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Hypothermia and the Electroencephalogram

To the Editor:—In their article on hypothermia and thiopental, Quasha *et al.*[1] observed burst-suppression in their control group, and attributed this EEG finding to the hypothermia common to all groups. This is remarkable, because neither our clinical experience nor the published reports of others[2,3] supports the observation that moderate hypothermia (25–30° C) *by itself* produces prominent burst-suppression. Their study was performed at the start of cardiopulmonary bypass, a period when many physiologic changes are being imposed upon the previously stable cerebral conditions. Included are thermal gradients (not just hypothermia) and acute hemodilution, with associated changes in serum proteins, electrolytes, glucose, osmolarity, and blood viscosity and oxygen-carrying capacity. Halothane was administered during the study period to maintain anesthetic level; however, the anesthetic potency of this agent doubles when the temperature is reduced to 27° C.[4] Thus, an anesthetic steady state may not have been present. The presence of an unusual, and possibly abnormal, control state raises questions about the general applicability of their conclusions. Accordingly, studies under stable hypothermic conditions seem to be indicated before these conclusions (no matter how reasonable) can be accepted.

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In reply:—Dr. Warren Levy's letter raises a number of important questions and indirectly suggests likely answers. First, as Dr. Levy indicated, it is known that hypothermia significantly increases the potency of halothane. As noted, this, plus the various transient changes that occur at the start of cardiopulmonary bypass, may well explain why we observed some transient burst suppression in our control group at moderate hypothermia (25–30° C) that by itself may not produce prominent burst suppression. Further, since hypothermia has been demonstrated to potentiate the effect of halothane, it should not be too surprising that it likely will also potentiate the effects of thiopental, which is the central point of our paper. Nonetheless, we appreciate the possible confounding effect that can be introduced by the various uncontrolled transient changes in numerous variables at the start of cardiopulmonary bypass. Therefore, although we consider our current conclusions as reasonable, we fully recognize them as tentative and in need of further verification based on more controlled studies, including basic animal studies, as well as clinical trials.