Letters to the Editor

Measuring Cerebral Autoregulation in Stroke Patients

To the Editor:

We have read with interest the article by Georgiadis et al in the December 2002 issue of Stroke.1 The authors have attempted to examine the integrity of cerebral autoregulation in hypothermic stroke patients. We would like to raise 2 methodological issues.

Despite measuring intracranial pressure, the authors have used mean arterial pressure rather than cerebral perfusion pressure to calculate the static rate of autoregulation in their patients. In patients with disturbed autoregulation, intracranial pressure will rise in response to an increase in mean arterial pressure and cerebral perfusion pressure will therefore change less than mean arterial pressure; this phenomenon is known as “false autoregulation” and has been recently reported as occurring frequently, at least in head-injured patients.2 Using mean arterial pressure instead of cerebral perfusion pressure may therefore lead to an overestimation of the static rate of autoregulation, ie, the patient seems to have better cerebral perfusion pressure may therefore lead to an overestimation of the static rate of autoregulation, definition of autoregulation. This contrasts with previous work showing that healthy normal volunteers typically have a static rate of autoregulation >85%.4–6 Admittedly these studies were performed under propofol anesthesia, whereas the patients in the authors’ study were sedated with midazolam. However, both of these drugs have cerebral vasoconstricting properties7 and it is unlikely that there are large differences between them in terms of their effects on autoregulation. Autoregulation should probably be graded, rather than treated as a yes-or-no phenomenon; therefore, comparison to a control group of “normals” is perhaps more appropriate than defining dysautoregulation in terms of an arbitrary threshold.

It is unclear why the authors expect autoregulation to fail in hypothermia. Theoretically, hypothermia should lead to an increase in the static rate of autoregulation as vasoconstriction results, even more so when alpha-stat management is used and PaCO₂ is kept constant.8 We have attempted to use the data given in the table to calculate the influence of hypothermia on static rate of autoregulation. However, the number of typographical mistakes made this methodologically impossible; for example, the calculated percentage changes in mean arterial pressure did not always tally with the mean arterial pressure measurements (this was especially so for patients 12 and 13). There were also discrepancies in the article over the vasoactive agent being used to increase mean arterial pressure, with both norepinephrine and epinephrine being mentioned. As these agents have varying adrenergic receptor efficacy and therefore potentially differing effects on cerebral blood flow and metabolism, it is important that they were not used interchangeably.

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Response

We would like to thank Drs Johnston and Czosnyka for their interest in our article. Here we respond to the issues raised.

(1) We agree that changes in arterial pressure could theoretically result in elevations in intracranial pressure, thus decreasing the actual cerebral perfusion pressure. Still, intracranial pressure was invasively monitored in all patients examined in this study, and no such elevations were observed, apart from the usual fluctuations (±2 mm Hg), so that this pathophysiological consideration did not alter our results.

(2) Cut-off for cerebral autoregulation: The >40% threshold was derived from the article of Tiecks et al.,1 in which static cerebral autoregulation (sCA) values as low as 42% were observed in patients without cerebrovascular or cardiac disease during elective orthopedic surgery under propofol anesthesia. Still, our conclusion that moderate hypothermia does not influence sCA was not based solely on the observation that all values measured were above this threshold, but rather on the fact that no significant differences or even a trend were observed between sCA values obtained under hypo- and normothermia in the same patients. We do not believe that a group of normals would in any way be appropriate as control group for mechanically ventilated, heavily sedated patients with severe cerebral infarction, as suggested by the authors. Moreover, as they point out, such control values are already available in the literature.

(3) Johnston and Czosnyka question the rationale of this study, stating that hypothermia results in vasoconstriction, so that sCA should theoretically increase. This statement is not entirely correct for a variety of reasons: (a) The only data currently available on this issue were derived from in vitro studies in isolated arteries; their applicability in humans in an in vivo situation should not be taken for granted. (b) We did not observe any changes in the flow velocities of the MCA when comparing baseline values to those obtained when target temperature was reached; furthermore, we never observed MCA velocity changes using continuous TCD-monitoring during hypothermia induction (D. Georgiadis, MD, et al, unpublished data). These findings dispute the presence of hypothermia-induced vasoconstriction. (c) Significant decreases of the cerebral autoregulation index were described by Doering et al in association with minimal (0.3°C) decreases in body temperature,2 suggesting that hypothermia does indeed decrease autoregulation, despite theoretical pathophysiological considerations.

(4) Typographical mistakes in the table: these do not especially concern patients 12 and 13, but solely patients 12 and 13. A number in the MAP values was erroneously duplicated, so that the values for baseline MAP and MAP after norepinephrine dosage was increased read 58/85, 68/85, 72/90, 74/93, 69/91, 92/84, 85/115, and 95/100, instead of 58/85, 68/90, 72/93, 74/91, 69/84, 92/115, 85/100, and 95/128. We are sorry for this mistake; the same is true for the vasoactive agents, where norepinephrine was the drug used in all cases.

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