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CORRESPONDENCE

Why chest compressions should start when systolic arterial blood pressure is below 50 mm Hg in the anaesthetised patient. Reply to *Br J Anaesth* 2020; 124: 234–8

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Editor—We read with interest the editorial by Harper and colleagues,[1] in which an emphasis was placed on pre-emptive cardiopulmonary resuscitation (CPR) at systolic BP (SBP) less than 50 mm Hg. We also believe that, with early institution of CPR, many life-threatening episodes of cardiac arrest can be prevented. However, its applicability needs to be classified in real-world situations, such as children *vs* adults *vs* older adults, perioperative *vs* intensive care, healthy *vs* critically ill patients, and patients with and without significant co-morbidities. We believe that the same dictum should not be applied to all groups of patients, and the specific high-risk patients and conditions need to be specified.

Most hypotensive episodes in the intraoperative period, particularly in young healthy patients, are transient and are corrected rapidly with a targeted oriented approach, for example, hypotension as a result of vagal stimulation. The trigemino-cardiac reflex is a common phenomenon during neurosurgery that manifests with varied autonomic disturbances, such as hypotension.[2] For such hypotensive episodes, it would be excessive to initiate CPR, which would create panic and disrupt the surgical procedure and potentially worsen outcome.

Selecting an arbitrary value for SBP of 50 mm Hg as a threshold for initiation of CPR also requires rationale. We feel that mean arterial pressure provides a stronger determinant when noninvasive BP is measured, as it overestimates SBP at low BP as the authors mentioned; the degree of such SBP overestimation is not known precisely.

Whilst the incidence of intraoperative cardiac arrest is very low (0.03–0.05%),[3,4] we presume that over-aggressive pre-emptive CPR potentially resulting in unwanted injuries will have a higher incidence. We also agree with the comments of Granfeldt and Anderson[5] that one arterial pressure does not fit all; rather, a threshold for CPR should be individualised. Unsynchronised CPR, especially during impaired ventricular filling, can be counterproductive. Finally, newer indices, such as the Hypotension Prediction Index that provides an accurate real-time and continuous prediction of impending intra-operative hypotension, can be used so that rescue measures can be instituted early.[6] In our opinion, high-risk patients (e.g. critically ill) should be identified for earlier intervention instead of using an arbitrary SBP value of 50 mm Hg for institution of pre-emptive CPR.

Declarations of interest

The authors declare that they have no conflicts of interest.

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Haemolysis index: validation for haemolysis detection during extracorporeal membrane oxygenation

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Editor—Extracorporeal membrane oxygenation (ECMO)-induced haemolysis is provoked by blood trauma, release of gaseous microbubbles from degassing, or cavitation of red cells exposed to negative pressure (in the pump head, for instance).[1,2] When plasma-free haemoglobin (fHb)-scavenging mechanisms are overwhelmed, fHb may cause damage to the kidneys and other organs.[2–5] Therefore, prompt identification of haemolysis is of utmost importance as changes in therapy could be implemented. Measurement of fHb via the conventional spectrophotometry lacks automation, is cumbersome, and is, at best, performed only once a day during working hours. This predisposes to delayed detection of life-threatening haemolysis episodes.[6] Newer clinical chemistry analysers display a haemolysis index (HI; a dimensionless value) to assess the reliability of measurements potentially flawed by haemolysis (e.g. K^+).

The aim of this prospective study was to assess whether the HI, an *in vitro* tool proposed to detect haemolysis related to sample collection, transport, and processing, could be used to assess *in vivo* haemolysis in patients on ECMO. We also assessed (1) inter-measurement variability of HI and fHb and (2) impact on HI measurements of icterus and lipaemia (possibly related to propofol infusion) that may interfere with spectrophotometric measurements.[7] The ethics committee of the French Society of Anaesthesia and Intensive Care approved the study protocol (00010254-2016-038). The purely observational nature of this study and the lack of clinical data collected enabled patient consent to be waived.

In both derivation (March 2015–December 2016) and validation (January 2017–September 2018) cohorts, a daily blood sample from consecutive adult patients on ECMO at our institution was analysed for electrolyte analysis, and lithium heparin tubes were centrifuged at 2200 *g* for 10 min before analysing the supernatant. HI was measured via a Roche®-Cobas 6000 analyser (light absorbance tested at 570 and 600 nm). fHb measurements relied on spectrophotometry (Unicam UV3 UV/Vis Spectrophotometer; PerkinElmer, Hopkinton, Massachusetts, United States); after dilution (1:11) of a plasma sample, the absorption spectrum was measured at different wavelengths before and after reducing oxyhaemoglobin with sodium hydrosulphite.[8] The relationship between fHb and HI was assessed (Lin's concordance correlation coefficient [CCC]). In the *derivation cohort*, using receiver operating characteristics (ROC) curve analysis, we evaluated the ability of HI to detect fHb exceeding a critical threshold of 100 mg dl^{-1}.[3,6] In the *validation cohort*, we tested the cut-off of HI we determined in the derivation cohort. Beforehand, the bootstrap technique created a large set of 1000 samples in each cohort.

To assess the inter-measurement variability, the coefficient of variation of HI and fHb was determined in 30 repeated measurements on four home-made fHb solutions (25, 50, 100, and 200 mg dl^{-1}). Those solutions were prepared and frozen for a few weeks at −20°C after we ensured they were stable for more than 12 months. They were then thawed in a random order, and measurement of fHb and HI started simultaneously.

The Roche®Cobas 6000 analyser provides spectrophotometric measurements of icterus and lipaemic indices, surrogates for plasma total bilirubin and triglycerides, respectively.[7] An *in vitro* experimental exploration was performed in five patients. First, we evaluated the impact on HI of a gradual