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Loss of Haemodynamic Coherence, Diagnosed Using a Continuous Monitoring of Oesophageal Photoplethysmography

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Abstract

We report the case of a 75-year-old man with appendicular peritonitis in whom we observed oesophageal tissue perfusion, using a new photoplethysmographic probe. As the patient suffered from lactic acidosis, we chose to increase the mean arterial pressure (MAP) using norepinephrine. Thanks to an oesophageal photoplethysmographic signal, we could observe a loss of haemodynamic coherence and a severe alteration of tissue perfusion triggered by the MAP increase. Considering new information regarding the tissue perfusion status, we were able to adjust our MAP targets accordingly and restore the initial perfusion index values. This case illustrates the loss of haemodynamic coherence and highlights the clinical relevance of continuous tissue perfusion monitoring to highlight potential adverse norepinephrine effects.

Keywords: Haemodynamic, haemodynamic coherence, photoplethysmography

Introduction

Haemodynamic management during a septic shock with standard techniques is mainly based on macrocirculatory parameters (heart rate, mean arterial pressure [MAP], cardiac index, echocardiography) (1). Current guidelines set only a minimal MAP target with no room for individualised perfusion goals.

However, during the septic shock, there is a dysregulation of the microcirculation, which leads to a loss of haemodynamic coherence. This loss of coherence due to dysregulation leads to an inefficiency of macrocirculation-driven procedures, failing to improve the microcirculatory perfusion and oxygen delivery to the parenchymal cells (2).

Since continuous monitoring of tissue perfusion is not available in routine practice, the loss of haemodynamic coherence often goes unnoticed and may mislead physicians when managing haemodynamics (3). We report the application of a continuous tissue perfusion monitoring system, using oesophageal photoplethysmography, which may allow substantial refinement of haemodynamic management.

Case Presentation

A 75-years-old man suffering from acute abdominal pain was admitted to the emergency department. He had no relevant medical history other than essential hypertension. The computed tomography showed intraperitoneal fluid effusion complicating a perforated appendicitis. An antibiotic treatment with cefotaxime, metronidazole and amikacin was administered. The patient was operated within the next 3 hours and underwent a laparoscopic appendectomy and peritoneal lavage. During the perioperative period, we used norepinephrine to maintain the MAP >65 mmHg after a fluid loading (2500 mL) with crystalloid solution assessed by echocardiography. At the end of the intervention, blood gas analysis revealed lactic acidosis (pH 7.28, lactic acid 3.7 mmol L^{-1}, pCO2 5.5 kPA).

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The patient was directly transferred into our intensive care unit (Centre Hospitalier Lyon Sud, France). Immediately after the admission, we monitored MAP using an invasive arterial catheter, and we placed a photoplethysmographic probe (IKORUS; APD, Lyon, France) in the oesophagus, which allowed us to monitor tissue perfusion through a perfusion index (PI) (Figure 1). The IKORUS probe is a standard duodenal probe with an optical sensor embedded in the shaft. The probe was inserted so that the sensor was in close contact with the oesophageal mucosa. The IKORUS monitor calculates the PI by analysing the changes of tissues absorbance of the photoplethysmography signal over time. This analysis is performed in real time, while requiring no additional action. The real-time constant monitoring is a major advantage over a video-microscopy device that requires the point of care use. Six hours following the ICU admission, MAP was 60 mmHg through 1.2 γ kg^{-1} min^{-1} of norepinephrine. Mechanical ventilation was optimised (7.2 mL kg^{-1} of VT, 7 cmH_2O PEEP, PaCO2 at 4.9 kPa). The patient's lactic acidosis worsened (pH 7.12, lactic acid 8 mmol L^{-1}). The PI was at 55. We decided to increase norepinephrine to raise MAP above 75 mmHg to improve tissue perfusion and correct lactic acidosis. At 1.9 γ kg^{-1} min^{-1}, MAP was 100 mmHg, and the PI dropped to 33, alerting us of a severe alteration in tissue perfusion, which reflected a loss of haemodynamic coherence (Figure 2). We lowered the norepinephrine perfusion to 1.5 γ kg^{-1} min^{-1}, which resulted in a decreased MAP and a restoration of the perfusion index.

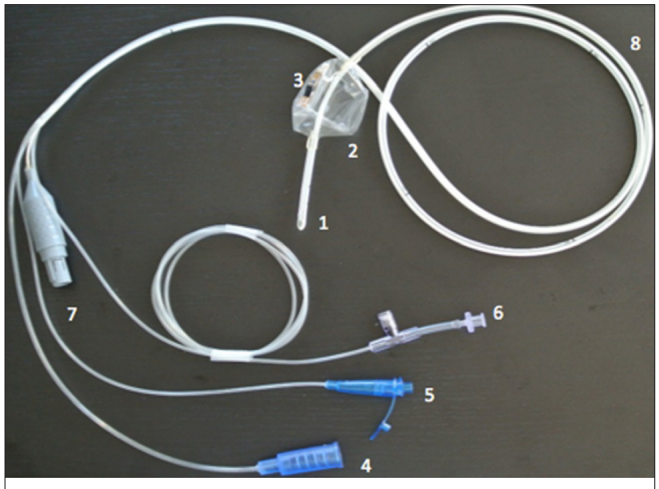

Figure 1. Photoplethysmographic probe. 1) Photoplethysmographic sensor on an inflatable balloon (the inflation of the balloon is only indicated for a post-pyloric position of the probe). 2) Feeding line. 3) Aspiration line. 4) Line dedicated to inflate and deflate the balloon

Finally, norepinephrine was stopped on Day 3, and extubating was done on Day 4. The patient was discharged on the 6th day, and the antimicrobial chemotherapy was maintained until Day 8.

The written consent of the patient was obtained for the use of the photoplethysmographic probe and the publication of the case, in accordance with the Declaration of Helsinki (1964) and ethical guidelines.

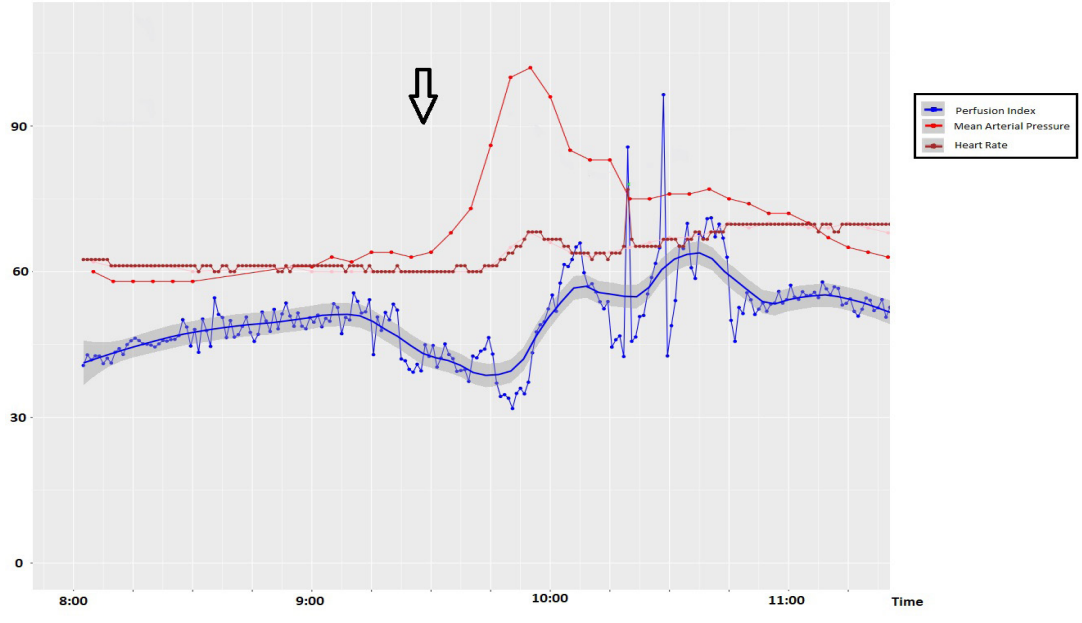

Figure 2. Illustration of the haemodynamic coherence loss

The arrow represents the ascension of the MAP using norepinephrine, with a conjoint diminution of the perfusion index. The decrease of the MAP allows a restoration of the PI and an improvement in microcirculation and tissue perfusion.
Brown dot: heart rate; Red dot: mean arterial pressure; Blue dot: perfusion index

Discussion

This case illustrates the need to monitor microcirculation to detect the loss of haemodynamic coherence and to allow an adequate MAP objective.

This patient with septic shock was treated according to guidelines: An adapted antimicrobial chemotherapy was administered, and a surgical control of the source of the infection and a monitoring of the macrocirculation (fluid loading assessed by echocardiography, invasive monitoring of MAP) were applied. The minimal MAP objective was obtained, and to improve a macrocirculatory part of the tissue dysoxia, we choose to increment MAP. This led to a severe alteration of tissue perfusion detected by the drop of the PI, which could have been unnoticed without a continuous monitoring of tissue perfusion. While the MAP decreased, we observed an improvement of the microcirculation, restoring PI and characterising a loss of haemodynamic coherence.

Microcirculation is regulated by the endothelial cells of the arterioles. It ensures an adequate oxygen delivery to tissues to meet the cell oxygen demand. Under physiological conditions, the regulation of microcirculation goes through the nitric oxide (NO), modulating the arteriolar smooth muscle cell tone. During sepsis, there is a dysfunction of the NO regulation system: the endothelial cells lose their ability to regulate the tissue perfusion due to an excessive NO production by inducible NO synthetase (4).

This alteration of the NO regulation is concomitant with an impairment of endothelial cells by changing their signal transduction pathway, losing the control over arteriolar smooth muscle cells. Associated with their loss of adrenergic sensitivity and tonus, the regulation of tissue perfusion by the arteriolar smooth muscle cells is severely disturbed. Red blood cells also fail at releasing vasodilators during hypoxia, and they easily aggregate to endothelial cells (in association with platelets). These mechanisms explain the constant microvascular dysfunction during the septic shock, which occurs within 6–24 hours (4).

This explains why the macrocirculatory haemodynamic-driven resuscitation procedures have failed improving the mortality rate during the septic shock (5). Standard haemodynamic parameters did not permit the detection of patient with a loss of haemodynamic coherence, leading to an inadequate MAP target. Thooft et al. (6) reported in their study a large variation of the microvascular response among patients when using near-infrared spectroscopy in septic shock. Continuous monitoring of microcirculation allows physicians to individualise the MAP targets for each patient.

Dubin et al. (7) have already showed the loss of haemodynamic coherence with the increase of MAP using norepinephrine. In 20 patients with septic shock, the authors showed that the increase of MAP is related to an increase of the cardiac index, pulmonary pressures and the left and right ventricular stroke work indices, but the lactate, VO_2 and sublingual capillary microvascular flow index remained unchanged. They reported considerable variations in the interindividual responses that seemed to depend on the basal condition of the microcirculation.

The analysis of oesophageal photoplethysmography waveform has been already documented, and it allows to obtain the information different than the SpO_2 measurements (8). The PI is calculated from the pulsatile part of the signal output. The area under the pulsatile signal output is proportional to the quantity of blood travelling in front of the photoplethysmographic sensor. This new index thus reflects the quantity of arterial oxygenated blood flow near the sensor. Additional information on the technology and experimental result of the device can be found in this paper (9).

Conclusion

This case report confirms the well-known fact that microcirculation cannot always be inferred from macroparameters. The MAP improvement did not enhance the tissue perfusion. Oesophageal photoplethysmography was able to highlight this situation, allowing us to improve our therapy. Other studies are required to set an appropriate 'tissue-perfusion goal therapy' and optimal range of PI to improve patient care.

Informed Consent: Verbal and written informed consent was obtained from patient who participated in this case.

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Conflict of Interest: The authors have no conflicts of interest to declare.

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