

Best evidence topic - Cardiac general

Should adrenaline be routinely used by the resuscitation team if a patient suffers a cardiac arrest shortly after cardiac surgery?

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Summary

A best evidence topic in cardiac surgery was written according to a structured protocol. The question addressed was whether adrenaline might be a useful addition to a protocol for the management of cardiac arrests for patients shortly after cardiac surgery. Altogether 889 papers were found using the reported search, of which 16 represented the best evidence to answer the clinical question. The authors, journal, date and country of publication, patient group studied, study type, relevant outcomes and results of these papers are tabulated. The quality and level of evidence was assessed using the International Liaison Committee on Resuscitation guideline recommendations. We conclude that the European Resuscitation Council and the American Heart Association both recommend 1 mg of adrenaline as soon as pulseless electrical activity or asystole is identified or after the second failed shock if the rhythm is VF/pulseless VT. However, they acknowledge that the evidence behind this recommendation is lacking and based entirely on animal studies which have as yet not been successfully replicated in human studies to show a benefit of survival to hospital discharge. They acknowledge that the current evidence is insufficient to support or refute the use of adrenaline in arrests and the International Liaison Committee on Resuscitation grade the recommendation to give adrenaline in cardiac arrests as 'indeterminate'. Thus, in the particular situation of a patient who arrests shortly after cardiac surgery where the chance of restoring sinus rhythm either by defibrillation or by an emergency re-sternotomy is high, and where adrenaline could in this situation be highly dangerous once sinus rhythm is restored, we recommend that 1 mg of adrenaline forms no part of the resuscitation protocol for patients who arrest after cardiac surgery.

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Keywords: Thoracic surgery; Cardiopulmonary resuscitation; Epinephrine; Adrenaline; Evidence based medicine**1. Introduction**

A best evidence topic was constructed according to a structured protocol. This is fully described in the ICVTS [1]. The quality of each study was assessed using the International Liaison Committee on Resuscitation 2005 protocol [2].

2. Three-part question

In [patients who have arrested shortly after cardiac surgery] does [the routine administration of 1 mg of adrenaline] improve [survival]?

3. Clinical scenario

A 72-year-old patient suffers a cardiac arrest 1 h after triple coronary artery bypass graft. The rhythm is pulseless electrical activity (PEA). The nursing staff commence cardiac massage and follow the European Resuscitation Council guideline which is to give 1 mg of adrenaline immediately for pulseless electrical activity (PEA) or asystole. The

surgeon is rapidly available and performs an emergency re-sternotomy within 3 min. On reopening there is a considerable gush of blood and a tamponade is relieved. Sinus rhythm returns but unfortunately the blood pressure rapidly rises to 250/150 due to the adrenaline, and several of the proximal graft anastomosis sutures cut through. After redoing both top-ends and oversewing the aortic and venous cannulation sites, you wonder what possible benefit adrenaline was in that arrest scenario.

4. Search strategy

Medline-1950 to Nov-2007 using OVID interface.

[adrenaline.mp OR epinephrine.mp OR exp Epinephrine/] AND [exp Resuscitation/OR resuscitation.mp OR exp Cardiopulmonary resuscitation/] AND [exp survival/OR survival.mp OR exp Patient discharge/OR discharge.mp].

Embase-1980 to Nov-2007 using the OVID interface. Search repeated replacing keywords epinephrine with adrenaline, patient discharge with hospital discharge, and cardiopulmonary resuscitation with resuscitation.

Cochrane DSR, ACP journal club and DARE searched using the term 'Adrenaline'.

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Table 1
Best evidence papers

Author, date and country Study type (level of evidence)	Patient group	Outcomes	Key results	Comments
Long et al., (2005), Circulation, USA, [5] Systematic review of experimental studies (level 6, excellent)	ILCOR worksheet Hypothesis: Epinephrine (at either standard doses or higher doses) is a safe and effective adjunct to defibrillation in cardiac arrests due to ventricular fibrillation (asystole also considered)	Recommendations	The use of epinephrine in cardiac arrest due to ventricular fibrillation is supported by animal studies: many in the past and 3 in the last 5 years. (level of evidence 6). It does provide adverse consequences as well, and subsequent doses do not appear to provide as dramatic an effect in raising coronary perfusion pressure (and thus chance of ROSC) as the first dose. However, due to the lack of human placebo-controlled trials, epinephrine is Class Indeterminate. For PEA and asystole, the use of epinephrine is also Class Indeterminate Epinephrine, 1 mg IV, given every 3–5 min, is generally accepted as useful in cardiac arrest from all rhythms although no human trials have compared epinephrine to placebo	
Cairns et al., (1998), Resuscitation, USA, [7] Experimental study (level 6, fair)	14 Dogs had induced VF and were left without CPR for 7.5 min CPR was then resumed and Epinephrine and countershocks were as per guidelines from 1998 Success measured as return of spontaneous circulation for 30 min – Epinephrine 1 mg was administered when indicated and at recommended time intervals	ROSC CPP	– $n=11$ animals could not be resuscitated – $n=3$ successfully resuscitated Only 1 animal survived after a second dose of adrenaline In those animals that could not be resuscitated the increase in CPP after EPI averaged only 3 ± 2 mmHg Subsequent doses produced no significant effect on CPP (2 ± 4 mmHg) In those animals successfully resuscitated ($n=3$), the change in CPP was 21 ± 11 mmHg after the first dose of EPI	The hemodynamic response to the first dose of EPI determines if the critical CPP needed for ROSC and survival will occur. Repeat doses of EPI do not appear to improve CPP to a degree to affect clinically meaningful measures of outcome, i.e. successful countershock and survival
Biondi-Zoccai et al., (2003), Resuscitation, Ireland, [13] Meta-analysis mainly experimental studies (level 6, fair)	Search for studies that compared treatment of cardiac arrest using vasopressin with either placebo or adrenaline 2 human studies and 33 animal studies found	ROSC in human studies ROSC in animal studies vasopressin vs. adrenaline ROSC in animal studies vasopressin vs. adrenaline	Vasopressin 63% (78/124) Adrenaline 59% (68/116) $P=0.43$ Vasopressin 84% (225/268) Adrenaline 59% (117/224) $P<0.001$ Vasopressin 93% (98/105) Placebo 19% (14/72) $P<0.001$	Did not search EMBASE Details of individual studies not presented in adequate detail
Klouche et al., (2003), Resuscitation, USA, [8] Experimental study (level 6, fair)	Twenty rats with ventricular fibrillation (VF) untreated for 8 min and then CPR (at a rate of 200 bpm and ventilation at 100 breaths per min) Drug treatment was with: Alpha-MNE in a dose of 100 μ g/kg, Vasopressin in a dose of 0.4 U/kg.	Survival in hours CPP (coronary perfusion pressure)	Alpha-MNE 57 ± 14 h VPN 41 ± 8 h Epinephrine 31 ± 10 h Control 15 ± 6 h Rise to around 30 mmHg in all drug groups. In controls rise was to 20 mmHg	Both post-resuscitation myocardial function and survival were most improved after administration of the selective $\alpha(2)$ -adrenergic agonist, intermediate after vasopressin and least after epinephrine and saline placebo CPR protocol differed to current resuscitation guidelines, as shocks given 4 min after drug

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Table 1 (Continued)

Author, date and country Study type (level of evidence)	Patient group	Outcomes	Key results	Comments
	Epinephrine in a dose of 30 µg/kg, or saline control	Myocardial function (left ventricular pressure, cardiac index)	Post-resuscitation myocardial function and survival were significantly better in animals treated with alpha-MNE (lower LVEDP)	
Lindberg et al., (2000), Resuscitation, Sweden, [9] Experimental study (level 6, fair)	18 Swedish bred pigs with full monitoring after a sternotomy, monitor insertion and chest closure. VF arrest without CPR for 1 min and 5 min of chest compressions using a chest compressor	Coronary perfusion pressure during arrest CO ₂ production during arrest	Control 7 ± 2 mmHg Adrenaline 45 ± 5 mmHg Noradrenaline 38 ± 5 mmHg Control 106 ± 15 ml/min Adrenaline 66 ± 5 ml/min Noradrenaline 62 ± 6 ml/min	Vasopressors increased coronary perfusion pressure and the likelihood of a return of spontaneous circulation, but decreased end-tidal CO ₂ concentration and induced a critical deterioration in cardiac output and thus oxygen delivery in this model of cardiopulmonary resuscitation Doesn't address question directly of whether adrenaline is good in cardiac arrest, just elucidates details of what its physiologic effects are
	Adrenaline or noradrenaline or control given at that stage			
	– Evaluation of changes in end-tidal CO ₂ concentration, CPP after injection of vasopressors during cardiopulmonary resuscitation. End-tidal CO ₂ concentration correlates with pulmonary blood flow	CO ₂ production 30 min after arrest	Control 211 ± 1 ml/min Adrenaline 233 ± 8 ml/min Noradrenaline 173 ± 14 ml/min	
Holmberg et al., (2002), Resuscitation, Sweden, [15] Retrospective cohort study (level 4, unsatisfactory)	A national survey of ambulance organisations in Sweden between 1990–1995 among 14065 patients with out-of-hospital cardiac arrest. Of these, resuscitation was attempted in 10966 cases	% patients who received adrenaline % patients who were intubated Survival in patients with ventricular fibrillation	Adrenaline was given in 42.4% of patients 47.5% of patients were intubated Independent predictor for adverse outcome (OR 0.43, CI 0.27–0.66)	Neither in total nor in any subgroup did they find results indicating beneficial effects of any of these two interventions Due to the retrospective cohort design of the study many factors determine administration of adrenaline including prolonged arrest and also certain ambulance crews were not licenced to give it, therefore, little inference can be drawn from these results
	Multivariate risk factors for adverse outcome analysed	Survival in patients with with a non-shockable rhythm	Independent predictor for lower survival (OR 0.30, CI 0.07–0.82)	
Behringer et al., (1998), Ann Int Med, Austria, [16] Retrospective cohort study (level 4, poor)	178 adults with non-traumatic, normothermic VF cardiac arrest brought to the emergency department with an unsuccessful initial defibrillation The median cumulative epinephrine dose administered was 4 mg (range, 0–50 mg)	ROSC Neurologic outcome assessed by cerebral performance category (CPC) within 6 months after cardiac arrest. A CPC of 1 or 2 was defined as favourable	In 178 patients: a) n = 151 (84%) spontaneous circulation was restored b) n = 63 (42%) had favourable neurologic recovery. Patients with an unfavourable CPC received a significantly higher cumulative dose of epinephrine than did patients with a favourable CPC (4 mg compared with 1 mg)	The authors controlled for the length of resuscitation as a confounder, but despite this, many other confounders might have caused this result. i.e. asystolic patients tend to receive more adrenaline as less time is spent on defibrillation, and asystole is known to have a poorer outcome compared to VF
Vandycke et al., (2000), Resuscitation, Belgium, [12] Meta-analysis of randomised trials (level 1, excellent)	5 randomised trials where high dose of adrenaline was compared vs. standard dose epinephrine in cardiac arrest identified from a	ROSC Survival to hospital admission	Odd ratio favoured High-dose adrenaline 1.14 (1.02–1.27) No difference 1.03 (0.86–1.24)	No statistically significant beneficial effect of high and/or escalating doses of epinephrine in comparison with standard dose of epinephrine in survival

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Table 1 (Continued)

Author, date and country Study type (level of evidence)	Patient group	Outcomes	Key results	Comments
	literature search from 1988–1998	Hospital discharge	Odd ratio was against high dose adrenaline 0.74 (0.53–1.03)	The authors do not describe whether the 5 studies used high-dose adrenaline as a first dose or only after one dose had failed
	Standard dose was usually 1 mg adrenaline			Did not distinguish between rhythms
	High-dose adrenaline was from 5 to 15 mg			
Chen et al., (2007), Am J Emerg Med, China, [10]	47 rabbits with cardiac arrest induced by clamping the endotracheal tube that did not recover with CPR	CPP in adrenaline group CPP in vasopressin group	4–36 mmHg at peak value, $P=0.000$ 9–18 mmHg at peak, value, $P=0.20$	Epinephrine, but not vasopressin, increases survival rates in this adult rabbit asphyxia model
Experimental study (level 6, good)	Randomised to epinephrine group ($n=24$) and vasopressin group ($n=23$)	ROSC after drug administration	Epinephrine 13 of 24 Vasopressin 2 of 23 $P<0.01$	
Ristagno et al., (2007), Crit Care Med, USA, [11]	10 Yorkshire-cross domestic pigs had untreated VF arrest for 3 min	Cerebral cortical microcirculatory blood flow and cortical tissue PO_2 and PCO_2 as indicators of cortical tissue ischaemia	Post resuscitation microvascular flows and P_aO_2 were greater and P_aCO_2 less after vasopressin when compared with epinephrine A significantly greater number of cortical microvessels were perfused after vasopressin	Cortical microcirculatory blood flow was markedly reduced after epinephrine, resulting in a greater severity of brain ischemia after the ROSC in contrast to the more benign effects of vasopressin
Experimental study (level 6, good)	Then randomized to receive central venous injections of epinephrine (30 $\mu\text{g}/\text{kg}$) or vasopressin (0.4 units/kg) at 1 min after the start of cardiopulmonary resuscitation using a thumper	CPP at 4 min of CPR ROSC	Vasopressin 20 ± 2 mmHg Adrenaline 21 ± 6 mmHg Resuscitation successful in all animals	Performed 3 sequential shocks 4 min after arrest and further shocks at 1 min intervals. Not current ACLS algorithm
Wenzel et al., for the European Resuscitation Council Vasopressor during Cardiopulmonary Resuscitation Study Group (2004), New Engl J Med, Austria, [17]	1186 patients with an out of hospital arrest were randomly assigned to: 2 doses of vasopressin (40 IU) or 1 dose of epinephrine (1 mg). Followed by additional treatment with epinephrine if needed	Hospital admission in patients with ventricular fibrillation, pulseless electrical activity Asystole	Vasopressin group 46.2% Epinephrine group 43.0% ($P=0.48$) Vasopressin group 33.7% Epinephrine group 30.5% ($P=0.65$) Vasopressin group 29.0% Epinephrine 20.3% ($P=0.02$)	The effects of vasopressin were similar to those of epinephrine in the management of ventricular fibrillation and pulseless electrical activity, but vasopressin was superior to epinephrine in patients with asystole. Vasopressin followed by epinephrine may be more effective than epinephrine alone in the treatment of refractory cardiac arrest
PRCT (level 2, excellent)	Endpoint: survival to hospital admission	Additional Epinephrine	Vasopressin group 25.7% Epinephrine group 16.4% ($P=0.002$)	
Pytte et al., (2006), Resuscitation, Norway, [18]	17 pigs had full monitoring, then 3 min of untreated VF arrest Adrenaline administered and types of CPR given:	Coronary perfusion pressure (CPP)	LabCPR 29 mmHg ClinicalCPR 12 mmHg $P<0.02$	The haemodynamic effects of adrenaline depend on chest compression quality. Peak dose of the delivery of adrenaline was significantly delayed when simulating clinically reported CPR quality compared to good quality CPR (150 s vs. 90 s)
Experimental study (level 6, excellent)	Clinical quality CPR manually performed chest compressions (30b- 38 mm depth) with a frequency of 100 min^{-1} interrupted by a 9 s break every 15 compressions	Cortical cerebral blood flow (CCBF) Femoral blood flow (FBF)	LabCPR 45% of baseline ClinicalCPR 35% of baseline LabCPR 1.2 ml/min ClinicalCPR 2.5 ml/min $P<0.02$	Adrenaline improved haemodynamics during good quality CPR in pigs, but not with quality simulating clinically reported CPR performance Historical CPR Ratio

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Table 1 (Continued)

Author, date and country Study type (level of evidence)	Patient group	Outcomes	Key results	Comments
	LabCPR automatic hydraulic chest compression device (Heartsaver 2000) maintaining consistent chest compressions of 45 mm depth, 100 min ⁻¹			

5. Search outcome

Three hundred and twenty-eight papers were found in MEDLINE, 499 in EMBASE and 62 in the Cochrane collection using the reported search. From these, 16 papers were identified that provided the best evidence to answer the question. These are presented in Table 1.

6. Results

The 2005 European Resuscitation Council Guidelines (ERC [3]) and the American Heart Association guidelines [4] state that for patients suffering a cardiac arrest with PEA or asystole, 1 mg of adrenaline should be given as soon as intravascular access is achieved and for every 3–5 min or every other loop of the algorithm. For VF/VT, adrenaline should be given after the second failed shock. However, the ERC state that *‘despite the widespread use of adrenaline during resuscitation, and several studies involving vasopressin, there is no placebo controlled study that shows that the routine use of any vasopressor at any stage during human cardiac arrest increases survival to hospital discharge. Current evidence is insufficient to support or refute the routine use of any particular drug or sequence of drugs. Despite the lack of human data, the use of adrenaline is still recommended, based largely on animal data.’* The evidence that they base this recommendation on are the worksheets produced by Long and Paradis [5] and Wenzel [6]. Long concludes that adrenaline use is supported by recent animal studies but that no human studies compare it to placebo. Also, they note that it produces adverse consequences and also subsequent doses are less effective. They give the level of evidence for adrenaline for VF or PEA/asystole as ‘indeterminate’ which is defined as *‘minimal evidence available, results inconsistent and contradictory and results not compelling’*

Cairns and Niemann [7] studied 14 dogs who had VF for 7.5 min prior to resuscitation attempts. Three dogs survived and adrenaline increased their coronary perfusion pressure (CPP) by 21 ± 11 mmHg. However, in the remainder, adrenaline only increased the CPP by 3 ± 2 mmHg. Also subsequent doses had minimal effect on CPP.

Klouche et al. [8] studied 20 rats using differing resuscitative drugs after VF. They found that adrenaline impaired post-resuscitation myocardial function more than vasopressin and a selective alpha-agonist, and this function was similar to saline-placebo controls. However, survival was superior with adrenaline than with controls.

Lindberg et al. [9] in 18 pigs who had a sternotomy and chest closure, and then VF, showed that while either adrenaline or noradrenaline increased CPP during the arrest up to 45 mmHg compared to only 7 mmHg for controls, it significantly impaired cardiac output and oxygen delivery after successful resuscitation.

Chen et al. [10], in 47 rabbits arrested after ET-tube clamping, found that adrenaline increased CPP from 4 to 38 mmHg whereas vasopressin failed to do this. Half the rabbits given adrenaline survived compared to 10% of the vasopressin group.

Ristagno et al. [11] showed significantly worse cerebral blood flows and oxygenation with adrenaline compared to vasopressin in 10 pigs after cardiac arrest.

Vandycke and Martens [12] performed a meta-analysis of five RCTs of high-dose adrenaline vs. standard dose adrenaline. They found a superior odds of return of spontaneous circulation but no difference to hospital admission and a poorer outcome to hospital discharge with higher doses of adrenaline. Biondi-Zoccai et al. [13] performed a meta-analysis of vasopressin vs. adrenaline, finding only two human studies but demonstrating superiority of vasopressin across 33 animal studies. Zhong and Dorian [14] performed a review of adrenaline and vasopressin in cardiac arrest stating that adrenaline had many adverse effects post-resuscitation including myocardial dysfunction, worsening arrhythmias and increased myocardial oxygen demand and that human studies in this area were urgently needed.

Holmberg et al. [15] in a survey of 11,000 patients in patient-care who had arrested out-of-hospital looked at risk factors for adverse survival. They found that adrenaline was a predictor of adverse outcome for asystolic and VF arrests. Behringer et al. [16] reported that in 178 patients who survived an out-of-hospital arrest that adrenaline cumulative dose was much higher in those patients with a poor neurological outcome. The best human study in this area compared vasopressin with adrenaline but had no placebo group. Wenzel et al. [17] randomized over 1000 patients who arrested out-of-hospital to vasopressin or adrenaline. There was no difference in VF arrests but asystole and combined vasopressin and adrenaline showed better survival to hospital admission.

Pytte et al. [18] was struck by the fact that the benefit of adrenaline seen in experimental studies had not translated into clinical studies and hypothesised that this may be due to the difference between clinical CPR and the CPR obtained in a laboratory by hydraulic-compression devices. They compared these types of CPR and found that while

labCPR produced significant haemodynamic effects with adrenaline, no haemodynamic increases were seen with clinicalCPR. Also the peak adrenaline level took 2.5 min to achieve in the clinicalCPR group after a single administration.

7. Clinical bottom line

The European Resuscitation Council and the American Heart Association both recommend 1 mg of adrenaline as soon as pulseless electrical activity or asystole is identified or after the second failed shock if the rhythm is VF/pulseless VT. However, they acknowledge that the evidence behind this recommendation is lacking and based entirely on animal studies which have as yet not been successfully replicated in human studies and thus the evidence for this recommendation is 'indeterminate'. Thus, in the particular situation of a patient who arrests shortly after cardiac surgery where the chance of restoring sinus rhythm either by defibrillation or by an emergency re-sternotomy is high, and where adrenaline could in this situation be highly dangerous once sinus rhythm is restored, we recommend that 1 mg of adrenaline forms no part of the resuscitation protocol for patients after cardiac surgery.

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eComment: Avoidance of administration of 1 mg of adrenaline in cardiac arrest after cardiac surgery

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Cardiac arrest in the early postoperative period after cardiac surgery is usually rapidly reversible by appropriate treatment of the underlying cause [1]. Excessive hypertension induced by adrenaline following return of spontaneous circulation in this setting may cause catastrophic disruption of surgical anastomoses. The intravenous administration of a relatively low dose of adrenaline in the peri-arrest scenario may have similar consequences. The evidence to support the use of adrenaline in cardiac arrest is weak [2].

The intravenous administration of 1 mg of adrenaline should not be recommended in cardiac arrest after cardiac surgery. Emphasis should instead be given to external cardiac compression, immediate defibrillation, rapid correction of reversible causes and early emergency re-sternotomy if appropriate. The European Resuscitation Council (ERC) should consider changing their guidelines for the resuscitation of patients who suffer cardiac arrest following cardiac surgery [3].

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eComment: Should adrenaline be routinely used by the resuscitation team if a patient suffers a cardiac arrest shortly after cardiac surgery?

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Most of the time emergency re-thoracotomy is the effective method to solve the problem causing a circulatory arrest after cardiac surgery. The authors state correctly that there is no evidence for injecting adrenaline to treat the loss of circulation [1]. If the cause of the cardiac arrest is not resolved by the re-thoracotomy, the surgeon can very easily inject adrenaline directly into the left ventricle of the heart. Moreover, this method in combination with internal cardiac massage guarantees that the adrenaline

reaches the coronary circulation in contrast to an intravenous injection of adrenaline in the circumstances of a circulatory arrest.

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eComment: Post CABG cardiac arrest

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I don't recommend adrenaline for post CABG arrest [1]. I think it is better to come back to the operating room promptly, if normal condition doesn't return after primary works. Placement of CPB, examination of grafts and redoing CABG (on pump, beating) is the best option. If grafts are apparently normal, then redo CABG from the most important target vessels (on pump, beating). After each graft we try to be off from CPB. If everything is OK, the chest is closed, if not, other important grafts will begin. I think immediate re-sternotomy for other post cardiac surgery arrest is the procedure of choice if external massage is ineffective.

Reference

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eComment: Avoidance of administration of 1 mg of adrenaline in cardiac arrest after cardiac surgery

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Regarding the administration of (intravenous) epinephrine for the early-post-operative cardiac surgical patient after the fact of the occurrence of complete circulatory arrest [1] I am an agnostic.

That said, I have found i.v. bolus epinephrine can occasionally avert the aforementioned scenario for the patient in a rapid downward 'death spiral'. In my experience this near-arrest physiology results from abrupt vasodilatory decompensation, for example when suddenly coughing (i.e. valsalva condition) as sedation wears off. And here is the point I would like to emphasize. The crucial form of epinephrine here is the 1 mg/10 cc (usually as a 'Bristoject' male leuc lock adapter). Push and flush 50 to 100 mg (0.5 to 1 cc) and the patient in jeopardy pulls out of the dive just skimming the treetops. Push the 'arrest dose' of 1 mg/1 cc and as amply noted by others on this concept, the vasodilatory crisis morphs into a hemorrhagic disaster of busted suture line(s) or cannulation site(s).

In summary: Ban the 1 mg/1 cc epinephrine syringes. Keep the 1 mg/10 cc dosage form handy.

Reference

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eComment: The moderate use of adrenaline in arrest of patient shortly after cardiac surgery

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Your article [1] is very interesting as it fills a gap of knowledge about the correct administration of adrenaline in postoperative cardiac patients suffering from cardiac arrest. According to the European Resuscitation Council [2] and the American Heart Association [3], a bolus of 1 mg of adrenaline is indicated as soon as pulseless electrical activity or asystole is identified or after the second failed shock, if the rhythm is VF or pulseless VT. The target of this administration is double: firstly to induce ventricular fibrillation or tachycardia (for a successful subsequent defibrillation), and secondly to increase the systemic vascular resistance and restore through this way, a better tissue perfusion. The most important of the targets mentioned above is the first, because the main demand in an asystolic patient in arrest is to retrieve any cardiac activity, even a ventricular fibrillation. However, the second target (to increase the systemic vascular resistance) is achieved much later, either after retrieving a normal rhythm or restoring the circulation by heart massage. In our opinion, adrenaline is indicated ONLY in the patients with no ventricular activity. If ventricular activity is recognized, the so called pulseless electrical activity (ventricular fibrillation or tachycardia), as in the patient in your scenario, the administration of adrenaline does not take place in CPR. Besides, in case of cardiac tamponade, we usually have an empty heart with no myocardial dysfunction, and the rhythm is mostly normal, or later (due to either myocardial hypoperfusion or/and metabolic acidosis) ventricular fibrillation is observed. In contrast, a myocardial dysfunction with dilation is observed in case of a rhythm characterized by junctional bradycardia, or asystolia. Especially in the cardiac surgical patient, the asystolic arrest is not rare. Systemic influences that increase extracellular K⁺ concentration, such as low PO₂, metabolic acidosis, renal failure, hypothermia, hemolysis and myocardial trauma, contribute to a partial depolarization of normal or already diseased His-Purkinje system [4]. In this case of arrest, the administration of adrenaline and the abrupt performance of external cardiac massage, may 'brake down' the vicious circle until the reopening of sternotomy for a more effective massage. Obviously, after recovery of VT or VF and an unsuccessful defibrillation, administration of 1 mg adrenaline every 3 to 5 minutes is clearly indicated [2, 3]. While the moderate use of adrenaline during post-cardiac surgery arrest is desirable; as we avoid all its adverse effects on the myocardium (increased myocardial oxygen consumption, sustained arrhythmias, and further dysfunction), as well as on the brain (decreased cerebral flow, worsening of brain ischemia) [5].

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