difference between the treatments. Then, through the use of the mathematically specious process of "relative risk" calculation, they deduce that desflurane is associated with 2.3 times the incidence of prebypass ischemia than sufentanil (17% *vs.* 7%).

These kinds of clinical studies have little practical moment to the ultimate consumer of them, the practitioner. Investigations that purport to test hypotheses with treatments involving insufficient subjects predictably, as in the present case, arrive at ambiguous conclusions. Rather than focusing on type I *versus* type II errors, I believe that the larger problem is one of investigators trying to do too much with too little. The investigation spanned a period of from August 1990 to April 1991—what, I ask, was the hurry? Investigators should ask themselves, if sufficient time, money, personnel, or energy are not available for the task at hand, perhaps the task should not be undertaken. Careful attention to these issues may help avoid ambiguous and confusing messages communicated to readers such as myself who depend heavily upon scientific publications for rational practice decisions.

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In Reply:—We thank Biddle for his comments regarding our paper and would like to respond to several of the comments made in his letter.

First, Biddle stated that we demonstrated that "desflurane, like the other potent inhalational agents can be used, with appropriate adjuncts, without unacceptable hemodynamic consequences in this patient population." This is an incorrect statement. In contrast, we concluded that during anesthetic induction, desflurane, when used with sodium thiopental as an adjunct, was associated with *more* hemodynamic changes and myocardial ischemia, when compared with sufentanil anesthesia. During anesthetic maintenance, however, the risk of myocardial ischemia was not significantly increased with desflurane (used as a *sole* anesthetic agent) when compared with sufentanil anesthesia when attempts at strict hemodynamic control were employed.

Second, Biddle commented on "the differences between the sufentanil and desflurane groups relative to adverse cardiac outcome" and then went on to abstract multiple sections from the manuscript to reach the conclusion that "these kinds of clinical studies have little practical moment to the ultimate consumer of them, the practitioner." In making these statements, Biddle appeared to be rather confused about the outcome measurements and the purpose of our study. To reiterate, the purpose of our study was to determine whether the risk of myocardial ischemia was increased in patients with coronary artery disease receiving desflurane as a primary anesthetic. We believed that this was an important question because desflurane, having a low blood solubility, would likely be used for rapid induction, emergence, and recovery in noncardiac surgery. Since a substantial proportion of patients undergoing noncardiac surgery have or are at risk for coronary artery disease, proving that desflurane is safe in this patient population is critical.

We chose patients undergoing coronary artery bypass graft (CABG) surgery as a model because these patients have known coronary artery anatomy and hemodynamics are already invasively monitored. Thus, the primary end-point in our study was myocardial ischemia as measured by electrocardiogram or echocardiography (precordial or transesophageal). Our assumption is that if the risk of myocardial ischemia is not increased under desflurane anesthesia, then it is unlikely that the risk of adverse cardiac outcomes such as myocardial infarction will be increased since myocardial ischemia is usually a harbinger of myocardial infarction. If our study had demonstrated an increased risk of myocardial ischemia associated with desflurane, then further studies examining adverse cardiac outcomes would be necessary. This type of study design is more feasible since outcome studies examining hard end-points such as death or infarction are generally very large in size. The inclusion of adverse cardiac outcome data (cardiac death, myocardial infarction, and ventricular failure) in our report was performed for completeness. Comparison of adverse outcomes between the two anesthetic groups would be inappropriate (as discussed in the limitation section of the manuscript) since the study sample size was calculated based on previous incidences of myocardial ischemia and *not* myocardial infarction rates.

To clarify issues regarding sample size calculation, we based our sample size calculation on previous studies of patients undergoing CABG surgery who received isoflurane anesthesia.[1] The sample sizes were calculated to give an 80% chance of detecting a 50% difference in the incidence of myocardial ischemia with a level of significance at 5%. Using a known incidence of myocardial ischemia of 20% (as detected by Holter electrocardiogram or transesophageal echocardiography), the sample sizes were 81 patients in each of the two treatment groups, for a total of 162 evaluable patients for the study. In anticipation of unevaluable data, the final sample size was 200 patients. In designing the study *a priori,* we chose not to perform multiple looks at the data before the conclusion of the study to avoid committing a type I error. Also, this type of approach would mandate adjusting the level of statistical significance required, necessitating larger sample size if the study were to be modified. Thus, in contrast to Biddle's charge of us "trying to do too much with too little," we have adhered to the study design and the *a priori* sample size calculation.

In our discussion of the potential limitations of this study, we indicated that our study could not exclude the possibility of a small difference in echocardiographic evidence of ischemia between the two anesthetics during the prebypass period since there was a trend

Table 1. Incidence of Myocardial Ischemia Detected by Electrocardiography (ECG) Alone, Echocardiography Alone (Precordial or Transesophageal) or ECG or Echocardiography in the Different Intraoperative Periods

	ECG			Echocardiography			ECG or Echocardiography	
Event	Desflurane	Sufentanil	P*	Desflurane	Sufentanil	P	Desflurane	Sufentanil
Induction (+ intubation)	9/99	0/98	.003	5/39	0/29	.067	14	0
Induction (− intubation)	6/99	0/98	.023	4/39	0/29	.13	10	0
Incision	3/99	1/98	.6	6/91	2/84	.3	9	3
Maintenance (− induction)	7/99	3/98	.3	15/91	6/84	.07	19	7
Maintenance (+ induction)	12/99	3/98	.03	20/91	6/84	.01	28	7
De novo maintenance + induction	8/99	1/98	.04					

De novo refers to those patients who developed new intraoperative ischemia without the occurrence of preoperative ischemia.

* Fisher's exact test.

for more echocardiographic ischemia (16%) in the desflurane group than in the sufentanil group (7%). We calculated the relative risk to be 2.3 (95% confidence interval 0.9–5.7). On further analysis of the ischemic data, it appears that we even may have underestimated the risk of myocardial ischemia under desflurane anesthesia if we combined the induction with the prebypass periods, or combined the electrocardiogram with the echocardiographic ischemic episodes (table 1). We took a conservative approach in the original analysis, *i.e.,* to separately analyze electrocardiographic and echocardiographic data, because it is not clear whether the true incidence of myocardial ischemia is the sum of the two. Nevertheless, these additional analyses further substantiate our conclusion that "further studies are necessary to investigate the induction and maintenance effects of desflurane in the at-risk patient undergoing noncardiac surgery" because extrapolation of the present data in the cardiac surgical patients to the noncardiac surgical patients is not warranted. Finally, and most importantly, why desflurane has a propensity to cause tachycardia and systemic and pulmonary hypertension remains unresolved. The mechanism of these changes and whether these effects can be blunted by adjunctive agents should be addressed by further studies.

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Perioperative Dislocation in a Patient with a Prosthetic Hip

To the Editor:—Hip dislocation is a known postoperative complication following total hip arthroplasty (THA). We present a case of dislocation 3 weeks following THA during the administration of a spinal anesthetic.

A 77-yr-old physician was scheduled for a transurethral resection of the prostate. His past surgery included bilateral THA with revision of the left hip prosthesis 3 weeks earlier.

He requested a spinal anesthetic and was placed in the sitting position with legs together, knees bent, and feet resting on a chair. He was assisted in leaning forward with his arms supported in front of him. During insertion of the spinal needle *via* a midline approach, he complained of sharp pain, which he described as "2 to 3 centimeters inferior and lateral to the puncture site." On physical examination, the patient had a noticeable deformity of the left hip. The orthopedics service was consulted, and radiographs of the left hip demonstrated posterior dislocation.

General anesthesia was induced, as requested by the patient, and the dislocation was reduced manually without difficulty; the prostatic resection proceeded under the same general anesthetic without complications.