functions, particularly at the level of the spinal cord, suggests that the time has come to stop using the term ‘DoA monitor’ to indicate a device for solely monitoring cortical functions.

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Hypothermia and cerebrovascular reactivity

Editor—Lavinio and colleagues1 have touched upon an important topic by suggesting that temperatures above 37°C may cause hyperaemic derangement of cerebrovascular reactivity in severely head-injured patients. However, one of the major limitations of their retrospective analysis lies in the fact that the effects of temperature could not be separated from any temporal changes that occurred in the brain after injury. Rewarming took place at a time when inflammatory responses in the brain presumably had evolved significantly compared with when the patients were cooled.2 Indeed, the authors acknowledge that the fever and loss of autoregulation observed in their patients may both, in fact, be epiphenomena of inflammation and suggest that prospective studies be performed. In this respect, I would like to draw attention to a study that may provide some additional insight into this matter.

In 2004, we reported on the use of systemic hyperthermia as an experimental therapy for patients with chronic hepatitis C virus infection, who are non-responders to conventional treatment with interferon.3 During 23 experiments at 41.8°C, we continuously measured cerebral oxygen extraction and middle cerebral artery blood flow velocity in patients. At regular temperature intervals, we formally assessed cerebral pressure-flow autoregulation by static tests using phenylephrine infusion, and by measuring the transient hyperaemic response to carotid compression and release. Our data showed that temperatures exceeding 40°C were associated with a marked decrease in cerebral oxygen extraction, a proportional increase in cerebral blood flow velocity, and a transient impairment of pressure-flow autoregulation. These associations remained after multivariate adjustment for variations in arterial partial pressure of carbon dioxide, mean arterial pressure, and propofol blood concentration.

The current findings by Lavinio and colleagues1 seem to confirm these earlier observations of transient cerebral vasoparalysis and hyperaemia during profound hyperthermia in patients with normal brains. However, their data suggest that similar effects become apparent at much lower temperatures in patients with head trauma, possibly reflecting an increased vulnerability of the injured brain to the deleterious effects of heat.

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Editor—we thank Dr Cremer for his comments. Cremer and colleagues3 previously demonstrated that systemic temperature exceeding 40°C is associated with partial vasoparalysis of cerebral vessels in normal brains. Our findings suggest that a similar scenario may happen in head-injured patients exposed to moderate hyperthermia for refractory intracranial hypertension.1

Cremer highlights that in our series of brain-injured patients, the hyperaemic derangement of cerebrovascular reactivity became apparent as soon as rewarming from moderate hypothermia exceeded the 37°C brain temperature threshold, reflecting a significantly increased vulnerability of cerebrovascular reactivity of the injured brain. We also emphasize that this phenomenon is commonly undetected by plain intracranial pressure (ICP) monitoring, which lacks explicit information about cerebral vasoreactivity. In this regard, a steady ICP after rewarming from moderate hypothermia is often falsely reassuring, inducing suboptimal control of systemic temperature in brain-injured patients.
To provide further insight into this topic, we would like to share our unpublished observations regarding a series of 15 patients with head injury, matched for age and severity of injury to the study population but never exposed to brain temperatures lower than 35°C. Although the cerebral pressure reactivity index (PRx) was demonstrated to be linearly related to brain temperature in the temperature range of 37–39°C in patients treated with moderate hypothermia (R=0.53; n=17, P=0.03), in this second group of patients, we did not observe any significant relationship between brain temperature and PRx (n=15, P=0.83). It must be noted that average PbCO2 was similar in the two groups [PbCO2 mean difference=2 mm Hg (0.21 kPa); P=0.36]. Similarly, PbO2 in patients treated with moderate hypothermia [PbO2=17 (10) mm Hg; 2.3 (1.4) kPa] was comparable with PbO2 in the group of patients not exposed to hypothermia [PbO2=20 (11) mm Hg; 2.6 (1.5) kPa, P=0.45]. However, although average PRx significantly correlated with PbO2 in patients exposed to moderate hypothermia (R=0.66; n=17, P=0.004), in the same temperature range there was no significant relationship between PRx and PbO2 in the group of patients who were not actively cooled (n=15, P=0.78). We also investigated the relationship between PRx and brain temperature within all individual patients. In patients exposed to hypothermia, PRx and brain temperature are positively correlated in 16 (67%) of 24 cases [median R=0.85; IQR (0.80–0.88)], whereas in controls a positive correlation between PRx and brain temperature was observed only in four (26%) out of 15 patients [median R=0.00; IQR (-0.88 to –0.87); P<0.01]. In summary, temperature-dependent hyperaemic derangement of cerebrovascular reactivity seems to be a phenomenon specifically related to therapeutic hypothermia for refractory intracranial hypertension. However, it must also be emphasized that none of our patients was exposed to temperatures exceeding 40°C. Therefore, our findings do not contradict Cremer’s experimental observations.

In conclusion, the exposure to moderate hypothermia seems to play an independent role in determining the vulnerability of cerebral vessels to rewarming in brain-injured patients.

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Regional anaesthesia in patients treated with aspirin and clopidogrel

Editor—I read the recent review on coronary artery stents and non-cardiac surgery with interest. Howard-Alpe and colleagues refer to the difficult clinical situation in which an anaesthetist wishes to perform a central neuroaxial block on a patient treated with antiplatelet therapy. The authors suggest preoperative platelet transfusion ‘if regional neuroaxial blockade is thought to be essential’ for emergency surgery. I wish to highlight three practical difficulties in transfusing platelets in order to allow a central neuroaxial block to be performed.

First, how many pools of platelets should be transfused? The authors cite French guidelines from 2003, which refer to platelet count. However, it is likely that patients on antiplatelet therapy will have a platelet count of >100 000 μl–1 and the platelet count tells us little about platelet function. A recent healthy volunteer study suggests that at least two to three pools of platelets may be required to normalize platelet function after clopidogrel and aspirin administration.

Secondly, how can platelet function be monitored after platelet transfusion to decide that a block may be safely performed? None of the platelet function tests described in the review will exclude the possibility of the very rare complication of haematoma after an epidural or spinal block. The platelet count may be normal despite abnormal platelet function, as demonstrated in pre-eclampsia. A case report of spinal anaesthesia in a patient taking clopidogrel and aspirin describes the use of platelet aggregometry to monitor the effect of platelet transfusion. However, as Howard-Alpe and colleagues state, this technique is laboratory-based and therefore may be unavailable to the clinician.

Finally, is the risk of platelet transfusion before central neuroaxial block offset by the perceived benefits of the block to the patient? Platelet transfusion is not without risk, including administration errors, and bacterial contamination of platelets.

Until anaesthetists have further data to support the safety (or otherwise) of epidural and spinal anaesthesia in patients taking both clopidogrel and aspirin, it is likely that those patients in ‘whom regional neuroaxial blockade is thought to be essential’ will be confined to a small group, such as those awaiting lung transplantation. 

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Editor—I would like to thank Dr Self for his interesting and informative letter. In our review article, we highlighted the difficulties and risks associated with