

CORRECTION

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Correction: A critical reappraisal of vasopressin and steroids in in-hospital cardiac arrest

Spyros D. Mentzelopoulos^{1,2*} and Athanasios Chalkias^{3,4}

Correction: Mentzelopoulos and Chalkias *Critical Care* (2024) 28:191

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Following publication of the original article [1], the authors identified an error within row 7 of Table 2. In Table 2, row 7, the lowest percentages of postresuscitation hypotension (i.e. 17% and 15%) actually correspond to the intervention group(s) and the highest (i.e. 28% and 29%) to control.

Table 2 row 7 currently reads:

Lowest MAP \leq 50 mmHg and SAP \leq 80 mmHg, intervention group(s) versus control (%)	28% versus 17%— $P=0.12$ and 29% versus 15%; $P=0.03^d$	NR but significant difference unlikely ^c
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The original article can be found online at <https://doi.org/10.1186/s13054-024-04962-8>.

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Table 2 Key differences between the Greek VSE trials and the Danish VAM IHCA trial

Key characteristic	VSE 1 and 2—pooled data	VAM-IHCA (Danish trial)	Potential effect
Time to study drugs, median (IQR)—min	4 (3–5)	9 (6–12)	Earlier, simultaneous activation of V1A-vasopressin and $\alpha 1$ -adrenergic receptors, with maximization of pressor effect and associated probability of prompt ROSC, ^a likely occurring only in intervention groups (vs. control) of the VSE 1 and 2 RCTs
Epinephrine and Vasopressin started and always given simultaneously	Yes	No	
Median time lag between first dose of epinephrine and study drugs (min)	0	3–4	
Significantly shorter median (IQR) time to ROSC (min) in intervention group(s) versus control	Yes; 14 (7–24) versus 20 (10–30)— $P < 0.001$	No; 16 (12–25) versus 18 (11–31)— P -value NR	Attenuated ischemia / reperfusion injury and lower risk of epinephrine-related adverse effects likely present only in intervention groups (vs. control) of the VSE 1 and 2 RCTs
Significantly lower median (IQR) total dose (mg) of epinephrine in intervention group(s) versus control	Yes; 4 (2–6) versus 5 (3–9)— $P < 0.001$	No; 3 (2–5) versus 3 (2–5)— P -value NR	
SAP < 90 mmHg within 20 min of ROSC, intervention group(s) versus control (%)	19% versus 44%— $P < 0.001$ ^b	NR but significant difference unlikely ^c	Early postresuscitation hypotension has been consistently associated with increased in-hospital mortality [1]; reported differences may partly explain the long-term benefit observed only in the VSE 1 and 2 RCTs
Lowest MAP \leq 50 mmHg and SAP \leq 80 mmHg, intervention group(s) versus control (%)	28% versus 17%— $P = 0.12$ and 29% versus 15%, $P = 0.03$ ^d	NR but significant difference unlikely ^c	
Postresuscitation use of steroids in intervention group(s) versus control (%)	99% versus 22% ^d	26% versus 46% ^e	In VAM-IHCA, there was non-protocolized, more frequent use of potentially beneficial interventions in the control group
Postresuscitation use of ECMO in intervention group(s) versus control (%)	0% versus 0% ^{df}	14% versus 30% ^e	

VSE, vasopressin-steroids-epinephrine; VAM, vasopressin-adrenaline-methylprednisolone; IHCA, in-hospital cardiac arrest; IQR, interquartile range; ROSC, return of spontaneous circulation; NR, not reported; RCT, randomized clinical trial; SAP, systolic arterial pressure; MAP, mean arterial pressure; ECMO, extracorporeal membrane oxygenation

Reported data originate from the total of the VSE 1 and 2 or VAM-IHCA participants, unless otherwise specified. For data reported as median (IQR), P -values were determined using the Mann Whitney U test. For data reported as percentages, P -values were determined by Fisher's exact test

^a, Defined as ROSC after ≤ 3 vasopressor doses

^b, VSE 1 and 2 data originate from the pooled subgroup of survivors for ≥ 4 h with postresuscitation shock and available SAP data (intervention, $n = 94$; control, $n = 72$)

^c, Analyses of VAM-IHCA hemodynamic data revealed very similar, early postresuscitation arterial pressures and vasopressor support [2]; consequently, there was likely no significant, between-group difference in the frequency of postresuscitation hypotension

^d, VSE 1 and 2 data originate from the pooled subgroup of survivors for ≥ 24 h (intervention, $n = 94$; control, $n = 69$)

^e, VAM-IHCA data originate from survivors for ≥ 24 h (intervention, $n = 63$; control, $n = 61$)

^f, There was no postresuscitation use of ECMO in the VSE 1 and 2 studies

Table 2 row 7 should read:

Lowest MAP \leq 50 mmHg and SAP \leq 80 mmHg, intervention group(s) versus control (%)	17% versus 28%— $P=0.12$ and 15% ver- sus 29%; $P=0.03^d$	NR but significant differ- ence unlikely ^c
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^e, VAM-IHCA data originate from survivors for ≥ 24 h (intervention, $n = 63$; control, $n = 61$)

^f, There was no postresuscitation use of ECMO in the VSE 1 and 2 studies

Table 2 has been updated in this correction and the original article [1] has been corrected.

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Reference

1. Mentzelopoulos SD, Chalkias A. A critical reappraisal of vasopressin and steroids in in-hospital cardiac arrest. *Crit Care*. 2024;28:191. <https://doi.org/10.1186/s13054-024-04962-8>.

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