



Short communication

Quality controlled manual chest compressions and cerebral oxygenation during in-hospital cardiac arrest[☆]Antti Kämäräinen^{a,b,*}, Marko Sainio^{a,c}, Klaus T. Olkkola^c, Heini Huhtala^d, Jyrki Tenhunen^a, Sanna Hoppu^a^a Critical Care Medicine Research Group, Department of Critical Care Medicine, Tampere University Hospital and University of Tampere, TAYS PL 2000, 33521 Tampere, Finland^b Emergency Medical Services, Emergency Department, Tampere University Hospital, TAYS PL 2000, 33521 Tampere, Finland^c Department of Anaesthesiology, Intensive Care, Emergency Care and Pain Medicine, University of Turku and Turku University Hospital, PO Box 52 (Kiinamyllynkatu 4-8), FI-20521 Turku, Finland^d School of Health Sciences, University of Tampere, 33014 University of Tampere, Finland

ARTICLE INFO

Article history:

Received 30 March 2011

Received in revised form 4 September 2011

Accepted 8 September 2011

Keywords:

Cardiac arrest

Resuscitation

Quality

Perfusion

Cerebral

Oximetry

Chest compressions

ABSTRACT

Aim: The quality of cardiopulmonary resuscitation (CPR) is associated with the rate of return of spontaneous circulation (ROSC) during human cardiac arrest. Current advances in defibrillator technology enable measurement of CPR quality during resuscitation, but it is not known whether this is directly reflected in cerebral oxygenation. In this descriptive study we aimed to evaluate whether the quality of feedback-monitored CPR during in-hospital cardiac arrest is reflected in near infrared frontal cerebral spectroscopy (NIRS).

Methods: Nine patients suffering an in-hospital cardiac arrest in a university hospital were included. All patients underwent quality-controlled CPR performed by a dedicated medical emergency team using a Philips HeartStart MRx defibrillator (Philips, Eindhoven, Netherlands) with a CPR quality (Q-CPR, Laerdal Medical, Stavanger, Norway) analysis feature. Simultaneously, bilateral frontal cerebral oximetry was measured using INVOS 5100c (Somanetics, Troy, MI, USA) NIRS.

Results: During quality controlled resuscitation, regional cerebral oxygenation (rSO₂) as measured with NIRS was low but it improved during CPR ($p = 0.043$) and 8 min after ROSC ($p = 0.022$). After the onset of NIRS recording, there were four episodes exceeding 30 s, during which the quality of CPR was substandard. When CPR technique was corrected and maintained for 2 min, a minor non-significant increase in rSO₂ was observed in two cases.

Conclusions: High quality CPR was not significantly reflected in cerebral oxygenation as quantified using NIRS. Even after ROSC and subsequent significant increase in cerebral oxygenation, rSO₂ readings were below previously suggested threshold of cerebral ischaemia. Improving CPR technique after an episode of low quality CPR did not significantly increase rSO₂.

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1. Introduction

High quality of cardiopulmonary resuscitation (CPR) is associated with the successful return of spontaneous circulation (ROSC),¹ cerebral perfusion² and cerebral oxygenation.³ Quality monitoring may be important also during CPR performed by health care professionals.⁴

Contemporary defibrillators enable real time CPR quality analysis. Although CPR aims to minimize ischaemic injury, cur-

rently there are limited techniques to quantify cerebral perfusion during CPR. We sought to evaluate whether CPR quality is reflected in cerebral oxygenation as a surrogate for cerebral perfusion.

2. Materials and methods

The study protocol was approved by the ethics committee of Tampere University Hospital (clinicaltrials.gov: NCT00951704). Following informed written consent from the next of kin, cardiac arrest patients in the Tampere University Hospital treated by a medical emergency team (MET) were enrolled (14 month period from November 2008 to January 2010). Exclusion criteria were age <18 years and unavailability of a dedicated study nurse for cerebral oximetry monitoring.

[☆] A Spanish translated version of the abstract of this article appears as Appendix in the final online version at doi:10.1016/j.resuscitation.2011.09.011.

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Table 1

Patient and resuscitation characteristics. AAA, abdominal aortic aneurysm; CA, cardiac arrest; PEA, pulseless electrical activity; ASY, asystole; VT, ventricular tachycardia; ROSC, return of spontaneous circulation; SD, standard deviation; IQR, interquartile range; rSO₂, regional haemoglobin oxygen saturation. The data are given as mean $\pm$ SD, median (interquartile range) or number.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8	Patient 9
Gender	Male	Female	Female	Male	Female	Female	Female	Female	Male
Age	75	81	72	87	84	83	84	79	78
Aetiology of arrest or preceding symptoms	Cardiac tamponade, hypotension	Unknown	Unknown	Cardiac, postoperative care following intravascular treatment of AAA	Acute renal failure, acidosis, respiratory depression	Postoperative hypoxia	Respiratory depression, hypoxia.	Postoperative hypoxia and chest pain	Postoperative hypoxia, aspiration of gastric contents
Pre-arrest chronic and subacute morbidity	Hypertension	Aortic stenosis, pulmonary fibrosis	Hypertension	Coronary artery disease, chronic atrial fibrillation	Inactive tuberculosis	Coronary artery disease, congestive heart failure, diabetes, renal carcinoma	Hypertension, Parkinson's disease, congestive heart failure, femoral fracture, intra-abdominal infection	Diabetes, hypertension	Aortic stenosis, mitral valve insufficiency
Time from CA to in-hospital resuscitation team arrival (s)	140	170	115	145	155	150	51	165	122
First rhythm	PEA	PEA	ASY	VT	ASY	PEA	PEA	PEA	ASY
Witnessed arrest?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Return of spontaneous circulation	ROSC	ROSC	No ROSC	ROSC	ROSC	ROSC	No ROSC	ROSC	ROSC
Alive after 6 months	No	No	No	Yes	No	No	No	No	No
Episode duration (min; s)	4:40	5:52	18:06	1:48	7:18	2:24	11:30	7:12	6:42
Total number of compressions	451	518	1769	196	620	283	1137	721	640
Compression depth (mm)	55 $\pm$ 7	52 $\pm$ 4	44 $\pm$ 4	38 $\pm$ 1	54 $\pm$ 6	57 $\pm$ 1	56 $\pm$ 9	49 $\pm$ 2	80 $\pm$ 8
Fraction of 30 s with compression depth <40 (mm)	0/10 = 0%	0/12 = 0%	4/37 = 11%	4/4 = 100%	0/15 = 0%	0/5 = 0%	0/24 = 0%	0/15 = 0%	0/13 = 0%
Compression rate min ⁻¹	96 $\pm$ 7	88 $\pm$ 11	98 $\pm$ 17	107 $\pm$ 3	85 $\pm$ 26	116 $\pm$ 3	97 $\pm$ 29	99 $\pm$ 24	103 $\pm$ 3
Fraction of 30 s with compression rate <90 or >120 min ⁻¹	2/10 = 20%	1/12 = 8%	0/37 = 0%	0/4 = 0%	0/15 = 0%	1/5 = 20%	9/24 = 38%	0/15 = 0%	0/13 = 0%
Compressions delivered (min ⁻¹)	98 $\pm$ 6	87 $\pm$ 10	97 $\pm$ 13	106 $\pm$ 0	84 $\pm$ 26	117 $\pm$ 0	97 $\pm$ 20	99 $\pm$ 17	94 $\pm$ 9
Incomplete chest release (% of total number of compressions)	1	1	10	10	55	1	31	0	18
Incomplete chest release per 30 s segments (%)	0 (0–0)	0 (0–0)	0 (0–0)	9 (2–14)	82 (0–90)	0 (0–3)	26 (19–40)	0 (0–0)	1 (0–18)
Compression as part of duty cycle (%)	39 (30–40)	41 (41–42)	44 (38–46)	45 (44–46)	42 (41–43)	41 (40–43)	46 (44–48)	41 (40–43)	41 (38–45)

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