

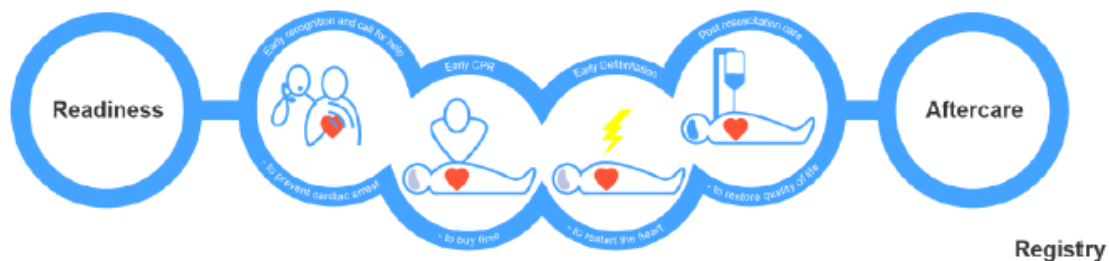
RIE OUT OF HOSPITAL CARDIAC ARREST CARE GUIDELINE

APPLICABLE TO ALL PATIENTS WHO HAVE ACHIEVED ROSC FOLLOWING OOHCA
INCLUDES ECPR ACTIVATION CRITERIA FOR REFRACTORY VF/VT

Date of OHCA	
Time of OHCA (or time found)	
Witnessed Arrest	
Was there bystander CPR?	
First recorded rhythm	VF/VT PEA ASYS
Time of first ROSC	
Time of sustained ROSC (if applic)	
Time of arrival in ED	
Time temperature management commenced	
Time temperature management completed	

Emergency Medicine SpR	Extension 23511
Critical Care SpR	Bleep 2306
Anaesthetic SpR	Bleep 2200
Cardiology SpR	Bleep 4028
CT Radiologist	Extension 23796
E-CPR Team	Extension 2222

This guideline brings together the latest guidance on the management of patients who have suffered a cardiac arrest and achieve ROSC in the pre-hospital or Emergency Department setting. It should be commenced in the ED and follow the patient throughout admission.

**Cardiac Arrest Care**

[Pre-Hospital or Emergency Department Response]

Ongoing resuscitation	As per ALS guidelines
E-CPR Call 2222 and state: "ECPR team to ED Resus" If ECPR team fail to appear within 5 minutes repeat activation	<p>Activate ECPR team if the following criteria met:</p> <ul style="list-style-type: none"> • Adult aged 70 or younger in cardiac arrest • Monday to Friday 0900 – 1700hrs • Initial rhythm VF/VT with no ROSC after 3 shocks • PEA if signs of life (lacrimation, reactive pupils, attempted respiration or spontaneous movements) and no ROSC at 10 mins of ALS • No rule-out criteria (life limiting pathology, trauma, known DNACPR, time from SAS arrival to anticipated cannulation > 60 mins) • Total CPR time < 45 minutes • Particularly pertinent to special circumstances: OD and hypothermia

Immediate Post Arrest Care [Emergency Department]	Guideline recommendations including investigation into cause of arrest (prior to patient leaving the emergency department)
Standard Post Arrest Care	Post arrest care should be commenced immediately in the context of sustained ROSC, regardless of location of patient
Airway	If patient remains comatose post ROSC secure airway by means of RSI
Bloods +/- toxicology	Routine bloods should be obtained including full haematology and biochemistry profile (FBC, U&E, LFT, CK, coag)
ECG	In post OOHCA patients with ST elevation, urgent angiography +/- PCI should be undertaken. Consider urgent angiography for patients with LBBB, severe haemodynamic or electrical instability
CT head and CTPA	In patients without evidence of myocardial ischaemia, a CT head and CTPA should be performed prior to admission to critical care to exclude a neurological or respiratory cause of arrest.
Debrief	STOP Hot Debrief Tool to include all relevant staff (ED, SAS, Critical Care)

ICU Care	General Management
Oxygenation	PO2 10-13, avoid hypoxaemia, avoid hyperoxaemia
Ventilation	Lung protective strategy PaCO2 4.5-6 Prophylactic antibiotics for aspiration are not recommended
Circulation	Arterial line and central venous access Early formal echo to quantify myocardial dysfunction (<i>within 24hrs</i>) Target MAP > 65, maintained with fluids, noradrenaline +/- inotropy (adrenaline/ dobutamine) dependent on individual patient need Dynamic measures of cardiac function are encouraged including serial echocardiography where indicated to assess trends
Mechanical Support	Consider mechanical circulatory support (IABP, VA ECMO) in patients with circulatory shock in whom treatment with vasopressors, fluids and inotropes is insufficient to maintain adequate organ perfusion
Neurology	Sedation with propofol/alfentanil should be commenced on admission, with the aim of stopping sedation after 48 hours Routine seizure prophylaxis is not recommended In clinical seizure activity following OOHCA, levetiracetam is recommended as the first line drug (see monograph). EEG evidence of seizure activity is a poor prognostic sign (see neuroprognostication section)
Routine Care	Glycaemic control (4-10mmol/L) Start feeding VTE prophylaxis
Temperature Management	Apply cooling pads on admission, target temperature of 37 degrees

Maintain temperature of 37 degrees for 72 hours following admission with aggressive avoidance of pyrexia > 37.7

Prognostication

No single predictor of outcome is accurate. A multimodal prognostication strategy is important, with prognostication occurring for most patient groups at least 72 hours from the time of arrest.

Discussions about patient prognosis and timings of neuroprognostication should also consider aspects other than brain injury such as age, co-morbidity, organ dysfunction and patient preferences.

Investigations useful in prognostication

The following investigations should be undertaken in the following timeframes to aid multimodal neuro-prognostication

EEG (Should be undertaken at any point 24 hours – 72 hours post arrest, ideally with sedation off)

EEG in this context gives prognostic information (See box at foot of page)

Hyperlink to EEG request form :

<http://intranet.lothian.scot.nhs.uk/Directory/Neurophysiology/PublishingImages/PAEDS%20EEG%20REQUEST%20FORM%20RHSC%202020.doc>

CT imaging (should be repeated at 48h post arrest)

SSEP (If available) – should be undertaken > 24 hours post arrest

Neuron Specific Enolase (NSE) (where available via biochemistry) should be undertaken at 24 and 48 hours post arrest

Clinical assessment and interpretation of investigations at 72 hours post arrest

At 72 hours, and confident sedation no longer contributing to clinical state, you should clinically assess the patient

In a comatose patient with M ≤ 3 at ≥ 72 h from ROSC, poor outcome is likely when two or more of the following predictors are present:

- no pupillary and corneal reflexes at ≥ 72 h
- bilaterally absent N20 somatosensory evoked potential (SSEP) wave at ≥ 24 h
- highly malignant EEG at >24h
- neuron specific enolase (NSE) > 60 mcg L⁻¹ at 48 h and/or 72 h
- status myoclonus ≤ 72 h
- diffuse and extensive anoxic injury on brain CT/MRI.

In patients in whom the above criteria is not met, there should be a further period of 24-48 hours of clinical assessment, with further additional ancillary investigations as necessary.

Prognostic Significance of EEG patterns following cardiac arrest

Presence of any highly malignant pattern or >2 malignant features on EEG has excellent (>95%) specificity for hypoxic brain injury. A benign EEG (without any malignant features) is highly predictive of a good neurological outcome.

Highly Malignant EEG Patterns (As per American Clinical Neurophysiology Society)

- Suppressed background (amplitude <10 µV) without discharges
- Suppressed background with continuous periodic discharges
- Burst-suppression background with or without discharges

Malignant EEG Patterns

- Malignant periodic or rhythmic patterns (abundant periodic discharges; abundant rhythmic polyspike-/spike-/sharp-and-wave; **presence of seizures**)
- Malignant background (discontinuous background; low-voltage background; reversed anterior-posterior gradient)
- Unreactive EEG (absence of background reactivity or only stimulus-induced discharges)

Rationale and evidence underpinning guideline update

European Resuscitation Council Guidelines

- Published 2021, includes expanded section on neuroprognostication, recommendation for angiography +/- CTPA and CT head in investigation of cardiac arrest

Tomahawk trial <https://www.nejm.org/doi/full/10.1056/NEJMoa2101909>

- In patients without ST elevation following OOHCA, immediate angiography no benefit over delayed strategy, although trial excluded patients with haemodynamic instability and LBBB

TTM2 trial <https://www.nejm.org/doi/full/10.1056/NEJMoa2100591>

- In OOHCA, induced hypothermia did not improve mortality, functional outcome or HRQoL

EEG in Prognostication

Westhall E, Rossetti AO, van Rootselaar AF, et al. Standardized EEG interpretation accurately predicts prognosis after cardiac arrest. Neurology. 2016;86(16):1482-1490. doi:10.1212/WNL.0000000000002462