Hyperinsulinaemic normoglycaemic clamp in coronary artery surgery

Editor—We congratulate Visser and colleagues on applying glucose–insulin–potassium (GIK) therapy using a hyperinsulinaemic normoglycaemic clamp. Their study confirms our findings that the clamp technique is an effective, and to date the only, method of maintaining normoglycaemia in patients undergoing coronary artery bypass grafting surgery. In addition, they demonstrated, for the first time in this population, the attenuation of systemic inflammation with perioperative GIK therapy. More importantly, this effect was prolonged beyond the time of insulin administration providing a rationale for insulin therapy to be started before the stress of surgery and cardiopulmonary bypass (CPB).

Several issues merit further comment. It is unfortunate that when initiating the clamp the authors were unable to prevent hypoglycaemia that is, a blood glucose <3.0 mmol litre⁻¹. Perhaps, if there had been more frequent measuring of blood glucose this complication may not have occurred. In our protocol, using an insulin dose three times that has been used in the present study, we avoided hypoglycaemia in diabetic and non-diabetic patients by measuring the blood glucose every 5 min at the start of the clamp.

We agree with the authors in stressing the importance of iatrogenic hyperglycaemia resulting from exogenous glucose administration. This is true of the metabolically unsupported control group (D5W at 30 ml h⁻¹) and may also be true of the treatment or clamp group. Although not stated in the manuscript, we assume that the five patients excluded, due to ‘insufficient insulin therapy during CPB’, belonged to the treatment group. If so, we suspect these patients were rendered hyperglycaemic secondary to the administration of additional exogenous glucose, as for example by transfusing blood products. Packed red blood cells and fresh frozen plasma contain high concentrations of glucose that are typically >20 mmol litre⁻¹. In anticipation of administering additional glucose, using a higher dose of insulin and therefore a higher glucose infusion rate (GIR), as proposed in our protocol, provides a larger buffer for adjusting the GIR and maintaining normoglycaemia.

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Editor—We thank Drs Carvalho, Leung and Schricker for their comment on our study. It is true that we did not succeed in preventing hypoglycaemia, (blood glucose <3 mmol litre⁻¹) in all our patients at all times. Glucose levels between 2.2 and 3.0 mmol litre⁻¹ were found in 7 out of 175 blood samples (4%) obtained during the first 5 h of the study. Their comment that more frequent glucose measurements taken at the start of the clamp may have prevented these individual episodes of hypoglycaemia is, therefore, gratefully acknowledged. However, measuring plasma glucose at 5 min intervals, as suggested, implies the continuous use of a blood gas analyzer, equipped with a glucose electrode. This is not realistic for the routine use of GIK, as each measurement takes ~5 min. The use of a faster ‘finger-stick’ glucose measuring device, as used by Carvalho and colleagues, is, in our opinion, not acceptable as an alternative, due to the well-known inaccuracy of those devices. The future availability of continuous glucose measuring sensors can potentially solve those problems. None of our patients showed effects after operation, that could be attributed to the short episodes of hypoglycaemia. Regarding the importance of iatrogenic hyperglycaemia, there appears to be a consensus among us. In addition, we agree that glucose, which is present in packed blood cells, may contribute to elevated plasma glucose levels. However, their assumption that five GIK patients were excluded from final analysis in our study because of hyperglycaemia following CPB is not correct. Those patients were excluded due to protocol violations when the insulin and glucose infusions were discontinued by mistake at the onset of CPB.
Finally, they suggested the use of much higher infusion rates of insulin, because it would lead to an increase in the infusion rate of glucose. In contrast to their data\(^2\) (which used variable infusion rates of both insulin and glucose) we actually measured plasma insulin concentrations, which were reported to be in the supra-physiological range of 600–800 pmol litre\(^{-1}\). We are not aware of any scientific evidence suggesting that the infusion rate of insulin described in our paper was too low. On the contrary, it cannot be excluded that their higher and varying insulin infusion rates may have led to a higher and more unpredictable rate of intracellular glucose usage, thus creating the necessity to check plasma glucose more frequently. To answer this interesting suggestion, a dose–response study in cardiac surgical patients is required. Such a study should be designed to investigate the effect of different high concentrations of insulin on the metabolic rate of glucose perioperatively.

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**Laryngospasm during subarachnoid block**

**Editor—** We thank Dr Chincholkar for his interest in our case report.\(^1\) To answer the questions raised by Dr Chincholkar,\(^2\) the level of block before the dressing was opened was T11, bilaterally to sharp pain. It may have been higher, but we did not test further as the level was satisfactory for the dressing removal. Only part of the outer layer of the dressing was removed, the patient’s leg was supported at his calf and the wound was not handled at any point. Thus, it is unlikely that removal of the dressing was the cause of laryngospasm. It took at least 3 min for the laryngospasm to break, that is, not immediately after the surgeons stopped removing the dressings. The measures described in our case report namely fluid loading and atropine prevented any further development of a high parasympathetic tone and this in our opinion explains the further uneventful course of the anaesthetic.

We employed sharp pain as a modality of testing as most clinicians would do. We do agree that a simple pin prick test may not equate to the complex mechanisms involved in perception of surgical pain, but this is the most common method of testing the level of block for regional anaesthesia in most clinical settings. Temporal summation is blocked in subarachnoid blocks\(^3\) but not so well in epidural blocks.\(^4\)

We disagree that the onset of bilateral blocks in lateral position is slow. There are multiple factors governing the spread and onset of subarachnoid block.\(^5\) Dr Chincholkar quotes a study of regional anaesthesia for Caesarean section\(^6\) but the complex changes during pregnancy are not comparable to our male patient undergoing foot surgery. Instead, I would draw his attention to a study,\(^7\) where the onset of bilateral sensory block to T10 with bupivacaine 0.5% (15 mg) with glucose 8% was a median of 2 min (range 2–10 min). The dose is slightly higher than the bupivacaine 0.5% (12.5 mg) with glucose 8% that we used. In our patient, the block was performed in the right lateral position and the patient was turned supine immediately and hence it is unlikely that the block was unilateral.

We too were perplexed when confronted with laryngospasm during a spinal anaesthetic. What we did was to follow ‘ABC’ as in any emergency. Haemodynamic optimization involved bolus of intravenous fluid, atropine and ephedrine. In our opinion, the laryngospasm responded to the above measures, which were mostly vagolytic.

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**S100B in Guillain–Barré syndrome**

**Editor—** Guillain–Barré syndrome (GBS), a subacute inflammatory demyelinating polyneuropathy, is the most common cause of acute neuromuscular paralysis. Up to one-third of patients with GBS require mechanical ventilation (MV)