

Anesthesiology
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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 established." 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.^{2,3} 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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Hypertension following Nitroprusside

To the Editor:—Khambatta *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 Khambatta *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

read "sodium nitroprusside and halothane," since the effects of the two drugs may be additive or even synergistic in some patients.

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Patient Acceptance of Orally Administered Antacid Therapy during Labor

To the Editor:—As anesthesiologists working on an active obstetric service, we have closely followed the reporting of the efficacies of various antacid-anticholinergic regimens, since aspiration of liquid vomitus is a leading cause of maternal death. We recognize that the pH of the aspirate is one critical factor in determining maternal outcome, and that much effort has been directed at controlling this variable. It is known that the percentage of patients "at risk," in terms of pH, can be reduced from 42 per cent to 0 per cent by the administration of magnesium trisilicate or to 13 per cent with aluminum hydroxide when given less than 45 min prior to anesthesia.¹ Roberts and Shirley also substantiated the efficacy of prophylactic alkalization of gastric contents by the routine use of antacids in labor.² We have followed this practice, but since previous studies have not considered patient acceptance of repeated doses of antacids, we examined the antacids commonly used at our institution in the following manner.

For a period of one month all patients received 30 ml of Gelusil®, Maalox®, or Mylanta II® every three hours throughout labor. On the first day after delivery the patients were visited by an anesthesiologist, who asked them whether they had experienced nausea and vomiting during labor or diarrhea since labor. They were also specifically asked whether they found the antacid objectionable, and what they thought the purpose of the antacid was.

TABLE 1. Palatability of Antacids

	Number of Patients	Vomiting (Per Cent)	Objections (Per Cent)
Mylanta®	155	9	5.2
Gelusil®	170	13	5.3
Maalox®	130	12	10.7
No antacid	123	20	—

There was no statistical difference between antacids in the incidences of nausea and vomiting or diarrhea, irrespective of parity, type of anesthesia, or administration of narcotics and tranquilizers. Regardless of the number of doses given, the majority of patients found the taste of the antacids to be either pleasant or neutral, and only 6.8 per cent of parturients objected to them. Although not statistically significant, there was a tendency for Maalox to be objectionable more often (table 1). We were surprised to find a higher incidence of vomiting in the control group compared with those receiving antacids ($P < 0.05$). Very few patients possessed a real understanding of the purpose of antacid administration.

We conclude that the administration of antacid during labor is acceptable to the vast majority of patients, and seems to decrease the incidence of vomiting. Since no difference in palatabilities exists and the buffering capacity of Gelusil is half that of