In Reply:—My co-authors and I appreciate Edmonds and colleagues' interest in our work. I would like to address their criticisms by pointing out the ways in which our approach differs from theirs and what I perceive to be significant weaknesses of their study.

Our work was an observational study. We sought first to characterize the influence of varying temperature on the electroencephalogram (EEG) during cardiopulmonary bypass in the "normal" situation and then to find associations between changes in EEG descriptors and postoperative neuropsychologic dysfunction. We found no consistent pattern of EEG changes with temperature, weak associations between changes in two of eight EEG descriptors and early postoperative neuropsychologic impairment, and no association at 7-month follow-up. Realizing the difficulty of proving what turned out to be an essentially negative result, we invested a great deal of human and computer time in diligently searching for underlying relationships that may not have been evident from simpler analysis of our data.

We were also careful to point out what we believe to be the major limitations of our work: two-channel monitoring, frequent roller pump interface, and our particular anesthetic technique.

Edmonds et al. tried to go a step further than we did. They made therapeutic interventions (increasing arterial pressure or perfusion pump flow) in response to prolonged focal increases in 5- to 6-hertz power as indicated by a commercial instrument that indexes the EEG descriptors to adaptively derived baseline values. According to their published article, these interventions resulted in a large reduction in the incidence of postoperative disorientation among their patients. Their letter also alludes to unpublished data supporting an additional claim of a reduction in major neurologic injury due to these interventions, which, taken at face value, suggests that they have made a major therapeutic breakthrough in the intraoperative management of cardiac surgical patients. Scrutiny of this portion of their work must await peer review and publication of the full details. I wish, however, to point out some of the major design flaws present in their already published work. (Similar flaws also may be found in the work of Arom et al., mentioned in Edmonds et al.'s letter.)

First, the Edmonds study was susceptible to bias because it was nonblinded, the assessors of outcome were supported by the manufacturer of the equipment tested, and the study used nonparallel controls. It is entirely possible that other differences in total perioperative care between the sequential patient groups may account for the observed differences in outcome. Second, their outcome measure (disorientation) was severely limited in scope and was assessed only on the fifth postoperative day, when residual effects of drugs, sleep deprivation, pain, etc. could have influenced the results. Third, few details were provided about their routine management of blood pressure and perfusion flow during bypass and the type, magnitude, and frequency of the interventions actually made. (In our study we employed high flow perfusion and carefully tried to keep mean arterial pressure in the range of 60- to 80-mmHg, and thus we may have avoided episodes that would have prompted interventions in Edmonds et al's patients.) Fourth, fluctuations in anesthetic depth caused by their technique of "repeated bolus doses of fentanyl or sufentanil" could have produced some of the EEG and blood pressure changes that they observed.

The lack of long-term follow-up is another major limitation of their work. Numerous studies have found one third or more of patients to exhibit some form of neuropsychologic impairment during the first week or so after cardiac surgery. However, a large majority of impaired patients in most series make a complete recovery within weeks or months. Without follow-up, it is not clear that the interventions advocated by Edmonds et al. confer any long-term benefit.

Our quoted statement about a "prohibitively large sample size" was based upon a simple statistical fact: given the approximately 5% severe neurologic injury rate in recent studies (references 11 and 32 in our paper), it would require approximately 500 patients each in treatment and control groups to have 80% confidence of detecting a 50% or greater reduction in the rate of impairment. Edmonds and his colleagues may have been lucky and acquired a set of data that happened to achieve statistical significance with a smaller sample size. But this occurrence could not have been lucky and acquired a set of data that happened to achieve statistical significance with a smaller sample size, but this occurrence could not have been expected a priori and was likely due to the large false positive rate (68%) that was accepted in choosing their criteria for intervention.

In summary, although our work essentially failed to find EEG correlates of postoperative neuropsychologic impairment, the possibility remains that others may yet succeed in this endeavor. However, on the basis of their work published to date, I do not believe that Edmonds et al. have made a convincing case for making therapeutic interventions based upon readings from their instrument (other than gross signal dropout). EEG monitoring is expensive, technically demanding, subject to confounding factors (e.g., of anesthetic type and depth, body temperature), and diverts the anesthesiologist's attention from other aspects of patient care. Thus, before routine monitoring can be advocated, the benefit of interventions based upon monitoring should be demonstrated in a properly designed clinical trial utilizing a randomized, prospective, double-blinded study design and including neuropsychologic and neuropsychologic assessment and follow-up.

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In Reply—The observations by Edmonds and Chabot and their colleagues raise several important issues. The technical limitations of Bashein’s work are recognized, and the suggestion of an eight-channel montage of bipolar pairs specifically selected for watershed areas is, from a neurophysiologic viewpoint, preferable to the more common two-channel montage. The suggestion of a statistical adaptive analysis has merit for dealing with the great variability in the electroencephalograph (EEG). Unfortunately, the history of EEG analysis during anesthesia contains many optimistic reports of new analysis techniques that subsequently proved less valuable than suggested by initial reports. Whether the proposed adaptive statistical approach suffers the same fate will depend upon prospective, randomized, blinded observer investigations using standard neurophysiologic tests, studies yet to be performed.

Anesthesia practice for cardiac surgery varies widely, and not all anesthesiologists eschew inhalational agents, bolus doses of opioids and sedatives, and other factors that are likely to increase EEG variability and complicate EEG interpretation. Until the implications of such variations are understood, the generalization to all patients of results derived from a restricted protocol is problematic. For the clinical anesthesiologist searching for a technique to improve patient care, such difficulties are an important disincentive to the use of EEG monitoring, and the data presented by the respondents are insufficient to alter this conclusion. While these data suggest a need for further studies, those who would engage in such research must be prepared for the effort and the difficulties.

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Potential Fresh Gas Flow Leak through Dräger Vapor 19.1 Vaporizer with Key-Index Fill Port

To the Editor.—Most newer anesthetic vaporizers are equipped with a fill and drain port. Malfunction or improper use of the fill and drain ports may allow a significant leak of anesthetic agents.1,2

The Dräger Vapor 19.1 anesthetic vaporizer equipped with a key-index fill and drain system prevents inadvertent use of the wrong anesthetic agent (fig. 1). However, under certain conditions, a significant leak can exist that may not be readily located. If the fill port lock screw is not tightly secured with the filler plug fully engaged prior to using the vaporizer, then total fresh gas flow through the anesthetic machine can leak through the vaporizer and out of the fill port. Importantly, it is inadequate to have only the filler plug in place; the fill port lock screw must be tightly secured to prevent such a leak. Closure of the fill valve has no effect on this leak.

It is our opinion the problem is one of design and therefore requires an additional step be included in testing the anesthesia circuit and breathing system. After closing the pop off valve and occluding the breathing system at the patient end, the system is filled via the O2 flush valve to 20 cmH2O pressure. Each vaporizer should be turned on to test the patency of the fill port filler plug and fill port lock screw apparatus. If this apparatus is not properly secured, then the 20 cmH2O pressure within the system rapidly falls and there is a detectable odor of the agent. Although the key-index fill and drain system prevents inadvertent filling with the wrong anesthetic agent, unless the fill port filler plug and fill port lock screw apparatus are properly secured and tested with the vaporizer turned on, a clinically significant leak may exist.

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