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Original Article

Advanced life support interventions during intra-operative cardiac arrest among adults as reported to the 7th National Audit Project of the Royal College of Anaesthetists

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Summary

Background Few existing resuscitation guidelines include specific reference to intra-operative cardiac arrest, but its optimal treatment is likely to require some adaptation of standard protocols.

Methods We analysed data from the 7th National Audit Project of the Royal College of Anaesthetists to determine the incidence and outcome from intra-operative cardiac arrest and to summarise the advanced life support interventions reported as being used by anaesthetists.

Results In the baseline survey, > 50% of anaesthetists responded that they would start chest compressions when the non-invasive systolic pressure was < 40–50 mmHg. Of the 881 registry patients, 548 were adult patients (aged > 18 years) having non-obstetric procedures under the care of an anaesthetist, and who had arrested during anaesthesia (from induction to emergence). Sustained return of spontaneous circulation was achieved in 425 (78%) patients and 338 (62%) were alive at the time of reporting. In the 365 patients with pulseless electrical activity or bradycardia, adrenaline was given as a 1 mg bolus in 237 (65%). A precordial thump was used in 14 (3%) patients, and although this was associated with return of spontaneous circulation at the next rhythm check in almost three-quarters of patients, in only one of these was the initial rhythm shockable. Calcium (gluconate or chloride) and 8.4% sodium bicarbonate were given to 51 (9%) and 25 (5%) patients, but there were specific indications for these treatments in less than half of the patients. A thrombolytic drug was given to 5 (1%) patients, and extracorporeal cardiopulmonary resuscitation was used in 9 (2%) of which eight occurred during cardiac procedures.

Conclusions The specific characteristics of intra-operative cardiac arrest imply that its optimal treatment requires modifications to standard advanced life support guidelines.

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Introduction

The circumstances under which peri-operative, particularly intra-operative, cardiac arrest occurs typically differ from most other in-hospital cardiac arrests. Although many advanced life support interventions applicable to in-hospital cardiac arrest are relevant to both the intra-operative and ward settings, in many cases, optimal treatment of intra-operative cardiac arrest may require modifcation of these generic advanced life support interventions [1].

While some expert groups have published consensus recommendations for the treatment of peri-operative cardiac arrest [2–5], the 2021 European Resuscitation Council guidelines include only a few sentences on the topic [6] and the 2020 American Heart Association guidelines make no reference to peri-operative cardiac arrest [7]. Patients undergoing surgical procedures are closely and continuously monitored, and causes of unexpected intra-operative cardiac arrest may be promptly reversible, particularly when detected immediately (e.g. a relative overdose of induction drug, a vagotonic response to pneumoperitoneum or sudden arrhythmia). If arterial blood pressure is monitored continuously using an indwelling cannula, sudden changes in blood pressure can be detected almost immediately and a pulsatile waveform may be seen even if peripheral and central pulses are no longer palpable. These are just some of the features of intra-operative cardiac arrest that may warrant adjustment of the doses of resuscitation drugs and indications for starting chest compressions.

The 7th National Audit Project of the Royal College of Anaesthetists (NAP7) studied peri-operative cardiac arrest in the four nations of the UK. The project provided an opportunity to study current practices in the resuscitation of patients with intra-operative cardiac arrest. We have analysed the resuscitation interventions delivered during intra-operative cardiac arrests reported to NAP7 and compared these with existing guidelines.

Methods

The methods of the NAP7 project are described in detail elsewhere [8]. In summary, all UK NHS hospitals and a subset of independent sector hospitals undertaking anaesthesia care in the UK were invited to take part in the project. A network of local co-ordinators was established to lead the project in each hospital. A national activity survey examined anaesthetic practices and complications in all cases undertaken in NHS hospitals over a 4-day period and provided a denominator for the NAP7 study of approximately 2.71 million anaesthetic interventions annually in the UK [9, 10]. The activity survey collected data only on the survival of the event and not overall hospital survival. A 1-year registry collected details of peri-operative cardiac arrests occurring from first anaesthetic contact until 24 h after discharge from immediate anaesthesia care via an anonymous, protected and encrypted online database. Detailed structured patient reports were anonymised, and each patient was reviewed by a panel of clinicians and lay representatives [8]. The included patients formed the numerator for the study and their review enabled quantitative and qualitative analyses. As part of the case review process, patients were classified by specialty and themes of importance enabling topic-specific analysis (with associated numerator and denominator data). The main results and full project are published separately [11–15]. This paper includes several new analyses not in the main report, but the primary analysis focuses on intra-operative cardiac arrest, defined as adult patients (aged > 18 y) having non-obstetric procedures who had arrested during anaesthesia (from induction to emergence) while under the care of an anaesthetist.

Results

The NAP7 baseline survey included hypothetical questions on when anaesthetists would consider starting chest compressions. Of the 10,746 respondents, approximately 80% indicated they would use a threshold blood pressure to determine when to start chest compressions (Fig. 1). For a patient who was ASA physical status 2, aged 50 y and who did not have invasive arterial blood pressure monitoring, > 50% of respondents would start cardiopulmonary resuscitation when the systolic blood pressure was < 40 mmHg. For a patient who was ASA physical status 3, aged 75 y, with a history of hypertension and who did not have invasive arterial blood pressure monitoring, > 50% of respondents would start cardiopulmonary resuscitation when the systolic blood pressure was < 50 mmHg. In both hypothetical cases, two-thirds of respondents indicated they would use a very low end-tidal carbon dioxide value as an indication to start chest compressions.

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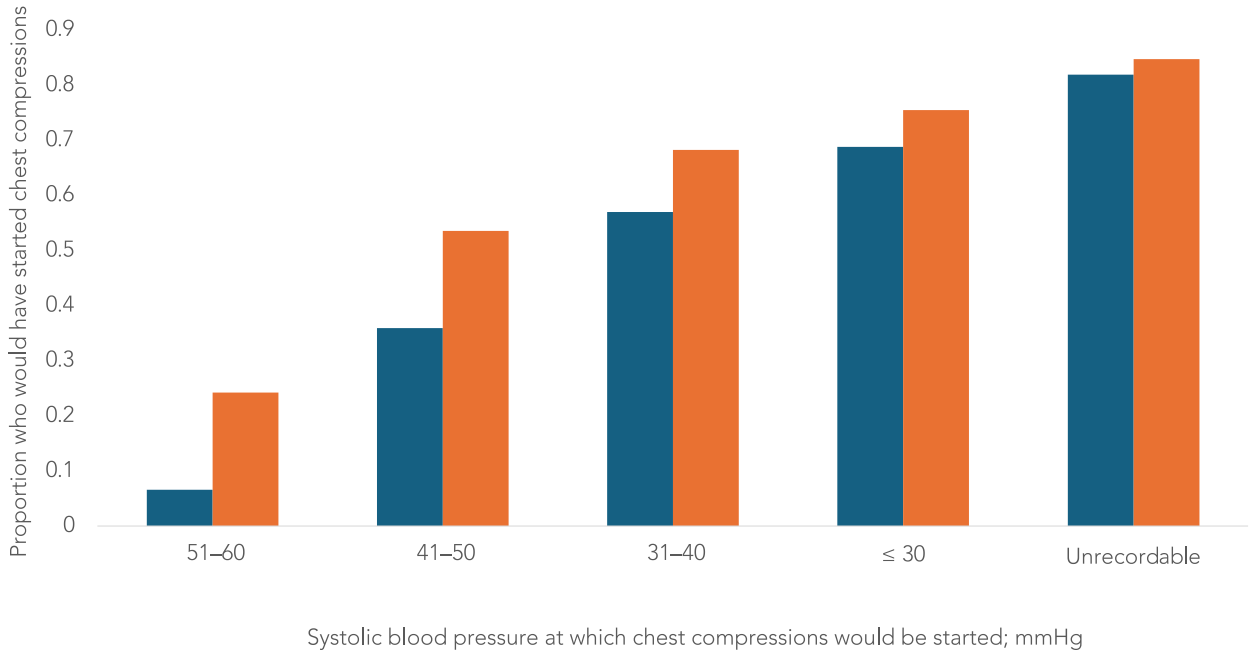


Figure 1 Comparison of systolic blood pressure triggers for starting chest compressions in a 50-year-old patient, ASA physical status 2, without invasive blood pressure monitoring (blue) (n = 8799) vs. a 75-year-old patient, ASA physical status 3, with a history of hypertension, without invasive blood pressure monitoring (orange) (n = 9098).

Table 1 Initial cardiac arrest rhythm for 548 adult intra-operative cardiac arrest patients. Values are number (proportion).

Initial cardiac arrest rhythm	
Pulseless electrical activity	298 (55%)
Asystole	93 (17%)
Bradycardia	67 (12%)
Ventricular fibrillation	32 (6%)
Pulseless ventricular tachycardia	38 (7%)
Unknown	20 (4%)

Of the 881 NAP7 patients, 548 were adult patients (aged > 18 y) having non-obstetric procedures who had arrested during anaesthesia (from induction to emergence) while under the care of an anaesthetist (online Supporting Information Appendix S2). The following results relate only to these patients. Based on NAP7 denominator data for the same case mix, the approximate incidence of intra-operative cardiac arrest is 3 in 10,000 (95%CI 2.6–3.0 in 10,000) [9]. Sustained return of spontaneous circulation (ROSC) was achieved in 425 (78%) patients, and 338 (62%) were alive at the time of reporting to NAP7 (online Supporting Information Appendix S3). Of the 548 patients, 458 (84%) had an initial non-shockable rhythm, 70 (13%) had a shockable rhythm and in 20 (4%), the initial rhythm was unknown (Table 1). Among the 365 patients with an initial rhythm of pulseless electrical activity (PEA) or bradycardia, the three most common triggers for starting cardiopulmonary resuscitation were: an impalpable pulse (208, 57%); severe hypotension (171, 47%); and a reduction in end-tidal carbon dioxide (91, 25%) (Fig. 2). In 159 (44%) patients in the PEA/bradycardic cohort, the blood pressure most proximate to starting chest compressions was documented (Fig. 3 and online Supporting Information Appendix S4).

The panel agreed causes of intra-operative cardiac arrest (primary and secondary) included: major haemorrhage (112, 20%); tension pneumothorax (7, 1%); and cardiac tamponade (5, 1%) (online Supporting Information Appendix S5).

Of the 548 patients with intra-operative cardiac arrest, 279 (51%) had invasive blood pressure monitoring. The proportion of patients who were ASA physical status 4 or 5 was greater among those with invasive blood pressure monitoring (123, 44%) compared with those without (59, 22%). Median (IQR [range]) systolic blood pressure before starting chest compressions among the PEA/bradycardia cohort was 50 (40–88 [0–160]) mmHg in 98 patients with invasive blood pressure monitoring and 60 (40–80 [0–190]) mmHg in the 61 patients with non-invasive blood pressure monitoring. Blood pressure was reported as 0 in 5 (5%) and 12 (20%) patients, respectively. Cardiopulmonary resuscitation was started within 1 min in 153 (81%) of the

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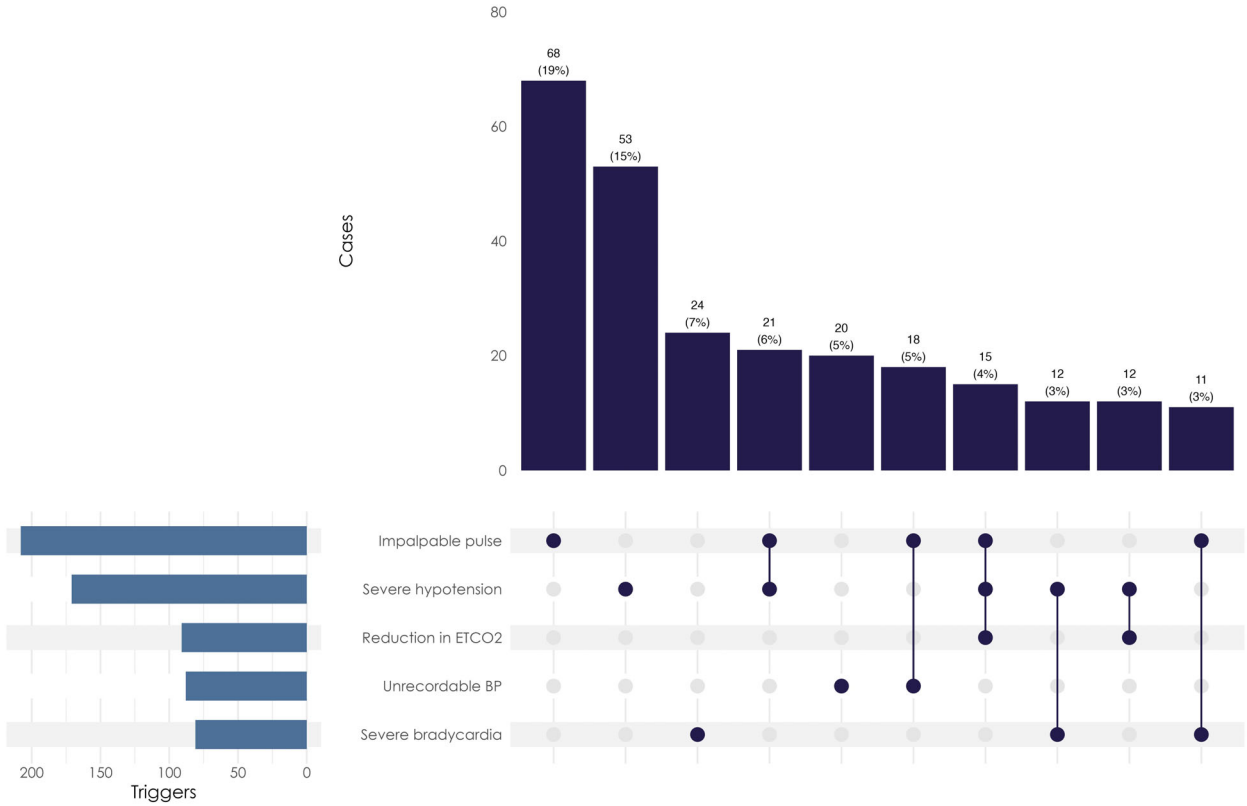


Figure 2 Indications for starting chest compressions in 365 adult, non-obstetric patients reported to NAP7 with intra-operative cardiac arrest and an initial rhythm of pulseless electrical activity or bradycardia. Horizontal bars represented the total number of patients for which a trigger was cited. The vertical bars show the 10 most frequent combinations of triggers. $ETCO_2$, end-tidal carbon dioxide; BP, blood pressure.

PEA/bradycardia cohort with invasive blood pressure monitoring compared with 123 (70%) of those without. There was no significant difference in the last recorded systolic blood pressure before starting chest compressions and rate of ROSC. Median (IQR [range]) systolic blood pressure with and without ROSC was 50 (40–80 [0–150]) mmHg vs. 60 (40–100 [0–190]) mmHg, respectively, $p = 0.113$. The rate of ROSC was lower among patients with invasive blood pressure monitoring as compared with those without: 197 (71%) vs. 228 (86%), respectively, $p < 0.001$. At the time of reporting, 147 (53%) patients who had invasive blood pressure monitoring were alive, compared with 191 (71%) who had non-invasive blood pressure monitoring.

Among the 548 patients with intra-operative cardiac arrest, cardiopulmonary resuscitation quality was guided by: waveform capnography in 446 (81%); arterial pressure waveform in 273 (50%); and a quality feedback device in 17 (3%).

A defibrillatory shock was delivered to 98 (18%) of the 548 patients who had intra-operative cardiac arrests, and 13 (13%) of these received more than four shocks. Delivery of a precordial thump was documented in 14 (3%) patients and was associated with ROSC at the next rhythm check in 10 (71%). A precordial thump was delivered in < 1 min to 13 (93%) patients and within 2–3 min in the remaining patients (Table 2).

Of the 435 (79%) patients given adrenaline, it was administered within a minute in 329 (76%). In the 365 patients with PEA/bradycardia, adrenaline was given as a 1 mg bolus in 237 (65%), titrated aliquots in 96 (26%), an infusion in 30 (8%) and no adrenaline was administered in 43 (12%) (online Supporting Information Appendix S6).

Overall, calcium and sodium bicarbonate were given to 51 (9%) and 25 (5%) patients, respectively. Of the 112 patients who had major haemorrhage causing cardiac arrest, calcium was given to 20 (18%) and sodium bicarbonate to 8 (7%). There were 12 patients where severe hyperkalaemia was reported, with 3 (25%) receiving calcium without sodium bicarbonate and 4 (33%) receiving both calcium and sodium bicarbonate. No patients included hypocalcaemia in the narrative. Among the 51 patients who

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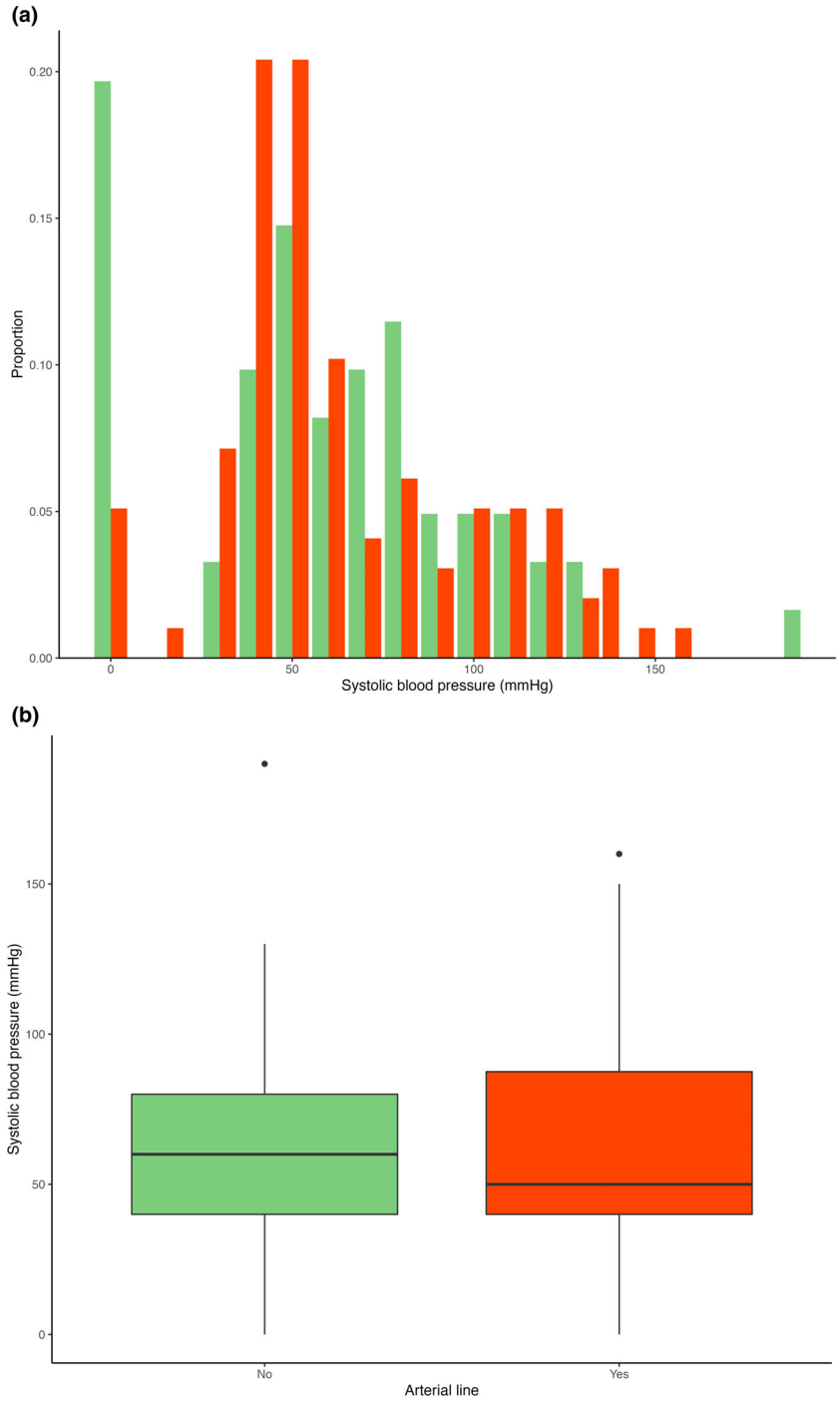


Figure 3 (a) Recorded blood pressure most proximate to starting chest compressions among 159 (44%) patients with a pulseless electrical activity/bradycardia cardiac arrest. (b) The same results displayed using a box and whisker chart. The lower and upper margins of the boxes correspond to the 25th and 75th percentiles, respectively. The whiskers extend to the farthest point within 1.5 times the IQR of the box margin. Red, with invasive blood pressure monitoring (n = 98); green, without invasive blood pressure monitoring (n = 61). See online Supporting Information Appendix S4 for results in tabular form.

received calcium there was a specific indication in 27 (53%). Patients receiving calcium were less likely to survive the resuscitation attempt compared with all other reported peri-operative cardiac arrests (56% vs. 78%) and less likely to leave hospital alive (26% vs. 46%). Among the 25 patients given sodium bicarbonate, a specific indication

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Table 2 Initial rhythms and use of the precordial thump in 548 adult intra-operative cardiac arrest patients.

Initial monitored rhythm	Cases of precordial thump	Return of spontaneous circulation at next rhythm check	Outcome reported to NAP7		
			Survived to hospital discharge	Alive in hospital	Died
All cases	14	10	8	6	0
Shockable rhythms	3	1	2	1	0
Ventricular fibrillation	2	1	1	1	0
Pulseless ventricular tachycardia	1	0	1	0	0
Non-shockable rhythms	11	9	6	5	0
Pulseless electrical activity	1	1	1	0	0
Asystole	8	6	4	4	0
Severe bradycardia	2	2	1	1	0

(hyperkalaemia or severe acidosis) was identified by the panel in 8 (32%). There were 19 patients in whom a severe metabolic acidaemia was reported, and 5 (26%) of these were given sodium bicarbonate.

A thrombolytic drug was administered to 5 (1%) of the 548 patients, two of whom had ROSC but all of whom died within 24 h. In all but one of the patients, the cause of cardiac arrest was thought likely to have been a pulmonary embolism, with the remaining case thought to have been caused by myocardial infarction (based on intra-operative echocardiography performed by a cardiologist). Echocardiography was used during resuscitation in 92 (17%) of the 548 patients. Of these, 14 (15%) were patients having cardiac surgery, and 19 (21%) occurred in the cardiac catheterisation laboratory. In the patients in whom echocardiography was used, cardiac tamponade was reported as the cause of cardiac arrest in 3 (3%) patients.

Of the 9 (2%) patients who underwent extracorporeal cardiopulmonary resuscitation (ECPR) with veno-arterial extracorporeal membrane oxygenation (VA-ECMO): five were having cardiac surgery (two survivors); two developed cardiac arrest in the cardiac catheterisation laboratory (both died); one had a cardiac arrest during a pacemaker change (and survived); and one went into cardiac arrest during liver transplant surgery (and died).

Discussion

The answers to hypothetical questions in the NAP7 baseline survey indicate that 80% of anaesthetists would use a threshold blood pressure to determine when to start chest compressions, but among the 548 NAP7 adult, non-obstetric patients with an initial rhythm of PEA or bradycardia, severe hypotension was said to be a trigger to start chest compressions in less than half of patients. Adrenaline was given as a 1 mg bolus in two-thirds of these patients. A precordial thump was used in only 3% but was associated with ROSC at the next rhythm check in almost three-quarters of patients. Calcium and sodium bicarbonate were given to 51 (9%) and 25 (5%) of patients, and ECPR was used in just 2% of cardiac arrests.

In adults during general anaesthesia, it has been suggested that chest compressions should be started if the systolic blood pressure decreases and remains < 50 mmHg despite interventions [16, 17]. The NAP7 project has provided the first data to indicate how often anaesthetists use profound hypotension as a threshold for starting chest compressions in clinical practice. A systolic blood pressure < 50 mmHg is thought likely to be associated with impalpable pulses [18], and the benefit from the additional cerebral and coronary blood flow produced by chest compressions is considered to outweigh the risk of harm from chest compressions [16].

Our data confirm that among those patients with invasive blood pressure monitoring, the median systolic blood pressure before starting compressions was 50 mmHg and in only five patients was the blood pressure documented as 0 mmHg before starting compressions. Among those patients without invasive blood pressure monitoring, although the last recorded blood pressure before starting compressions was slightly higher (median 60 mmHg), there were more patients with a pre-compression blood pressure of 0 mmHg. It is possible that in the presence of invasive blood pressure monitoring, anaesthetists are more confident in letting the blood pressure decrease before starting compressions but are more likely to intervene before the blood pressure approaches zero.

Although several observational studies using CT imaging have documented rib fractures in approximately

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70% of patients receiving cardiopulmonary resuscitation for out-of-hospital cardiac arrest [19, 20], a study that included 247 individuals who received bystander chest compressions but were not in cardiac arrest, reported only six (2%) patients who experienced resuscitation-related injuries [21]. However, we are unaware of any data on the incidence of injuries from intra-operative chest compressions. The rate of ROSC and proportion of patients alive at the time of reporting were lower among patients with invasive blood pressure monitoring, but this most likely reflects differences in case mix (as evidenced by the higher proportion of patients who were ASA physical status 4 or 5).

Although standard advanced life support guidelines recommend a dose of 1 mg adrenaline, which is injected after the third shock in ventricular fibrillation/pulseless ventricular tachycardia (VF/pVT), or immediately after cardiac arrest from a non-shockable rhythm [22], the optimal dose of adrenaline to treat cardiac arrest is unknown [23]. Smaller doses are likely appropriate when adrenaline is first given for profound hypotension, when there is a high probability of a low flow state during PEA or severe bradycardia, or when there is a very short time between the onset of cardiac arrest and the injection of adrenaline. With continuous intra-operative monitoring, cardiac arrest should be diagnosed immediately and treated rapidly, with the likelihood of restoring spontaneous circulation very quickly. Under these circumstances, giving a 1 mg bolus of adrenaline may lead to severe hypertension and tachyarrhythmias, and this was reported in at least one of the NAP7 patients. The Resuscitation Council UK suggests that for peri-operative cardiac arrest, adrenaline is given initially in increments (e.g. 50–100 μg intravenously) rather than a 1 mg bolus, but if 1 mg in total has been given with no response, that further intravenous adrenaline doses of 1 mg are given [24]. Australasian guidelines for resuscitation after cardiac surgery include similar recommendations [25]. It appears that many anaesthetists are unaware of the guidance for smaller intravenous doses of adrenaline when used very early in a PEA peri-operative cardiac arrest, as most NAP7 patients were given a 1 mg dose.

The European Resuscitation Council [22] and Resuscitation Council UK [24] do not recommend routine use of a precordial thump to treat cardiac arrest but suggest that a single precordial thump may be appropriate for a witnessed and monitored VF/pVT cardiac arrest while awaiting a defibrillator. The International Liaison Committee on Resuscitation (ILCOR) does not recommend use of the precordial thump in established cardiac arrest [26]. A single precordial thump has a very low success rate for cardioversion of a shockable rhythm. Two evaluations of the precordial thump delivered to patients undergoing electrophysiological studies showed that it successfully terminated malignant ventricular arrhythmias (VF or VT) in just 1 of 80 patients [27] and 2 of 155 patients [28], respectively. In each of these patients, the arrhythmia was VT. None of the 49 patients with VF in these two studies were terminated by a precordial thump. In a study of VF/pVT out-of-hospital cardiac arrests, a precordial thump delivered to 103 patients resulted in ROSC in five patients (three VF and five pVT), but the rhythm deteriorated in another 10 patients [29]. In an Italian study of a precordial thump in the treatment of 144 patients with an out-of-hospital cardiac arrest, the intervention resulted in ROSC in just three patients, and in all of these patients, the initial rhythm was asystole [30]. Among the NAP7 patients, a precordial thump was associated with ROSC at the next rhythm check in 10 of 14 patients, but 9 of the 10 patients were initially non-shockable rhythms. Given that other interventions, including chest compressions, were also taking place, it is not clear if the precordial thump contributed to ROSC or whether this occurred irrespective of this intervention. Our observational data add very low certainty evidence to support the use of a precordial thump for witnessed and monitored non-shockable rhythm cardiac arrest.

Calcium is recommended as a treatment for cardiac arrest associated with hyperkalaemia, hypocalcaemia or calcium channel blocker overdose, but is not recommended as a routine treatment during cardiac arrest [22, 31]. Among NAP7 patients with intra-operative cardiac arrest, the largest group (39%) receiving calcium were patients with a major haemorrhage as a cause of their cardiac arrest. Low ionised calcium can be caused by the rapid transfusion of blood components containing citrate, although this is uncommon when liver function is normal [32]. Calcium is used to protect the heart in severe hyperkalaemia [33], and this accounted for about 14% of cardiac arrest patients where calcium was given. That calcium increases the rate of ROSC in hyperkalaemic cardiac arrest has been challenged by a blinded randomised study showing no effect of calcium on hyperkalaemic cardiac arrest in pigs [34]. Among the 51 NAP7 patients in which calcium was administered, there was no specific indication reported in almost half of these. Our finding is consistent with data from a study of in-hospital cardiac arrests in the USA, which showed that calcium is given in approximately 25% of patients [35]. Calcium was administered in 20% of the remaining 333 NAP7 patients not included in this intra-operative cohort, which aligns

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closely with the findings of the study from the USA. A recent randomised controlled trial documented a decreased rate of ROSC with calcium compared with placebo in out-of-hospital cardiac arrest (19% vs. 27%, RR 0.72, 95%CI 0.49–1.03, p = 0.09) [36]. During ischaemia, adenosine triphosphate depletion results in high cytosolic and mitochondrial concentrations of calcium, which may contribute to ischaemic and reperfusion injury [37]. The injection of exogenous calcium may exacerbate this injury and could reduce the rate of ROSC. Although NAP7 data cannot rule out benefits or harms from the use of calcium for intra-operative cardiac arrest, the available evidence suggests that it should not be used unless there is a clear indication.

Sodium bicarbonate is recommended as a treatment for cardiac arrest associated with hyperkalaemia or caused by an overdose of drugs with quinidine-like effects [6]. Despite not being recommended for routine use during cardiac arrest, it is given in almost 50% of in-hospital cardiac arrests in the USA [35]. Five of the NAP7 patients were given sodium bicarbonate to treat severe metabolic acidosis, but whether the correction of acidaemia with sodium bicarbonate during cardiopulmonary resuscitation is helpful or harmful is unknown. The potentially harmful effects of sodium bicarbonate include: a negative inotropic effect on an ischaemic myocardium; the delivery of a large, osmotically active, sodium load; and a shift to the left in the oxygen dissociation curve. In a blinded randomised study, sodium bicarbonate increased the rate of ROSC in a porcine model of hyperkalaemic cardiac arrest [34].

In two of the four NAP7 cardiac arrest patients given a thrombolytic drug for presumed pulmonary embolism, the diagnosis was made on the basis that intra-operative echocardiography showed right ventricular dilatation. However, the right ventricle frequently dilates during cardiac arrest, even in the absence of pulmonary embolism [38], and the decision to give thrombolysis intra-operatively is made even more challenging because of the risk of bleeding. Under these circumstances, if it is available, VA-ECMO will enable restoration of the circulation and oxygenation while definitive treatment is organised [2, 6]. Catheter-guided mechanical thrombectomy has a greater role in treating this type of case in centres where it is feasible [39]. The use of ECPR was uncommon among the NAP7 patients, and in all but one of the nine patients, it was used to treat patients undergoing cardiac procedures.

The NAP7 baseline survey documented that VA-ECMO or ECPR was available in only 18 (9%) hospitals, and all but three of these were cardiac surgery centres [14]. The most recent ILCOR recommendation for ECPR for in-hospital cardiac arrest suggests that it may be considered as a rescue therapy for selected patients when conventional cardiopulmonary resuscitation is failing to restore spontaneous circulation in settings where this can be implemented [40]. Recent European guidelines on cardiac arrest in the peri-operative period suggest that open chest cardiac massage be considered if ROSC has not been achieved with closed chest cardiac compression and ECPR is not available [2]. These European guidelines also suggest immediate use of resuscitative endovascular balloon occlusion of the aorta (REBOA) in patients with exsanguinating infra-diaphragmatic haemorrhage uncontrollable by other means. Neither open chest cardiac massage nor REBOA was used in any of the intra-operative cardiac arrests reported to NAP7. A recent randomised controlled trial of the use of emergency department REBOA in patients with trauma and who were exsanguinating showed increased mortality compared with standard care [41]. Depending on the cause and timing of the haemorrhage, these trial findings may not be generalisable to intra-operative major haemorrhage when treatments may start very early.

The incidence of intra-operative cardiac arrest among adults having non-obstetric procedures is 3 in 10,000 (95% CI 2.6–3.0 in 10,000). More than 80% of intra-operative cardiac arrests have an initial non-shockable rhythm. Among the NAP7 patients with PEA or bradycardia, despite guidelines that suggest giving intravenous adrenaline initially in increments of 50–100 μg, adrenaline was given as a 1 mg intravenous bolus in two-thirds of patients. We suggest that the specific characteristics of intra-operative cardiac arrest warrant modifications to standard advanced life support guidelines.

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Supporting Information

Additional supporting information may be found online via the journal website.

Appendix S1. NAP7 collaborators.

Appendix S2. Characteristics and case details for 548 included patients.

Appendix S3. Cardiac arrest characteristics and outcomes for 548 included patients.

Appendix S4. Results from Figure 3 in tabular form.

Appendix S5. Panel-agreed primary cause of intra-operative cardiac arrest in 548 adult patients.

Appendix S6. Drugs given to treat intra-operative cardiac arrest.

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