We are aware of strategies that clinicians have developed to enable them to use open TCI systems with the Schnider model for TCI in morbidly obese patients. Although we do not recommend these practices, we feel that it is important to mention this issue, to illustrate the inherent and significant dangers. One option for clinicians who wish to use the Schnider model in morbidly obese patients is to input a ‘corrected’ TBW value—typically the user will input the maximum TBW value that the system will allow for a patient of that height (i.e. a falsely low TBW is used). As illustrated by the dotted lines in Figure 2A and B, the result of this strategy is that all morbidly obese patients of a given height and gender will be assigned the same TBW, LBM, and $k_{10}$ values, and all will thus receive the same amount of propofol for a given target concentration profile. By fixing $k_{10}$ this strategy fixes the only Schnider model parameter that usually varies with weight (the other parameters with co-variates are the fast re-distribution rate constants which vary with age). Thus, anaesthetists who use the Schnider model in morbidly obese patients should know that by inputting falsely low TBW values, they have generated a model where the infusion rates no longer scale according to the patient weight. Since propofol is a highly lipid-soluble agent, maintenance doses are likely to be related to TBW. There is thus a real danger that with this strategy insufficient maintenance doses will be administered to morbidly obese patients. At present, clinicians who wish to administer i.v. anaesthesia to morbidly obese patients are ‘between the devil and the deep blue sea’ (we promise not to re-invoke the satanic analogy!). Our personal view is that with current evidence and knowledge, the most prudent approach is to manually administer an induction bolus based on an estimate of LBM, and thereafter to administer a manually controlled infusion, carefully titrated to clinical effect.

We have focused on obese patients, but there are several other groups who fall outside of the boundaries of the original research, such as the very young or old, and the critically ill, and in whom the utility of different models is uncertain. In his letter, Engbers has also touched on the issue of which model is best in ‘normal’ patients. The study of Glen and Servin\textsuperscript{5} which he mentions, is a valuable contribution, but cannot be regarded as conclusive evidence. A goal of the ‘WorldSIVA Open TCI’ initiative (www.opentci.org) is to attempt to answer precisely these sorts of questions, by harnessing the statistical benefits of combining data sets derived from different studies. A recent study resulting from this initiative, involving a very large data set, has shown results at odds with those of Glen and Servin.\textsuperscript{11} There is clearly some way to go yet.

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Raised triglycerides and propofol infusion in H1N1 patients

Editor—We write concerning the treatment of patients with H1N1 infection on our intensive care unit (ICU). We recently looked after three young female patients who presented with H1N1 influenza and went on to develop acute respiratory distress syndrome (ARDS). All three showed disproportionately raised serum triglycerides after receiving standard doses (up to 3.85 mg kg\textsuperscript{-1} h\textsuperscript{-1}) of propofol 1%. Serum triglycerides decreased after stopping the propofol infusion but increased later in the illness, despite no lipid containing infusion being used. None received parenteral nutrition or insulin, and none had liver or renal failure. This is not usually observed in our other ICU patients of similar age. All three patients also received oseltamivir 150 mg twice a day.

Case 1: A 23-yr-old patient who presented with an H1N1 pneumonitis, which lead to ARDS. She was sedated with propofol and alfentanil. Her baseline serum triglycerides were 2.1 mmol litre\textsuperscript{-1} and increased to 5.2 mmol litre\textsuperscript{-1}. Midazolam was substituted in place of

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propofol and 2 days later triglycerides had returned to baseline. On day 12, triglycerides had increased again to 4.6 mmol litre\(^{-1}\). She died of a pulmonary embolus.

**Case 2:** A 16-yr-old patient who presented with pneumonia after H1N1 influenza. Her serum triglycerides increased from 1.0 to 3.3 mmol litre\(^{-1}\) and reduced after stopping propofol. When it was re-introduced, her serum triglycerides increased again and correlated closely to the propofol infused, reducing after propofol was stopped. Triglycerides increased again on day 12 to 3.9 mmol litre\(^{-1}\) without any propofol infusion.

**Case 3:** A 31-yr-old patient who was 34 weeks pregnant when she contracted H1N1. After 3 days of propofol, her triglycerides had increased from 3.5 to 9.8 mmol litre\(^{-1}\) when propofol was replaced with midazolam. They gradually reduced to 4.3 mmol litre\(^{-1}\) but on day 11 they increased again to 7.3 mmol litre\(^{-1}\). She died of multi-organ failure.

As these patients did not display any features of propofol infusion syndrome,\(^1\) we think the lipid load due to propofol is not being appropriately handled by these patients which is unusual in this young population. There is no mention of triglyceride metabolism disturbance in the oseltamivir data sheet, but this does not seem to have been specifically studied. As routine measurement of triglycerides is not generally carried out in the critically ill on a daily basis, other ICU clinicians may not be aware of this phenomenon. We are not sure of the clinical significance of this observation; however, given that one of our patients died of a pulmonary embolus, we believe that the clinical community should be made aware of disturbed lipid metabolism in these patients.

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Transversus abdominis plane block: a note of caution!

Editor—There have been recent publications and an editorial in the *British Journal of Anaesthesia* regarding transversus abdominis plane (TAP) block.\(^1\)–\(^3\) I wish to sound a note of caution. A cadaver study by Rosario et al.\(^4\) examining the mechanism of femoral nerve palsy after ilio-inguinal nerve block showed that the transversalis fascia (immediately deep to transversus abdominis) is continuous posteriorly with the iliacus fascia, which is itself immediately deep to the femoral nerve. That is, the femoral nerve lies in the same tissue plane as the space deep to transversus abdominis. They demonstrated that as little as 1 ml of injectate placed between transversus abdominis and transversalis fascia tracks postero-medially to surround the femoral nerve. The needle needed to be advanced only 2–3 mm to penetrate transversus abdominis. With an injection point 3 cm medial to the anterior superior iliac spine, they measured the distance to the femoral nerve at 4.5 cm in females and 3.2 cm in males. The injection point for TAP block is more posterior and is likely to be even closer to the femoral nerve. TAP block is a new technique with no track record of complications. It is highly likely that too deep placement of even a portion of the injectate for a TAP block could cause a femoral nerve palsy. Being new to ultrasound guidance, I find it difficult to reliably identify the layers of the abdominal wall and use small volume injections to locate and determine the depth of the needle. I am unconvincod that ultrasound guidance will eliminate the possibility of this potential complication. As TAP blocks are being recommended for day-case surgery, it is imperative that the injection is performed with a high degree of accuracy and also that accidental femoral nerve palsy is reported. In our unit, an accidental femoral palsy caused a patient to fracture their ankle in the day unit when trying to mobilize after an inguinal hernia repair.

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Effect of short-term exercise training on aerobic fitness in patients with abdominal aortic aneurysms

Editor—Kothmann and colleagues\(^1\) have shown that a 6 week hospital-based exercise programme for patients under surveillance with abdominal aortic aneurysm’s