Different Levels of Ventilation Are a Plausible Explanation for Different Outcomes of Acute Stroke Patients Undergoing Endovascular Therapy

To the Editor:
I read with interest the report of Davis et al.1 and the accompanying editorial,2 which describe and discuss the observation that the outcome of endovascular therapy for acute stroke is much worse when accompanied by general anesthesia compared with local anesthesia with sedation. The systolic blood pressures were higher in the sedated patients, and it was suggested that adequate blood pressure control could ameliorate the outcomes observed in the general anesthesia group.

Although I agree that adequate blood pressure control is always to be recommended, I wish to propose that there was another important difference between the two groups that was not addressed in these two reports and could well be significant in contributing to the outcome of the two groups: the partial pressure of arterial carbon dioxide (PaCO2).

The PaCO2 of the sedated patients would have been greater than normal because of (hopefully mild) respiratory depression, whereas the PaCO2 would have been lower than normal in the patients during general anesthesia because patients are traditionally hyperventilated, especially in neurosurgical cases. In response to PaCO2, there would be cerebral vasodilatation in the sedated, hypercarbic group with spontaneous ventilation and cerebral vasodilatation in the anesthetized, hypocarbic with controlled ventilation.

It has been shown that hyperventilation and hypocapnia in head-injured patients result in poor clinical outcome.3 Similarly, it is quite plausible that hyperventilation is detrimental to patients with acute stroke. In fact, the report by Davis et al. could be interpreted as showing that hypercarbia might have a salutary effect on the outcome for these patients.

It is unlikely that arterial blood gases were measured often enough in this retrospective study for meaningful comparisons between the groups. However, prospective studies could be designed to compare the effect of different levels of ventilation on the outcome of acute stroke patients requiring general anesthesia for endovascular therapy.

Until the results of such a study become available, I suggest that the difference in outcome between the two groups of patients (local anesthesia with sedation vs. general anesthesia) could, at least partially, be explained by the difference in PaCO2 between the two groups, and therefore should have been discussed in the article and editorial.

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References

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The Time When Hypotension Occurs May Be Important in the Management of Intraarterial Thrombolysis for Stroke

To the Editor:
The contributions of general anesthesia and blood pressure management, either independently or together, to adverse outcome from intraarterial thrombolysis for stroke may be important. Davis et al. have attempted to study this through a retrospective analysis of their patient experience.1 They concluded “that patients managed with general anesthesia, and its concomitant relative systolic hypotension, during endovascular therapy for acute ischemic stroke have a much lower likelihood of good neurologic outcome, compared to patients managed with local anesthesia.” We note, however, that the lowest average systolic blood pressure in the general anesthesia group, 104 ± 17 mmHg, is exactly the same as the baseline systolic blood pressure. Baseline measurements are usually those made at some time-point before the initiation of general anesthesia, e.g., in the emergency department, the preoperative area, or the first blood pressure on arrival in the operating room.
We don’t dispute the potential detrimental effect of low blood pressure, but if the lowest blood pressure was indeed the baseline value, *i.e.*, before anesthesia, then the conclusion needs to focus not just on intraprocedural blood pressure but on the contributions of hypotension from the onset of the stroke, emergency medical services care, and management in the emergency department. These may be longer periods of hypotension than the actual procedure.

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Reference


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In Reply:

We thank Drs. Williams, Gelb, and Talke for their interest and comments on our paper.1

Dr. Williams has highlighted a potential contributor to secondary brain injury that we were unable to control for in our retrospective study. Both hypocapnia and hypercapnia have plausible mechanisms for worsening blood flow to the critically ischemic penumbra. Hypocapnia may result in further cerebral vasoconstriction and, as Dr. Williams has pointed out, may be associated with poor outcome after head trauma. We are aware that studies have shown that hypercapnia may have a neuroprotective effect after ischemia in immature animals, but we are not aware of any clinical evidence in humans to support this finding. The proposed mechanism is that of improved collateral flow due to vasodilation; however, a consequence of vasodilation may ultimately be brain edema and increases to intracranial pressure. Normocapnia is probably a safe goal at this time. Unfortunately, we did not have periprocedural blood-gas tensions available to us but we acknowledge the importance of this information.

We agree with Drs. Gelb and Talke that blood pressure management throughout the precanalization period is likely to be a critical issue. Our interventional team is currently trying to develop institutional guidelines for management of blood pressure in this setting, because current national guidelines are not particularly helpful for this group of patients.

We must apologize for the title of table 1; the use of ‘Baseline’ is misleading—it does not apply to the blood pressure measurements. In this article we did not report any ‘baseline’ (preintervention) blood pressure values. The values in table 1 were those obtained during the procedure—the same values that were reported in the ‘Results’ section (page 400). We did not attempt to define a ‘baseline blood pressure value,’ for the reasons that are outlined in the discussion (page 403, top).

This confusion generated by the misleading title does not detract from the justified concern of Drs. Gelb and Talke that blood pressure management may be important in all phases of acute stroke treatment.

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Reference


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In Reply:

We would like to thank Dr. Williams for responding to our editorial,1 which appeared in the February 2012 issue of ANESTHESIOLOGY.

Dr. Williams makes the point that patients sedated for endovascular treatment of acute ischemia may have higher arterial carbon dioxide partial pressure levels than patients treated for the same problem but receiving a general anesthetic. There are two points to this argument. The first is that patients receiving general anesthesia have a lower arterial carbon dioxide partial pressure than patients sedated without a general anesthetic. The second is that vasodilation from re- tention of carbon dioxide in the sedated patients will dilate the cerebral vasculature and protect penumbral areas by that mechanism.

First, with a general anesthetic the partial pressure of carbon dioxide can be regulated to whatever level is required. It is incorrect to assume that the patient will be hyperventilated and thereby have a lower carbon dioxide partial pressure than will be achieved without intubation, although that may be the case if the anesthesiologist hyperventilates the patient. Dr. Williams is also correct that sedation may cause the patient to hypoventilate and retain carbon dioxide.

Second, it is assumed that ischemic cerebral regions dilate anyway. Cerebral blood flow is probably pressure dependent in the penumbra. The issue of where to keep the partial pressure of carbon dioxide has been discussed extensively in management of patients receiving carotid endarterectomy under general anesthesia.2–4 What was found was that it was difficult to predict the effect of dilating or constricting the surrounding healthy tissue on the ischemic cerebral areas. If you increase the partial pressure of carbon dioxide, dilate the noninvolved cerebral areas, you may shunt blood to normal brain tissues.