EMS ECHO 111


RECOGNIZE,
REACT & RESCUE.
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Mr. Arinaitwe Jonan, EMT at KCCA, Paramedic Student at Lubaga Training School.
Chat Questions
Dr. Sarah Oworinawe, EM Physician at Yumbe RRH
This session will delve into areas such as;
1 Early recognition & emergency team activation.
2.Early initiation of high quality CPR.
3.Rhythm recognition and early defibrillation.
4.Identification of reversible causes.
5.Post-cardiac arrest care for enhanced recovery & survival.
scan to register
FRIDAY
13th March 2026
2-4pm EAT
Use link;
https://shorturl.at/jS99a

RECOGNIZE, REACT & RESCUE

Cardiac Arrest Management

Based on ERC 2025 Guidelines

Dr. Daniel Olinga · EM Physician & Lecturer, MUST

Friday 13 March 2026 · ECAU × Ministry of Health × Seed Global Health × ECHO

SESSION OVERVIEW

01 Chain of Survival

02 Recognising Cardiac Arrest

03 Cardiac Arrest Rhythms + ECG

04 Shockable Algorithm (VF / pVT)

05 Non-Shockable Algorithm (PEA / Asystole)

06 4Hs & 4Ts — Reversible Causes

07 CPR Decision Framework

08 Drugs in CPR + Timing

09 Pre-Hospital Transfer & Traumatic Arrest

10 Pupils, Post-Arrest Care, ERC vs AHA, Pearls & Takeaways

Let's have a Deep Dive...

CHAIN OF SURVIVAL — ERC 2025

1

Early Recognition

& Call for Help

2

Early CPR

High-Quality Compressions

3

Early Defibrillation

AED or Manual Defib

4

Advanced Care

ALS & Team Resuscitation

5

Recovery

Rehabilitation & Support

ERC 2025: Survival doubles with bystander CPR. Every minute without defibrillation in VF reduces survival by ~10%.

RECOGNISING CARDIAC ARREST

1

UNRESPONSIVE

No response to voice or sternal rub

2

NO NORMAL BREATHING

Absent or agonal gasps — both = cardiac arrest

3

CALL FOR HELP & START CPR

Do NOT wait for pulse confirmation

⚠ AGONAL BREATHING

Agonal breathing (gasping, slow laboured breaths) is a sign OF cardiac arrest — not a sign of life. Treat it as arrest and start CPR immediately.

💡 ERC 2025 — DA-CPR

Dispatcher-Assisted CPR: Emergency dispatchers should actively coach bystanders through CPR. This alone doubles survival in some settings.

MYTH:

"Fixed dilated pupils mean the patient is dead or unsalvageable."

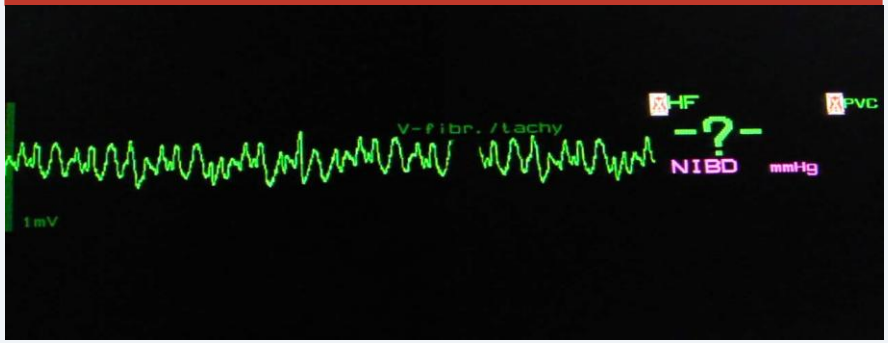
FACT:

- ✓ Pupils dilate within 15–30 seconds of cardiac arrest — this is normal physiology.
- ✓ Adrenaline, atropine, cocaine and many other drugs cause fixed dilated pupils.
- ✓ Pupils are NOT used to decide whether to start, continue, or stop CPR.
- ✓ Reactive pupils can and do return with successful resuscitation.

CARDIAC ARREST RHYTHMS — THE 4 RHYTHMS

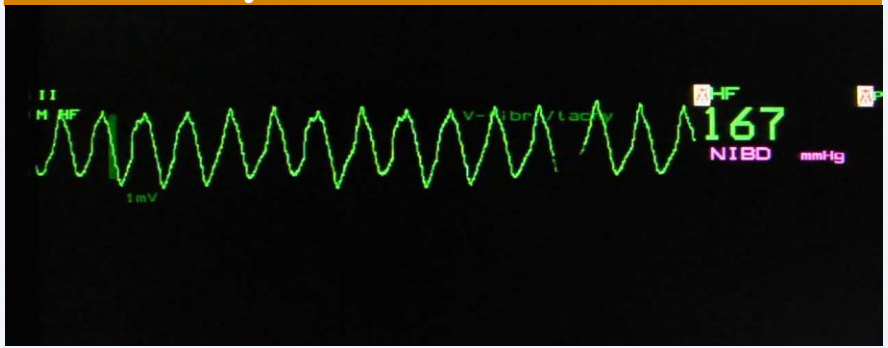
VF Ventricular Fibrillation

SHOCKABLE



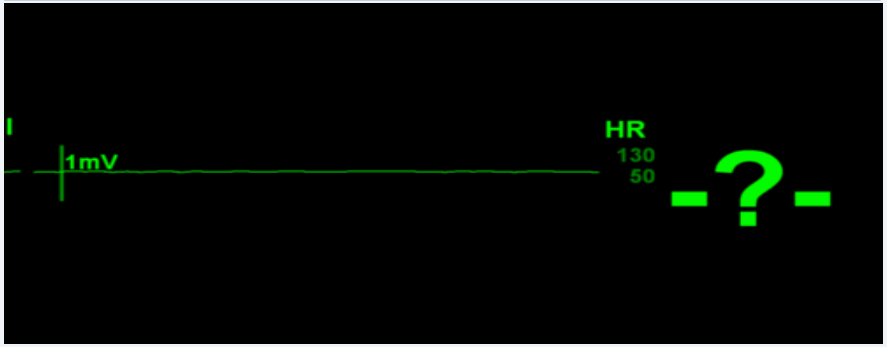
pVT Pulseless Ventricular Tachycardia

SHOCKABLE



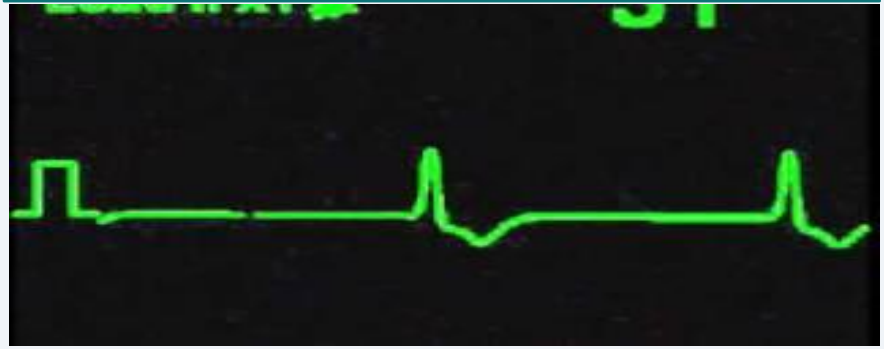
AS Asystole

✕ NO SHOCK

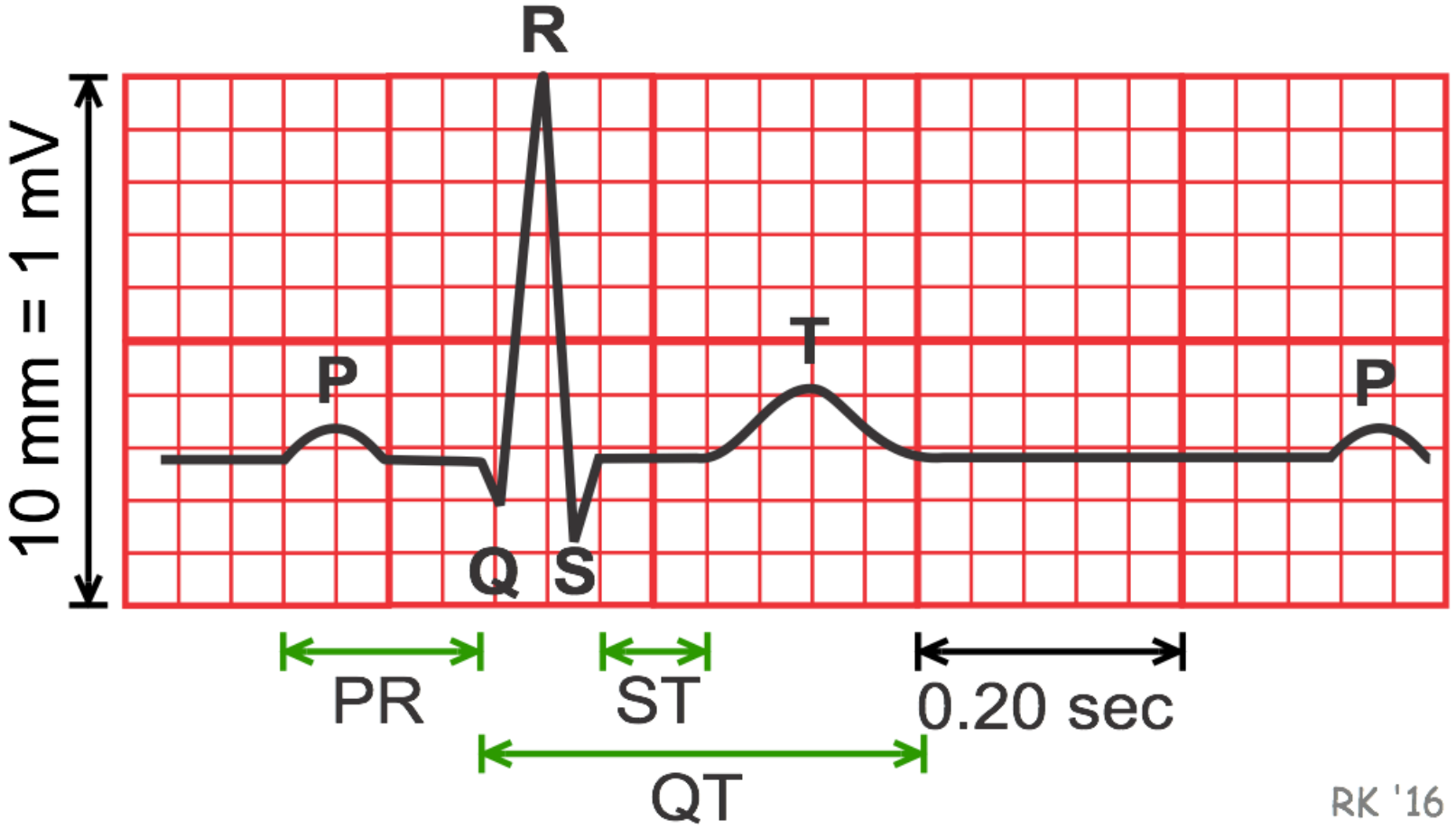


PEA Pulseless Electrical Activity

✕ NO SHOCK

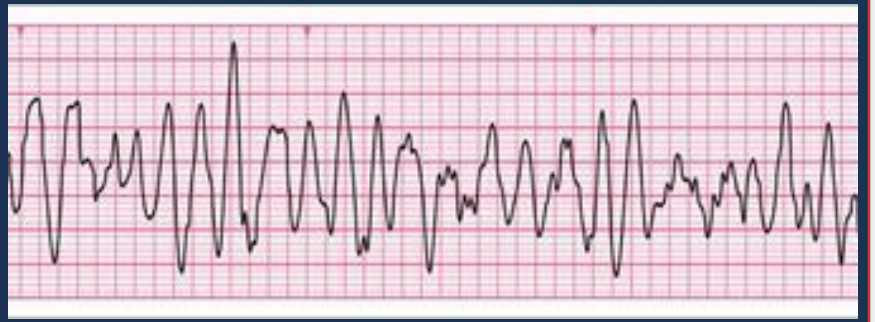


But what is a Normal ECG Tracing..



SHOCKABLE RHYTHMS — VF & pVT

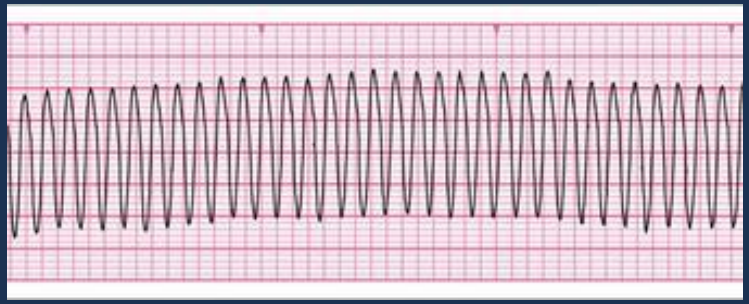
Ventricular Fibrillation (VF)



Irregular grid lines shown — rate indeterminate

- **Chaotic, irregular baseline — no discernible QRS complexes**
- **No P waves, no T waves**
- Amplitude may be coarse (>5mm) or fine (<5mm)
- Fine VF can mimic asystole — increase gain to check
- Coarse VF = more likely to respond to defibrillation

Pulseless VT (pVT)



Rate ~180–250 bpm — wide QRS (>120ms)

- **Regular, wide QRS complexes (>120 ms) — monomorphic**
- **Rate usually 150–250 bpm**
- P waves absent or dissociated
- PULSE CHECK confirms 'pulseless' — do not delay
- Polymorphic VT (Torsades) — consider IV magnesium 2g

NON-SHOCKABLE RHYTHMS — ASYSTOLE & PEA

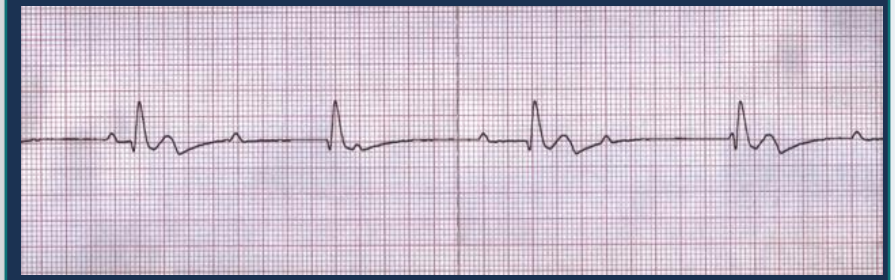
ASYSTOLE

NO SHOCK

Flat or near-flat baseline — confirm in ≥ 2 leads

- **Flat or near-flat baseline — confirm in ≥2 leads**
- **Check: lead connections, gain, correct lead selection**
- Do NOT diagnose asystole from a single lead
- P-wave asystole: occasional P waves — still no output
- CAUSE: Hypoxia, hyperkalaemia most common

PULSELESS ELECTRICAL ACTIVITY (PEA)



Organised QRS — but NO pulse on examination

- **Organised electrical activity — any morphology possible**
- **The KEY: no palpable central pulse despite rhythm**
- Commonest cause = untreated reversible cause (4Hs & 4Ts)
- Narrow PEA: tamponade, tension PTX — act fast
- Wide PEA: hyperkalaemia, severe hypoxia, drug OD

HIGH-QUALITY CPR — THE NUMBERS THAT MATTER

100–120 /min	**5–6 cm** depth	**30:2** ratio
Compression Rate	Compression Depth	Ratio (until advanced airway)
>60% CCF	**<10 s** pause	**500 mL** tidal
Chest Compression Fraction	Max Pre-Shock Pause	Ventilation Volume

Allow FULL chest recoil after every compression — do NOT lean on the chest. Rotate rescuers every 2 minutes to prevent fatigue.

ALS ALGORITHM — SHOCKABLE (VF / pVT)

SHOCK ENERGY

Biphasic 1st shock

150–200 J

Biphasic subsequent

200 J

Monophasic all

360 J

AED

Device-specific

Paediatric

4 J/kg

CARDIAC ARREST
Shockable Rhythm (VF / pVT)

START CPR 30:2
Attach Defibrillator

↓

CHARGE & DELIVER
Shock ×1

↓

IMMEDIATELY RESUME CPR
2 minutes — No pulse check first

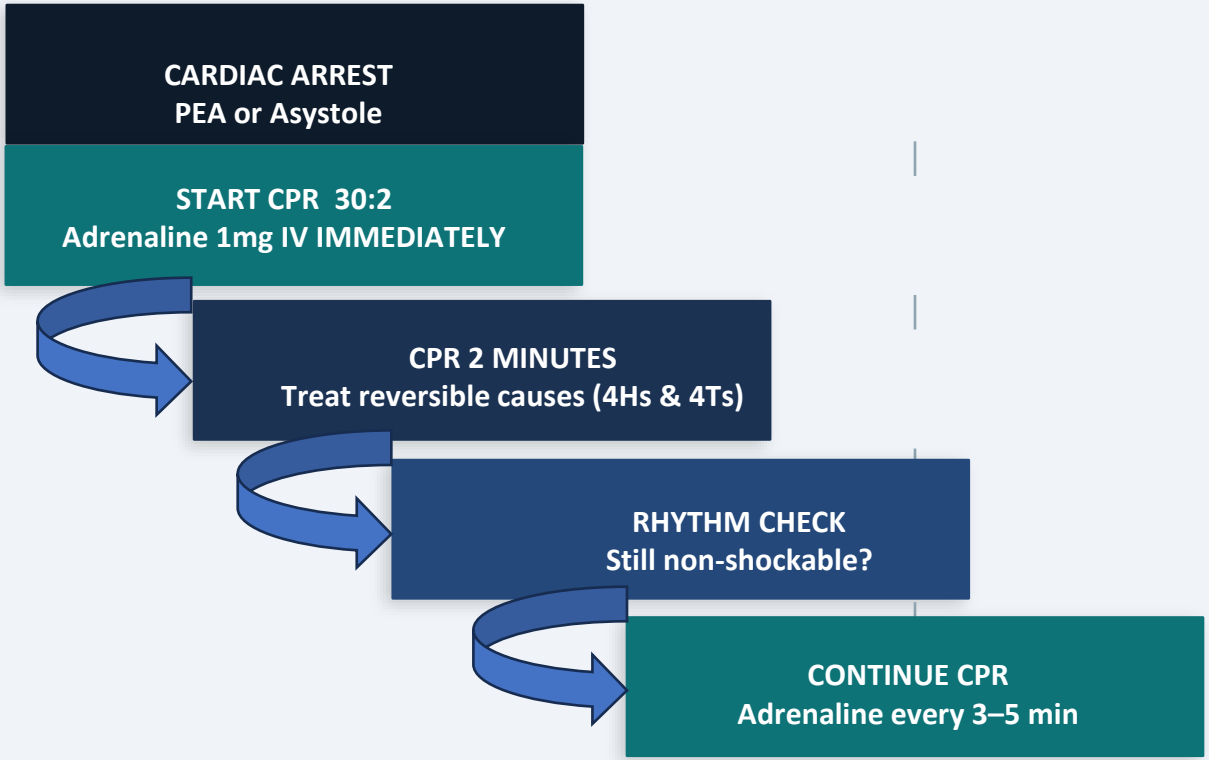
↓

RHYTHM CHECK
Shockable? → Repeat loop

DRUG TIMELINE

After 3rd Shock	Adrenaline 1mg IV Amiodarone 300mg IV
Every 3–5 min	Repeat Adrenaline 1mg
After 5th Shock	Amiodarone 150mg IV
Consider	Lidocaine 100mg IV if no amiodarone available

ALS ALGORITHM — NON-SHOCKABLE (PEA / ASYSTOLE)



KEY DIFFERENCES

- → No defibrillation
- → Adrenaline given immediately
- → No amiodarone
- → Find & treat the cause
- → Check rhythm every 2 min
- → If VF/pVT appears → switch to shockable arm

REVERSIBLE CAUSES — THE 4 Hs

HYPOXIA

Most common reversible cause

FIX: 100% O_2 via bag-valve mask • Secure airway • Ventilate adequately • Check ET tube position & bilateral chest rise

HYPOVOLAEMIA

Haemorrhage or volume depletion

FIX: Identify & stop haemorrhage • IV/IO large-bore access • Rapid crystalloid bolus • Packed red cells if haemorrhagic arrest

HYPO / HYPERKALAEMIA (& other metabolic)

Electrolyte disturbances & glucose

FIX: Point-of-care ABG/electrolytes • Hyperkalaemia: Calcium chloride 10 mL 10% IV • Hypoglycaemia: 50 mL 50% dextrose IV

HYPOTHERMIA

'Not dead until warm and dead'

FIX: Warm IV fluids (40°C) • Active external rewarming • ECMO rewarming for severe cases (<28°C) • Prolong CPR until core temp ≥35°C

REVERSIBLE CAUSES — THE 4 Ts

TENSION PNEUMOTHORAX

Commonest reversible arrest cause in trauma

FIX: Clinical diagnosis: deviated trachea, absent breath sounds, distended veins • Immediate bilateral needle decompression 2nd ICS MCL • Follow with chest drain

TAMPONADE (Cardiac)

Suspect after penetrating chest trauma or pericarditis

FIX: POCUS confirms: pericardial effusion + RV collapse • Pericardiocentesis ultrasound-guided • Surgical drainage/thoracotomy if refractory

TOXINS / OVERDOSE

Drug or toxic substance causing arrest

FIX: Identify agent (history, Toxbase) • Specific antidotes: Naloxone (opioids), Flumazenil (BZD caution), Lipid emulsion (LA toxicity) • Prolonged CPR — toxins are cleared

THROMBOSIS (PE or ACS)

Massive PE or STEMI precipitating arrest

FIX: PE: Thrombolysis (tenecteplase) then CPR 60–90 min • ACS: Primary PCI if ROSC; ECMO-assisted PCI in refractory arrest • ECMO: bridge to definitive treatment

WHEN TO START CPR

START CPR if the patient is: Unresponsive + Absent / Abnormal Breathing + No contraindication

- ✅ Unresponsive to verbal and physical stimulation
- ✅ Absent or agonal breathing (gasps count as cardiac arrest)
- ✅ No valid DNACPR order or advance directive in place
- ✅ Witnessed collapse or brief unwitnessed arrest
- ✅ No signs of irreversible death — *No rigor mortis, no livor mortis, no decapitation*
- ✅ Rescuer safety can be maintained

REMEMBER: Do NOT delay CPR to confirm pulse — pulse checks are inaccurate and waste critical time.

WHEN NOT TO START CPR

CPR may be withheld when: There is a VALID reason not to resuscitate

Valid DNACPR (Do Not Attempt CPR) order or advance care directive	*Must be confirmed before withholding*
Unequivocal signs of irreversible death	*Rigor mortis, livor mortis, decapitation, incineration*
Prolonged unwitnessed arrest (normothermic) with asystole — no reversible cause found	*After thorough assessment*
Non-survivable traumatic injury — e.g. blunt trauma with prolonged asystolic arrest, no reversible cause	
Clinician safety cannot be assured — e.g. ongoing mass-casualty incident, hazardous environment	
Patient's previously expressed and documented wish to not be resuscitated	*Document and respect wishes*

When in doubt — START CPR. It is always easier to stop than to justify not starting. Err on the side of action.

WHEN TO STOP CPR

Stopping CPR is a clinical decision — made after team discussion, considering all factors below

💚 **ROSC (Return of Spontaneous Circulation) achieved**	*→ Move to post-arrest care pathway*
⏱️ Prolonged normothermic arrest with no reversible cause identified	*≥20–30 min without ROSC after complete algorithm*
😓 Rescuer exhaustion or safety compromise	*No rotation available; unsafe environment*
📋 DNACPR discovered or confirmed during resuscitation	*Stop with dignity. Document decision.*
🫀 No electrical activity (asystole) despite >20 min CPR, optimised treatment, no reversible cause	*Team consensus required*

COMMUNICATION: Involve family if possible before stopping. A clear, compassionate statement: 'We have done everything we can. We are going to stop resuscitation.' Document time of death.

WHEN TO PROLONG CPR — SPECIAL CIRCUMSTANCES

HYPOTHERMIA

Core temperature < 30°C

'Not dead until warm and dead'
Continue CPR until rewarmed to ≥35°C — ECMO rewarming preferred in severe cases.

DROWNING

Especially cold water submersion

Excellent neurological outcomes reported even after prolonged submersion in cold water (<5°C).
Aim for rewarming — do not give up early.

TOXIC ARREST / OVERDOSE

Drug-induced cardiac arrest

Toxins are metabolised during CPR.
Lipid emulsion for local anaesthetic toxicity. Sodium bicarbonate for TCA.
Extended CPR — neurological recovery is possible.

POST-THROMBOLYSIS

Thrombolytics given during arrest

Continue CPR for 60–90 minutes after thrombolytic administration.
Thrombus lysis takes time — do not stop prematurely.

eCPR CANDIDATE (ECMO)

Extracorporeal CPR

Young patient, witnessed arrest, no ROSC after 20 min of optimal CPR, reversible cause.
Transport to ECMO centre — mechanical CPR device for transfer.

DRUGS IN CPR — ADRENALINE & AMIODARONE

ADRENALINE (Epinephrine)

Dose	1 mg IV / IO
Route	IV or IO (flush 20 mL NS)
Shockable	After 3rd shock, then every 3–5 min
Non-shockable	IMMEDIATELY, then every 3–5 min
Mechanism	α-agonist → ↑ coronary & cerebral perfusion pressure
Evidence	Improves ROSC but NO benefit to neurological survival to discharge (PARAMEDIC-2 trial)

AMIODARONE

1st Dose	300 mg IV bolus
2nd Dose	150 mg IV bolus
When	After 3rd shock (1st dose) After 5th shock (2nd dose)
Indication	Shockable rhythm ONLY (VF / pVT)
Dilution	In 5% dextrose (NOT normal saline)
Caution	Potent vasodilator on ROSC — watch for hypotension Alternative: Lidocaine 100mg IV if unavailable

DRUGS IN CPR — OTHER AGENTS

LIDOCAINE **100 mg IV**	Alternative if amiodarone unavailable — shockable rhythm only	*0.5–0.75 mg/kg boluses. 2nd line. ERC 2025 acceptable.*
SODIUM BICARBONATE **50 mmol IV**	Hyperkalaemia-induced arrest • TCA overdose • Prolonged arrest (>20 min)	*NOT routine — worsens intracellular acidosis. Guided by ABG.*
CALCIUM CHLORIDE **10 mL of 10%**	Hyperkalaemia • Hypocalcaemia • Calcium channel blocker overdose	*Can cause coronary artery spasm — correct indication essential.*
MAGNESIUM SULPHATE **2 g IV slow**	Torsades de Pointes (polymorphic VT) Hypomagnasaemia-induced arrest	*Dilute and give over 5–10 min if not in arrest.*
ATROPINE **No longer recommended**	ERC 2025: NOT recommended for asystole or PEA	*Removed from ALS algorithm. No evidence of benefit.*

PRE-HOSPITAL TRANSFER & CARDIAC ARREST

✅ LOAD AND GO	✕ AVOID STAY AND PLAY
• Continuous high-quality CPR during transport	✕ Extended airway attempts on scene
• Use mechanical CPR device (LUCAS/AutoPulse) if available	✕ Multiple IV access attempts before moving
• Pre-alert receiving hospital early (MINCARD protocol)	✕ Lengthy scene assessment without CPR
• Minimise scene time — time = brain	✕ Waiting for senior EMS before transporting
• Consider ECMO centre for refractory arrest	✕ Unnecessary procedures that delay transport
• Transport on backboard, not stretcher for CPR quality	✕ Stopping CPR during stretcher loading

IO access is acceptable and equivalent to central venous access. Use it if IV access is delayed by >1 minute during arrest.

TRAUMATIC CARDIAC ARREST — ERC 2025

In traumatic arrest, treat REVERSIBLE CAUSES simultaneously with CPR — do NOT delay to treat one at a time

1	HAEMORRHAGE CONTROL	Direct pressure, tourniquets, haemostatic dressings — major haemorrhage kills fast
2	AIRWAY & VENTILATION	Airway opening, BVM, ET tube — hypoxia is the #1 reversible cause in trauma
3	BILATERAL NEEDLE DECOMPRESSION	Both sides — tension PTX is common and rapidly fatal. 2nd ICS MCL bilaterally
4	PERICARDIOCENTESIS / THORACOTOMY	Penetrating chest trauma with tamponade → resuscitative thoracotomy at major trauma centre
5	VOLUME RESUSCITATION	Massive transfusion protocol: 1:1:1 ratio. Permissive hypotension (SBP 80–90 in penetrating)

Blunt arrest + asystole on scene + no reversible cause found: consider termination. Penetrating arrest: transport rapidly for OR thoracotomy.

POST-CARDIAC ARREST CARE — HAEMODYNAMICS & AIRWAY

HAEMODYNAMICS

Target MAP	≥ 75 mmHg
Avoid SBP	< 90 mmHg
Vasopressors	Noradrenaline 1st line Adrenaline if refractory
12-lead ECG	Immediately — STEMI? → Cath lab within 90 min
Echo (POCUS)	LV function, effusion Wall motion abnormalities
Fluids	IV fluid if hypotensive Avoid excess — pulmonary oedema risk

OXYGENATION & VENTILATION

SpO_2 target	94–98% — titrate O_2 down
Avoid hyperoxia	Worsens neurological outcome
$PaCO_2$ ($EtCO_2$)	Normocapnia: 4.5–6.0 kPa Avoid hypo/hypercapnia
Intubation	ET tube if not secured Confirm with waveform capnography
$EtCO_2$ >10 mmHg	Suggests effective CPR Sudden rise → ROSC
RR target	10–12 breaths/min post-intubation

POST-CARDIAC ARREST CARE — NEURO PROTECTION & ICU GOALS

🧠 NEUROPROTECTION (ERC 2025)

Temperature target	**36–37.5°C (NO routine cooling to 33°C — ERC 2025 update)**
AVOID	Fever (>37.8°C) worsens outcome — treat aggressively
TTM duration	≥24 hours temperature management
Neuroprognostication	Do NOT before 72h post-ROSC Multimodal: EEG, imaging
Sedation	Light sedation preferred — allow neuro reassessment
Seizures	EEG monitoring; treat status epilepticus aggressively

🏥 ICU GOALS

Blood glucose	6–10 mmol/L — avoid hypoglycaemia
Electrolytes	K^+ 4.0–4.5 mmol/L target Mg^{2+} replete if low
Coronary angio	STEMI → immediate; NSTEMI → within 24h Consider even without STEMI if suspected ACS
CT Brain / imaging	When haemodynamically stable Exclude intracranial event as cause
Family comms	Daily update; involve in decisions Palliative pathway if appropriate
Rehabilitation	Early mobilisation; physio; cognitive follow-up

CLINICAL PEARLS

- $EtCO_2$ is your best real-time CPR quality monitor. >10 mmHg = adequate; sudden rise = ROSC.
- In hypothermic arrest, use ECMO rewarming. CPR for 60+ minutes can result in neurologically intact survival.
- POCUS during CPR identifies tamponade, pneumothorax, and hypovolaemia — but keep pauses <10 seconds.
- Feedback devices (metronomes, CPR feedback monitors) improve compliance with rate and depth — use them.
- Agonal breathing is cardiac arrest. Treat it. Do not waste time re-assessing it.
- For young patients with witnessed refractory VF: think eCPR / ECMO early and pre-alert the ECMO centre.
- Rotate compressors every 2 minutes — fatigue degrades depth within 60–90 seconds, even in trained providers.
- Post-ROSC coronary angiography should be considered even without STEMI if the history suggests ACS.

COMMON PITFALLS ⚠️

- Interrupting CPR for >10 seconds — each interruption dramatically reduces coronary perfusion pressure.
- Excessive ventilation (hyperventilation) — increases intrathoracic pressure, reduces venous return, drops coronary perfusion.
- Not confirming asystole in 2 leads — fine VF can look like asystole. Increase gain and check 2 leads.
- Delaying defibrillation to secure IV access — charge the defibrillator FIRST, then vascular access.
- Using pupils to decide on resuscitation — always a pitfall. Pupils are NOT arrest determinants.
- Giving amiodarone without confirming shockable rhythm — amiodarone is for VF/pVT only.
- No team leader assigned — unclear roles = chaos = worse outcomes. Assign roles BEFORE arrest.
- Stopping CPR during defibrillator charging — continue compressions until shock delivery (hands-off ≤5 sec).

TEAM DYNAMICS IN RESUSCITATION

TEAM LEADER	Situational awareness, directs team, calls rhythm, decides drug/shock, communicates
COMPRESSOR 1 & 2	High-quality compressions. Rotate every 2 minutes. Call own fatigue.
AIRWAY MANAGER	BVM ventilation, intubation, capnography, confirms ET tube placement
VASCULAR ACCESS	IV/IO access, drug preparation, administer drugs with 20 mL flush
RECORDER / TIMEKEEPER	Log all events, drug times, rhythm checks. Announce 2-min intervals.

Closed-loop communication: Name → Task → Acknowledge. 'John, give adrenaline 1mg now.' — 'Adrenaline 1mg, given.'

5 KEY TAKEAWAYS

01	ACT FAST: Immediate recognition + bystander CPR + early defibrillation. Every minute in VF without defibrillation = 10% less survival.
02	HIGH-QUALITY CPR IS THE MOST IMPORTANT INTERVENTION: Rate 100–120/min, depth 5–6 cm, full recoil, CCF >60%, minimal pauses.
03	FIND THE CAUSE: Treat 4Hs & 4Ts simultaneously during CPR — don't wait for ROSC. The cause is usually treatable.
04	KNOW YOUR ALGORITHM: VF/pVT = shock + adrenaline after 3rd shock + amiodarone. PEA/asystole = immediate adrenaline, no shock.
05	ROSC IS NOT THE FINISH LINE: Post-arrest care — temperature 36–37.5°C, haemodynamics, oxygenation, neuroprotection — determines survival.

THANK YOU

Questions

Dr. Daniel Olinga

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Emergency Care Association of Uganda · Seed Global Health · ECHO · Ministry of Health Uganda