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Letter to the Editor

Cerebral microdialysis after cardiac arrest – Misinterpretations based on a misconception

To the Editor,

With interest, we read the article by Putzer *et al.* reporting on the effects of adrenaline on regional cerebral blood flow, cerebral oxygenation and cerebral metabolism during cardiopulmonary resuscitation in a porcine cardiac arrest model.[1] In their study the authors evaluate cerebral energy metabolism from intracerebral microdialysis and analysis of lactate, pyruvate and glucose.

The authors describe that after 8 min of cardiac arrest the lactate/pyruvate ratio (LPR) exhibited a non-significant increase compared with baseline: 39 [22–47] and 30 [13–38], respectively. During the subsequent extracorporeal life support (ECLS) LPR increased dramatically to reach 307 [229–445] 10 min after start of recirculation. After commencement of adrenaline administration LPR slowly decreased but mean values of lactate remained high while pyruvate and glucose were low during the following 20 min. No significant differences regarding LPR, lactate or pyruvate were obtained between animals in mean arterial pressure groups 40 mmHg and 60 mmHg. The observation that LPR remained unchanged during ischemia to increase during ECLS was explained as: "*perfusion pressure remained relatively low and presumably always below a certain critical threshold considered necessary to maintain organ blood flow, the so-called critical closing pressure*".

The changes in variables reflecting cerebral energy metabolism during complete ischemia and recirculation have been known for a long time. Within seconds after complete interruption of blood flow cerebral LPR increases to reach a maximal value after less than 10 min due to the increase in lactate and a complete lack of pyruvate.[2,3] Following adequate recirculation LPR returns to close to normal within 30–60 min while lactate and pyruvate may remain elevated longer as an indication of mitochondrial dysfunction.[4,5]

The described observation that LPR was unchanged during ischemia and increased following ECLS is due to a misconception of the microdialysis technique. With a perfusion rate of 2 μL/min the perfusate in the microdialysis probe will, depending on the length of the tubing, reach the collecting micro-vial after approximately 5–10 min. The perfusate will contain a mixture of the chemical variables collected since the previous sample. Obviously, this technical fact fully explains why biochemical signs of energy crisis was not observed during complete ischemia but increased to a maximum during the latter part of ECLS and the initial period after start of adrenalin infusion. Accordingly, it is also incorrect to use the biochemical pattern at the end of ECLS as baseline for evaluation of the effects of adrenaline infusion.

It is very likely that the mean arterial pressure (25–30 mmHg) and estimated cerebral perfusion pressure (8–13 mmHg) attained during ECLS were too low for adequate cerebral recirculation. It is also reasonable to assume that the increase in both these variables after adrenaline infusion was beneficial for cerebral energy metabolism. However, due to the misconception regarding the principles of the microdialysis technique a correct evaluation of the adrenaline effect is not obtained in this study.

Declaration of Competing Interest

The authors have no financial relationships

The authors have no Conflicts of Interest to disclose.

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