
Sir,—In commenting on our paper [1], Kardos, Rudas and Simon described their own use of the analysis of short-term heart rate variability to assess the effect of varying posture on cardiac autonomic control. Essentially they have confirmed that head-up tilt causes sympathetic activation (as evidenced by an increase in the low frequency variability) in comparison with supine and head-down positions. This phenomenon is well described [2, 3]. Their letter also indicates that increased parasympathetic tone was found when their subjects went from head-up tilt (+70°) to head-down tilt (–30°). However, they give no indication of any comparison between the supine and head-down positions, which is the finding in which we were interested. Without this comparison we cannot comment further on their letter as their results seem consistent with our own.

We would be very happy to reply to the letter of Kardos, Rudas and Simon if a statistical comparison can be given between the flat supine position and head-down tilt (with exclusion of order bias).

D. C. Galletty
G. J. McHugh
*Department of Anaesthesia*
*Wellington Hospital*
*Wellington South, New Zealand*


**Post-reperfusion coagulopathy**

Sir,—The case reports of Bayly and Thick [1] on reversal of post-reperfusion coagulopathy by protamine sulphate 0.75 mg kg−1 in the presence of a “straight line” thrombelastogram, 5 min after reperfusion of the new liver; an empirical dose was used because of the fact that the patients were still receiving veno-venous bypass without the use of therapeutic anticoagulation or heparin bonded tubing.

In each case, coagulation was re-established with a minimally prolonged reaction time. However, in each case, fibrinolysis was apparent and the resultant thrombelastogram produced the “Taj Mahal” pattern. The combination of reformation of coagulation following administration of protamine and the more usual presence of a prolonged reaction time after reperfusion led us to believe that either heparin itself or a heparin-like substance released from the newly perfused liver was responsible for the coagulopathy consistently seen after reperfusion. In support of this, the partial thromboplastin time (PTT) was also specifically prolonged at this stage of the procedure.

Since our more standardized use of aprotinin (a bolus of 2 Mu followed by 50 ml per hour until after reperfusion) severe coagulopathy at this stage is now unusual, even in the “at risk” groups, namely alcoholic liver disease, long standing autoimmune disease and regrafs.

While protamine may have a place in the diagnosis of reperfusion coagulopathy, I would advise caution in its use for complete correction of a heparin-like effect. Not only is the bypass tubing at risk of clot formation, but the hepatic artery and portal vein may also be at risk of occlusion.

We accept an International Normalized Ratio (INR) of < 2 and a PTT of < 90 s at the end of the procedure in order to protect the vascular anastomoses.

J. HUGGINS
*Featherstone Department of Anaesthetics*
*Queen Elizabeth Hospital*
*Edgbaston, Birmingham*


Sir,—It is gratifying that our observations have been confirmed by others, who also appear to have reached the same conclusions on the cause of the phenomenon.

In contrast with Dr Huggins’ experience, the appearance of fibrinolysis, as detected by thrombelastography, is rare in our unit. In fact, I have seen evidence of fibrinolysis only twice in the last 35 liver transplants. In neither of the reported cases was fibrinolysis apparent, as evidenced by the thrombelastogram in figure 2. On the other hand, experience over the past year has shown that a heparin-like effect is demonstrable in up to 50% of all liver transplants, although not all of these required treatment with protamine.

I agree with Dr Huggins that severe coagulopathy after reperfusion is becoming less common, although we do not routinely use aprotinin or prophylaxis. When treating a coagulopathy, it remains important to aim to stop unacceptable bleeding, rather than to normalize numbers. Therapy should be directed at the cause of the problem: thus if a heparin-like effect is suspected, then protamine would seem to be the logical treatment. If this is used injudiciously, then there are major risks of vascular thromboses, just as if platelets or other procoagulants are used inappropriately. We do not wish to risk clot formation in the bypass tubing, and resultant emboli.

P. J. M. BAYLY
*Freeman Hospital*
*Newcastle upon Tyne*

**Hyperventilation-induced unconsciousness during labour**

Sir,—We read with interest the case report by Burden, Janke and Brighouse [1], describing the management of hyperventilation-induced unconsciousness during labour with extradural block. Although we applaud the fact that the outcome of this case was positive, we question the wisdom of initiating major conduction block in an unconscious parturient who presumably had an unprotected airway. The authors reported that at the time of arrival in the hospital, this patient was “unconscious” and scored 7 on the Glasgow coma scale. They were so sure that the patient’s problems were secondary to pain-related hyperventilation that they opted to treat her unconsciousness with an extradural anaesthetic. This patient’s principal problem was not unconsciousness, but rather the potential for aspiration pneumonitis. If she was indeed unconscious, her airway should have been the paramount consideration. Additionally, we question the advisability of initiating an extradural anaesthetic in a patient who has just lost consciousness. If her altered mental state was caused by increased intracranial pressure, a “wet-tap” could have had devastating consequences; additionally, in her comatose state, the patient could not have identified paresthesiae, and a total spinal or intravascular injection might have been misdiagnosed.

We suggest that this case report sends the wrong message to your readers. Parturients at term are known to have “full anaesthetic. This patient’s principal problem was not unconsciousness, but rather the potential for aspiration pneumonitis. If she was indeed unconscious, her airway should have been the paramount consideration. Additionally, we question the advisability of initiating an extradural anaesthetic in a patient who has just lost consciousness. If her altered mental state was caused by increased intracranial pressure, a “wet-tap” could have had devastating consequences; additionally, in her comatose state, the patient could not have identified paresthesiae, and a total spinal or intravascular injection might have been misdiagnosed.

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R. A. BOURLIER
D. J. BIRNBACK
*Department of Anaesthesiology*
*St Luke’s-Roosevelt Hospital Center*
*Columbia University College of Physicians and Surgeons*
*New York, NY, USA*