? II

IN A STUDY by Bland and Lowenstein[1] we were told, "Do let the blood pressure drop and do use myocardial depressants."[2] In defense of this thesis, Dr. Hamilton and co-workers have now demonstrated that this approach is indeed feasible.[3] They have treated 12 patients who responded to surgical stress with increased systemic, pulmonary arterial, and left ventricular filling pressures with the myocardial depressant anesthetics halothane and enflurane. Six of their patients also showed evidence of myocardial ischemia during the stress by ST-segment analysis which reverted to normal with the anesthetic treatment. Thus, anesthesia, specifically halothane and enflurane, clearly was beneficial for those ischemic hearts.

However, the same Dr. Lowenstein, with different coworkers, now suggests that the decrease in blood pressure and global ventricular function seen with increasing concentrations of halothane, although clearly not detrimental to the normal heart, may be associated with regional myocardial ischemia in a heart with coronary stenosis and "may be an important mechanism for perioperative infarction."[4] Clearly such an effect is not beneficial for the ischemic heart!

Can we resolve this quandary? In fact, I do not believe that there is any contradiction between the two studies. As noted previously, "tachycardia, sympathetic hyperactivity and perhaps even increased output of hormones such as angiotensin and vasopressin are deleterious to the ischemic heart."[5] Although tachycardia was not a significant part of the syndrome treated by Roizen *et al.*,[3] they documented sympathetic hyperactivity with increased plasma norepinephrine concentrations. Thus, they were restoring the myocardial oxygen supply-demand balance by decreasing demand with the depressant anesthetics. However, as Hamilton has suggested, other pharmocologic regimens in other people's hands may be just as beneficial.[2]

On the other side of the coin, "if myocardial perfusion and oxygenation are adequate without anesthesia, then the production of decreased coronary perfusion pressure by anesthesia in a heart where the flow may be highly pressure dependent is unlikely to benefit that heart."[5] Lowenstein *et al.* intentionally prepared an animal with a "critical stenosis" (so that no ischemia was evident at low anesthetic concentrations and high perfusion pressures) in the left anterior descending coronary artery leaving the circumflex coronary artery normal. As anesthetic concentration was increased and diastolic arterial pressure decreased, progressive dysfunction appeared in the region supplied by the stenosed coronary artery signifying myocardial ischemia. On the other hand, there was no such dysfunction in the segment of myocardium supplied by the normal circumflex artery. Thus, Lowenstein and co-workers have demonstrated for the first time that the oxygen supply-demand balance of myocardium supplied by a stenosed artery is not well-preserved during increasing concentrations of halothane. On the basis of the present work, it seems likely that the decreased perfusion pressure was responsible for this imbalance. This hypothesis is supported by the observations of Behrenbeck *et al.* who observed no difference in regional myocardial function between ischemic and non-ischemic areas as halothane concentration was increased when perfusion was controlled at the different anesthetic concentrations by an extra corporeal pump.[6]

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Thus, in one instance, adequate myocardial oxygen balance was disturbed by surgical stress and restored by anesthesia. In the other instance, adequate (barely) myocardial oxygen balance was disturbed by increasing anesthetic concentrations producing more decrease in oxygen supply than demand. The conclusion voiced in a previous editorial still appears to be valid. "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."[5]

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