0.05; this makes no sense to me. Either one accepts the statistical approach to analyzing data or not. Rightly or wrongly, I believe the former, it is the commonly accepted scientific approach for linking conclusions to data.

The San Diego group, while accepting the scientific validity of the study, question its clinical application on the grounds that the management of the control group was not representative of the usual standard of practice for such patients.[4] Specifically, they fault the failure to use hypothermia, membrane oxygenators, and arterial filters during bypass as well as the use of glucose-containing solutions in the pump prime. Accordingly, they speculate that the incidence of neuropsychiatric deficits in the control group may have been artificially high, thus exaggerating the beneficial effects of thiopental therapy. Because this is far from my area of expertise, I cannot effectively respond to this criticism and will instead rely on my colleagues who specialize in cardiac anesthesia to thrash this one out.

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In reply:—Neither of these two thoughtful letters finds fault with our scientific methods or conclusions. Both, however, express discomfort with our clinical recommendation. Although Scheller *et al.* acknowledge our incidence of neuropsychiatric dysfunction in the control group was well within the range reported by others, they remarkably suggest that if we perfused patients properly, we would have no neurologic dysfunction in the control group and would not need barbiturate protection. For support, they cite three studies of cardiopulmonary bypass in humans, not one of which claims to show objective reduction in cerebral dysfunction by filters, membranes, or hypothermia, and three reports of temporary global cerebral ischemia in animals, a model not related to that considered here. Until someone can demonstrate an absence of neurologic dysfunction by optimal perfusion of the population we studied, thiopental seems to be indicated for patients managed with "suboptimal" perfusion.

Shanks *et al.* should know that truth is not established by a *P* value. Our recommendation was based not on this "significant" difference alone, but rather on: 1) the growing evidence that embolus is the commonest cause of cerebral dysfunction after open heart operations[1,2]; 2) the striking similarity between these temporally predictable events and animal models of focal cerebral ischemia[3,4]; 3) a wealth of data describing the benefit of barbiturates in the animal model[3–5]; and 4) the beneficial dose–response effect found in our two investigations.

Although the risk of thiopental therapy (greater hemodynamic instability after cardiopulmonary bypass and slow postoperative awakening) is not insignificant, the benefit of reducing the incidence and severity of this potentially devastating complication is compelling. We, too, anxiously await the appearance of data from other centers as the only way to establish the wisdom of our recommendation.

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