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LETTERS TO THE EDITOR AND REPLIES

Changes in cerebral blood flow and oxygen extraction during the post-resuscitation syndrome

Sir,

Anoxic neurological injury is a major cause of morbidity and mortality in cardiac arrest patients. After restoration of spontaneous circulation pathophysiological changes in cerebral perfusion occur, known as the post-resuscitation syndrome. Recently, Lemiale et al. studied cerebral blood flow and oxygen extraction in patients after cardiac arrest during mild therapeutic hypothermia.[1] In this elegant study, the authors observed low cerebral blood flow velocities during the first 24 h of hypothermia, with an increase during subsequent re-warming. These results are slightly different from our previous work in which a gradual increase in cerebral blood flow velocities was found during the first 24 h after cardiac arrest in normothermic patients.[2] The authors attribute these differences to the fact that mild therapeutic hypothermia may delay a change in blood flow velocities.

We think that another explanation may be present. It is well known that changes in arterial pCO_2 have a profound effect on cerebral blood flow. In the experiments by Lemiale et al the arterial pCO_2 decreased from 45 ± 6 mmHg on admission to 36 ± 6 and 37 ± 7 mmHg at 12 and 24 h, increasing again after 36 h.[1] We have shown previously that changes in arterial pCO_2 correlate very well with changes in flow velocities in the middle cerebral artery, indicating that CO_2 reactivity is preserved after cardiac arrest during normothermia.[3] We are currently studying the cerebral CO_2 reactivity in hypothermic patients after cardiac arrest. Preliminary results of these experiments indicate that CO_2 reactivity is also preserved during hypothermia and that blood flow velocity in the ACM actually increases within the first 24 h. Therefore, the prolonged decrease in pCO_2 compared to baseline levels may have reduced the cerebral blood flow, thus masking an increase in flow velocities as was found by Buunk et al. This precludes valid conclusions regarding blood flow velocities during hypothermia after cardiac arrest. To measure cerebral blood flow velocities after in patients after cardiac arrest treated with mild therapeutic hypothermia, pCO_2 should be kept stable.

Conflict of interest

All authors disclose that they have no financial or personal relationships with other people or organisations that could inappropriately influence their work.

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Reply to Letter: Changes in cerebral blood flow and oxygen extraction during the post-resuscitation syndrome

Sir,

We thank Bisschops, Hoedemaekers and colleagues for their helpful comments. In our study, we showed that

cerebral blood flow (CBF) is low immediately after cardiac arrest and remains low during the 36 first hours.[1] We agree with the fact that arterial CO_2 pressure (PCO_2) may have an impact on CBF in post-cardiac arrest patients. Indeed, arterial PCO_2 decreased between admission and H12 ($p = 0.002$), then remained stable. Even if we agree that the decrease in PCO_2 between admission and hypothermia period could have contributed to CBF changes, we do not think that it could be the main explanation for the diminished CBF. Three reasons support our own interpretation. First, immediately on admission, a low diastolic velocity (below 20 cm/s) was noticed in 12/18 patients without any simultaneous hypocapnia and/or hypothermia ($PCO_2 = 45.5$ [38—49.5] mmHg). Second, since our local practices target a strict adherence to normal ranges for arterial PCO_2, we did not observe a severe hypocapnia during the hypothermia period (36 ± 6 at H12, 37 ± 7 at H24, 41 ± 8 at H36). On this point, our results are close to those reported by Buunk et al.[2] which showed that a low CBF was gradually increased after the first 24 h. Lastly, the diminished pulsatility index that we observed during the early period is contradictory with a negative effect of PCO_2 on CBF.[3]

Conflict of interest statement

None.

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Capnography—Reliable technique for identifying correct tube placement in cardiac arrest endotracheal intubations

I read with great interest the article by Nolan[1] on strategies to prevent unrecognised oesophageal intubation during out-of-hospital cardiac arrest (OHCA). The author describes very exactly the problem of verification of the position of the tracheal tube, the early detection of malposition and alternative airway devices in OHCA The author suggested two principle strategies for eliminating unrecognised oesophageal intubation in the OHCA patient: (A) reliable method of confirming the position tube (transthoracic impedance changes measured through defibrillator pads) and (B) alternative airway techniques (supraglottic airway devices). Dr. Nolan concluded in his contribution that exhaled CO_2 detectors are unreliable in cardiac arrest. Sayah et al.[2] demonstrated in an experimental study that capnometry is reliable for the early detection of oesophageal intubation during cardiac arrest. In our prospective study[3] in prehospital setting we observed all adult patients who were intubated by an emergency physician in the field. We analysed 246 patients in cardiac arrest. In this study capnography had a 100% sensitivity and specificity, compared to capnometry which had 88% sensitivity and 100% specificity in the arrest population. This study showed that if capnographic waveform during cardiac arrest and resuscitation is present, regardless of its amplitude, the tube can be confidently judged to be correctly placed. Our study confirmed that the capnographic waveform monitor is the most reliable technique for identifying correct tube placement after intubation in both arrest and non-arrest situations. Based on the results presented, we suggested an integral algorithm for tracheal tube confirmation and the prevention of dislodgment after emergency intubation. Capnography can also be used for assessment of the quality of resuscitation and it has prognostic value for determining the outcome of CPR. In our prospective studies[4,5] we showed correlation between the initial, mean and final end-tidal CO_2 values and return of spontaneous circulation (ROSC), admission to hospital and survival. Prehospital data from these studies provide strong support for an initial end-tidal CO_2 of 1.33 kPa to be the resuscitation threshold in the field. In our opinion, the initial value of end-tidal CO_2 should be included in every Utstein style analysis.

Confirmation of tube placement and CPR are a dynamic process, requiring ongoing patient assessment with capnography. In our clinical practice and research we find capnography very useful tools, which help us to monitor CPR and prevent unrecognised oesophageal intubation during OHCA.

Conflict of interest

None.

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