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Editorial

EDITORIAL

Why chest compressions should start when systolic arterial blood pressure is below 50 mm Hg in the anaesthetised patient

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We ask a deceptively simple question: when in the setting of profound hypotension, despite treatment, should chest compressions be started during general anaesthesia? The question arises out of discussions and recommendations in the 6th National Audit Project of the Royal College of Anaesthetists (NAP6), which studied perioperative anaphylaxis.[1] The issues discussed apply to all causes of profound hypotension and pre-cardiac arrest low flow states with two exceptions: cardiac tamponade, in which the value of cardiac compressions has been questioned,[2] and haemorrhagic shock with uncontrolled bleeding, in which chest compressions might accelerate blood loss and lead to worse outcomes.[3] The aim of chest compressions is to maintain organ perfusion while the cause of severe hypotension or cardiac arrest is treated. Based on the limited evidence available, we propose an answer to the question 'at what blood pressure should chest compressions be started in the anaesthetised patient?'

The notion that chest compressions should be started only during cardiac arrest is an oversimplification, not least because cardiac arrest, defined as 'absence of signs of circulation',[4] can be difficult to diagnose in the anaesthetised patient. In ventricular fibrillation there is a clear need for immediate defibrillation and chest compressions, and during asystole there is a clear need for chest compressions. What are less clear are the clinical criteria, particularly during isolated severe hypotension, that should prompt chest compressions in other situations.

Current resuscitation guidelines de-emphasise the role of the pulse check to confirm cardiac arrest,[5] recognising varying ability to assess the presence or absence of a pulse. Therefore, most rescuers should start chest compressions when the patient is unresponsive and not breathing normally, and only those experienced in resuscitation should assess the pulse whilst simultaneously looking for signs of circulation. The guidelines for in-hospital cardiac arrest recommend that responders skilled in pulse checks, which clearly include anaesthetists, should start cardiopulmonary resuscitation (CPR) 'if there is no pulse or other signs of life'.[6]

In the context of anaesthesia, as almost all patients are unresponsive and not breathing normally, not least because about half of patients receive a neuromuscular blocking drug,[7] initiation of chest compressions relies on cardiovascular assessment. Anaesthetists respond to multiple possible sources of cardiovascular information including arterial pressure, pulse waveform and oximetry, capnography, electrocardiogram, and measurements of cardiac output. Primary interventions include haemostasis, intravenous fluids, and vasoactive drugs, but when these are failing, the anaesthetist must decide when to start chest compressions.

Peripheral pulses

The modality used for triggering chest compressions should be as objective as possible: one option is failure to detect a palpable pulse. Historically, the anaesthetist's assistant would palpate the radial pulse during induction of anaesthesia and report a weak or absent pulse. The use of noninvasive blood pressure (NIBP) monitoring has superseded this practice.

However, even trained clinicians cannot assess the pulse with sufficient reliability to confirm cardiac arrest.[8] In hypovolaemic pre-hospital trauma patients,[9] reported weak or normal radial pulses were associated with mean systolic

arterial pressures of 102 and 128 mm Hg, respectively, suggesting that a weak radial pulse was over-reported. In palliative care patients, a normal radial pulse, as distinct from weak or absent, was associated with a systolic arterial pressure of 90 mm Hg or more with a sensitivity and specificity of 84% and 82%, respectively.[10]

Central pulses

The absence of a carotid or femoral pulse might appear to be a logical threshold for starting chest compressions in anaesthetised patients, but reliability is still relatively poor. Dick and colleagues[11] investigated the ability of emergency medical technicians and paramedics to detect a carotid pulse in anaesthetised patients before (pulsatile) and during (non-pulsatile) cardiopulmonary bypass. Only 16.5% could determine the carotid pulse status within 10 s, and only one (2%) participant correctly identified within 10 s that a carotid pulse was absent. In pulseless patients, the absent pulse was not detected within 60 s in 10% of checks. In patients with a radial systolic intra-arterial pressure higher than 80 mm Hg, 45% of participants did not detect a central pulse. There was some evidence that training and experience improved this clinical skill. The European Resuscitation Council guidelines state 'Only those experienced in ALS should try to assess the carotid pulse whilst simultaneously looking for signs of life. This rapid assessment should take no more than 10 s. Start CPR if there is any doubt about the presence or absence of a pulse'.[8] Absence of a femoral or carotid pulse is also not necessarily diagnostic of absent aortic blood flow: in human cardiac arrest, chest compressions are frequently started in the presence of an ongoing pulsatile aortic pressure. Paradis measured aortic pressures during a brief pause in chest compressions in patients with presumed pulseless electrical activity (PEA) cardiac arrest in whom there was no femoral or carotid pulse.[12] In 20% of patients there was spontaneous pulsatile aortic pressure (mean 5–58 mm Hg), although the pulse pressure was only 2–14 mm Hg.

Hypotension

As the reliability of pulse palpation is relatively poor, systolic arterial pressure below a certain value is arguably a more certain and objective indicator of inadequate circulation and the need for chest compressions. NAP6 examined more than 260 cases of life-threatening anaphylaxis, all of which were associated with hypotension; chest compressions were universal when anaesthetists deemed cardiac arrest had occurred, but uncommon during severe hypotension.[1,13] Chest compressions were performed in 100% of patients deemed to have PEA cardiac arrest, in 50% with an unrecordable blood pressure, and in 9% and 2% of patients whose lowest recorded blood pressure was <50 and 51–60 mm Hg, respectively. In almost 40% of cardiac arrests, the submitting anaesthetist reported preceding 'prolonged hypotension' (although neither prolonged nor hypotension were defined). Considered logically, this begs the question: what degree of hypotension should trigger chest compressions in an anaesthetised patient?

After reviewing this large cohort, and consultation with experts in resuscitation, the NAP6 authors recommended that chest compressions be started if systolic arterial pressure decreases to less than 50 mm Hg,[1] and described this state as *profound hypotension*. The NAP6 panel noted that this degree of hypotension is very likely to be associated with pulselessness. A small study of patients with hypotension caused by hypovolaemic shock reported that pulses became impalpable in the order of radial, carotid, then femoral.[14] In the group with all three pulses present, radial systolic intra-arterial pressure was invariably above 50 mm Hg, whereas radial systolic intra-arterial pressure below 50 mm Hg was invariably associated with an absent radial pulse. Therefore, in the *unanaesthetised* but unconscious patient, a blood pressure below 50 mm Hg would meet the criteria for initiating chest compressions. Should this not also be the case when the patient is anaesthetised? At present this is not routine practice. Using a systolic blood pressure of below 50 mm Hg as a threshold for starting chest compressions, the NAP6 authors reported that chest compressions were delayed in more than 50% of cases.[1]

Cerebral perfusion and hypotension

Chest compressions to augment blood flow and blood pressure should logically start at the arterial pressure below which organ (especially brain) perfusion is significantly impaired. It is well established that organ blood flow exhibits some degree of autoregulation, such that perfusion tends to be preserved over a range of MAP values.[15,16] Co-morbidities and concurrent medications, including anaesthetic agents, can adversely affect these compensatory mechanisms.[17] Autoregulation of the cerebral vasculature can be inferred from multichannel continuous-wave near-infrared spectroscopy. A recent review challenged the common belief that cerebral blood flow is constant when MAP is 60–150 mm Hg.[18] The available data indicate that the relationship is not flat and that rapid changes in MAP are more likely to defeat autoregulation, which is further impaired by hypoxaemia and hypercarbia.

The impact of anaesthesia on cerebral autoregulation is further complicated as it varies with agent and setting, and much of our knowledge comes from animal experiments.[16] General anaesthesia reduces cerebral oxygen requirement and might have neuroprotective effects.[19] Co-morbidities and acute brain disease or injury further affect thresholds for reduced brain perfusion or risk of hypoxic–ischaemic injury. There is increasing evidence that hypotension during anaesthesia is associated with adverse outcomes; a recent systematic review suggested that an episode of MAP below 50 mm Hg lasting as little as 5 min is associated with a high risk of organ injury.[20] Importantly, if MAP decreases to less than 40 mm Hg, however briefly, the risk of acute kidney injury increases almost four-fold, and if it persists for longer than 5 min the risk of myocardial injury increases more than four-fold and of death two-fold. A MAP of 40 mm Hg, the reported threshold for organ injury during anaesthesia, is likely to be associated with a systolic arterial pressure of approximately 55 mm Hg, although this can vary a little according to the algorithm used by the measurement device and clinical factors.

Radial artery systolic pressure overestimates systolic pressure in the ascending aorta by up to 35 mm Hg,[21] further supporting the proposed NIBP systolic pressure threshold of 50 mm Hg to initiate immediate chest compressions.

How reliable is NIBP measurement in low blood pressure states?

The thesis that profound hypotension should be used as a threshold for starting chest compressions in anaesthetised

patients presupposes that NIBP measurements are accurate during such hypotension. This is important as arterial pressure monitoring during anaesthesia is most frequently noninvasive, with intra-arterial pressure monitoring used in only a minority of cases. Lehman and colleagues[22] performed more than 27 000 pairwise comparisons of concurrent invasive and NIBP measurements in critically ill patients. Below 95 mm Hg, systolic pressure was over-estimated by noninvasive measurement in comparison with invasive arterial pressure, and this disparity was more marked if hypotension was severe: at a systolic intra-arterial of 60 mm Hg, the systolic NIBP was 10 mm Hg higher. Thus, when an NIBP monitor records a systolic pressure of 50 mm Hg, the true value is probably significantly lower and likely closer to 40 mm Hg. This is not merely of theoretical importance; in patients with a systolic NIBP of 70 mm Hg or less the prevalence of acute kidney injury was greater than with the same arterial pressure measured invasively.[22]

Capnography

Capnography is a valuable tool for assessing cardiac output and is a potential contender as a threshold modality for starting cardiac compressions during anaesthesia. The effect of changes in cardiac output on end-tidal carbon dioxide ($PECO_2$) has been studied. Jin and colleagues[23] investigated the relationship between cardiac index (CI), MAP, and $PECO_2$ in several forms of shock in a porcine model and showed a correlation in haemorrhagic, septic, and cardiogenic shock, with the highest correlation in the latter. However, the confidence intervals of the differences were large, and $PECO_2$ began to decrease only when the CI had decreased by about a third. At the peak of hypovolaemia, $PECO_2$ had decreased by approximately 30%, and MAP and CI by ~50%. Further work is necessary to characterise the relationship between arterial pressure and $PECO_2$ before one is able to propose a threshold value of $PECO_2$, below which cardiac compressions should be started in severe perioperative cardiovascular collapse. Ventilatory factors may further complicate the issue, for example if there is concurrent bronchospasm.

Will chest compressions cause harm?

Although among hypovolaemic patients, a palpable pulse will be unlikely if systolic arterial pressure is less than 50 mm Hg,[14] it remains possible that some patients, for instance if they are vasodilated, will have a weak pulse. In such patients, it is unlikely that chest compressions will cause significant harm. Informed by expert consensus, not primary evidence, European Resuscitation Council guidelines state: 'Delivering chest compressions to a patient with a beating heart is unlikely to cause harm. However, delays in diagnosing cardiac arrest and starting CPR will adversely affect survival and must be avoided'.[8] A study of out-of-hospital CPR in the USA investigated possible harm associated with chest compressions in situations in which a trained emergency dispatcher gave telephone instructions to a bystander.[24] The telephone criteria for cardiac arrest were unconsciousness and abnormal breathing. In 247 patients who had received chest compressions but were not in cardiac arrest, none experienced visceral injury, only 2% sustained any injury (mostly rib fractures), and 12% experienced subsequent discomfort. The overall risk of harm would appear to be very low, compared with the undoubted risk of delaying chest compressions when they are needed.

There are likely many caveats to our recommendation. The appropriate threshold may vary with the intravascular volume status and the cause of cardiovascular collapse. Echocardiography, both transthoracic and transoesophageal, is increasingly available and, in skilled hands, can aid diagnosis and guide resuscitation once cardiac compressions have started. Further considerations also include human factors challenges, such as persuading or leading the surgical team in starting chest compression. Despite these challenges, we believe the fundamental logic described above holds true, as supported by two recent publications. First, the recently updated International Liaison Committee on Resuscitation (ILCOR) Utstein framework for reporting in-hospital cardiac arrest data and outcomes discusses using a systolic arterial pressure of less than 50 mm Hg for identifying PEA cardiac arrest, and using a pressure above 50 mm Hg as a marker for return of a spontaneous circulation.[25] Second, recent international consensus recommendations for the management of perioperative allergic reactions include a recommendation to start chest compressions when systolic arterial pressure is less than 50 mm Hg.[26] Finally, we accept that chest compressions and ongoing advanced life support will not guarantee improved survival. Current guidelines suggest extracorporeal CPR should be considered,[8] but in most anaesthetic settings this is not available.

To summarise, we suggest that relying on the absence of a pulse to diagnose the need for chest compressions in an anaesthetised patient is unreliable and unlikely to be practically useful. It is logical that there is a blood pressure in the anaesthetised patient below which the balance of risk *vs* benefit favours starting chest compressions. There are no studies that define blood pressure, nor are they likely to be undertaken, so it is necessary to extrapolate from existing evidence. Treatment to reverse the cause of profound hypotension should of course be initiated first. In an anaesthetised patient whose arterial pressure is measured noninvasively, the degree of hypotension will be underestimated, and when NIBP is less than 50 mm Hg the actual systolic arterial pressure is likely to be ~10 mm Hg lower, and the patient is likely to be pulseless. The MAP is therefore likely to be below 40 mm Hg, and this level of hypotension is associated with increased organ injury and mortality. Although currently the threshold systolic arterial pressure below which chest compressions should start is uncertain, we propose that a threshold of 50 mm Hg is logical.

Authors' contributions

Writing of the first draft of the manuscript: NJNH.
Writing of the subsequent and final drafts of the manuscript: NJNH.
Co-wrote subsequent and final drafts: JPN, JS, TMC.
Original proposal: TMC.

Declarations of interest

JS is chair of the International Liaison Committee on Resuscitation Advanced Life Support Task Force (unpaid) and receives payment from Elsevier as an editor of *Resuscitation*. JPN is chairman of the European Resuscitation Council and receives a payment from Elsevier as editor-in-chief of

Resuscitation. TMC is an associate editor of the *British Journal of Anaesthesia*. NJNH has no conflicts to declare.

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