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# Re: Neural injury after use of vasopressin and adrenaline during porcine cardiopulmonary resuscitation

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**LETTER TO THE EDITOR**

# Re: Neural injury after use of vasopressin and adrenaline during porcine cardiopulmonary resuscitation

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Dear Editor,

Halvorsen and colleagues (1) investigated cerebral and cardiac tissue injury subsequent to use of vasopressin and adrenaline in combination compared with vasopressin alone during cardiopulmonary resuscitation (CPR). They concluded that combined use of vasopressin and adrenaline caused greater signs of cerebral and cardiac injury than use of vasopressin alone during experimental CPR. However, given that epinephrine was administered after 12 min of untreated ventricular fibrillation (VF), these results are not surprising.

First, neurologically intact survival to hospital discharge after in-hospital cardiac arrest has been found to be significantly more likely after earlier epinephrine administration (2). This recent retrospective evaluation of more than 25,000 patients (at 570 US hospitals), who were not in intensive care units or emergency departments and who exhibited initial rhythms of asystole or pulseless electrical activity (PEA), found that delayed administration of epinephrine (>3 min) was associated significantly with lower chance for survival to hospital discharge, in stepwise fashion (12%, 10%, 8%, and 7% survival, respectively, for patients receiving their first epinephrine dose ≤3, 4–6, 7–9, and >9 min after arrest ($p < 0.001$).

Second, as the authors already understand from their previous work in a swine model of hemorrhagic circulatory arrest that applied 15 min of open-chest CPR after 8 min of untreated VF, intracranial pressure (ICP) in the post-resuscitation phase is greater with use of adrenalin versus vasopressin, but with no significant difference in neuronal injury (3). However, one possible explanation for the lack of difference in neuronal injury could be that open-chest CPR does not raise intrathoracic pressure; therefore, it does not raise ICP. It has been postulated that without effective-depth chest compressions with complete recoil and/or gasping to maintain lower ICP, adrenaline administration has a strong potential to result in poor survival and neurological outcome (4). The elevation of intrathoracic pressure during chest compression generates carotid pressure and flow but also increases ICP, which may be what limits cerebral blood flow (5-7). The lower the ICP is, the lower is the resistance to forward blood flow to the brain. Allowing complete sternal recoil after effective-depth compressions results in less intrathoracic pressure; this in turn produces greater coronary and cerebral perfusion (because of the greater perfusion pressures produced by the effective-depth chest compressions) and lower increases in ICP (because of the complete sternal recoil). While Lund University Cardiopulmonary Assist System (LUCAS) CPR ensures effective-depth compression with complete recoil, it may not mitigate enough the increased ICP generated during chest compressions without the aid of gasping, unless perhaps an inspiratory impedance valve is used (8). Gasping alone during VF arrest improves cerebral perfusion and decreases ICP (9). Therefore, without a means to mitigate intracranial pressure adequately during CPR, late adrenaline administration resulted in greater neural injury and, as evidenced, has a strong potential to result in poor survival and neurological outcome.

***Declaration of interest:*** The author reports no conflicts of interest.

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