Myocardial Protection and Oxyhemoglobin Dissociation Curve

To the Editor.—In an excellent review of myocardial protection during anesthesia, Warltier et al.1 mention the possibility of improving oxygen availability to the myocardium by a right shift of the oxyhemoglobin dissociation curve.2–4 The beneficial effects of β-blocking drugs also are reviewed. However, the correlation of the well-known fact that β-blockers shift the curve to the right is omitted. This rightward shift may promote oxygen delivery to the myocardium during ischemia. The clear reduction in perioperative infarction rates after β-blockade rests on this fundamental effect.

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Prevention of Perioperative Myocardial Ischemia

To the Editor.—I read with interest the Clinical Concepts and Commentary section of the January 2000 issue of Anesthesiology.1 It was useful, informative, educative, and of practical value in day-to-day anesthetic practice.

The evolving modern concept of “ischemic preconditioning” is emphasized rightly. I could not understand clearly why the authors, while explaining the shortcomings of general anesthesia, said that high concentrations of volatile anesthetics may cause increases in sympathetic activity, whereas in the next paragraph, they explained the cardioprotective effects of volatile anesthetics. I suppose that should be read as decreases in sympathetic activity, rather than increases in sympathetic activity.

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Perioperative Myocardial Ischemia: Pathophysiology and Does it Really Matter

To the Editor.—The excellent review detailing approaches to the prevention of perioperative myocardial ischemia1 includes a number of assertions and implications that conflict with published studies. First, the authors define coronary perfusion pressure as the difference between aortic diastolic pressure (DBP) and left ventricular end-diastolic pressure. I have been unable to find any peer-reviewed documentation stating that DBP is the upstream pressure for coronary perfusion pressure. This seems illogical because flow into the coronary arteries, coronary blood flow, is at or near its nadir at DBP.2–3 Presumably, this statement crept into textbooks because of the high coronary blood flow during ventricular diastole. However, the observed nadir in coronary blood flow at DBP is expected because DBP corresponds to the onset of ejection, which occurs during ventricular systole when coronary blood flow is impeded because left ventricular intracavitary and intramyocardial pressures are at least as high as aortic root pressure. Moreover, to use the left ventricular end-diastolic pressure as the downstream pressure for coronary perfusion pressure, one must assume that there is a vascular waterfall across the left ventricle. Although this seems logical, and early data seemed to confirm this, later studies that incorporated the effects of vascular compliance did not confirm an arterial waterfall.4 It is an area of some controversy, but most data do not support this concept. Consequently, the authors’ definition of coronary perfusion pressure does not seem to be supported by data.

Second, it is interesting that no prospective study has shown that intraoperative management of ischemia affects the myocardial infarction rates after β-blockade rests on this fundamental effect.

Richard B. Weiskopf, M.D., Editor

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tation rate in the immediate postoperative period. This may be the result of difficulties in designing and implementing such studies, or it may be that such interventions do not affect this outcome, which is plausible for the following reasons. There is abundant evidence that the majority of events that constitute the acute coronary syndromes are related to plaque rupture or ulceration. Plaques that are vulnerable to such disruption tend not to be at the sites of high-grade stenoses, but high-grade stenoses cause stable angina and are at the sites of rate-related ischemia, which is not part of the acute coronary syndromes. Consequently, the regions at risk for a postoperative myocardial infarction might differ from those that generate rate-related ischemia. Therefore, rate-related ischemia might be totally unrelated to the risk of postoperative myocardial infarction, \textit{i.e.}, this type of ischemia may be the equivalent of stable angina induced with exercise, which occurs frequently in patients with high-grade stenoses but rarely causes infarction. In contrast, unstable angina, which is part of the acute coronary syndromes, reflects transient occlusion or embolization from an unstable plaque and may be unrelated to rate-related ischemia. This is not to say that such ischemic episodes should not be treated, especially because stress or exercise seems capable of disrupting vulnerable plaques. However, if the ST-T depression seen in the acute perioperative period is the equivalent of stable angina, failure of treatment to alter perioperative myocardial infarction rates and difficulties in predicting the risk of an adverse outcome would be expected. Perhaps we should focus more on preventing plaque rupture or alterations in the coagulation system that might predispose to thrombosis at the sites of unstable plaques.

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ative infarction. Not all patients with myocardial infarction have plaque rupture. Plaque rupture is only one element of a continuum of multifactorial etiologies that cause irreversible tissue damage. Because of the multifactorial nature of myocardial infarction, it can be treated by any of a variety of means, including classic manipulations of oxygen supply and demand, as well as by interference with the coagulation cascade. No treatment should be considered in isolation from the others. β-Blockers have been proven to reduce the reinfarction rate after acute myocardial infarction, have been proven to decrease cardiac morbidity and mortality after surgery, and should be used in the perioperative period. New avenues for reduction of the incidence and severity of myocardial infarction are being explored, but this does not negate use of β-adrenergic blocking agents.

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