STEMI WITH CARDIOGENIC SHOCK

RECOGNIZE IT — DEFINITION & FIRST LOOK

- **Definition** A cardiac disorder causing **clinical AND biochemical evidence of sustained tissue hypoperfusion** — **irrespective of blood pressure** (ACC Concise Clinical Guidance, CCG 2025)
- **How common** About **10% of STEMI,** with **early mortality 40–50%** (ACC/AHA 2025). AMI-CS is 7–10% of all ACS
- **What counts** STEMI **or** NSTEMI; LV, RV or biventricular failure; **mechanical complications;** and shock from brady- or tachyarrhythmia, heart block or cardiac arrest in the setting of AMI (SHARC definitions)

- **Suspect it SBP <90** or **MAP <60–65** or a **>30 mm Hg fall** for >30 min · **HR >100** · **pulse pressure <25% of SBP** · cold, clammy · capillary refill >2 s · **urine <30 mL/h** (<0.5 mL/kg/h) · confusion
- **Perfusion labs Lactate >2 mmol/L** · ALT >200 U/L or >3× ULN · creatinine ≥2× ULN · **pH <7.2** · unexplained metabolic acidosis
- **Mnemonic** SUSPECT CS = **S** symptoms and signs · **U** urine output · **S** sustained hypotension · **P** perfusion · **E** ECG and echo · **C** congestion · **T** triage — shock-team activation or transfer (CCG)
- **First tests** **12-lead ECG and TTE or point-of-care ultrasound in every suspected case.** ST elevation → **cath lab now.** Urgent echo is expected in shock or a suspected mechanical complication (ACC/AHA 2025 text)
- **Invasive? Not needed to make the diagnosis** — but it defines the phenotype and guides devices (CCG)
- **Trap *Normotensive shock:*** *hypoperfusion* ***without*** *hypotension carries a* ***higher*** *mortality than hypotension with preserved perfusion (SCAI 2022). Do not wait for the blood pressure to fall*
- **Trap *A normal lactate does not exclude hypoperfusion,*** *and lactate rises for other reasons (mesenteric ischaemia, compartment syndrome)*

PHENOTYPE — THE PA CATHETER NUMBERS

- **Why measure** Invasive haemodynamics define the **congestion profile,** grade severity and guide **escalation and weaning** of support (CCG). **No RCT** in shock; ESCAPE studied non-shock HF and does not apply; PACCS (HF-CS) is testing it
- **SCAI C** If measured: **cardiac index <2.2 L/min/m²** · **PCWP >15 mm Hg**
- **Cardiac power CPO = MAP × CO / 451** (watts). The **strongest independent haemodynamic correlate of in-hospital death** in the SHOCK registry (Fincke, n=541). At 24 h, **CPO <0.6 W** predicted 30-day death (INOVA)

- **LV-dominant PCWP or LVEDP >15 mm Hg**
- **RV-dominant RA (CVP) >15 mm Hg** with a **relatively normal PCWP;** an **RA:PCWP ratio >0.6** is adverse
- **Biventricular** Both RA and PCWP raised — common, and linked to death, durable LVAD and transplant in registries
- **PAPi (PA systolic − PA diastolic) / RA.** Most useful when **RA >10** and PA systolic is **not >50.** Low = **<0.9 in AMI-CS** (Korabathina: PAPi ≤0.9 identified death or RV-support need in proximal RCA occlusion — n=20). At 24 h, **PAPi <1.0** predicted death (INOVA)
- **Pearl** *CPO is flow × pressure: it catches the patient whose pressure is held up by vasopressors while the output is failing.*

WHY IS THIS HEART FAILING? — THE CAUSE

- **SHOCK registry** n=1,190 AMI-shock patients (1993–97): **predominant LV failure 78.5%** · severe MR 6.9% · VSR 3.9% · isolated RV shock 2.8% · tamponade 1.4%
- **1 in 8 A mechanical complication caused 12%** — and VSR carried **87.3% in-hospital mortality** vs 60% overall
- **Pearl** *Echo before you choose a device: the treatment of a ruptured papillary muscle, a VSR or tamponade is surgical, not a bigger pump.*

- **Iatrogenic COMMIT** (n=45,852): early IV then oral metoprolol cut reinfarction and VF but caused **11 more shocks per 1,000** (5.0% vs 3.9%), mostly on days 0–1. ACC/AHA 2025 lists **low-output state or risk of shock** as a contraindication to early β-blockade
- **Also** **Avoid IV nitroglycerin with SBP <90, a fall >30 mm Hg, or suspected RV infarction** (ACC/AHA 2025 Table)

RIGHT VENTRICULAR SHOCK

- **Recognize Every inferior STEMI → record V4R** (Zehender's conclusion). ST elevation in V4R: **sensitivity 88%, specificity 78%** for RV infarction; in-hospital death **31% vs 6%** (RR 7.7) — Zehender, n=200
- **Haemodynamics RA >15 with a relatively normal PCWP; RA:PCWP >0.6; PAPi ≤0.9** (the RV-failure signature in proximal RCA occlusion)
- **Do not** **No IV nitroglycerin in suspected RV infarction** (ACC/AHA 2025) — the RV lives on preload
- **Rate & rhythm Chronotropes may be trialled in bradycardia-induced shock** (CCG); treat heart block — see the STEMI sheet for AV block by territory
- **Ventilation** PEEP **raises pulmonary vascular resistance** and lowers preload — its effect depends on whether RV failure is present (CCG)
- **Devices** **A microaxial LV pump needs adequate RV function to fill the LV,** and DanGer **excluded overt RV failure** — the 2a does not cover RV shock. Percutaneous RV support exists; **no randomized trial** in the sources used
- **Pearl** *Volume and AV-synchrony come from physiology, not a graded recommendation — neither ACC/AHA 2025 nor the CCG grades them.*

SCAI SHOCK STAGES — SEVERITY, NOT DESTINY (SCAI 2019, UPDATED 2022)

Stage	Definition	Bedside — typically	Biochemistry — typically	Haemodynamics — typically	May also include
PRE-SHOCK — PERFUSION PRESERVED					
A · At risk	No signs or symptoms of shock, but at risk — e.g. a **large acute MI**	Normal JVP · warm, well perfused · strong pulses · normal mentation	**Normal lactate**	Normotensive (SBP ≥100 or baseline)	If measured: CI ≥2.5 · CVP ≤10 · PCWP ≤15 · PA sat ≥65%
B · Beginning	**Haemodynamic instability WITHOUT hypoperfusion** — relative hypotension or tachycardia	**Elevated JVP** · still warm and well perfused	**Normal lactate**	**SBP <90** or MAP <60 or >30 mm Hg drop · **HR ≥100**	Rales · minimal renal impairment · raised BNP
SHOCK — HYPOPERFUSION PRESENT					
C · Classic	**Hypoperfusion** needing **one intervention** (drug or device) beyond volume. Hypotension is typical, **not required**	Volume overload	**Lactate ≥2 mmol/L**	If measured (strongly recommended): **CI <2.2 · PCWP >15**	Looks unwell · altered mentation · cold, clammy, mottled · **urine <30 mL/h** · creatinine ×1.5 · raised LFTs
D · Deteriorating	Like C but **worse**: the initial strategy has **failed to restore perfusion**	Worsening or non-improving hypoperfusion despite initial therapy	**Lactate rising and persistently >2**	**Escalating doses or more pressors, or an added MCS device**	Worsening renal function and LFTs · rising BNP
E · Extremis	**Actual or impending circulatory collapse** — including CPR in progress	Typically unconscious	**Lactate ≥8 mmol/L**	**Profound hypotension despite maximal support**	Near-pulselessness · multiple defibrillations · **pH <7.2** · base deficit >10 · bolus pressors

Therapy-intensity rule (SCAI 2022): one vasoactive drug or device to reverse hypoperfusion = C; needing another drug or device after an adequate trial = D; perfusion not restored despite multiple or very high-dose agents = E. Operational cut-offs for each stage were tested by the Cardiogenic Shock Working Group (Kapur 2022, n=3,455).

A-modifier = cardiac arrest WITH coma (GCS <9 or not following commands). Stage measures shock severity, not mortality: age, arrest, phenotype and comorbidity modify risk (the 3-axis model). Improving one stage is a strong favourable sign; failing to improve is a strong adverse one.

Most patients change stage within the first 24 h, and stage B carries the highest risk of worsening (ACC CCG 2025) — restage repeatedly.

THE FIRST HOURS — STEMI WITH CARDIOGENIC SHOCK

recognize → find the cause → open the culprit → support by phenotype → restage

1 · IS THIS SHOCK? — hypoperfusion, with or without hypotension

STEMI + HYPOPERFUSION
lactate >2 · cold · oliguria · confusion →

ACTIVATE — IN PARALLEL, NOT IN SEQUENCE
Cath lab AND shock team now; call the Level 1 centre early
ECG · echo/POCUS · lactate · arterial line · assign a SCAI stage
Time from symptom onset does NOT matter in shock

DO NOT
Wait for the blood pressure to fall
Give IV β-blocker or nitroglycerin to a patient at risk
Choose a device before the echo has excluded a rupture

2 · WHAT IS FAILING? — the echo and the PA catheter decide

CAUSE & PHENOTYPE
SHOCK registry shares →

LV PUMP FAILURE (~79%)
PCWP >15 · CI <2.2 · CPO falling
→ culprit PCI, then lane 4
DanGer phenotype if LVEF <45%, not comatose

RV-DOMINANT (~3%)
RA >15, PCWP near normal, RA:PCWP >0.6
PAPi ≤0.9 · ST↑ in V4R
No nitrates · treat bradycardia · RV support

MECHANICAL (~12%)
MR 6.9% · VSR 3.9% · tamponade 1.4%
Surgical centre (COR 1) · IABP/MCS bridge (2a)
Free wall rupture → emergency surgery

3 · WAS THERE A CARDIAC ARREST? — neurology decides how far to go

RESUSCITATED ARREST
go to a PCI centre (COR 1) →

STEMI · AWAKE or FAVOURABLE
Primary PCI — COR 1, B-NR
Outcomes like STEMI without arrest

STEMI · COMATOSE, POOR FEATURES
PCI may be reasonable — COR 2b
CPR or ROSC >30 min · pH <7.2 · lactate >7
DanGer excluded these patients

NO STEMI · COMATOSE · STABLE
NO immediate angiography — COR 3: No Benefit
COACT · TOMAHAWK

4 · SUPPORT BY RESPONSE — restage the SCAI class repeatedly in the first 24 h

CULPRIT-ONLY PCI
- Emergency, any delay since onset
- CABG if PCI fails or unsuitable
- Non-culprit PCI now = HARM

→

DRUGS
- Norepinephrine for hypotension
- Add dobutamine or milrinone for low output
- Diurese congestion

→

MICROAXIAL PUMP — 2a
- Refractory, SCAI C–E, LV-dominant
- LVEF <45%, not comatose
- Large-bore access possible

→

BEYOND
- VA-ECMO only selectively — never routine
- Level 1 centre · advanced HF team
- Recovery, LVAD, transplant or palliation

THE INVARIANT — true in every branch above

Opening the culprit artery is the backbone in every phenotype (COR 1, whatever the delay since onset). Every device choice is phenotype-specific — the echo and the haemodynamics decide, not habit.
No device is COR 1. Routine IABP and routine VA-ECMO are COR 3: No Benefit; the microaxial pump's 2a covers only the DanGer-like STEMI patient.

Educational quick-reference for cardiology fellows — ACC/AHA 2025 ACS guideline (JACC 2025;85:2135) and ACC 2025 Concise Clinical Guidance on cardiogenic shock (consensus). ESC grades not reproduced.

STEMI WITH CARDIOGENIC SHOCK

OPEN THE ARTERY — REVASCULARIZATION

- **Always Emergency revascularization of the culprit vessel by PCI or CABG, irrespective of time from symptom onset** — **COR 1, LOE B-R** (ACC/AHA 2025)
- **SHOCK trial** n=302, LV-failure shock. **30-day mortality 46.7% vs 56.0% (P=0.11) — the primary endpoint was MISSED. 6-month 50.3% vs 63.1% (P=0.027),** sustained to 1 and 6 years. The foundation of the COR 1
- **Speed Ideally within 90 min** (ACC/AHA text). FITT-STEMI (n=12,675): each **10-min delay** (60–180 min) cost **3.31 deaths per 100** in shock without arrest vs **0.34** in stable STEMI
- **CABG** Emergency CABG when PCI is **not feasible or fails** (observational support; part of the COR 1)

- **Culprit only Routine PCI of a non-culprit artery at the index procedure should NOT be performed — COR 3: Harm, LOE B-R** (separate rows for STEMI and NSTE-ACS)
- **CULPRIT-SHOCK** n=706 (~60% STEMI). **30-day death or RRT 45.9% vs 55.4%** (RR 0.83); death alone RR 0.84. **At 1 year, death 50.0% vs 56.9% (RR 0.88, 95% CI 0.76–1.01)** — no longer significant — while repeat revascularization was **32.3% vs 9.4%** and HF readmission 5.2% vs 1.2%
- **Scope** Enrolled only patients with an **identifiable culprit.** An unstable-looking non-culprit or an **uncertain culprit** → individualize (ACC/AHA text)
- **Trap *Complete revascularization is COR 1 in haemodynamically stable STEMI — shock is the exception, not the rule***
- **Access** Radial preferred in ACS — **COR 1, LOE A** (all ACS, not shock-specific). Large-bore femoral access for a device: **ultrasound + fluoroscopy** guidance (ACC/AHA text)

WHO GETS A DEVICE — AND WHO DOES NOT

- **The one 2a Microaxial flow pump in SELECTED STEMI with severe or refractory shock** — **COR 2a, LOE B-R.** The only temporary support with a survival benefit in an RCT
- **"Selected" DanGer Shock entry:** STEMI + shock **<24 h; SBP <100** or vasopressor; **lactate ≥2.5 mmol/L and/or SvO_2 <55%** with normal PaO_2; **LVEF <45%. Excluded: comatose (GCS <8) after out-of-hospital arrest, and overt RV failure.** 14 specialized centres
- **In practice** ACC/AHA: **SCAI C, D or E, non-comatose,** with **vessels that take large-bore access.** CCG: **LV-dominant** shock with hypoperfusion or haemodynamic deterioration
- **Why not COR 1 Safety composite 24.0% vs 6.2% (RR 4.74); RRT 41.9% vs 26.7%** — bleeding, limb ischaemia, haemolysis. The COR balances those against the deaths prevented
- **When?** Timing was **not dictated** by protocol → the preferred timing is **unclear.** DanGer post hoc: benefit seemed to **fade beyond ~11 h** from symptoms (OR 0.51 vs 0.92; interaction P=0.26 — hypothesis only)

- **Not routine Routine IABP or VA-ECMO is not recommended — COR 3: No Benefit, LOE B-R** (one shared row). CCG: **routine tMCS in all shock is strongly discouraged**
- **Pooled** IPD meta-analysis (9 RCTs, n=1,114): early routine MCS **HR 0.87 (0.74–1.03)** for 6-month death; **STEMI-CS without risk of hypoxic brain injury HR 0.77 (0.61–0.97);** major bleeding **OR 2.64,** vascular **OR 4.43**
- **Bridge Short-term MCS as a bridge to surgery for a mechanical complication** — **COR 2a, LOE B-NR**
- **Escalate** Under-supported → **more flow or a bigger device** (e.g. CP → 5.5). Not unloaded → **address afterload** (LV venting on VA-ECMO) and congestion (CCG)
- **Wean Daily readiness check;** step down **0.5–1 L/min every 2–4 h** (≈2 P-levels on a microaxial pump); ask advanced HF first — recovery, durable LVAD or transplant? (CCG)
- **Trap *The 2a is shock-only.*** *STEMI-DTU (JACC 2026, n=527): 30 min of unloading before PCI in **anterior STEMI WITHOUT shock** — infarct size **30.8% vs 31.9%** (P=0.50), with more major bleeding and vascular complications*

CARDIAC ARREST + STEMI

- **Where** Resuscitated arrest with STEMI → **EMS straight to a PCI centre** — **COR 1, LOE C-LD**
- **Awake Non-comatose, or comatose with favourable features** + STEMI → **primary PCI** — **COR 1, LOE B-NR.** About **one-third** arrive neurologically intact; their outcomes match STEMI without arrest
- **Comatose, poor** PCI **may be reasonable after individualized assessment** — **COR 2b, LOE C-LD**
- **Poor features** Unwitnessed · no bystander CPR · non-shockable rhythm · **CPR >30 min** · **ROSC >30 min** · **pH <7.2** · **lactate >7 mmol/L** · **age >85** · dialysis-dependent ESRD
- **No STEMI Comatose, electrically and haemodynamically stable, no ST elevation → NO immediate angiography — COR 3: No Benefit, LOE A** (six trials)
- **COACT / TOMAHAWK** COACT (n=552): 90-day survival **64.5% vs 67.2%.** TOMAHAWK (n=554): 30-day death **54.0% vs 46.0%** (HR 1.28, 1.00–1.63); death or severe neurological deficit **64.3% vs 55.6%** — trending the wrong way
- **Pearl** *The SCAI "A" modifier means arrest **with coma** (GCS <9 or not following commands) — a brief defibrillated arrest with normal neurology does not change the prognosis.*

VASOACTIVE DRUGS — LOWEST DOSE, SHORTEST TIME

Agent	Receptors · dose (CCG Table 2)	SVR · BP · CO · HR	Use when	What the trials show	Caution
INOPRESSORS					
Norepinephrine	α1+++ β1++ β2+ · **0.05–1 µg/kg/min**	↑↑ · ↑↑ · ↑ · ↑	**Reasonable first choice for most hypotensive shock** (CCG consensus)	**SOAP II** (n=1,679, all shock): vs dopamine, arrhythmias **12.4% vs 24.1%**; in the **cardiogenic subgroup (n=280)** dopamine had higher 28-day mortality (P=0.03, subgroup)	Arrhythmia and ↑ myocardial O_2 demand as doses climb (CCG, class caution)
Epinephrine	β1+++ α1++ β2++ · 0.01–0.5 µg/kg/min	↑↑ · ↑↑ · ↑↑ · ↑↑	No preferred role in the sources used	**OptimaCC** (n=57, AMI-CS): same CI and BP as norepinephrine but **refractory shock 37% vs 7%** → stopped early; more tachycardia, double product and lactic acidosis	**Not the first pressor in AMI shock** — the only RCT points the other way
Dopamine	D1+++ β1++ α1+ · 2–5 / 5–10 / 10–20 µg/kg/min	↑↑ · ↑↑ · ↑ · ↑↑	Bradycardia-driven shock (chronotrope)	SOAP II: **twice the arrhythmias** of norepinephrine	**Arrhythmia; worse survival signal in cardiogenic shock**
INODILATORS AND INOTROPES — LOW OUTPUT, ADEQUATE PRESSURE					
Dobutamine	β1+++ β2++ · **2–10 µg/kg/min**	↓↔ · ↓↔ · ↑↑ · ↑	Low output with adequate pressure	**DOREMI** (n=192, single centre, SCAI B–E): vs milrinone, composite **54% vs 49%** (RR 0.90, P=0.47); in-hospital death 43% vs 37% — **no difference**	Hypotension · tachyarrhythmia · ↑ myocardial O_2 demand
Milrinone	PDE-3 inhibitor · **0.125–0.5 µg/kg/min**	↓↓ · ↓↓ · ↑↑ · ↔↑	Low output with high SVR	DOREMI — equivalent to dobutamine	**Renally cleared, long half-life — use judiciously as creatinine rises**; hypotension
Levosimendan	Calcium sensitizer · 0.05–0.2 µg/kg/min	↓ · ↓ · ↑ · ↔	—	Cochrane: no agent proven superior for mortality	**Not FDA-approved in the US**
PURE PRESSORS, DILATORS, RATE					
Vasopressin	V receptor · 0.01–0.04 U/min	↑↑ · ↑↑ · ↔↓ · ↔↓	—	No AMI-shock RCT cited by the CCG	Raises afterload without inotropy
Phenylephrine	α1+++ · 0.1–10 µg/kg/min	↑ · ↑↑ · ↔↓ · ↔↓	—	—	**Strongly discouraged as the single first-line agent — reflex bradycardia, falling output**
Nitroprusside · nitroglycerin	NO donors · 0.3–10 µg/kg/min · NTG 25–200 µg/min (start 10 µg/min)	↓ · ↓ · ↑↔ · ↑↔	**Normotensive shock with high SVR**; acute MR or VSR if pressure allows	Afterload reduction lowers regurgitant and shunt flow (AHA 2021 statement)	**NTG: not with SBP <90, a >30 mm Hg fall, or suspected RV infarction**
Isoproterenol	β1+++ β2+++ · 2–20 µg/min	↓ · ↔ · ↑ · ↑↑	**Bradycardia-induced shock**	—	Tachyarrhythmia; vasodilation

Doses are as printed in the ACC 2025 Concise Clinical Guidance (Table 2) — a consensus table, not FDA labelling; check your institution's protocol. ACC/AHA 2025 makes NO vasopressor or inotrope recommendation.
Target MAP >60–65 mm Hg in the optimization phase (CCG). Treat congestion too: IV loop diuretic, add a thiazide, ultrafiltration if refractory.

TEMPORARY MECHANICAL SUPPORT IN AMI SHOCK — WHAT THE RANDOMIZED TRIALS SHOW

Device	What it does	Randomized evidence in AMI shock	ACC/AHA 2025	Harms · caveats
DEVICES				
IABP	Diastolic inflation **raises coronary perfusion**; systolic deflation **lowers afterload**. Small profile → fewer access complications	**IABP-SHOCK II** (n=600): 30-day death **39.7% vs 41.3%** (RR 0.96); **6.2 years 66.3% vs 67.0%**. No difference in lactate, catecholamines or ICU stay	**Routine: COR 3 No Benefit (B-R) · bridge for a mechanical complication 2a**	Bleeding 3.3% vs 4.4%, limb ischaemia 4.3% vs 3.4% — **safe but ineffective**. Still useful in **VSR and acute MR** (cuts shunt/regurgitant flow)
Microaxial flow pump	Drains the LV into the ascending aorta — **unloads the LV**	**DanGer Shock** (n=355): 180-day death **45.8% vs 58.5%**, **HR 0.74 (0.55–0.99), NNT 8**; ~10-year follow-up **52.5% vs 68.8%** (HR ≈0.70). Earlier: **ISAR-SHOCK** (n=26) raised CI more than IABP (+0.49 vs +0.11) with 30-day death 46% in both; **IMPRESS** (n=48, ventilated) 30-day death 46% vs 50% vs IABP	**COR 2a (B-R) in selected STEMI with severe or refractory shock**	**Needs adequate RV function and oxygenation**. Safety composite **24.0% vs 6.2%**; RRT 41.9% vs 26.7%. **Not tested in comatose post-arrest or overt RV failure**
VA-ECMO	Provides **flow AND oxygenation** — but **raises LV afterload**	**ECLS-SHOCK** (n=417): 30-day death **47.8% vs 49.0%** (RR 0.98); moderate/severe bleeding **23.4% vs 9.6%**, vascular complications **11.0% vs 3.8%**. **ECMO-CS** (n=117): composite 63.8% vs 71.2% (HR 0.72, P=0.21) — 39% of controls got ECMO later	**Routine: COR 3 No Benefit (B-R)**	**Bleeding, limb ischaemia, LV distension — may need venting**. HYPO-ECMO: 33–34 °C for 24 h did not significantly cut 30-day death (42% vs 51%, P=0.15)
ALL DEVICES POOLED				
IPD meta-analysis	9 RCTs, n=1,114 — loading (VA-ECMO) and unloading devices	6-month death **HR 0.87 (0.74–1.03)** overall; unloading 0.80 (0.62–1.02); loading 0.93 (0.75–1.17). **STEMI-CS without risk of hypoxic brain injury: HR 0.77 (0.61–0.97)**	**—**	Major bleeding **OR 2.64**, vascular complications **OR 4.43**. The benefit lives in the patient DanGer enrolled

DanGer 10-year figures are from the NEJM 2025 correspondence as summarized by the ACC (letter not read directly). ECMO-CS enrolled mixed-cause shock; its AMI subgroup (n=74) was consistent (ACC/AHA text).
Large-bore access (≥12 Fr): pulse checks every 1–2 h, avoid excessive anticoagulation, re-examine after every turn or transfer; remove in the cath lab or OR when possible; uncontrollable bleeding or limb ischaemia usually means the device comes out (CCG).

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SYSTEMS OF CARE — SHOCK TEAMS & TRANSFER

- **Levels (CCG) Level 1:** 24/7 medical and surgical expertise, all tMCS, high volume, often LVAD/transplant. **Level 2/3:** PCI or diagnostic capability, limited devices. No agreed national tiering yet
- **Transfer** **Refractory AMI shock after local revascularization → almost always to a Level 1 centre** (CCG)
- **Call early** Contact the regional centre when **≥1 vasoactive drug** is started, a **device is being considered,** or after **arrest with tenuous neurology** — "never too early"
- **Five questions** SCAI stage and SHARC class? · Needs escalation now? · Any limit on escalation (DNR, terminal illness)? · Do we have the beds, skills and devices? · **Stable enough to move?**
- **Shock teams CCCTN** (24 CICUs, 10 with teams): shock-team centres used **more PA catheters** (60% vs 49%), **less MCS overall** but more advanced types, and had **lower CICU mortality 23% vs 29%** (adjusted OR 0.72). **INOVA** single centre: 30-day survival **47% → 58% → 77%** over 3 years — observational, before/after
- ⚠ **Grade** ACC/AHA 2025 has **no graded transfer recommendation** for shock; the CCG cites a **Class 2b** transfer statement from the 2022 HF guideline. The CCG itself is **consensus without COR/LOE**

THE FIRST 24 HOURS IN THE CICU

- **Where** Shock → **cardiac ICU** — **COR 1, LOE C-EO** (ACC/AHA 2025)
- **Target MAP >60–65 mm Hg** (optimization phase). Framework: **Recognize/Rescue → Optimize → Stabilize → De-escalate/Exit**
- **Reassess** **Urine hourly** · **perfusion labs every 2–4 h early** (6–8 h once stable) · **pulse checks every 1–2 h with large-bore access** · echo daily (POCUS acceptable) · shock-team review at least daily
- **Congestion IV loop diuretic,** add a thiazide, **ultrafiltration** if refractory — untreated congestion injures kidney, liver and gut
- **Ventilation** Positive pressure **lowers LV afterload** and helps LV failure with or without severe MR

- **Bleeding** **Major bleeding may reach 60% in AMI shock; limb ischaemia is 4× higher with tMCS** and **doubles in-hospital death**
- **Palliative Early palliative care** when devices go in — some patients will not be candidates for recovery, LVAD or transplant

TRAPS THAT KILL — OR MISLEAD

- **Trap *Waiting for hypotension.*** *Normotensive hypoperfusion is shock, with worse survival than hypotension alone*
- **Trap *Reading SHOCK as negative.*** *It missed its 30-day endpoint; the survival benefit appeared at 6 months and lasted 6 years*
- **Trap *Stenting every lesion.*** *Non-culprit PCI at the index procedure in shock is COR 3: Harm*
- **Trap *Reflex IABP or ECMO.*** *Both are COR 3: No Benefit when routine*
- **Trap *Extending DanGer to everyone.*** *It excluded comatose post-arrest patients and overt RV failure, and raised serious device events almost five-fold*
- **Trap *Dopamine, epinephrine or phenylephrine first.*** *Each has a trial signal or a consensus warning against it as the opening agent*
- **Trap *"No murmur, so no papillary rupture."*** *The murmur can vanish when LA and LV pressures equalize — get the echo (TEE if needed)*
- **Trap *Nitroglycerin in inferior STEMI with hypotension.*** *Suspect RV infarction first*
- **Trap *Immediate angiography for every comatose arrest.*** *Without ST elevation and with stable haemodynamics it is COR 3: No Benefit*
- **Trap *Early IV β-blocker in a patient at risk of shock*** *(COMMIT: 11 extra shocks per 1,000)*
- **Trap *Equating SCAI stage with prognosis.*** *Age, arrest with coma and phenotype move risk within every stage*

GUIDELINE SILENCES — AND WHAT IS COMING

- ⚠ **Silent ACC/AHA 2025 makes no vasopressor, inotrope or PA-catheter recommendation.** Cite the **ACC CCG 2025** (consensus) for norepinephrine first and for invasive haemodynamics
- ⚠ **Not here ESC 2023 grades are not printed here** — its full text could not be opened for this build, and it predates DanGer Shock. The STEMI sheet carries the ESC positions it could verify
- **DanGer, long term ~10-year** mortality **52.5% vs 68.8%** (HR ≈0.70; NEJM 2025 letter, as summarized by the ACC) — the benefit did not fade
- **MCS centres FITT-STEMI** (2013–22, n=5,604 STEMI-CS): hospital mortality **44.7% vs 45.0%** at centres with vs without MCS on site — having devices is not the same as selecting patients well
- **Stopped RECOVER IV** (US microaxial-pump RCT, NCT05506449) — **terminated** after 4 patients
- **Watch ANCHOR** (VA-ECMO in AMI-CS, NCT04184635) · **RESCUE-SHOCK** (culprit-only vs multivessel PCI on VA-ECMO, NCT05527717) · a Leipzig trial of routine microaxial pump and PA-catheter-guided care (NCT07762560) · **PACCS** (PA catheter, HF-CS)

MECHANICAL COMPLICATIONS — <1% OF AMI, MOSTLY IN THE FIRST WEEK, MOSTLY SURGICAL

Complication	When · who	Clues	Confirm	Bridge	Definitive · mortality
RUPTURES					
Papillary muscle rupture	**Within days**; 0.05–0.26%. **Posteromedial** (single supply — RCA or Cx) → **inferior/lateral MI**; anterolateral (dual supply) is rare	Sudden **pulmonary oedema** (~half) → shock. **The murmur may be ABSENT** (LA and LV pressures equalize). LVEF often normal; often 1–2-vessel disease	TTE — **TEE if partial rupture** (TTE can miss it)	Positive-pressure ventilation; NTG or nitroprusside if pressure allows; IABP; **short-term MCS (COR 2a)**	**Surgical emergency** — usually **valve replacement**. Hospital mortality **10–40%**
Ventricular septal rupture	**Day 3–5**; ~0.3%. Older, female, **delayed reperfusion**. Anterior/apical (LAD) **70%** vs inferior 29% — inferior VSRs are more complex	**New pansystolic murmur** at the lower left sternal edge; hypotension, cool, oliguric	Echo · RHC: **O_2 step-up RA → PA**; Qp:Qs up to 8:1	**Afterload reduction**. IABP: SBP **81 → 102 mm Hg** within 30 min (SHOCK registry). ECMO for multi-organ failure — venting has specific hazards here	Untreated: **~80% dead at 30 days**; SHOCK registry in-hospital **87.3%**. Early shock WITHOUT organ failure = best emergency-surgery candidate; percutaneous closure if surgery is prohibitive
Free wall rupture	True incidence unknown — often **sudden out-of-hospital death**. Risk with **delayed or ineffective reperfusion**	**Sudden collapse**: JVD, pulsus paradoxus, **PEA**, muffled sounds; may follow chest pain and nausea; new ST elevation	**Bedside echo**	ECMO may stabilize — but tamponade limits venous return	**Emergency surgery**; mortality **>35%** even after repair. Watch for the **oozing** variant
Contained rupture (pseudoaneurysm)	Rupture sealed by pericardial adhesions — usually **inferior or lateral** wall	HF, chest pain, dyspnoea, arrhythmia or embolism — **none pathognomonic**	Imaging	—	High rupture risk → most experts favour **prompt surgery**

ACC/AHA 2025: manage any mechanical complication in a centre with cardiac surgical expertise — COR 1, LOE C-EO; short-term MCS as a bridge to surgery — COR 2a, LOE B-NR. Overall surgical mortality ~40%; delayed surgery (>7 days) looks better, but selection and survivor bias drive much of that. Details from the AHA 2021 scientific statement on mechanical complications (Damluji). Heart Team from the moment of diagnosis — deterioration is unpredictable and can be precipitous.

ACC/AHA 2025 RECOMMENDATIONS FOR ACS WITH CARDIOGENIC SHOCK

REVASCULARIZATION			
1	B-R	ACS with cardiogenic shock or haemodynamic instability: emergency revascularization of the culprit vessel by PCI or CABG to improve survival, irrespective of time from symptom onset.	ACC/AHA 2025
3: Harm	B-R	ACS (STEMI or NSTE-ACS — two separate rows) complicated by cardiogenic shock: routine PCI of a non-infarct-related artery at the time of the index PCI should NOT be performed — higher risk of death or renal failure.	ACC/AHA 2025
1	A	ACS undergoing PCI: radial approach preferred over femoral to reduce bleeding, vascular complications and death (all ACS, not shock-specific).	ACC/AHA 2025
MECHANICAL CIRCULATORY SUPPORT AND MECHANICAL COMPLICATIONS			
2a	B-R	Selected patients with STEMI and severe or refractory cardiogenic shock: a microaxial intravascular flow pump is reasonable to reduce death ("selected" = DanGer Shock-like, per the supportive text).	ACC/AHA 2025
3: No Benefit	B-R	AMI with cardiogenic shock: ROUTINE intra-aortic balloon pump or VA-ECMO is not recommended — no survival benefit.	ACC/AHA 2025
2a	B-NR	Mechanical complication of ACS: short-term MCS devices are reasonable for haemodynamic stabilization as a bridge to surgery.	ACC/AHA 2025
1	C-EO	Mechanical complication of ACS: manage in a facility with cardiac surgical expertise.	ACC/AHA 2025
1	C-EO	ACS with haemodynamic instability, uncontrolled arrhythmia, suboptimal reperfusion or cardiogenic shock: admit to a cardiac ICU.	ACC/AHA 2025
CARDIAC ARREST			
1	C-LD	Resuscitated cardiac arrest with STEMI: EMS should preferentially transport to a primary-PCI-capable centre.	ACC/AHA 2025
1	B-NR	Resuscitated, non-comatose — or comatose with favourable prognostic features — with STEMI: primary PCI to improve survival.	ACC/AHA 2025
2b	C-LD	Comatose with unfavourable prognostic features and STEMI: primary PCI may be reasonable after individualized assessment.	ACC/AHA 2025
3: No Benefit	A	Comatose after arrest, electrically and haemodynamically stable, WITHOUT STEMI: immediate angiography is not recommended.	ACC/AHA 2025

1 *is recommended* · 2a *is reasonable* · 2b *may be considered* · 3: No Benefit *is not recommended* · 3: Harm *is potentially harmful*

Educational quick-reference for cardiology fellows — ACC/AHA 2025 ACS guideline (JACC 2025;85:2135) and ACC 2025 Concise Clinical Guidance on cardiogenic shock (consensus). ESC grades not reproduced.