Correspondence

Anaesthetic simulation

To the Editor:

We appreciate the editorial "Anaesthetic simulation and lessons to be learned from aviation" by Drs. Helmreich and Davies¹ published in the September issue of the Canadian Journal of Anaesthesia and offer the following comments:

Helmreich and Davies go into a lengthy discussion of content and ask: "How can the authors reassure us that these are indeed the right things to do and not just "clinically reasonable "without some form of validation, other than that described in this article?"

The concept of test validity is entirely different from test reliability and was not the purpose of our study.² Our study did not evaluate the content or the validity of the simulator testing process whatsoever. Furthermore, our scenarios were designed to address the controversial proposed role of the simulator to assess the ability of new trainees and practicing clinicians to practice clinical skills and not to evaluate Crisis Management Skills.

Helmreich and Davies describe the evaluation and assessment process in the aviation industry in detail and claim: "Devitt et al. are using a medium to high fidelity simulator to provide part-task training to individual anaesthetists. "...In contrast, true CRM addresses team-interface issues..."

Their description of the aviation industry Crew Resource Management (CRM) is interesting but bears little relevance to our published results. Our project to evaluate the simulator as a potential assessment tool is in no way connected to the ongoing Crisis Management Training Program (which actually does use teams including nurses and surgeons since 1995).³

Matt M. Kurrek MD

Hugh Devitt MD

Canadian Simulation Centre for Human
Performance and Crisis Management Training
Sunnybrook Health Science Centre
University of Toronto

REFERENCES

- 1 Helmreich RL, Davies JM. Anaesthetic simulation and lessons to be learned from aviation. *Can J Anaesth* 1997; 44: 907-12.
- 2 Devitt JH, Kurrek MM, Cohen MM, et al. Testing the raters: inter-rater reliability of standardized anaesthesia simulator performance. *Can J Anaesth* 1997; 44: 924-8.

3 Anaesthesia Patient Safety Foundation (APSF)
Newsletter Fall 1995.

(<http://gasnet.med.yale.edu/apsf/newsletter/1995/fall/simulator.html>)

REPLY

We thank Drs. Kurrek and Devitt for their response to our editorial. We feel that our editorial reflects our concern with the direction described for the use of simulators in anaesthesia.

R.L. Helmreich PhD

University of Texas Aerospace Crew Research Center
Austin, Texas, USA

J.M. Davies MD, FRCPC

Foothills Medical Centre
Calgary, Alberta

Metabolic events with spontaneous malignant hyperthermia

To the Editor:

In their very interesting paper (Kozak-Ribbens G. *et al.*, "Metabolic events with spontaneous malignant hyperthermia...", 1997; 44: 757-64), the authors describe an MH episode that begins during recovery from one of several periods of electrical stimulation, and characterise this as spontaneous MH during non-triggering anaesthesia. But, electrical stimulation *per se* does indeed trigger MH episodes in susceptible pigs during non-triggering anaesthesia.¹ Further, as with carbachol, the effect of electrical stimulation may be a direct acetylcholine-type arousal of skeletal muscle, which is exaggerated in susceptible skeletal muscle.² Sympathetic arousal related to stimulation is unlikely to be a direct factor,^{3,4} although any increase in temperature related to sympathetic factors or to triggering may help to further advance the MH event.^{2,5,6}

The authors elegantly document the intracellular acidosis associated with MH within skeletal muscle and amplify our information about the gradients between muscle and blood. As ionized lactate ion diffuses poorly out of the cell, the intracellular milieu, due to the MH episode, is more acidotic than is reflected by arterial blood gas analysis.⁷ As to hepatic temperature being greater than that in skeletal muscle, it is likely that the very high liver blood flow is, in part,

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.

Gerald A. Gronert MD
Department of Anesthesia TB 170
University of California
Davis CA 95616

REFERENCES

- 1 Ahern CP, Milde JH, Gronert GA. Electrical stimulation triggers porcine malignant hyperthermia. *Res Vet Sci* 1985; 39: 257-8.
- 2 Gronert GA, Milde JH, Taylor SR. Porcine muscle responses to carbachol, α - and β -adrenoceptor agonists, halothane or hyperthermia. *J Physiol* 1980; 307: 319-33.
- 3 Maccani RM, Wedel DJ, Hofer RE. Norepinephrine does not potentiate porcine malignant hyperthermia. *Anesth Analg* 1996; 82: 790-5.
- 4 Gronert GA, White DA. Failure of norepinephrine to initiate porcine malignant hyperthermia. *Pflug Arch* 1988; 411: 226-8.
- 5 Ording H, Hald A, Sjontoft E. Malignant hyperthermia triggered by heating in anaesthetized pigs. *Acta Anaesthesiol Scand* 1985; 29: 698-701.
- 6 Denborough MA, Hopkinson KC. Death caused by overheating in piglets susceptible to malignant hyperthermia (Letter). *Med J Aust* 1994; 160: 731-2.
- 7 Gronert GA, Ahern CP, Milde JH. Treatment of porcine malignant hyperthermia: lactate gradient from muscle to blood. *Can Anaesth Soc J* 1986; 33: 729-36.

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

G. Kozak-Ribbens A. Miri
D. Bendahan A. Talmant
L. Rodet G. Monin
S. Confort-Gouny P.J. Cozzone
Marseille, France

REFERENCES

- 1 Haggendal J, Johsson L, Carlsten J. The role of sympathetic activity in initiating malignant hyperthermia. *Acta Anaesthesiol Scand* 1990; 34: 677-82.
- 2 Haggendal J, Jonsson L, Johansson G, Bjurström S, Carlsten J. Disordered catecholamine release in pigs susceptible to malignant hyperthermia. *Pharmacol Toxicol* 1988; 63: 257-61.
- 3 Williams CH, Stubbs DH, Payne CG, Benedict JD. Role of hypertension in fulminant hyperthermia-stress syndrome. *BMJ* 1976; 1: 628.
- 4 Williams CH, Buzello W, Dozier SE, Chandra P. Hemodynamics, cardiac function, oxygen use and catecholamines during porcine malignant hyperthermia. (Abstract) *Anesth Analg* 1985; 64: 300.
- 5 Maccani RM, Wedel DJ, Hofer RE. Norepinephrine does not potentiate porcine malignant hyperthermia. *Anesth Analg* 1996; 82: 790-5.
- 6 Gronert GA, White DA. Failure of norepinephrine to initiate porcine malignant hyperthermia. *Pflug Arch* 1988; 411: 226-8.
- 7 Kozak-Ribbens G, Bendahan D, Rodet L, et al. Metabolic events with spontaneous malignant hyperthermia crisis in an anaesthetized pig. *Can J Anaesth* 1997; 44: 757-64.
- 8 Iainzo PA, Sessler DI. Skeletal muscle is responsible for heat production in porcine malignant hyperthermia - Reply. *Anesth Analg* 1996; 83: 1133-4.
- 9 Iainzo PA, Kehler CH, Zink RS, Belani KG, Sessler DI. Thermal response in acute porcine malignant hyperthermia. *Anesth Analg* 1996; 82: 782-9.