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Duplication of Technique

To the Editor:—The recent article by Bourke *et al.*[1] attributed to them a technique previously described in a paper from Memorial Sloan-Kettering Cancer Center.[2] In that paper we stated that the technique, "is indicated whenever topical anesthesia of the airway is appropriate."[2]

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In reply:—We apologize to Dr. Rooney for failing to discover her article in our literature search and consequently not acknowledging it.[1] Hopefully, between the two articles,[1,2] more anesthesiologists and patients will benefit from this valuable technique.

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Are Recommendations Regarding Barbiturate Protection during Bypass Justified?

To the Editor:—We congratulate Dr. Nussmeier and her colleagues on their fine work demonstrating the usefulness of barbiturates in providing cerebral protection during cardiopulmonary bypass (CPB) in patients in whom a heart chamber has been opened.[1] Furthermore, we applaud Dr. Michenfelder's candor regarding the final clinical recommendations stemming from the study of Nussmeier *et al.*[2] However, the recommendations ultimately put forth by the authors and summarized by Dr. Michenfelder, *i.e.*, "that this 'proven' therapeutic intervention not be denied patients undergoing cardiopulmonary bypass procedures requiring an open ventricle," may be inappropriate in light of some of the design elements of the study.

Of particular concern is that management of CPB may have been suboptimal with respect to protection of the brain from focal ischemic damage. The authors plainly state that arterial filters and membrane oxygenators were not used, that bypass was carried out under essentially normothermic conditions, and that a glucose-containing solution was used in the pump prime. While these practices

may be standard in their institution, they are by no means universal. There is evidence to suggest that the interposition of a micropore filter on the arterial line of the pump may reduce gross morphologic abnormalities of the brain induced by CPB.[3] The use of membrane oxygenators may be associated with a reduction of particulate emboli and perhaps with fewer neuropsychiatric sequelae than bubble oxygenators.[4,5] Hypothermia has been demonstrated to protect the brain in numerous models of global or focal ischemia.[6] The use of glucose-containing solutions during bypass may elevate blood glucose, and elevations of blood glucose may exacerbate focal neurologic insults.[7,8] These points were either not addressed by the authors or, with respect to hypothermia, were dismissed as not producing cerebral protection relevant to the circumstances of open heart surgery. With respect to the latter, the authors argued that the patient is hypothermic only during CPB and that the events responsible for the production of focal cerebral ischemia probably occur either at the time of cannulation or at the end of bypass when the patient is normothermic. However, this would appear to contradict the strong, positive correlation between total CPB time and postoperative neurologic dysfunction that is nicely demonstrated by the authors' data as well as those of others.

Dr. Michenfelder's editorial did not discuss these potentially confounding factors. Could it be that the combined use of arterial filtering, membrane oxygenation, a nonglucose-containing pump prime, and hypothermia would have lowered the overall incidence of neurologic sequelae in the control group to the point where the potential benefit of barbiturates might be reduced to a level that no longer justifies the added hemodynamic risk? It is difficult to compare the control data of Nussmeier *et al.* with those reported by other investigators. The incidence of neurologic dysfunction following CPB when a cardiac chamber has been entered differs greatly among institutions, depending on the methods used to identify dysfunction and the type of analyses performed, *e.g.*, prospective *versus* retrospective.[9] However, their stroke rates appear well within acceptable limits, although this may relate to their comparatively short total CPB times and/or surgical practices.

We do not dispute the apparently valid conclusions of Nussmeier *et al.* that barbiturate protection during CPB may be entirely reasonable in the circumstance of their local clinical practices. However, it may be premature to recommend a change in clinical practice of cardiovascular teams that currently manage CPB in a manner which differs substantially from that of Nussmeier *et al.* We eagerly await the results of other well-controlled studies from large centers concerning barbiturate protection during CPB.

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