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# Controversies in perioperative cardiac arrest



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### Key findings

- A precordial thump was used in 18 (2%) of cases of perioperative cardiac arrest reported to NAP7 and there was a pulse at the next rhythm check in 13 (72%) of these cases.
- A precordial thump was most effective when there was a witnessed and monitored non-shockable cardiac arrest (12 of 14 cases).
- Calcium was used in 117 (13.3%) of the 881 NAP7 cases.
- Bicarbonate was used in 63 (7.2%) of the 881 NAP7 cases.
- Nineteen (2.2%) of the 881 NAP7 cases received extracorporeal CPR (eCPR) and all were in specialist adult or paediatric cardiac surgery centres.
- A thrombolytic drug was injected in 9 (1%) of the 881 NAP7 cases.
- Echocardiography was used during resuscitation in 160 (18.2%) of the 881 NAP7 cases.

### Precordial thump

### What we already know

Since 2015, the European Resuscitation Council guidelines (Soar 2015) and Resuscitation Council UK (RCUK 2021) guidelines have not recommended routine use of a precordial thump but have suggested that a single precordial thump may be appropriate for a witnessed and monitored ventricular fibrillation (VF) or pulseless ventricular tachycardia (pVT) cardiac arrest while awaiting a defibrillator. This guidance remains in current guidelines and the current Resuscitation Council UK Advanced Life Support (ALS) course manual. This is because a single precordial thump has a very low success rate for cardioversion of a shockable rhythm but the chances of causing harm are very small (Amir 2007; Haman 2009; Pellis 2009; Kohl 2005; Nehme 2013; Dee 2021).

### What we found

Over the one-year case reporting period, delivery of a precordial thump was documented in 18 (2%) adult cases of 881 perioperative cardiac arrest cases and was associated with a return of spontaneous circulation (ROSC) in 13 cases (72%) (Table 15.1). Beyond this observation, there are too few cases to make detailed observations on the specific characteristics of patients and settings where a precordial thump was used. Of the 18 cases reported:

- the ages ranged from 26 to 85 years
- 12 patients (71%) were male
- 13 occurred during general anaesthesia
- 16 patients also had chest compressions
- in 13 cases, there was ROSC at the next pulse check following delivery of the precordial thump; however, as other interventions were also taking place it is not clear whether the precordial thump contributed to ROSC or whether this would have occurred irrespective of this intervention
- duration of cardiac arrest was generally shorter than other cardiac arrests (< 10 minutes, 89% vs 67%).</li>

We did not identify any evidence of harm caused by a precordial thump. Outcomes in this group were generally good. This was despite it being a group judged to have had poor prearrest care by the NAP7 panel (22% poor vs 11% for all cases and overall poor care 17% vs 2.1%). Of the 18 patients who had a precordial thump, all survived the resuscitation attempt and 8 (44%) went home, 9 (50%) were still in hospital and 1 (6%) died.



#### Table 15.1 Initial rhythms and use of precordial thump

Initial monitored rhythm	Cases of precordial thump (n)	Return of spontaneous circulation at next rhythm check		Outcome when reported to NAP7: survived to
		(n)	(%)	hospital discharge/alive in hospital/died (n)
All cases	18	13	72	8/9/1
Shockable rhythm				
VF/pVT	4	1	25	2/2/0
VF	3	1	33	2/1/0
ρVT	1	0	0	0/1/0
Non-shockable rhythms	14	12	86	6/7/1
PEA	4	4	100	1/2/1
Asystole	8	6	75	4/4/0
Severe bradycardia	2	2	100	1/1/0
PEA pulsaless alactrical activity: pVT pulsaless vantricular tachycardia: VE vantricular fibrillation				

PEA, pulseless electrical activity; pVT, pulseless ventricular tachycardia; VF, ventricular fibrillation.

### Discussion

Given that the precordial thump is no longer routinely taught in life support courses and its use has been de-emphasised in recent guidelines, we were not surprised that it was used in only 2% of cardiac arrest cases reported to NAP7. Furthermore, our findings are in keeping with previous studies that suggest a precordial thump may be more useful for witnessed nonshockable cardiac arrest rhythms. A review of 103 cases of ventricular fibrillation (VF)/pulseless (pVT) out-of-hospital cardiac arrest (OHCA) given a precordial thump documented a rhythm change in 17 cases, but immediate ROSC occurred in just 5 cases; the rhythm deteriorated in 10 cases (Nehme 2013). There are several reported cases of the successful use of a precordial thump in witnessed asystole (Pellis 2010), which is consistent with the cases we documented in NAP7. We cannot be certain whether the precordial thump was responsible for ROSC in the 13 NAP cases or whether ROSC occurred in response to other interventions (eg chest compressions) before the next rhythm check. Our observational data add some very low certainty evidence to support the use of a precordial thump for witnessed and monitored non-shockable rhythm cardiac arrest (eq severe bradycardias progressing to asystole).

A patient having general anaesthesia developed asystole during a pacemaker change. The cardiologist gave several precordial thumps, but no chest compressions or drugs, before a heart rhythm was restored. A new pacing wire was then inserted. The reviewers thought that the precordial thumps were probably beneficial in this case. When several precordial thumps are given, this could be a form of percussion pacing. An elderly patient having elective upper-limb surgery developed a profound bradycardia and became pulseless after induction of general anaesthesia. The patient was treated with a single precordial thump, chest compressions, ephedrine and glycopyrrolate and had a palpable pulse at the next rhythm check a few minutes later. The patient made a good recovery and was discharged. The reviewers thought that any additional benefit of the precordial thump was uncertain.

### Recommendations

### National

Resuscitation guideline writers should review the role of the precordial thump given the potential for benefit in witnessed and monitored non-shockable rhythm cardiac arrest.

#### Individual

 Precordial thump should not delay other more evidencebased methods of resuscitation including cardiopulmonary resuscitation (CPR) and administration of relevant drugs.

### Calcium What we already know

Calcium is currently recommended as treatment for cardiac arrest associated with hyperkalaemia, hypocalcaemia or calcium channel blocker overdose. However, calcium is not recommended as a routine treatment for cardiac arrest (Soar 2021b). Major haemorrhage with massive transfusion is associated with hypocalcaemia mainly because of the citrate in fresh frozen plasma and blood.



A 2023 systematic review of administration of calcium compared with no calcium during cardiac arrest in adults or children identified three randomised controlled trials (RCTs) with 554 adult patients with OHCA, eight observational studies with 2,731 adult cardiac arrest patients, and three observational studies with 17,449 children with in-hospital cardiac arrest (IHCA; Hsu 2023). This review found that calcium use did not improve outcomes in adults or children.

Even though current guidelines do not advise the routine use of calcium in cardiac arrest, it is given in approximately 25% of IHCAs in the United States and its use is increasing (Moskowitz 2019). In the Calcium for Out-of-hospital Cardiac Arrest (COCA) RCT, 397 patients with OHCA received up to two doses of 5 mmol calcium chloride or saline (Vallentin 2021). The primary outcome, ROSC, occurred in 19% of patients in the calcium group compared with 27% in the saline group (risk ratio, RR, 0.72, 95% confidence interval, CI, 0.49 to 1.03; P = 0.09). A prespecified subanalysis of patients with pulseless electrical activity (PEA), who are more commonly given calcium, revealed that ROSC occurred in 20% of patients in the calcium group compared with 39% in the saline group (RR 0.51, 95% CI 0.26 to 1.0; (Vallentin 2022). During ischaemia, adenosine triphosphate depletion results in high cytosolic and mitochondrial concentrations of calcium, which may contribute to ischaemic

**Table 15.2** Causes of cardiac arrest in patients receiving calcium

 and with five or more cases reported

Den el e en e d'erene	Cases		
Panei-agreed cause	(n)	(%)	
Specific indication:			
Major haemorrhage	34	29	
Significant hyperkalaemia	15	13	
Hypocalcaemia	2	1.7	
No specific indication:			
Septic shock	16	14	
Isolated severe hypotension*	13	11	
Cardiac ischaemia	11	9.4	
Bradyarrhythmia	7	6	
Cardiac tamponade 7 6			
* Central vasopressors considered/started.			

and reperfusion injury. The injection of exogenous calcium may exacerbate this injury and could account for the reduced rates of ROSC in the COCA trial.

In children, data from the American Heart Association's Get With the Guidelines – Resuscitation registry and ICU-RESUScitation project showed that calcium use during CPR for children with and without heart disease having an IHCA was common and associated with worse survival (Dhillon 2022; Cashen 2023).

### What we found

Calcium use was documented in 117 (13.3%) of the 881 NAP7 case reports. The panel-agreed causes of cardiac arrest in cases when calcium was given are shown in Table 15.2. Two cases included hypocalcaemia in the narrative. There were 167 cases of major haemorrhage causing cardiac arrest and calcium was given in 34 (20%) of these cases. There were 23 cases where severe hyperkalaemia was reported: 15 (65%) received calcium and 7 received both calcium and bicarbonate. Overall, in 58 (49.6%) reports in which calcium was administered there was a specific indication and in 59 (50.4%) there was not.

A patient underwent a rapid sequence induction and tracheal intubation. The heart rhythm changed to VT with a heart rate above 170 beats/minute soon after induction. There was initially a pulse, but this quickly deteriorated to pVT. The patient's preoperative plasma potassium value was 3.0–3.4 mmol/l. A return of spontaneous circulation was achieved with a single shock from a defibrillator. A dose of 10 ml 10% calcium chloride was also injected. The reviewers could find no indication for this calcium administration.

The surgical specialties of patients receiving calcium for cardiac arrest are shown in Figure 15.1. The cardiac arrest rhythms for the 117 patients receiving calcium were similar to the whole group of patients with cardiac arrest (Table 15.3). Compared with reports to NAP7 in which calcium was not given, patients who did receive it were more likely to be young children (age < 5 years 13.7% vs 5.1%), highly comorbid (ASA 4–5 50% vs 34%) and of non-white ethnicity (22% vs 11%). Cardiac arrests that included administration of calcium were more likely to occur after leaving recovery (32% vs 18%), in critical care (27% vs 12%) and to be prolonged (> 20 minutes 47% vs 20%).

Patients receiving calcium were less likely to survive the resuscitation attempt compared with all other reported perioperative cardiac arrests (56% vs 78%) and less likely to leave hospital alive (26% vs 46%). There were similar proportions of survivors still admitted (15% vs 17%) and more in-hospital deaths in patients receiving calcium (59% vs 37%).



Figure 15.1 Surgical specialty of patients receiving calcium for cardiac arrest. ENT, ear nose and throat; GI, gastrointestinal; NA, not answered.

Table 15.3 Initial monitored rhythm in patients receiving calcium

Rhythm	Patients receiving calcium		All cases
	(n)	(%)	(%)
Asystole	12	10	15
Bradycardia	15	13	15
Pulseless electrical activity	6	59	52
Pulseless ventricular tachycardia	7	6.0	5.6
Unknown	7	6.0	5.1
Ventricular fibrillation	7	6.0	6.5

An elderly patient having an elective laparoscopic procedure under general anaesthesia had a severe vasovagal episode on intra-abdominal insufflation. The patient was given chest compressions, adrenaline and calcium. The patient was resuscitated successfully and survived to go home. The panel could not identify any indication for the calcium in this case.

An elderly patient developed pulseless ventricular tachycardia following a rapid sequence induction that included thiopentone and suxamethonium. The patient was successfully resuscitated following defibrillation and a dose of calcium. There was no hyperkalaemia. The panel could not identify any indication for the calcium in this case.

#### Discussion

In the majority of perioperative cardiac arrests reported to NAP7, there was no clear and obvious indication for calcium during cardiac arrest. Although we did not specifically ask about hypocalcaemia in the NAP7 case review form, it is unlikely that these large numbers of cases were associated with hypocalcaemia.

The largest group (29%) receiving calcium was patients with a major haemorrhage as a cause of their cardiac arrest. A low ionised calcium can be caused by rapid transfusion of blood components containing citrate, although this is uncommon when liver function is normal (UK Blood Services 2014). However, in haemorrhagic shock requiring massive transfusion, liver function is often impaired by hypoperfusion (Rossaint 2023). Calcium is used to protect the heart in severe hyperkalaemia (Alfonzo 2020), and this accounted for about 8.5% of cardiac arrest cases where calcium was given.

The specialty using calcium most commonly during cardiac arrest is cardiac surgery. Calcium is used during cardiac anaesthesia to improve cardiac function because of its inotropic effects when weaning patients from cardiopulmonary bypass (Lomivorotov 2020). However, there are also concerns that the use of calcium may be harmful. A multicentre RCT studying whether intravenous calcium chloride reduces the need for inotropic support after cardiopulmonary bypass weaning is currently in progress (Lomivorotov 2021).

Calcium use was seen disproportionately in paediatric cases, in cases where the arrest took place on critical care, including paediatric critical care, and in prolonged resuscitation.

NAP7 data suggest a relative two-fold overuse of calcium compared with guidelines. Overall, these patients had a poorer outcome than other NAP7 cases, although this may be confounded by case mix. Calcium is not part of current guidelines for cardiac arrest outside the specific circumstances of severe hyperkalaemia and hypocalcaemia (Hsu 2023). The most recent RCT of calcium in cardiac arrest suggested possible harm from calcium use (Vallentin 2021, 2022). Although the NAP7 data cannot rule out benefits or harms from the use of calcium for perioperative cardiac arrest, the available evidence suggests that it should not be used unless there is a firm indication.

### Recommendations

#### Individual

Calcium should not be given to patients in cardiac arrest unless there is a very specific indication such as hyperkalaemia or hypocalcaemia (Lott 2021).

### **Bicarbonate**

### What we already know

Bicarbonate is not recommended as a routine treatment for cardiac arrest (Soar 2021b). It is currently recommended as treatment for cardiac arrest associated with hyperkalaemia or caused by overdose of drugs with quinidine-like effects (eq tricyclic antidepressants, neuroleptics; Lott 2021). Even though current quidelines do not advise the routine use of bicarbonate in cardiac arrest, it was given in almost 50% of IHCAs in the United States in 2016 and its use is increasing (Moskowitz 2019). Observational studies of the impact on outcome of bicarbonate use in OHCA have reported conflicting results (Kawano 2017; Kim 2016) but these studies are subject to significant confounding, not least because of resuscitation time bias (the longer the resuscitation attempt, the worse the outcome but the more likely that advanced life support interventions are to be delivered; Andersen 2018). A systematic review and metaanalysis of the effectiveness of bicarbonate in OHCA and IHCA included six observational trials (18,406 patients) and documented no significant differences between bicarbonate and no bicarbonate groups in ROSC (OR 1.19; 95% CI 0.68-2.07) or survival to hospital discharge (odds ratio, OR, 0.3; 95% CI 0.07 to 1.32; Wu 2020). Bicarbonate is frequently given to correct severe acidaemia in critically ill patients, although there is very little evidence that this beneficial (Coppola 2021).

### What we found

Administration of bicarbonate was documented in 63 (7.2%) of the 881 NAP7 cases. Of these 63 cases, the panel-agreed cause of cardiac arrest was significant hyperkalaemia in 5 (8%) cases. The panel-agreed causes of cardiac arrest in cases when bicarbonate was given are shown in Table 15.4.

There were 40 NAP7 cases for which a severe metabolic acidosis was reported and 10 (24%) of these were given bicarbonate. Of 23 cases where severe hyperkalaemia was reported, 7 (30%) received bicarbonate. All 7 also received calcium.

**Table 15.4** Causes of cardiac arrest in patients receiving bicarbonate and with five or more cases reported, and frequency of these causes in all NAP7 reports

Panel-agreed cause	Patients receiving bicarbonate		All cases
	(n)	(%)	(%)
Major haemorrhage	16	25	14
Septic shock	13	21	6.3
Cardiac ischaemia	12	19	6.7
Significant hyperkalaemia	7	11	1.2
Bradyarrhythmia	5	8	8.6

Compared with patients not receiving bicarbonate, those given bicarbonate were more commonly very young (age < 1 year, 17% vs 3.3%), severely comorbid (ASA 5 17% vs 6.5%), of nonwhite ethnicity (25% vs 9%) and their treatment involved cardiac surgery (11% vs 1%) or interventional cardiology (18% vs 0.5%). Arrests in this group occurred more commonly in locations external to theatres (60% vs 27%). including critical care or paediatric intensive care (33% vs 12%) and were more prolonged (> 20 minutes, 50% vs 30%). The surgical specialties are shown in Figure 15.2, duration of cardiac arrest in Figure 15.3. Patients receiving bicarbonate tended to have a longer duration of cardiac arrest than those patients not receiving bicarbonate.

Initial cardiac arrest rhythms were similar to those among patients not receiving bicarbonate. Patients receiving bicarbonate were less likely to survive the resuscitation attempt compared with all other reported perioperative cardiac arrests (62% vs 75%) and less likely to leave hospital alive (25% vs 45%). There were similar proportions of survivors still admitted (14% vs 17%) and more in-hospital deaths in patients receiving bicarbonate (60% vs 38%).

Quality of care, as judged by the panel, was similar for patients given or not given bicarbonate.



### Discussion

Bicarbonate was used in a significant proportion (7.2%) of cardiac arrests reported to NAP7. Indications for its use were rare and in most cases the panel identified no clear indication for its use. Bicarbonate was used more in haemorrhage and sepsis and in cardiac arrests outside the operating theatre (including adult and paediatric intensive care).

On reperfusion of the donor liver during liver transplant surgery, the patient developed a very high potassium value on blood gases and had an asystolic cardiac arrest. Cardiopulmonary resuscitation was started and bicarbonate was given during advanced life support that also included chest compressions, fluids, adrenaline and calcium – this was associated with a return to a normal potassium value. ROSC was achieved after 10–15 minutes of resuscitation and the patient survived. An insulin and glucose infusion was not required. The reviewers identified this as a case where there was an indication for giving bicarbonate – severe hyperkalaemia in the setting of a severe metabolic acidosis.

An adult classed as ASA 2 undergoing general anaesthesia for an elective urological procedure developed a PEA cardiac arrest. CPR was commenced and ROSC was achieved after 11 minutes. Drugs given during resuscitation included adrenaline 5 mg and bicarbonate 100 ml. The panel could see no indication for the bicarbonate. The NAP7 data show that use of bicarbonate was associated with longer duration of cardiac arrest – patients who have a prolonged cardiac arrest are more likely to have a severe metabolic acidosis. Whether correction of acidaemia with bicarbonate during CPR is helpful or harmful is unknown. The potentially harmful effects of bicarbonate include (Neumar 2010):

- a negative inotropic effect on an ischaemic myocardium
- the delivery of a large, osmotically active, sodium load to an already compromised circulation and brain
- a shift to the left in the oxygen dissociation curve, further inhibiting release of oxygen to the tissues.

Further data may be provided by the continuing Bicarbonate for In-Hospital Cardiac Arrest trial, a randomised, double-blind, placebo-controlled trial (<u>clinicaltrials.gov</u> NCT05564130). Pending the results of this trial, treatment should focus on highquality CPR and treating the underlying cause of cardiac arrest; bicarbonate is probably not helpful.

### **Recommendations**

### Individual

Bicarbonate should not be given to patients in cardiac arrest unless there are specific indications, such as hyperkalaemia and overdose of drugs with quinidine-like effects (eg tricyclic antidepressants, neuroleptics; Lott 2021).



Figure 15.2 Surgical specialty of patients receiving bicarbonate for cardiac arrest. ENT, ear nose and throat; GI, gastrointestinal; NA, not answered.

Specialty



**Figure 15.3** Duration of cardiac arrest in patients receiving bicarbonate. The blue bars represent cases of cardiac arrest receiving bicarbonate and the purple line all cases of cardiac arrest not receiving bicarbonate. A blue bar extending above the purple ling indicates over representation of bicarbonate use in that group, and under the line, underrepresentation.

### Extracorporeal CPR

### What we already know

Extracorporeal CPR (eCPR) should be considered for a patient in refractory cardiac arrest where there is a potentially reversible cause and when the expertise to deliver eCPR is available (Soar 2021b). Three recent RCTs of the use of eCPR for OHCA have produced conflicting results. Two of these trials were terminated prematurely after predetermined interim analyses: one because of superiority of eCPR (Yannopoulos 2020) and the other because of its futility (Belohlavek 2022). The most recent trial showed no difference in 30-day favourable functional outcome, the primary outcome (Suverein 2023). The effectiveness of eCPR is likely highly dependent on patient selection and the experience of clinicians and centres delivering the intervention; as such, it is a challenging intervention to study in an RCT. Intraoperative cardiac arrest is usually a monitored event and so there should be minimal delay in starting CPR and, in many cases, there are potentially reversible causes. Under these circumstances, if cardiac arrest is refractory to appropriate treatment, and if eCPR is available, it may enable perfusion of organs while the precipitating cause is treated (Lott 2021). The most recent International Liaison Committee on Resuscitation recommendation for eCPR for in-hospital cardiac arrest suggests that it may be considered as a rescue therapy for selected patients when conventional CPR is failing to restore spontaneous circulation in settings where this can be implemented (weak recommendation, very low certainty evidence; Berg 2023). The most recent international data from the Extracorporeal Life Support Organization Registry for the year 2022 documented 14,509 adult eCPR cases with a 30% survival to discharge, 6,179 paediatric eCPR cases with a 41% survival to discharge, and 2,619 neonatal eCPR cases with a 43% survival to discharge (ELSO 2023).

 Table 15.5 Centres providing extracorporeal membrane oxygenation/

 extracorporeal cardiopulmonary resuscitation (ECMO/eCPR) services in UK

	Survey response (n)	ECMO/ eCPR (n)	Proportion (%)
Cardiac surgery centres (total)	27	15	56
Adult	22	10	46
Paediatric (with PICU)	10	8	80
Non-cardiac surgery centres (total)	168	3	1.8
Adult	164	3	1.8
Paediatric hospitals	144	1	0.7
Paediatric hospitals with PICU	11	1	9.1
Total	195	18	9.2

ECMO, extracorporeal membrane oxygenation; eCPR, extracorporeal cardiopulmonary resuscitation; LC, Local Coordinator; PICU, paediatric intensive care unit.

### What we found

### **Baseline Survey**

Extracorporeal membrane oxygenation (ECMO) or eCPR) was available in 18 (9.2%) hospitals (Table 15.5). Of the 27 (13.7%) hospital sites that reported being cardiac surgery centres, 15 (55.6%) offered ECMO or eCPR.

### Case reports

Of the 881 NAP7 cases, 19 (2.2%) received eCPR; 10 were children (18% of paediatric NAP7 cases): 4 neonates and 5 children aged 1–17 years. All but one of these cases involved cardiac surgery and were placed on cardiopulmonary bypass while in cardiac arrest. The remaining child treated with eCPR went into pVT after scoliosis surgery. Of the nine adults (1.1% of adult NAP7 cases) who underwent eCPR, five were cardiac



Figure 15.4 Time from cardiac arrest to establishing extracorporeal cardiopulmonary resuscitation eCPR – cumulative number of cases

surgical patients (two survived), two developed cardiac arrest in the cardiac catheterisation laboratory (both died), one had a cardiac arrest during a pacemaker change (survived) and the other went into cardiac arrest during liver transplant surgery (died).

In total, 15 patients (79%) had a circulation restored and resuscitation efforts were stopped in 4 patients. Four patients (21%) survived to hospital discharge, nine (47%) died and six (32%) were still in hospital at time of reporting.

In 4 cases (21%) the decision for eCPR was made immediately; in 10 cases (53%) it was made within the first 10 minutes. The cumulative time from cardiac arrest to establishing eCPR flow is shown in Figure 15.4.

The aorta was the most common site of arterial cannulation and was used in 10 cases (53%), followed by the right femoral artery (5 cases, 26%), left femoral artery (3, 16%), common carotid artery (1, 5.3%) and other sites (2, 11%). The duration of ECMO support is shown in Table 15.6.

The reasons for stopping ECMO were recovery (9 cases, 47%), diagnosis incompatible with life (5 cases, 26%) or multiple organ failure (4 cases, 21%).

Some 11 complications of ECMO were reported in 9 (42 %) cases: leg ischaemia (1 case), compartment syndrome (2), surgical



site bleeding requiring return to theatre (2), intracranial haemorrhage (2), hypoxic ischaemic brain injury (1), multiple thrombus (1), unable to achieve flows (1) and uncertain complication (1). Six cases had one complication, one case had two complications, and one case had three complications. Table 15.6 Duration of extracorporeal membrane oxygenation

	Patients		
Duration	( <i>n</i> )	(%)	
< 24 hours	7	37	
24 to < 48 hours	1	5.3	
48 to < 72 hours	3	16	
3-5 days	2	11	
6-7 days	3	16	
> 7 days	1	5.3	
N/A (continuing)	2	11	

A patient with complex heart disease had a PEA cardiac arrest, most likely caused by anaphylaxis, during an interventional cardiology procedure. Initial resuscitation was unsuccessful and eCPR was started about 30 minutes after cardiac arrest. The patient remained on ECMO for the next few days and recovered from the cardiac arrest. The panel commented that this case highlighted the potential value of eCPR in refractory cardiac arrest in settings where it is feasible.

### Discussion

To the best of our knowledge, very few hospitals in the UK have the facilities to provide eCPR outside a specialist cardiac centre. Although eCPR is of considerable interest, only 19 (2.2%) of NAP7 cases received it.

eCPR was notably more common in children (18%) compared with adults (1.1%). Of the 10 children or neonates, all but one was in a cardiac surgery setting. Given the high cost and complexity of setting up an eCPR programme, in contrast to the many other high-income countries, it is very unlikely that the provision of eCPR will change in the UK in the near future.

### Recommendations

### Individual

In patients with perioperative cardiac arrest who are refractory to conventional resuscitation and who have a potentially reversible cause, consider eCPR if it is available and feasible.

### Thrombolysis

### What we already know

Despite only very low certainty and conflicting evidence from observational studies, thrombolysis is recommended if cardiac arrest is known or suspected to have been caused by pulmonary embolism (PE; Lott 2021; Konstantinides 2020). It is usually difficult to definitely diagnose PE as a cause of cardiac arrest: the circumstances of the cardiac arrest may increase the likelihood of PE as a cause (eq sudden collapse in the relatively immobile postoperative patient with additional risk factors for thromboembolism) and echocardiography findings of right ventricular dilatation may be suggestive, but neither of these provides a definitive diagnosis. The right ventricle frequently dilates during cardiac arrest even in the absence of PE. Another challenge facing clinicians in deciding whether to treat perioperative cardiac arrest with a thrombolytic drug is the risk of bleeding. If a thrombolytic drug is given to treat cardiac arrest caused by a PE, CPR may need to be continued for up 60–90 minutes to give sufficient time for the clot to dissolve (Lott 2021; Konstantinides 2020). This is a situation where a mechanical chest compression device would be useful if staff are familiar with its use and can deploy it without prolonged interruption to CPR. The use of mechanical thrombectomy by interventional radiology or eCPR may, in settings where these are feasible, also have a role in treating cardiac arrest caused by a PE (Soar 2021a).

### What we found

A thrombolytic drug was injected in nine (1%) of the 881 NAP7 cases. ROSC was achieved in four of these patients, but all died within 24 h. In all but one of the cases, the cause of cardiac arrest was thought likely to have been a PE; the remaining case was thought to have been caused by myocardial infarction (based on intraoperative echocardiography performed by a cardiologist). The diagnosis of PE was made with CT pulmonary angiography in just one case (in which cardiac arrest occurred after cemented hemiarthroplasty). In two cases of intraoperative cardiac arrest, the diagnosis of PE was presumed because echocardiography demonstrated right ventricular dilatation. In four cases, cardiac arrest occurred on the ward or ICU within 24 h postoperatively. In all these cases, thrombolysis was given because of presumed, but not definitively confirmed, PE. In one case, PEA cardiac arrest occurred during liver transplantation and the cause was blood clot seen in the right heart; ROSC was achieved after thrombolysis.

There was a single case of a postoperative sudden cardiac arrest in a ward patient who died. There was a suspicion of a PE, but thrombolytic drugs were not readily available and were therefore not given.

A patient had a ward cardiac arrest following major abdominal and pelvic cancer surgery. There was a focused ultrasound during CPR and thrombolysis was given. There was a prolonged resuscitation attempt (1-2 h) without restoration of circulation and the patient died. The panel view was that thrombolysis was reasonable in this patient given the risk factors for thromboembolism.

### Discussion

In at least one of the NAP7 cases, PE was thought to be a possible cause of intraoperative cardiac arrest but the extensive surgery being undertaken (with associated bleeding) was thought to make the bleeding risk too high. If a thrombolytic drug is given to treat cardiac arrest caused by a PE, CPR may need to be continued for up 60–90 minutes to give sufficient time for the clot to dissolve (Lott 2021; Konstantinides 2020). All the patients who had thrombolysis died within 24 h. It is likely that, in the future, catheter-guided mechanical thrombectomy will have a greater role in treating this type of case in centres where it is feasible (Soar 2021a).

### **Recommendations**

#### Local

Thrombolytic drugs should be readily available to give in an emergency – their location and guidance for use should be signposted on resuscitation trolleys or cardiac arrest drug boxes.

### Individual

In patients with perioperative cardiac arrest in whom pulmonary embolism is a likely cause, consider giving a thrombolytic drug. The decision to do this will depend on the balance between the likelihood of massive pulmonary embolism as a cause of cardiac arrest and the risk of uncontrollable bleeding.



Figure 15.5 Specialties where echocardiography was used during cardiac arrest. ENT, ear nose and throat; GI, gastrointestinal; NA, not answered.

## Echocardiography during resuscitation

### What we already know

Although point of care ultrasound is being used increasingly during cardiac arrest to diagnose the cause of cardiac arrest and to provide information on reversibility, there is no high-certainty evidence for its benefit for either of these indications (Reynolds 2022; Wyckoff 2022; Soar 2020). Ultrasound equipment is likely to be more available at the site of a perioperative cardiac arrest than in many other locations in hospital; this is particularly the case for cardiac surgical patients and those undergoing interventional cardiological procedures, where there is also greater expertise in the use of cardiac ultrasound.

### What we found

Echocardiography was used during resuscitation in 160 (18.2%) of the 881 NAP7 cases. Of these 160 cases, 38 (23%) were cardiac surgical cases and 27 (17%) occurred in the cardiac catheterisation laboratory (Figure 15.5). In the cases in which echocardiography was used, cardiac tamponade was reported as the cause of cardiac arrest in six (3.8%) cases and was thought to be contributory in two (1.2%) cases. In addition, a tension pneumothorax was identified as causing cardiac arrest in one case (0.6%).

### Discussion

The increasing use of echocardiography during cardiac arrest parallels the increasing availability of point of care ultrasound. In our cases, it was uncertain whether the use of echocardiography improved the care of the patient and identified a reversible cause (eight cases had cardiac tamponade and one case had a pneumothorax, about 6% of cases where echocardiography was used). It may be helpful for diagnosing the cause of cardiac arrest and/or identifying any pathology that may be treatable, although this will depend on circumstances. In postoperative cardiac surgical patients where there is a high pretest probability of cardiac tamponade, the diagnostic utility of echocardiography may be high but in other contexts it may merely interrupt chest compressions while having a very low diagnostic yield. There are important pitfalls. For example, during cardiac arrest there is commonly right ventricular dilatation, which may lead to the incorrect assumption that the cardiac arrest has been caused by PE (Reynolds and Del Rios 2020). Echocardiography can be used to distinguish true PEA (in which there is electrical activity on the ECG and no cardiac motion on echocardiography) from a low-flow pseudo-PEA (in which some cardiac motion is seen on echocardiography, but the cardiac output is insufficient to generate a palpable pulse). Cardiac arrest caused by severe hypovolaemia, including anaphylaxis for example, is likely to result in 'pseudo-PEA' (see Chapter 25 ALS for perioperative cardiac arrest). In general, the prognosis of pseudo-PEA is better than that of true PEA (Gaspari 2021).

### **Recommendations**

### Individual

If point of care echocardiography and staff experienced in its use and limitations are immediately available, consider its use to diagnose reversible causes of perioperative cardiac arrest.



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