The following is the abstract of the article discussed in the subsequent letter:

**Bergersen TK, Hartgill TW, and Pirhonen J.** Cerebrovascular response to normal pregnancy: a longitudinal study. *Am J Physiol Heart Circ Physiol* 290: H1856–H1861, 2006.—We used a longitudinal study design (gestational weeks 8, 15, 22, 29, and 36 and 12 wk postpartum) to investigate the effect of normal pregnancy on cerebral autoregulation and pressor response. Blood flow velocities in the right internal carotid artery, end-tidal CO₂, and mean arterial pressure (MAP) were simultaneously and continuously recorded in 16 healthy pregnant women during standardized hyperventilation and handgrip. Blood flow velocities were recorded using Doppler ultrasound sampled beat by beat using the ECG signal. The results demonstrate that the vasoconstrictor response to hyperventilation is unchanged during pregnancy. During standardized handgrip, MAP showed a statistically significant increase during pregnancy that did not affect cerebral blood flow. A statistically significant reduction in the MAP response to handgrip was seen in week 36. In conclusion, pregnancy has no impact on cerebral autoregulation. There is an impact on the pressor response resulting in a blunted reaction at week 36, probably caused by a fall in the baroreflex set point.

**Cerebral Autoregulation and CO₂ Responsiveness of the Brain**

*To the Editor:* Bergersen et al. (1) describe the effect of normal pregnancy, from 8 wk gestation to 3 mo postpartum, on the pressor response to static handgrip and changes in PCO₂ for evaluation of cerebral autoregulation. Changes in carotid artery blood velocity (FV) are related to mean arterial pressure and end-tidal PCO₂ (PETCO₂), but to manipulate PETCO₂ to gauge cerebral autoregulation is problematic. The tendency of cerebral blood flow (CBF) to remain constant over a range of systemic blood pressures is termed cerebral autoregulation, for which both local mechanisms and autonomic control participate. Forbes (2) demonstrated that pial vessels of the cat are under both vasomotor and chemical control (4). The cerebrovascular responsiveness to arterial PCO₂ or PETCO₂ (3), as estimated by CBF or FV, operates independently from cerebral autoregulation and is known as the CO₂ reactivity of the brain. Cerebral autoregulation and its CO₂ reactivity are two distinct mechanisms that interact in a complex way (5, 6). For example, the limits of arterial blood pressure within which cerebral autoregulation operates are modified by the PCO₂ (7). What Bergersen et al. (1) show elegantly is not that cerebral autoregulation but that cerebral CO₂ reactivity is preserved during and after pregnancy.

**REFERENCES**