Microcirculatory monitoring of a Jehovah's Witness suffering from haemorrhagic shock

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EDITOR:

A 33-yr-old previously healthy female patient presented to our department after a car accident. She was intubated and ventilated and was suffering from haemorrhagic shock with an initial blood pressure of 70/40 mmHg. Injuries included head injury, nasal fracture, bilateral femoral fractures, right patella fracture, distal lower left leg fracture, distal left radius fracture and a central liver haematoma. History revealed that she belonged to the group of Jehovah's Witnesses. Before induction of anaesthesia she had insisted on refusing any blood transfusion.

After initial surgical stabilization of the fractures by external fixators, the patient was highly dependent on catecholamines. She was admitted to the ICU with the following blood gas analysis: pO_2 33.3 kPa, pCO_2 5.4 kPa, SaO_2 99.8%; pH 7.26; BE −7.8; bicarbonate 17.9 mmol L^{-1}; lactate 7.0 mmol L^{-1}, Hb 3.1 mmol L^{-1}. Therapeutic objectives were to avoid further blood loss, optimize oxygenation, stabilize circulatory function with catecholamines and to stimulate erythropoiesis.

During her ICU stay, she was ventilated with inspiratory oxygen between 80 and 100% and with a positive end-expiratory pressure (PEEP) of 8–10 mmHg not only to optimize oxygenation but also because of aspiration pneumonia. A tracheostomy was considered but not performed, since any further blood loss should be avoided.

Norepinephrine (0.02–0.12 μg kg^{-1} min^{-1}) and dobutamine (3–6 μg kg^{-1} min^{-1}) were needed to maintain mean arterial pressure between 60 and 90 mmHg, central venous pressure at 10–16 mmHg and to maintain sufficient organ perfusion. Diuresis was maintained above 80 mL h^{-1}, with fluid excess in the range of 1000–3000 mL 24 h^{-1}. Further invasive haemodynamic monitoring was considered but not performed to avoid any additional blood loss. Recombinant human erythropoietin was injected (40 000 I.U. intravenously every 3 days) together with daily folic acid, iron and vitamin B_{12}. The liver haematoma was observed daily by ultrasound and did not show any progression in size. Between days 3 and 5 of her ICU stay, the patient developed hypermenorrhoea and to stop this bleeding a GnRH analogue was given successfully.

Due to the critical haemodynamic situation and the lack of invasive haemodynamic monitoring, we looked into non-invasive methods to obtain information on microcirculation and tissue oxygenation. For this purpose, a flat probe (LF-1™; Lea Company, Giessen, Germany) was placed daily on the patient's tongue and connected to a device allowing simultaneous measurement of laser Doppler flowmetry and tissue spectrophotometry (O2C™; Lea Company, Giessen, Germany). This provided microvascular haemoglobin oxygen saturation, relative haemoglobin concentration and relative blood flow at a tissue depth of 200 and 400 μm every 2 s over a time period of 5 min. Results of representative days are presented in Table 1.

Surprisingly, our data showed that though our patient suffered from severe anaemia, local tissue oxygenation was not compromised throughout her whole ICU stay. Our therapy maintained sufficient blood flow in capillaries and consequently balanced oxygen supply and consumption. These findings correlate well with the results of blood gas analyses and lactate concentration, which were all in a moderate range throughout the 16 days of her ICU stay (Table 1). On day 16 of her ICU stay, the patient suddenly developed resistant circulatory failure and died. Autopsy revealed that her cerebellum had incarcerated in the foramen magnum as a result of general brain swelling. Histological analysis of the lung showed an acute lung injury with massive infiltrates.

We used a new device, the O2C™, which combines white light spectrophotometry and laser Doppler flowmetry, enabling non-invasive monitoring of tissue oxygenation and microcirculatory blood flow in various depths of about 100 μm to 16 mm [1], depending on the probe used. The blood flow, post-capillary oxygen saturation and the amount of haemoglobin in the capillaries can be determined. For this purpose, white light in the range of 500–800 nm is applied simultaneously with laser light at a wavelength of 830 nm via a flexible fibreoptic probe [1–3]. Unlike arterial oxygen saturation, the capillary–venous oxygen saturation shows the balance between oxygen

Correspondence to: Axel Menzebach, Department of Anaesthesiology and Intensive Care Medicine, University Hospital Rostock, Schillingallee 35, 18055 Rostock, Germany. E-mail: axel.menzebach@uni-rostock.de; Tel: +49 381 4946411; Fax: +49 381 4946402

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Table 1. Microcirculatory, laboratory, haemodynamic variables and catecholamine doses taken during the ICU stay.

	Day 1	Day 5	Day 9	Day 15
Tissue depth 200 μm				
μHbO_2 (%)	58±5	60±6	70±4	63±2
rHb	23±2	20±3	20±1	30±2
Flow	92±23	80±30	85±8	135±71
Tissue depth 400 μm				
μHbO_2 (%)	73±1	75±1	74±1	85±1
rHb	42±2	39±1	38±1	43±1
Flow	276±11	231±19	236±9	186±28
Laboratory variables				
Hb (mmol L^{-1}]	1.9	1.5	1.3	1.5
Hct (%)	10	8	8	9
pO_2 (kPa)	20.5	55	40	40
F_iO_2	0.9	0.9	0.9	0.8
Lactate (mmol L^{-1})	2.5	2.2	1.5	2.3
pH	6.01	7.48	7.34	7.37
Temperature (°C)	38.2	37.4	37.2	37.8
Haemodynamic variables				
CVP (mmHg)	16	14	13	10
Catecholamines				
Norepinephrine (μg kg^{-1} min^{-1})	0.12	0.06	0.02	0.03
Dobutamine (μg kg^{-1} min^{-1})	6	6	3	4

Data are means ± SD; μHbO_2 (%): microvascular haemoglobin oxygen saturation; rHb [AU]:relative haemoglobin concentration; flow: relative blood flow [AU] obtained by laser Doppler.

delivery to the tissue and oxygen consumption by that tissue. Therefore, microcirculatory oxygen saturation measurement is an ideal variable to determine local tissue hypoxia not detectable by global measurements. Unlike the mixed-venous oxygen saturation, shunt-blood flow in the tissues only plays a minor role. As a consequence, capillary–venous oxygen saturation can be lower than mixed-venous [4]. Hence, microcirculatory haemoglobin oxygen saturation is an excellent indicator of tissue hypoxia since it reflects oxygen extraction processes along the capillaries.

Relative haemoglobin concentration shows the haemoglobin amount in the tissue monitored and represents the filling of the microvessels, which is proportional to capillary density, capillary recruitment and venous filling. Since approximately 80% of the haemoglobin is to be found in the microcirculation on the venous side, this is an indicator of venous congestion. Relative blood flow shows volume flow in vessels below 100 μm in the tissue of interest. Unfortunately, there are no reports of normal values of either of these variables obtained in healthy volunteers.

We did not observe any significant changes in microcirculatory oxygen saturation or local haemoglobin concentration throughout her 16-day ICU stay, indicating adequate tissue oxygenation. Between days 1 and 5, we found a 30% decrease in flow at a tissue depth of 400 μm, indicating a progression of anaemia. We chose to observe tissue oxygenation and microcirculation of the tongue because it might reflect the situation in the gastrointestinal tract [4]. Care must of course be taken in extrapolation of these results to other regions of the body. This additional monitoring was very useful in guiding our therapy allowing, optimization of oxygen delivery through modulation of catecholamine therapy, proving that the consequences of anaemia on the microcirculation could be partly compensated.

Tissue oxygenation was not compromised over nearly 15 days (both in superficial and in deeper tissue layers) and it is possible that microcirculatory monitoring may provide helpful information about efficacy of intensive care therapy in patients suffering from massive acute anemia [1–3]. Although erythropoetin has been used to reduce the need for blood transfusions, its effectiveness is uncertain in the critically ill and the period until a significant increase in haemoglobin concentrations occurs may be as long as 10–30 days [5]. Jehovah's Witnesses can create perplexing treatment problems by their refusal of blood transfusions. Donor blood transfusion is the gold standard in the treatment of unstable patients suffering from severe anemia [6,7]. The O2C™

provides a new online bedside monitoring system, which may be of value in such patients.

A. Menzebach, C. Mutz, T. W. L. Scheeren
Department of Anaesthesiology and Intensive Care Medicine
University Hospital Rostock
Schillingallee, Rostock Germany

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The use of nicorandil in cardioplegia solution

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EDITOR:
We would like to comment on the article by Chinnan and co-workers [1] written on myocardial protection by nicorandil during open-heart surgery under cardiopulmonary bypass (CPB).

In relation to cardioplegia, coronary artery spasm (CAS) is frequently underestimated during CPB. Until now, CAS was not detectable with current monitoring techniques during CPB and its impact on cardiac surgery has not been elucidated in previous studies. During CAS the blood flow to the myocardium decreases depending on the degree of coronary vasoconstriction, thereby increasing anaerobic metabolites and myocardial ischaemia. Consequently, CAS could be responsible for right and left ventricular failure after leaving CPB. Therefore, apart from being a potassium channel opening drug preventing intracellular Ca^{2+} loading [2], the fact that nicorandil is a coronary vasodilator might have contributed to favourable results in your study.

The link between vasospastic angina and vulnerability for ventricular tachycardia or ventricular fibrillation outside the setting of cardiac surgery has already been described in several case reports [3–5]. Iida and colleagues [5] reported that ventricular fibrillation improved and disappeared with the start of treatment with nitrates and suggested that CAS should be considered as a differential diagnosis in the presence of ST-segment changes and/or intractable ventricular arrhythmias. Extending this conclusion into CPB surgery, this could imply that rather than giving an anti-arrhythmic drug, a coronary vasodilator should be used to prevent and to treat arrhythmias secondary to ischaemic episodes caused by CAS. As your results show, a link between CAS and ventricular fibrillation seems to be apparent. After cross-clamp removal in coronary artery bypass grafting (CABG) patients, two patients in the nicorandil group developed significant cardiac dysrythmias vs. six patients in the placebo group. Unfortunately, the number of your patients is limited and it would be interesting to pursue your study as a multicentre trial on a larger scale.

The relationship between nicorandil and coronary vasodilatation also explains the enhanced distribution of cardioplegia solution, a faster time until electromechanical arrest in the nicorandil group and a faster return of electromechanical activity after aortic cross clamp removal in patients under nicorandil in the mitral valve replacement group.

Interestingly, although not significantly different, in CABG patients four nicorandil patients compared to two placebo patients had creatine kinase (CK-MB) levels higher than $75\,IU\,L^{-1}$. Within the patients treated with nicorandil in the CABG group, as

Correspondence to: Gabor Kiss, University Hospital of Brest, Anaesthesia and Surgical Intensive Care, Bd. Tanguy Prigent, Brest, France. E-mail: gaborkiss2001@hotmail.com; Tel: +33 2 98 34 74 28; Fax: +33 2 98 34 74 41

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