

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<sup>2</sup> (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<sup>-1</sup>. 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.

H. B. Van Wezel\*

C. J. Zuurbier

J. van Dijk

F. J. Hoek

E. de Jonge

B. A. de Mol

Amsterdam, The Netherlands

\*E-mail: H.B.vanwezel@amc.uva.nl

1 Visser L, Zuurbier CJ, Hoek FJ, et al. Glucose, insulin and potassium applied as perioperative hyperinsulinaemic normoglycaemic clamp: effects on inflammatory response during coronary artery surgery. *Br J Anaesth* 2005; **95**: 448–57

2 Carvalho G, Moore A, Qizilbash B, Lachapelle K, Schrickler T. Maintenance of normoglycemia during cardiac surgery. *Anesth Analg* 2004; **99**: 319–24

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### Laryngospasm during subarachnoid block

Editor—We thank Dr Chincholkar for his interest in our case report.<sup>1</sup> To answer the questions raised by Dr Chincholkar,<sup>2</sup> 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<sup>3</sup> but not so well in epidural blocks.<sup>4</sup>

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.<sup>5</sup> Dr Chincholkar quotes a study of regional anaesthesia for Caesarean section<sup>6</sup> 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,<sup>7</sup> 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.

K. Subramani

Weston-Super-Mare, UK

E-mail: ksubramani9@hotmail.com

1 Subramani K, Paul A. Laryngospasm during subarachnoid block. *Br J Anaesth* 2005; **94**: 668–70

2 Chincholkar MG. Laryngospasm during subarachnoid block. *Br J Anaesth* 2005; **95**: 277

3 Curatolo M, Petersen-Felix S, Arendt-Nielsen L, Zbinden AM. Spinal anaesthesia inhibits central temporal summation. *Br J Anaesth* 1997; **78**: 88–9

4 Curatolo M, Petersen-Felix S, Arendt-Nielsen L, Fischer M, Zbinden AM. Temporal summation during extradural anaesthesia. *Br J Anaesth* 1995; **75**: 634–5

5 Hocking G, Wildsmith JA. Intrathecal drug spread. *Br J Anaesth* 2004; **93**: 568–78

6 Lewis NL, Ritchie EL, Downer JP, Nel MR. Left lateral vs. supine, wedged position for development of block after combined spinal-epidural anaesthesia for Caesarean section. *Anaesthesia* 2004; **59**: 894–8

7 Whiteside JB, Burke D, Wildsmith JAW. Comparison of ropivacaine 0.5% (in glucose 5%) with bupivacaine 0.5% (in glucose 8%) for spinal anaesthesia for elective surgery. *Br J Anaesth* 2003; **90**: 304–8

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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)

in the ICU,<sup>1</sup> although outcome is generally good,<sup>2</sup> acute mortality remains relatively high and ~20% of hospitalized patients may have a long-term disability.<sup>3</sup>

Serum protein S100B is a recognized marker of traumatic brain injury. We observed an increase in S100B levels in two GBS patients, pointing to a potential acute influence of peripheral neuropathy on levels not related to CNS injury.

Serum S100B was measured from venous blood samples in two GBS patients requiring MV, on admission to ICU and after 10 days. S100B values  $>0.2 \mu\text{g litre}^{-1}$  are considered abnormal. The patients were examined by a neurologist and the diagnosis was based on the National Institute of Neurological and Communicative Disorders and Stroke diagnostic criteria.<sup>4</sup>

The first patient was treated with a total of 10 plasma exchanges and steroids and required 13 days of MV. The patient was discharged from hospital at 40 days with severely limited lower limb activity and confined to bed. On admission, the S100 serum level was raised at  $0.495 \mu\text{g litre}^{-1}$ , and at 10 days the level had reduced to  $0.093 \mu\text{g litre}^{-1}$ .

The second patient was treated with plasma exchange and steroids. The patient recovered spontaneous ventilation after 62 days and was discharged, still confined to a chair, 90 days after SGB onset. On ICU admission, the S100B serum level was very high ( $2.61 \mu\text{g litre}^{-1}$ ), and was still abnormal 10 days later ( $0.891 \mu\text{g litre}^{-1}$ ).

The serum S100B increases observed in these two GBS patients exceed those attributed to brain tissue damage patients with mild traumatic head injury.<sup>5</sup> The physiological role of this neuroprotein is not yet clear, but in GBS it may act as a regeneration stimulus or as marker of neuronal damage. A comparison of CSF and serum levels of S100B

would be useful for the understanding of the role played by this protein in GBS. S100B in GBS might be an expression of neurotrophic and neuroprotective activity but no conclusion should be made without extensive and focused clinical and experimental studies.

O. Piazza\*  
G. Esposito  
E. De Robertis  
G. Servillo  
R. Tufano  
Naples, Italy  
\*E-mail: orpiazza@unina.it

- 1 Sharshar T, Chevret S, Bourdain F, Raphaël J. Early predictors of mechanical ventilation in Guillain-Barré syndrome. *Crit Care Med* 2003; **31**: 278–83
- 2 Henderson RD, Lawn ND, Fletcher DD, McClelland RL, Wijdicks EF. The morbidity of Guillain-Barré syndrome admitted to the intensive care unit. *Neurology* 2003; **60**: 17–20
- 3 Dornonville de la Cour C, Andersen H, Stalberg E, Fuglsang-Frederiksen A, Jakobsen J. Electrophysiological signs of permanent axonal loss in a follow-up study of patients with Guillain-Barré syndrome. *Muscle Nerve* 2005; **31**:70–7
- 4 National Institute of Neurological and Communicative Disorders and Stroke ad hoc Committee (NINCDS). Criteria for diagnosis of Guillain-Barré syndrome. *Ann Neurol* 1978; **3**: 565–6
- 5 Ingebrigtsen T, Waterloo K, Jacobsen EA, Langbakk B, Romner B. Traumatic brain damage in minor head injury: relation of serum S-100 protein measurements to magnetic resonance imaging and neurobehavioral outcome. *Neurosurgery* 1999; **45**: 468–75

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