Preoperative Invasive Monitoring and Coronary-artery Disease

To the Editor:—In a recent article,1 Lunn and associates conclude: "The data indicate that invasive monitoring in patients with coronary-artery disease not taking propranolol should be minimized or performed after a reasonable depth of anesthesia is achieved before inserting an arterial catheter." We agree in principle with the conclusion as regards pulmonary-artery catheterization, but do not agree with the conclusion when radial arterial catheterization is considered. Our disagreement is based on the following. For the past 13 years we have inserted radial-artery catheters in unanesthetized patients the day before their cardiovascular operations. The insertion is done by a specially trained nurse or technician in the patient’s room after a careful explanation of the procedure. This routine procedure is much less threatening to the patient than a similar procedure performed in the operating suite. Using this procedure, we have inserted more than 11,000 arterial catheters in patients with coronary-artery disease.5,4 We have encountered no serious angina in any of these patients. With patients known to be intolerant of even minor stress, we have taken special time and care. Even with this group of patients, arterial catheter insertion has not initiated angina. Since one of the most crucial times to monitor these patients is during induction of anesthesia, we think it unwise to wait until a reasonable depth of anesthesia is achieved before inserting an arterial catheter.

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References

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Hypertension following Nitroprusside

To the Editor:—Kambatta et al.1 have recently speculated that nitroprusside-induced hypotension caused a decreased hepatic arterial blood flow which, in turn, caused a decreased metabolism of renin, an increased half-life of renin, and an increased plasma level of renin. The logic employed was based on the presumed absence of hepatic autoregulation, thus rendering hepatic arterial flow dependent on systemic blood pressure.

We see two problems with this logic. First, the concept that the liver is not autoregulated has been proposed before,2 but there is evidence that indicates that hepatic blood flow is altered by myogenic, neural, metabolic, and hormonal influences.3,4 In addition, hemorrhage in animals causes total hepatic blood flow to decrease while hepatic arterial blood flow concomitantly increases.5,6 Thus, the concept that hepatic arterial blood flow is passively altered by blood pressure is doubtful, and assumptions based on this concept should be reexamined.

Second, a marked selective reduction in hepatic arterial flow in systemically normotensive patients has been demonstrated to occur in some patients receiving halothane anesthesia.7,8 It is possible that hepatic arterial blood flow was indeed decreased in the series of Kambatta et al., but it may have been due in part to halothane and in part to nitroprusside infusion. Thus, the statement that "the hepatic circulation has not been studied in man during hypotensive anesthesia with sodium nitroprusside" should be extended to

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