[HOSPITAL / HEALTH AUTHORITY NAME]

SHOCK AND HYPOTENSION PATHWAY

Protocol 15: Rapid Recognition, Shock Classification, Perfusion-Guided Resuscitation, Cause-Directed Treatment, Reassessment, and Safe Disposition

DRAFT FOR EMERGENCY MEDICINE, INTERNAL MEDICINE, ANAESTHESIA, CRITICAL CARE, SURGERY, OBSTETRICS, PAEDIATRICS, CARDIOLOGY, NURSING, PHARMACY, LABORATORY, BLOOD BANK, IMAGING, TRANSFER, AND CLINICAL-GOVERNANCE REVIEW

IMMEDIATE SAFETY RULE: Shock is acute circulatory failure. It may be present before marked hypotension develops. Treatment must restore tissue perfusion while the cause is identified and corrected.

Document control	Details
Document owner	Emergency Department / Medical Services / Nursing Services / Clinical Governance
Policy number	ED-CLIN-015
Version	Draft 1.0
Effective date	[To be assigned after approval]
Review date	[Normally 12-24 months, or earlier after major evidence or service change]
Supersedes	New protocol
Applies to	All staff involved in the assessment, resuscitation, transfer, and continuing care of patients with suspected shock or clinically important hypotension
Related protocols	Protocols 1-12; Protocol 13 Acute Chest Pain and Suspected ACS; Protocol 14 Acute Shortness of Breath and Respiratory Distress; local sepsis, major haemorrhage, trauma, obstetric, anaphylaxis, toxicology, paediatric, blood-transfusion, and resuscitation algorithms

STATUS: This is a draft clinical-governance document. Exact medication doses, fluid volumes, blood-component ratios, vasopressor concentrations, haemodynamic targets, paediatric parameters, and procedural techniques must be confirmed against the hospital formulary, available equipment, approved algorithms, and staff competencies before implementation.

1. Purpose

To provide a standardized, time-sensitive, and auditable pathway for recognizing and treating adults, adolescents, and children with suspected shock, hypotension, or tissue hypoperfusion. The protocol aims to prevent delayed recognition, indiscriminate fluid administration, delayed haemorrhage control, delayed vasoactive support, and unsafe transfer by requiring repeated assessment of perfusion, physiology, likely mechanism, response to intervention, and need for definitive source control.

2. Scope

This protocol applies from first clinical contact in the Emergency Department until shock has resolved, responsibility has transferred to a suitably monitored inpatient or critical-care team, or the patient has been transferred to another facility. It includes undifferentiated shock and hypoperfusion due to sepsis, haemorrhage, dehydration or fluid loss, cardiac pump failure, pulmonary embolism, tension pneumothorax, cardiac tamponade, anaphylaxis, neurogenic shock, endocrine crisis, poisoning, and mixed mechanisms. Cardiac arrest, neonatal resuscitation, and definitive specialty management remain governed by their specific protocols.

3. Core policy statements

- Every patient with suspected shock shall be classified Red / immediate and moved to a resuscitation-capable area. Registration, payment, imaging, or transfer paperwork must not delay life-saving treatment.
- Shock shall be suspected from evidence of inadequate tissue perfusion or organ dysfunction, not from blood pressure alone. A patient may be in shock with a normal or transiently normal blood pressure.
- ABCDE assessment, monitoring, vascular access, bedside glucose, focused history, examination for bleeding and reversible causes, and early senior review shall occur in parallel.
- Resuscitation shall use repeated small, defined interventions followed by documented reassessment. Fluids are medicines; they require an indication, dose, rate, endpoint, and stop rule.
- Balanced crystalloids are generally preferred for non-haemorrhagic resuscitation unless a condition-specific reason supports another fluid. Starches shall not be used for shock resuscitation.
- Active haemorrhage requires immediate haemorrhage control and early blood-product support. Crystalloid must not be used as a substitute for blood in ongoing major bleeding.
- In left-sided cardiogenic shock, routine fluid loading as the primary treatment is unsafe. A small test bolus may be considered only when hypovolaemia or right-sided preload dependence is plausible and reassessment is immediate.
- Vasopressor support shall not be delayed when hypotension and hypoperfusion persist despite appropriate initial fluid or when further fluid is unsafe. Peripheral initiation may be used through a suitable, closely monitored line under an approved protocol while definitive access is arranged.
- Definitive treatment of the cause—haemorrhage control, antibiotics and source control, coronary reperfusion, relief of obstruction, intramuscular adrenaline for anaphylaxis, antidote, or obstetric intervention—must proceed alongside haemodynamic support.
- Each resuscitation cycle shall reassess mental status, capillary refill and skin perfusion, pulse, blood pressure or MAP, respiratory status, urine output when available, lactate trend where indicated, and evidence of fluid intolerance.
- Transfer to critical care, theatre, catheterization, obstetric services, or a higher-level facility shall be activated early when definitive capability is unavailable locally.

4. Definitions

Term	Operational definition
Shock	Acute circulatory failure causing inadequate cellular oxygen delivery, impaired oxygen utilization, or both, with threatened or established organ dysfunction.

Term	Operational definition
Hypotension	Blood pressure below the expected range for the patient. It is a warning sign, not a complete definition of shock.
Cryptic or compensated shock	Tissue hypoperfusion despite a blood pressure that is not markedly low, often signalled by tachycardia, altered mentation, oliguria, delayed capillary refill, mottling, rising lactate, or a narrow pulse pressure.
Fluid responsiveness	A clinically meaningful increase in stroke volume or cardiac output after a reversible preload challenge. Fluid responsiveness does not by itself prove that more fluid is beneficial.
Fluid tolerance	The ability to receive fluid without causing harmful pulmonary, systemic venous, abdominal, or tissue congestion.
Perfusion target	A patient-specific clinical endpoint reflecting restored organ perfusion, such as improving mentation, capillary refill, skin temperature, urine output, lactate trend, and stable haemodynamics.
Major haemorrhage	Bleeding that is immediately life-threatening, causes haemodynamic instability or critical organ compromise, or is expected to require rapid blood-component replacement and definitive haemorrhage control.
Vasopressor	A medicine used primarily to increase vascular tone and arterial pressure.
Inotrope	A medicine used primarily to augment myocardial contractility and cardiac output.
Source control	A definitive intervention that removes or controls the cause of infection, bleeding, obstruction, or contamination.

5. Roles and accountability

Role	Minimum accountability
Triage / receiving nurse	Recognizes shock indicators, assigns Red acuity, activates the resuscitation response, starts permitted standing actions, and documents the time of recognition.
Resuscitation nurse	Applies monitoring, obtains access and specimens, administers authorized treatment, tracks fluids and medicines, trends perfusion, and immediately escalates deterioration.
ED clinician / team leader	Leads ABCDE assessment, identifies probable shock mechanism, orders and reviews treatment, defines endpoints and stop rules, activates specialty pathways, and retains responsibility until explicit handover.
Senior ED / acute-care physician	Reviews persistent, mixed, refractory, or diagnostically uncertain shock; approves advanced vasoactive, procedural, and transfer decisions within local governance.
Anaesthesia / critical care	Supports airway, ventilation, invasive monitoring, vasoactive treatment, organ support, and critical-care disposition.
Surgery / obstetrics / interventional services	Provide urgent definitive haemorrhage or source control and participate in major haemorrhage activation.

Role	Minimum accountability
Cardiology	Supports suspected cardiogenic shock, acute coronary syndrome, arrhythmia, mechanical complication, echocardiography, reperfusion, and transfer decisions.
Laboratory / blood bank	Prioritizes shock specimens, communicates critical results, activates major haemorrhage support, and maintains component traceability.
Pharmacy	Supports vasopressor preparation, emergency medication safety, antidotes, concentrations, compatibility, and stock readiness.
Transfer / bed-management team	Coordinates critical-care admission, theatre, interfacility transfer, transport platform, escort, and receiving-facility acceptance without interrupting active resuscitation.

6. Pathway activation and triage

Activate this pathway for hypotension, collapse, syncope with instability, unexplained tachycardia or bradycardia, altered mental status, cool or mottled skin, delayed capillary refill, weak pulse, oliguria, major bleeding, severe infection with organ dysfunction, chest pain with hypoperfusion, severe allergic reaction, suspected pulmonary embolism, tension pneumothorax, tamponade, or any clinician concern for circulatory failure.

- Red / immediate: signs of shock or threatened circulatory collapse, active major haemorrhage, rapidly falling blood pressure, severe respiratory compromise, altered consciousness, severe chest or abdominal pain with instability, anaphylaxis, or suspected obstructive shock.
- Do not downgrade solely because a single blood pressure is normal or improves transiently.
- Place the patient in a resuscitation-capable area; apply infection precautions as indicated without delaying treatment.
- Use age-appropriate paediatric and pregnancy-adjusted assessment. Maternal hypotension may be a late sign of major blood loss.

DO NOT MISS: A deteriorating patient with warm skin, bounding pulse, normal blood pressure, or isolated tachycardia may still have early distributive or compensated shock. Trend the whole patient.

7. The first 5 minutes

Target	Required action
0-1 minute	Call for resuscitation help; begin ABCDE; assess responsiveness, airway, breathing, major external bleeding, pulse quality, skin perfusion, and immediately reversible causes.
0-3 minutes	Attach ECG, pulse oximetry, and cycling non-invasive blood pressure; record temperature; obtain bedside glucose; position appropriately; provide oxygen or ventilatory support when indicated.
0-5 minutes	Establish two suitable IV lines or IO access if needed; draw urgent bloods; assess for bleeding, infection, cardiac failure, and obstruction; activate major haemorrhage, sepsis, ACS, obstetric, anaphylaxis, or transfer pathway as indicated.

Target	Required action
Within the first resuscitation cycle	Give a cause-appropriate intervention—haemorrhage control and blood, defined crystalloid challenge, vasopressor, intramuscular adrenaline, decompression, rhythm treatment, or other definitive action—and document the response.
Continuous	Assign a team leader and recorder; communicate the working shock type, immediate threats, treatments, endpoints, and next review time.

8. Recognition of shock and tissue hypoperfusion

No single observation is sufficiently sensitive. Use serial findings and clinical trajectory.

Domain	Concerning findings
Neurological	Anxiety, agitation, confusion, reduced responsiveness, new weakness, or collapse.
Skin / microcirculation	Cool or clammy skin, mottling, cyanosis, delayed capillary refill, or paradoxically warm flushed skin in distributive shock.
Cardiovascular	Tachycardia or inappropriate bradycardia, weak or bounding pulse, narrow pulse pressure, hypotension, arrhythmia, raised or flat neck veins, chest pain.
Respiratory	Tachypnoea, hypoxaemia, increased work of breathing, pulmonary oedema, or respiratory fatigue.
Renal	Reduced urine output or no urine; urine output is a delayed marker and should not postpone treatment.
Metabolic / laboratory	Raised or rising lactate, metabolic acidosis, base deficit, worsening renal or hepatic function, coagulopathy, or hypoglycaemia / hyperglycaemia.
Context	Bleeding, trauma, pregnancy, infection, myocardial infarction, heart failure, pulmonary embolism, allergy, toxin exposure, endocrine disease, dehydration, or recent procedure.

9. Immediate stabilization: ABCDE

9.1 Airway and breathing

- Open and protect the airway; use cervical-spine precautions when indicated.
- Give oxygen for hypoxaemia, respiratory distress, peri-arrest, major trauma, or another clear indication; titrate when reliable monitoring is available.
- Assist ventilation early when mental status, fatigue, acidosis, or respiratory failure threatens oxygen delivery.
- Recognize that positive-pressure ventilation can worsen venous return and precipitate collapse in severe hypovolaemia, right-ventricular failure, tamponade, or tension pneumothorax. Optimize the circulation and have rescue treatment immediately available.

9.2 Circulation

- Control compressible external bleeding immediately with direct pressure, packing, tourniquet, pelvic binder, or other locally approved method.

- Obtain two large-bore peripheral IV lines when feasible. Use IO access if critical treatment is delayed. Do not delay vasopressor or blood administration while pursuing central access.
- Obtain blood samples without delaying resuscitation: full blood count, electrolytes, renal and liver function, glucose, blood gas and lactate, coagulation profile, group and screen / crossmatch, cultures where infection is suspected, pregnancy test where relevant, and cause-specific tests.
- Start a cause-appropriate resuscitation intervention and reassess immediately. Avoid automatic repeated fluid boluses.
- Consider early arterial-line placement and central access in refractory shock when expertise and monitoring are available, but do not delay emergency treatment.

9.3 Disability and exposure

- Check glucose and treat severe abnormality.
- Assess pupils, GCS or AVPU, temperature, pain, and seizure activity.
- Fully expose as clinically necessary to find haemorrhage, rash, swelling, burns, trauma, or infection while preventing hypothermia and preserving dignity.
- In pregnancy, assess gestation, vaginal bleeding, abdominal pain, fetal concern, and signs of ectopic pregnancy or postpartum haemorrhage; call obstetric support early.

10. Rapid classification of shock

Shock is commonly mixed. The initial classification directs the safest first intervention and must be revised as new data emerge.

Mechanism	Common causes	Typical clues	Immediate treatment emphasis
Hypovolaemic / haemorrhagic	External or internal bleeding, GI bleed, ruptured ectopic or aneurysm, dehydration, vomiting, diarrhoea, burns, third-space loss	Flat neck veins, cool peripheries, narrow pulse pressure, blood loss or fluid-loss history; ultrasound may show low filling but is not definitive	Stop losses; haemorrhage control and early blood if bleeding; defined crystalloid replacement for non-haemorrhagic volume loss; prevent hypothermia
Distributive	Sepsis, anaphylaxis, neurogenic shock, vasoplegia, endocrine crisis	Warm or flushed skin early, wide pulse pressure, fever or hypothermia, rash / airway signs, spinal injury; may become cold later	Treat cause immediately; appropriate crystalloid assessment; early vasopressor when hypotension persists or fluid is unsafe
Cardiogenic	Acute MI, myocarditis, severe heart failure, mechanical complication, acute valve disease, arrhythmia	Pulmonary oedema, raised JVP, cool skin, chest pain, new murmur, ECG / echo abnormalities	Avoid routine fluid loading; treat rhythm and ischaemia; early cardiology / critical care; vasopressor / inotrope guided by phenotype
Obstructive	Massive PE, tension pneumothorax, cardiac tamponade, dynamic hyperinflation	Raised JVP, sudden dyspnoea, unilateral absent breath sounds, pulsus / echo clues, right-heart strain, peri-arrest physiology	Immediate relief of obstruction or urgent transfer for definitive intervention; cautious fluids only in selected preload-dependent states
Mixed	Sepsis with cardiomyopathy, trauma plus tension pneumothorax, haemorrhage with anaesthetic vasodilation, PE with RV failure	Overlapping findings and changing response	Treat each mechanism; use serial examination, ultrasound, dynamic response, and senior review

11. Focused diagnostic assessment

11.1 History and examination

- Determine onset, trajectory, preceding symptoms, fluid or blood loss, infection, chest pain, dyspnoea, allergy, pregnancy, trauma, toxin or medication exposure, comorbid cardiac / renal / hepatic disease, anticoagulant use, recent surgery, and baseline functional status.
- Examine for source of bleeding or infection; heart sounds and murmurs; lung fields; jugular venous pressure; abdominal, pelvic, back, and limb findings; rash or angioedema; neurological deficit; and signs of endocrine crisis.
- Review prehospital observations and treatments. A transient response may conceal ongoing bleeding or deterioration.

11.2 Investigations

- Obtain a 12-lead ECG early in undifferentiated shock and immediately when cardiac ischaemia or arrhythmia is possible.
- Use bedside ultrasound, when trained staff are available, to identify pericardial effusion, ventricular dysfunction, right-heart strain, pneumothorax, free fluid, aortic pathology, and response to a reversible preload challenge. Ultrasound supplements but does not replace clinical assessment or definitive imaging.
- Choose imaging according to stability. An unstable patient should not be sent to a remote imaging area unless the result is immediately necessary, transport risks are controlled, and definitive treatment will follow.
- Trend lactate when elevated or when ongoing hypoperfusion is suspected. Interpret lactate in context because adrenergic stimulation, seizures, liver dysfunction, drugs, and other mechanisms may elevate it.

12. Perfusion goals, monitoring, and reassessment

Use a patient-specific set of endpoints rather than a blood-pressure number alone. A typical initial MAP target in adult septic shock is approximately 65 mmHg, but targets must be individualized for chronic hypertension, pregnancy, traumatic brain injury, aortic disease, cardiogenic shock, age, and response.

Reassess after every intervention	Document
Macrocirculation	Heart rate and rhythm, blood pressure / MAP, pulse pressure, shock index trend where appropriate, and vasoactive dose.
Microcirculation	Capillary refill, skin temperature and mottling, peripheral pulse quality, and mental status.
Organ perfusion	Urine output when measurable, cognition, chest pain, respiratory effort, renal function, and lactate / acid-base trend where indicated.
Fluid tolerance	New crackles, increasing oxygen need, B-lines or pulmonary oedema, rising JVP, peripheral oedema, hepatomegaly, abdominal pressure, or worsening right-heart congestion.
Cause control	Bleeding stopped or continuing; infection source addressed; obstruction relieved; reperfusion or procedure underway.
Next decision	Continue, change, or stop fluid; start or titrate vasopressor / inotrope; give blood; perform procedure; escalate; transfer.

PERFUSION PRINCIPLE: A rise in blood pressure without improving perfusion is not successful resuscitation. Conversely, a lower pressure may be acceptable when mentation and perfusion are improving and the clinical context supports it.

13. Fluid resuscitation and stewardship

- Confirm the indication: suspected preload-responsive hypovolaemia or distributive shock. Do not give fluid solely because the blood pressure is low.
- Use a defined bolus or reversible preload challenge with a documented endpoint. In adults, a small aliquot is often appropriate when the diagnosis is uncertain or fluid intolerance is possible. Paediatric volumes must follow an approved age- and condition-specific pathway.
- Balanced crystalloids are generally preferred for sepsis and many non-haemorrhagic states. Use 0.9% saline when specifically indicated, including selected neurocritical situations, and follow local policy.
- In sepsis or septic shock, initial crystalloid up to approximately 30 mL/kg may be considered with adjustment for clinical context and frequent reassessment; it is not a mandate to give an unreviewed fixed volume to every patient.
- After the initial phase, use dynamic assessment and individualized fluid strategy. Stop fluid when there is no meaningful perfusion response, when fluid intolerance appears, or when a different shock mechanism is more likely.
- Avoid routine fluid loading as primary treatment in left-sided cardiogenic shock. Use caution in right-ventricular failure, pulmonary embolism, tamponade, renal failure, cirrhosis, pregnancy, frailty, and severe anaemia.
- Record all resuscitation fluid, blood products, oral or prehospital intake, and output. Move to de-escalation and active removal after shock resolves when fluid overload is clinically important and the patient is stable.

14. Haemorrhagic shock and major bleeding

- Activate the local major haemorrhage protocol early when bleeding is life-threatening, ongoing, concealed, or associated with shock.
- Control bleeding immediately. Definitive surgery, endoscopy, interventional radiology, obstetric intervention, or transfer takes priority over normalization of laboratory values.
- Use warmed blood components and a locally approved balanced component strategy or whole-blood pathway where available. Transition to laboratory- or viscoelastic-guided therapy as soon as feasible.
- Avoid excessive crystalloid and prevent the lethal triad / diamond of hypothermia, acidosis, coagulopathy, and hypocalcaemia.
- Use permissive hypotension or a restrictive fluid strategy in selected bleeding trauma patients until haemorrhage control, but not when traumatic brain injury, pregnancy, spinal cord perfusion concerns, or another contraindication requires a higher pressure target.
- Administer tranexamic acid and anticoagulant reversal when indicated under local protocols and within evidence-based time windows. Do not delay haemorrhage control.
- Monitor ionized calcium, temperature, fibrinogen / coagulation, haemoglobin trend, and clinical bleeding. A normal early haemoglobin does not exclude major acute blood loss.

15. Vasoactive and inotropic support

- Start vasopressor support when hypotension with hypoperfusion persists after an appropriate initial fluid assessment, or earlier when further fluid is unsafe or unlikely to help.
- Norepinephrine is the preferred first-line vasopressor for adult septic shock. The preferred agent in cardiogenic, neurogenic, obstetric, paediatric, and toxicological shock depends on phenotype and approved specialty guidance.
- Use a standardized concentration, infusion pump, dedicated labelled line, and continuous ECG and blood-pressure monitoring. Record the indication, start time, concentration, route, dose, titration target, site checks, and response.
- A suitable proximal peripheral IV may be used for urgent initiation under an approved peripheral-vasopressor protocol. Inspect the site frequently and have an extravasation response plan. Arrange central access when ongoing or escalating support is expected.

- Escalating vasopressor requirement is a danger sign requiring urgent senior and critical-care review, reassessment of shock mechanism, occult bleeding, cardiac function, source control, acidosis, and drug delivery.
- Use an inotrope only when cardiac dysfunction with inadequate output and persistent hypoperfusion is suspected despite adequate arterial pressure and volume assessment. Inotropes do not replace vasopressors when MAP is inadequate.

16. Cause-directed emergency treatment

16.1 Septic and other distributive shock

- Recognize sepsis as infection with organ dysfunction; shock may be present with warm skin and a normal pressure early.
- Obtain cultures promptly when this does not materially delay antimicrobial therapy. Administer empiric antimicrobials according to severity, likely source, local resistance, allergy, renal function, and stewardship policy.
- Identify anatomical sources requiring urgent drainage, debridement, device removal, surgery, or transfer.
- Use crystalloids, dynamic fluid assessment, serial lactate where elevated, capillary refill, and norepinephrine-led vasopressor support in accordance with the approved sepsis pathway.
- Consider corticosteroid support in ongoing vasopressor-dependent septic shock under the local critical-care protocol.

16.2 Hypovolaemic and haemorrhagic shock

- Replace the material lost: blood for major bleeding; appropriate crystalloid and electrolyte replacement for dehydration or non-haemorrhagic losses.
- Search for concealed thoracic, abdominal, pelvic, retroperitoneal, gastrointestinal, obstetric, and aortic bleeding.
- Do not interpret transient blood-pressure improvement as haemorrhage control. Continue direct observation, serial examination, and definitive planning.

16.3 Cardiogenic shock

- Obtain ECG and urgent cardiac imaging; identify acute coronary syndrome, mechanical complication, myocarditis, acute valve disease, severe ventricular failure, or arrhythmia.
- Activate cardiology, anaesthesia / critical care, and interfacility transfer early. Revascularization or correction of the cause must not be delayed by repeated fluid challenges.
- Avoid routine nitrates, beta-blockers, or diuretics when profound hypotension or hypoperfusion makes them unsafe. Choose vasoactive and inotropic support according to blood pressure, rhythm, ventricular phenotype, and senior specialist guidance.
- Consider mechanical circulatory support only through an approved pathway and in consultation with a capable receiving centre.

16.4 Obstructive shock

- Tension pneumothorax: perform immediate decompression based on clinical diagnosis when unstable; do not await imaging.
- Cardiac tamponade: urgent drainage or transfer for definitive intervention; cautious fluid may be used only as a temporary bridge when appropriate.
- Massive / high-risk pulmonary embolism: activate the pulmonary embolism and reperfusion pathway, assess contraindications and available thrombolysis / embolectomy options, and transfer urgently when required.
- Dynamic hyperinflation: disconnect briefly and allow exhalation when clinically appropriate, reduce ventilation-related air trapping, and treat the underlying bronchospasm under an advanced airway protocol.

16.5 Anaphylactic, neurogenic, endocrine, and toxicological shock

- Anaphylaxis: give intramuscular adrenaline immediately and repeat according to the approved anaphylaxis algorithm; support airway, breathing, circulation, and arrange observation based on severity.
- Neurogenic shock: protect the spine, exclude haemorrhage, support perfusion, treat clinically important bradycardia, and obtain urgent specialist guidance.
- Adrenal crisis or severe endocrine disturbance: obtain appropriate specimens when feasible but do not delay corticosteroid, glucose, electrolyte, and supportive treatment.
- Poisoning: contact toxicology / poison services early, use cause-specific antidotes and decontamination where appropriate, and anticipate delayed cardiovascular collapse.

17. Paediatric, pregnancy, frailty, and other special considerations

- Children may maintain blood pressure until late. Use age-specific heart rate, blood pressure, capillary refill, mental status, pulse quality, urine output, and work of breathing. Weight-based therapy must use a measured or approved estimated weight.
- In paediatric septic shock, give smaller aliquots with frequent reassessment and stop for signs of overload; the total early volume and timing of vasoactive support depend on local critical-care capability and the approved paediatric sepsis pathway.
- In pregnancy, account for aortocaval compression, increased circulating volume, fetal implications, ectopic pregnancy, placental abruption, uterine rupture, and postpartum haemorrhage. Use left uterine displacement when appropriate and call obstetrics / anaesthesia early.
- Older or frail adults may have atypical vital signs, limited fluid tolerance, polypharmacy, occult bleeding, infection without fever, or treatment limitations. Frailty must prompt individualized care, not therapeutic neglect.
- In severe anaemia, sickle-cell disease, renal failure, cirrhosis, pulmonary hypertension, or chronic heart failure, define individualized perfusion and transfusion goals with senior input.
- Confirm advance directives and treatment limitations without delaying immediately necessary care when wishes are unknown. Document goals-of-care discussions and responsible decision-makers.

18. Escalation, disposition, and transfer

18.1 Immediate senior / critical-care escalation

- Persistent shock after the first resuscitation cycle; recurrent hypotension; rising lactate or worsening acidosis; escalating oxygen, ventilation, vasopressor, or blood requirement; suspected cardiogenic or obstructive shock; major haemorrhage; severe paediatric or obstetric shock; or diagnostic uncertainty.
- Need for invasive airway, arterial monitoring, central access, vasoactive infusion beyond ED capability, emergency procedure, theatre, catheterization, dialysis, extracorporeal support, or transfer.

18.2 Disposition standards

- Patients with ongoing or recently resolved shock generally require critical-care or high-dependency monitoring. Ward transfer is appropriate only after explicit senior review, sustained stability, a clear cause and treatment plan, and an environment capable of required monitoring.
- Discharge directly from the ED is inappropriate after true shock except in rare, clearly documented circumstances after complete resolution of a benign reversible cause, an adequate observation period, senior review, and safe follow-up.
- For interfacility or overseas transfer, stabilize as far as possible, obtain explicit acceptance, choose a transport platform and escort able to continue blood, ventilation, monitoring, and vasoactive support, and document contingency plans.

18.3 Stop-transfer criteria

- New or worsening airway, breathing, or circulatory instability not manageable during transport.
- Unsecured vascular access, inadequate oxygen / blood / medication / battery supply, unavailable trained escort, or absent receiving acceptance.
- Need for an immediate life-saving intervention that should occur before departure, unless departure itself is the only route to that intervention and the transfer risk has been explicitly accepted by senior clinicians.

19. Documentation requirements

- Time shock was recognized and pathway activated; initial and serial observations; perfusion findings; working mechanism and differential diagnosis.
- All fluids, blood products, medicines, procedures, source-control actions, and their start times.
- For every bolus or vasoactive change: indication, amount / dose, endpoint, reassessment time, response, adverse effect, and next decision.
- Results reviewed, critical results communicated, pending-result ownership, consultation and acceptance details, transfer decisions, and responsibility handover.
- Treatment limitations, consent or emergency authority, patient / family communication, and reason for any deviation from this protocol.

20. Quality indicators and audit

Indicator	Suggested measure
Recognition	Percentage of patients meeting local shock criteria assigned Red and moved immediately to a resuscitation area.
Initial assessment	Time from arrival / recognition to documented ABCDE, full vital signs, glucose, vascular access, and senior notification.
Reassessment	Percentage of fluid boluses, blood-product phases, and vasoactive changes followed by documented perfusion reassessment.
Cause control	Time to major haemorrhage activation, antibiotic administration when indicated, definitive haemorrhage / source control, reperfusion, or obstruction relief.
Medication safety	Compliance with standardized vasopressor concentration, pump, line labelling, site monitoring, and extravasation plan.
Transfer	Time from decision to acceptance and departure; percentage with complete transfer checklist and documented arrival handover.
Outcome / balancing	Unexpected cardiac arrest, unplanned ICU transfer, fluid overload, extravasation injury, delayed haemorrhage control, delayed antibiotics, transfer incidents, and mortality review.

21. Training, equipment, and implementation

- All ED clinicians and nurses shall receive competency-based training in shock recognition, ABCDE, haemorrhage control, fluid challenge and reassessment, peripheral vasopressor safety, sepsis, anaphylaxis, paediatric shock, obstetric haemorrhage, and transfer.
- Resuscitation areas shall have immediate access to monitoring, pumps, pressure bags, blood warmers where available, rapid infusers where governed, IO equipment, haemorrhage-control devices, ultrasound, emergency medicines, standardized vasopressor preparations, major haemorrhage packs, and transfer equipment.

- Implementation shall include simulation of septic shock, occult haemorrhage, cardiogenic shock, tension pneumothorax, anaphylaxis, paediatric shock, postpartum haemorrhage, and interfacility transfer.
- Local leadership shall approve exact haemodynamic thresholds, fluid aliquots, blood-product strategy, vasopressor concentrations, peripheral-line standards, sepsis timing, paediatric pathways, major haemorrhage activation, and referral contacts before launch.

22. References and evidence base

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7. Sinha SS, Morrow DA, Kapur NK, et al. 2025 ACC Expert Consensus Statement on the Evaluation and Management of Cardiogenic Shock. Journal of the American College of Cardiology. 2025. doi:10.1016/j.jacc.2025.02.018.

8. Relevant locally approved adult, paediatric, neonatal, obstetric, trauma, transfusion, sepsis, anaphylaxis, toxicology, cardiac, procedural, and transfer standards.

ANNEX A. One-page shock and hypotension workflow

RECOGNIZE → RESUSCITATE → CLASSIFY → CORRECT THE CAUSE → REASSESS → ESCALATE / TRANSFER

1. Identify shock or tissue hypoperfusion; assign Red; move to resuscitation.

2. ABCDE, monitoring, glucose, temperature, two IV / IO routes, urgent bloods, and immediate bleeding control.

3. Call senior help and activate the appropriate pathway: major haemorrhage, sepsis, ACS / cardiogenic shock, obstructive shock, anaphylaxis, obstetric, paediatric, toxicology, or transfer.

4. Classify the probable mechanism: hypovolaemic / haemorrhagic, distributive, cardiogenic, obstructive, or mixed.

5. Give one cause-appropriate intervention with a defined endpoint: blood and haemorrhage control; crystalloid challenge; vasopressor; intramuscular adrenaline; decompression; rhythm treatment; source control.

6. Reassess perfusion, pressure, respiratory status, fluid tolerance, lactate trend, and cause control.

7. Continue, change, or stop treatment. Escalate early if shock persists or resources are insufficient.

8. Document responsibility, pending actions, transfer acceptance, transport plan, and handover.

ANNEX B. Shock danger-sign card

Immediate danger sign	Required response
Altered mental status, collapse, or peri-arrest physiology	Resuscitation activation; ABCDE; senior and anaesthesia / critical-care help.
Major active or suspected concealed haemorrhage	Direct haemorrhage control; major haemorrhage activation; surgery / obstetrics; blood bank.
Persistent hypotension or worsening perfusion after initial intervention	Reclassify mechanism; start / escalate vasoactive support as appropriate; critical-care review.
Chest pain, new ECG change, pulmonary oedema, new murmur, or severe ventricular dysfunction	Cardiogenic-shock pathway; urgent cardiology / reperfusion / transfer.
Sudden dyspnoea with raised JVP, unilateral absent breath sounds, or tamponade / PE features	Treat or urgently exclude obstructive shock; do not delay decompression for imaging when tension pneumothorax is clinically diagnosed.
Rash, airway swelling, wheeze, or collapse after exposure	Immediate intramuscular adrenaline and anaphylaxis pathway.
Pregnancy or postpartum state with pain, bleeding, or shock	Obstetric emergency activation; haemorrhage protocol; left uterine displacement where appropriate.
Child with weak pulse, delayed refill, altered behaviour, or hypotension	Paediatric resuscitation; age-specific parameters; early senior / transfer escalation.

ANNEX C. First-5-minute checklist

☐ Resuscitation response activated and team leader identified

☐ ABCDE completed; airway and ventilation risk assessed

☐ Major external bleeding controlled

☐ ECG, SpO2, cycling BP, temperature, and glucose obtained

☐ Two IV lines or IO access established

☐ Blood gas / lactate, FBC, chemistry, coagulation, group and screen / crossmatch, and cause-specific tests sent

☐ Pregnancy status considered where relevant

☐ Working shock mechanism verbalized

☐ Major haemorrhage / sepsis / ACS / anaphylaxis / obstetric / paediatric / transfer pathway activated as indicated

☐ First cause-appropriate intervention given

☐ Reassessment time and perfusion endpoints stated

ANNEX D. Shock classification matrix

Finding	Hypovolaemic / haemorrhagic	Distributive	Cardiogenic	Obstructive
Skin / pulse	Cool, weak; narrow pulse pressure	Often warm, bounding early; may cool later	Cool, clammy; weak pulse	Cool; pulse may be weak; sudden collapse
Neck veins	Usually low / flat	Variable	Often raised	Often raised except some PE states
Lungs	Usually clear unless aspiration / injury	Variable; infection or ARDS possible	Crackles / pulmonary oedema common	Unilateral absent sounds in tension PTX; may be clear in PE / tamponade
POCUS clues	Low filling, free fluid, aortic / obstetric source	Hyperdynamic or septic cardiomyopathy; source clues	LV / RV dysfunction, mechanical complication	Tamponade, RV strain, absent lung sliding
Fluid approach	Blood for bleeding; crystalloid for non-haemorrhagic loss	Defined crystalloid with reassessment	Avoid routine loading; small test only if plausible need	Cautious bridge in tamponade or selected RV preload dependence; definitive relief
Definitive priority	Stop loss / haemorrhage control	Antimicrobials and source control; vasopressor	Reperfusion / rhythm / pump support	Decompression, drainage, reperfusion / embolectomy

ANNEX E. Fluid challenge / preload test record

Field	Record
Date / time	
Indication and suspected mechanism	
Fluid, volume, route, and planned duration	
Baseline HR / rhythm, BP / MAP, SpO2, capillary refill, mental status, respiratory findings	
Dynamic assessment used (e.g., passive leg raise, stroke-volume change, pulse-pressure response)	
Defined endpoint / expected response	
Stop rules (e.g., no response, pulmonary congestion, rising JVP, worsening oxygenation)	
Post-intervention findings and response	
Decision: repeat / stop / vasopressor / blood / procedure / escalate	
Clinician name and signature	

ANNEX F. Perfusion reassessment record

Time	HR / rhythm	BP / MAP	Mental status	Capillary refill / skin	Respiratory / fluid tolerance	Urine / lactate	Treatment and next decision

ANNEX G. Major haemorrhage activation checklist

- ☐ Life-threatening bleeding or shock recognized; major haemorrhage pathway activated
- ☐ Bleeding source and definitive-control team identified
- ☐ Blood bank notified; group / screen / crossmatch sent; emergency-release process understood
- ☐ Blood warmer / rapid delivery system prepared within local governance
- ☐ Temperature, calcium, coagulation / fibrinogen, haemoglobin trend, and acid-base status monitored
- ☐ Anticoagulant / antiplatelet use and reversal plan documented
- ☐ Tranexamic acid considered under approved pathway and time window
- ☐ Crystalloid minimized; warmed components used; component / whole-blood strategy follows local protocol
- ☐ Surgery / obstetrics / endoscopy / interventional radiology / transfer activated
- ☐ Every product documented and transfusion reaction surveillance maintained

ANNEX H. Vasopressor initiation and safety checklist

- ☐ Indication and target MAP / perfusion endpoint documented
- ☐ Shock mechanism and fluid status assessed; further fluid judged ineffective or unsafe
- ☐ Standardized drug concentration and smart-pump / infusion-pump settings used
- ☐ Dedicated, labelled line; compatibility confirmed
- ☐ Peripheral route: suitable proximal vein, good blood return, frequent site checks, and extravasation plan
- ☐ Continuous ECG, frequent BP / arterial pressure, SpO2, and perfusion monitoring
- ☐ Dose, start time, titration, response, and adverse effects documented
- ☐ Senior / critical-care review and plan for definitive access
- ☐ Escalating dose triggers reassessment for occult cause, cardiac dysfunction, acidosis, delivery problem, and source control

ANNEX I. Septic shock immediate-action card

1. Recognize infection with organ dysfunction or circulatory failure; activate sepsis pathway.

2. Obtain cultures when feasible without materially delaying treatment; send lactate and repeat if elevated / clinically indicated.

3. Give empiric antimicrobials according to severity, source, allergy, renal function, local resistance, and stewardship policy.

4. Use balanced crystalloid as first-line resuscitation fluid, with frequent individualized reassessment.

5. Start norepinephrine when hypotension persists or further fluid is unsafe; target perfusion and an initial adult MAP near 65 mmHg unless individualized.

6. Identify and obtain urgent source control.

7. Reassess capillary refill, mentation, urine, lactate trend, respiratory status, and fluid tolerance.

8. Escalate persistent or refractory shock to critical care and transfer services.

ANNEX J. Cardiogenic shock checklist

- ☐ ECG obtained; ACS / STEMI and arrhythmia pathways considered
- ☐ Bedside echocardiography / ultrasound obtained when available
- ☐ Pulmonary oedema, RV failure, mechanical complication, valve emergency, myocarditis, or tamponade assessed
- ☐ Routine fluid loading avoided; any test bolus has explicit indication and immediate reassessment
- ☐ Cardiology, anaesthesia / critical care, and transfer centre contacted early
- ☐ Reperfusion / definitive correction plan documented
- ☐ Vasoactive / inotropic choice individualized to pressure, rhythm, and ventricular phenotype
- ☐ Mechanical circulatory support discussed only through approved pathway
- ☐ Transport plan can safely continue monitoring, ventilation, and infusions

ANNEX K. Obstructive shock checklist

Suspected cause	Immediate action
Tension pneumothorax	If unstable and clinically diagnosed, immediate decompression followed by definitive pleural management; do not wait for imaging.
Cardiac tamponade	Urgent echo if this does not delay treatment; call procedural / surgical expertise; arrange drainage or immediate transfer.
High-risk pulmonary embolism	Activate PE pathway; support RV perfusion; urgent reperfusion / embolectomy decision and transfer.
Dynamic hyperinflation	Recognize after ventilation / severe obstructive disease; allow exhalation, correct ventilator strategy, and treat bronchospasm.

ANNEX L. Paediatric shock safety prompts

- ☐ Use age-specific reference ranges; hypotension is a late sign
- ☐ Obtain actual or approved estimated weight in kilograms
- ☐ Reassess after every fluid aliquot for pulse, refill, mentation, liver size, crackles, and oxygen need
- ☐ Stop boluses for signs of overload or no perfusion response
- ☐ Use age- and weight-specific vasoactive dosing and standardized concentrations
- ☐ Consider sepsis, dehydration, haemorrhage, anaphylaxis, cardiogenic causes, duct-dependent congenital disease, and toxins
- ☐ Involve paediatrics / critical care and transfer service early

ANNEX M. Obstetric shock safety prompts

- ☐ Pregnancy and postpartum status established
- ☐ Ectopic pregnancy, abruption, uterine rupture, placenta-related bleeding, and postpartum haemorrhage considered
- ☐ Obstetrics, anaesthesia, blood bank, theatre, and neonatal team activated as indicated
- ☐ Left uterine displacement used when clinically appropriate
- ☐ Haemorrhage control and blood support prioritized; maternal stabilization takes priority
- ☐ Fetal assessment performed when feasible without delaying maternal life-saving care

☐ Rh status, coagulation, fibrinogen, calcium, temperature, and massive transfusion needs addressed under local protocol

ANNEX N. Shock pathway audit tool

Audit item	Yes	No	N/A / comments
Red triage and immediate resuscitation-area placement			
ABCDE and complete baseline observations documented			
Shock mechanism / differential documented			
Appropriate access, specimens, ECG, and glucose completed			
Cause-specific pathway activated without delay			
Every fluid / blood / vasoactive intervention followed by reassessment			
Definitive haemorrhage / infection / cardiac / obstructive source plan documented			
Senior / critical-care escalation appropriate and timely			
Transfer acceptance, escort, equipment, and handover complete			
Outcome and complications reviewed			

ANNEX O. Local configuration table

Item requiring local approval	Approved local standard / contact
Adult and paediatric activation thresholds	
Initial perfusion / MAP targets by clinical group	
Adult crystalloid aliquot and reassessment standard	
Paediatric fluid aliquot, maximum early volume, and vasoactive trigger	
Approved crystalloid solutions and exceptions	
Major haemorrhage activation criteria and blood-component / whole-blood strategy	
Tranexamic acid and anticoagulant-reversal protocols	
Standard vasopressor / inotrope concentrations and infusion charts	

Item requiring local approval	Approved local standard / contact
Peripheral vasopressor site, duration, observation, and extravasation standard	
Sepsis antimicrobial timing and local empiric formulary	
POCUS credentialing and image-governance standard	
Cardiology, surgery, obstetrics, anaesthesia, critical care, blood bank, and transfer contacts	
Critical-care admission and interfacility / overseas transfer triggers	
Audit frequency and responsible committee	

ANNEX P. Approval and sign-off

Role	Name	Signature	Date
Clinical lead, Emergency Department			
Director of Medical Services			
Director of Nursing Services			
Anaesthesia / Critical Care lead			
Surgery / Trauma lead			
Obstetrics and Gynaecology lead			
Paediatrics lead			
Cardiology / Internal Medicine lead			
Blood Bank / Laboratory lead			
Pharmacy lead			
Clinical Governance / Quality Committee			

Implementation note: Before approval, the multidisciplinary team should conduct tabletop and simulation testing using septic shock, occult haemorrhage, cardiogenic shock, tension pneumothorax, paediatric shock, postpartum haemorrhage, and transfer scenarios. All locally configured thresholds, contact details, medicine concentrations, and equipment lists must be validated at least annually and after any service change.