responsible as skeletal muscle heat is relatively rapidly removed through increased blood flow, with the total lower body venous return carrying a cumulative amount of heat to the liver via the inferior vena cava. I agree with the authors that rigidity develops when the intracellular calcium concentration increases to mechanical threshold, generally considered to be around 5×10^{-5} M.

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REPLY

We appreciate the comments made by G.A. Gronert about our recent study on the metabolic changes associated with a non rigid and spontaneous porcine MH crisis.

We suggested that heat production associated with this MH crisis could have been triggered by electrical stimulation or by abrupt production of catecholamines. Sympathetic arousal related to stimulation has been widely documented¹⁻⁶ and the results are still controversial. A clear statement as to whether catecholamines are primarily involved or not in the development of porcine MH crisis has not been proposed so far.

Blood gas analysis not only reflect extracellular events but also an averaged wholebody response associated with localised muscle effort. On the contrary, MRS

measurements provide direct insight into intracellular alterations in a selected organ, namely the exercising muscle under examination.

The role of temperature as a possible triggering agent of porcine MH crisis is a valid issue that we discussed. We have positioned temperature probes into the muscle and deeply into the piglet mouth and observed that muscle and central temperatures varied in the same way during the MH crisis.⁷ The primary site of heat production (muscle and/or other organs such as liver) during MH crisis is still debated.⁸⁻¹⁰ It should be kept in mind that i) skeletal muscle is the most abundant tissue in mammals, about 40% body weight and ii) intramuscular pressure of isometrically exercising muscle increases thereby reducing blood flow and probably the capacity of heat removal.¹²

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Metallic microparticles in the needle-through-needle technique

To the Editor:

Eldor attempted to lay the blame for two cases of aseptic meningitis following regional anaesthesia on metal particles that formed because of the needle-through-needle combined spinal-epidural technique.¹ He quoted our investigation of the formation of metallic particles when we passed pencil-point needles over the curved orifice of Tuohy or Hustead epidural needles² to support his argument. In fact, we found similar notching at the tip of epidural needles that Eldor reported in his original letter.³ However, he neglected to state that we did not find metal particles after needle-through-needle passage that he espouses to explain this notching defect. Our report and that of another⁴ contradict his contentions.

Until Eldor can provide substantial evidence for the existence of microparticles, beyond the ones we found on every freshly opened, unused Tuohy needle inspected in our study, he should not be given an unrefereed forum to express unsupported claims.

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REPLY

I thank Dr. Herman and his group for their continuous interest in my 1991 observation that the needle-through-needle technique produces metallic microparticles. However I need to clarify some facts related to Dr. Herman's article:

A "notching defect" is a notching defect. That they did not detect metal filings or chips in either flush solutions does not mean they were not there. They have progressed from "No metal particle contamination"^{1,2} to "No additional metal particle formation."^{1,3}

Dr. Herman is mixing two things: His "notching defect" is evidence of friction between the spinal needle and the bent epidural needle tip in the needle-through-needle technique. It is another whether these metallic microparticles cause or serve as a nidus for aseptic meningitis.

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Persistent intraoperative myoclonus during propofol-fentanyl anaesthesia

To the Editor:

A 63-yr-old otherwise healthy man presented for shoulder surgery. Three minutes after induction of anaesthesia with 1 µg·kg⁻¹ fentanyl and 2 mg·kg⁻¹ propofol, the patient developed myoclonic jerking of his legs. These spasmodic contractions persisted for the next 10 min, during which propofol was infused at 6 mg·kg⁻¹·hr⁻¹. Because there were no changes in nature of the myoclonic movements, propofol was discontinued and 10 min later, the patient was fully awake and the myoclonic movements completely disappeared.