

Differences in Adult Medical Cardiac Arrest Treatment

Paramedic	Intensive Care	Extended Care	Specialist
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Method

Produced August 2022. This poster is a descriptive analysis and comparison of a specific and discrete cluster of primary sources. All of the ten jurisdictional services have open access Clinical Practice Guidelines (CPGs). Content was extracted by paramedics, with oversight from two senior lecturers in paramedicine. Scope of practice was classified as 'Paramedic' (undergraduate degree, represented by a ✓), 'Intensive Care Paramedic' (intensive care postgraduate degree), 'Extended Care Paramedic' (primary care postgraduate degree), or 'Specialist' (all other advanced roles, e.g. Retrievalist). Standard, routine cares such as defibrillation, CPR, oxygen, metronome, CPR feedback device, and EtCO₂ monitoring were omitted for brevity, as were other conditions covered under their own, separate CPG (such as 'special circumstances' including ventricular assist devices). This comparison does not review the peer-reviewed, published literature to determine current best practice in treatment. Consequently, no CPG is inferred to be superior or inferior to any other, nor that the most common treatment is necessarily optimal. This resources is created purely to assist making paramedics aware of current Australasian treatment options across JASs.

Jurisdiction (Service)f	Intervention											Pharmacology												
	Circulation							Airway			Access	Gastric aspiration	Adrenaline	Amiodarone	Calcium Gluconate / Chloride	Lignocaine	Isotonic, acidic volume filler	Magnesium sulphate	Sodium bicarbonate	CPRIC Sedation				
	Compression-Ventilation ratio	Pad placement other than anterior-lateral	Defibrillation mode	Stacked shocks	Dual defibrillation	Mechanical compression device	Precordial thump	OPA / NPA	LMA	ETT (unassisted)	IO									Midazolam	Fentanyl	Ketamine	Rocuronium	
Aus. Capital Territory (ACTAS)	30:2 with pause 15:1 no pause – LMA/ETT						✓ (a)	✓	✓	ICP	✓		✓	ICP (b)	ICP (c)		✓	ICP (d)	ICP (e)	(u)				
New South Wales (NSWA)	30:2 with pause		Manual			✓ (v)		✓	✓	ICP	ICP	ICP	✓	ICP (f)	ICP (c)	ICP (f)	✓ (g)		ICP (p)	✓			ICP	
New Zealand (SJNZ)	30:2 with pause 15:2 with pause – respiratory arrest 10/min no pause - LMA/ETT (w)	Anterior- posterior for persistent VF/VT	Manual		Under medical advice only	✓	✓ (a, h)	✓	✓	ICP	✓		✓ (i)	✓ (j)			✓ (k)					ICP	ICP (l)	
New Zealand (WFA)	30:2 with pause 15:2 with pause – respiratory arrest 10/min no pause - LMA/ETT (w)	Anterior- posterior for persistent VF/VT	Manual		Under medical advice only	✓	✓ (a, h)	✓	✓	ICP	✓		✓ (i)	✓ (j)			✓ (k)					ICP	ICP (l)	
Northern Territory (SJNT)	30:2 with pause					✓		✓	✓	ICP	✓	✓	✓	✓ (b, j, m)	✓ (c)		✓ (n)	✓ (o)	✓ (p)			ICP		
Queensland (QAS)	30:2 with pause 6-10/min no pause – LMA/ETT	Alternate position after 3 shocks	AED for first shock	✓ (q)		ICP		✓	✓	ICP	ICP	✓	✓	✓ (r)	ICP (c)			ICP (o)	ICP (p)	ICP	ICP			
South Australia (SAAS)	30:2 with pause 6-10/min no pause – LMA/ETT					✓		✓	✓	ICP	✓	✓	✓	✓ (b)			✓ (s)			✓				
Tasmania (AT)	30:2 with paus 8-10/min no pause – LMA/ETT							✓	✓	ICP	✓		✓	ICP			✓	ICP (o)	ICP (p)					
Victoria (AV)	30:2 with pause 15:1 no pause – LMA/ETT		Manual			✓ (t)		✓	✓	ICP	ICP	✓	✓	ICP (b, m)	ICP (c)		✓ (n)		ICP (p)		✓	✓		
Western Australia (SJWA)	30:2 with pause Continuous – LMA/ETT		Manual			✓		✓	✓	✓	✓	ICP	✓	✓ (b, m)										

AED – Automatic external defibrillator CPRIC = Cardiopulmonary resuscitation induced consciousness ETT = Endotracheal intubation ICP = Intensive care paramedic IO = Intraosseous LMA = Laryngeal mask airway NPA = Nasopharyngeal airway OPA = Oropharyngeal airway PEA = Pulseless electrical activity VF = Ventricular fibrillation VT = Ventricular tachycardia

(a) For paramedic-witnessed and monitored VF/VT arrest (b) After third shock if in a shockable rhythm (c) Cardiac arrest secondary to hyperkalaemia (d) First drug if Torsades; fourth drug if VF (e) >15 minutes arrest, hyperkalaemia, cardioactive drug overdose (f) VF, VT, or Torsades only (g) Arrest secondary to anaphylaxis (h) Where defibrillator is not immediately available (i) Cease after 15-20 minutes if VF persists and administer further amiodarone instead (j) If in VF or VT after first shock (k) 2-3 litres if in PEA (l) If intubated; sedative also required (m) Repeat after fifth shock (n) 20 ml/kg if in PEA due to hypovolaemia, anaphylaxis, or asthma (o) Torsades (p) Hyperkalaemia, crush, or cardioactive drug overdose (q) Paramedic-witnessed shockable arrest (r) Refractory to three shocks; again after five shocks (s) Up to 30 ml/kg if hypovolaemic or obstructive cause (t) Available to all paramedics, however, in practice only carried by ICP units (u) No CPRIC guideline, however, ICP may sedate to permit basic airway management (v) Currently being installed in all vehicles (w) With LMA in situ, if compressions are causing ineffective ventilations, a pause in compressions may be inserted

Treatment rationale

- Defibrillation
- Triggers widespread depolarisation of the myocytes, potentially terminating dysrhythmias and allowing a perfusing rhythm to be restored.
 - Stacked shocks is the provision of multiple defibrillations directly after each other.
 - Dual defibrillation is the placement of two different defibrillators (four sets of pads) with two stacked shocks provided in different anatomical locations.

- External compressions
- Compressions facilitate some blood flow, speculated to be either by the cardiac pump theory (external compression of the ventricles mimics systole) or by the thoracic pump theory (increased thoracic pressure relative to abdominal pressure pulls blood distally).
 - Mechanical compression devices provide external compression via a pumping mechanism placed over the sternum.

- Precordial thump
- A single strong blow by the base of a closed fist to the sternum, believed to produce a minor depolarisation that may terminate dysrhythmias.

- Gastric aspiration
- Aspiration of the gastric contents via a catheter placed through either an LMA or ETT to reduce regurgitation and maintain a secure airway.

- Adrenaline
- Alpha-1 agonism causes peripheral vasoconstriction.
 - Alpha-2 agonism increases glucagon and decreases insulin, raising serum glucose.
 - Beta-1 agonism causes positive inotropy, chronotropy, dromotropy, and lusitropy.
 - Beta-2 agonism induces bronchodilation, offsetting obstructive gas trapping and improving tidal volume.
 - Beta-3 agonism triggers lipolysis, raising serum glucose.
 - Stabilises mast cells, reducing degranulation and release of inflammatory mediators.

- Amiodarone
- An anti-dysrhythmic working in all Vaughan-Williams classes, most importantly in prolonging the sinoatrial, atrioventricular, and ventricular repolarisation periods, and in slowing conduction in the sinoatrial and atrioventricular nodes.

- Calcium
- An electrolyte that stabilises the cardiac membrane immediately.

- Lignocaine
- Local anaesthetic and antidysrhythmic that function in the nervous system primarily by temporarily attaching to the intracellular side of sodium channels, binding them to their open state; this prevents repolarisation and inhibits neural transmission, potentially terminating dysrhythmias.

- Magnesium sulphate
- An electrolyte that is used as a cofactor in multiple processes, with antidysrhythmic, anticonvulsive, vasodilative, and bronchodilative effects.

- Sodium bicarbonate
- Sodium bicarbonate reduces potassium's cardiotoxic effects and is alkalising, increasing Ph and moving potassium into cells.

- CPR Induced Consciousness
- A phenomenon where sufficient cerebral perfusion is established by CPR to illicit life-like responses from a patient (such as blinking, hand movements, and similar) despite an ongoing non-perfusing rhythm. In some instances, this may interfere with external compressions or airway maintenance.