A STANDALONE CLINICAL COMPANION MANUAL

Advanced Life Support Manual

Eight core resuscitation and life-support courses in one bedside reference



BLS/ACLS CARDIOVASCULAR	**PALS** PEDIATRIC	**ATLS** TRAUMA	**ADLS** DISASTER
NRP/NALS NEONATAL	**AHLS** HAZMAT	**ENLS** NEUROLOGICAL	**PHTLS** PREHOSPITAL TRAUMA

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Advanced Life Support Manual

BLS/ACLS · PALS · ATLS · ADLS · NRP/NALS · AHLS · ENLS · PHTLS — eight core resuscitation and life-support courses in one bedside reference

This manual distills eight internationally recognized life-support courses — Basic and Advanced Cardiovascular Life Support, Pediatric Advanced Life Support, Advanced Trauma Life Support, Advanced Disaster Life Support, the Neonatal Resuscitation Program, Advanced Hazmat Life Support, Emergency Neurological Life Support, and Prehospital Trauma Life Support — into a single, consistently formatted companion volume. Each chapter follows the same structure: a clinical snapshot and learning objectives, the core algorithm or sequence for that discipline, weight- or age-based dosing tables, callout boxes for decision points, red flags and practice pearls, a worked clinical case, and a chapter-end quick-reference summary table.

This manual is an educational reference synthesized from current published guidelines and course curricula as of 2026. It is not a substitute for enrollment in, or certification through, the official provider courses it summarizes, and it does not replace institutional protocols, hands-on skills verification, or the full current-version source guidelines, which should always be consulted directly for clinical decision-making.

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CHAPTER 1

BLS/ACLS

Basic and Advanced Cardiovascular Life Support

1 Clinical Snapshot

Cardiac arrest remains one of the leading causes of death in industrialized nations, with roughly 350,000 out-of-hospital cardiac arrests (OHCA) and over 290,000 in-hospital cardiac arrests (IHCA) occurring annually in the United States alone. Survival to hospital discharge after OHCA remains under 10 percent in most systems, while IHCA survival is somewhat higher at 15-25 percent, reflecting the benefit of immediate monitoring and rapid response. Outcomes are directly tied to how quickly a bystander recognizes the arrest, begins high-quality chest compressions, and applies a defibrillator — every minute without CPR and defibrillation after a witnessed shockable arrest reduces survival by roughly 7-10 percent. Basic Life Support (BLS) and Advanced Cardiovascular Life Support (ACLS) form the backbone of resuscitation training for lay rescuers and healthcare professionals respectively, and the 2025 American Heart Association (AHA) Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care (CPR and ECC) represent the most significant restructuring of this framework since 2010, unifying what were previously separate adult, pediatric, in-hospital, and out-of-hospital pathways into a single coherent model.

Why this matters · Typical patient · Where you meet it

Why it matters: Cardiac arrest is unforgiving of delay — brain injury begins within minutes of no-flow, and every clinician, from first responder to intensivist, must execute a well-rehearsed, algorithm-driven sequence without hesitation.

Typical patient: Adults with sudden collapse and no pulse, ranging from a witnessed ventricular fibrillation (VF) arrest in a previously healthy middle-aged patient with coronary disease, to an unwitnessed asystolic arrest in an elderly patient with multiple comorbidities.

Common settings: Public spaces and homes (OHCA, lay-rescuer initiated), general wards, monitored telemetry units, emergency departments, intensive care units, and pre-hospital EMS encounters (IHCA and advanced-provider settings).

2 Learning Objectives

By the end of this chapter the reader should be able to:

- Describe the unified 2025 AHA Chain of Survival and explain why the previous separate adult/pediatric and IHCA/OHCA chains were consolidated.
- Perform and teach the technical parameters of high-quality CPR, including current compression rate, depth, recoil, and ventilation targets.
- Execute the BLS sequence correctly for adult, child, and infant victims, including AED application and rhythm interpretation.
- Manage adult foreign-body airway obstruction using the current cyclic sequence.
- Run the ACLS adult cardiac arrest algorithm for both shockable (VF/pulseless VT) and non-shockable (asystole/PEA) rhythms, including correct drug timing.

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- State current doses, routes, and timing for the core resuscitation drugs and current defibrillation/cardioversion energies for each rhythm.
- Recall and systematically search for the H's and T's of reversible cardiac arrest causes.
- Apply the bradycardia and tachycardia algorithms, including when to cardiovert versus defibrillate.
- Implement structured post-cardiac-arrest care, including temperature control and hemodynamic targets.
- Recognize special circumstances — pregnancy, opioid-associated arrest, and hypothermic arrest — that modify standard algorithms.

3 The Unified Chain of Survival (2025)

For over two decades the AHA maintained parallel Chains of Survival — separate versions for adult and pediatric victims, and separate versions for in-hospital and out-of-hospital arrest. The 2025 Guidelines for CPR and ECC replace these with a single unified chain that applies across all ages and all settings, reflecting evidence that the underlying sequence of actions — prevention, recognition, high-quality CPR, defibrillation, advanced care, and recovery — is conceptually identical regardless of where or in whom the arrest occurs, even though the specific techniques (compression depth, drug doses) still vary by age and setting within that shared framework.

- Link 1 — Recognition and activation of the emergency response system: rapid identification of unresponsiveness and abnormal or absent breathing, immediate activation of EMS or the hospital rapid-response/code team.
- Link 2 — High-quality CPR: immediate, minimally interrupted chest compressions at the correct rate and depth, begun by the first available rescuer, lay or professional.
- Link 3 — Defibrillation: earliest possible rhythm analysis and shock delivery for a shockable rhythm, using a public-access AED or a manual defibrillator.
- Link 4 — Advanced resuscitation: ACLS-level interventions — advanced airway management, vascular access, vasopressor and antiarrhythmic administration, and treatment of reversible causes.
- Link 5 — Post-arrest care: structured critical care after return of spontaneous circulation (ROSC), including temperature control, hemodynamic optimization, and coronary reperfusion when indicated.
- Link 6 — Recovery: rehabilitation, neuroprognostication, psychological support for patients and families, and survivorship follow-up, now formally recognized as part of the chain rather than an afterthought.

The unification also carries a practical training implication: a single core algorithm is now taught with age- and setting-specific modifications layered on top, rather than as entirely separate curricula, which the AHA anticipates will improve retention and cross-training between BLS and ACLS providers.

4 High-Quality CPR: Technical Parameters

The physiologic benefit of CPR is entirely dependent on technique. Compressions that are too shallow, too slow, too fast, or too frequently interrupted fail to generate adequate coronary and cerebral perfusion pressure. The 2025 update reaffirms the core 2020 compression targets while adding new emphasis on rescuer positioning, real-time feedback, and minimizing pre- and post-shock pauses.

Key Numbers — High-Quality CPR

Compression rate: 100-120 per minute, all ages.

Compression depth, adult: at least 2 inches (5 cm), not exceeding 2.4 inches (6 cm).

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Compression depth, child: approximately 2 inches (5 cm), at least one-third the anteroposterior chest diameter.

Compression depth, infant: approximately 1.5 inches (4 cm), at least one-third the anteroposterior chest diameter.

Allow full chest recoil between compressions; avoid leaning on the chest.

Minimize interruptions: keep any pause under 10 seconds; maximize chest-compression fraction.

Ventilation with an advanced airway in place (adult): 1 breath every 6 seconds (10/min), asynchronous with compressions.

Avoid excessive ventilation — hyperventilation raises intrathoracic pressure and reduces venous return and coronary perfusion.

- Rescuer positioning (2025 update): position the patient's torso at approximately the level of the rescuer's knees on a firm surface — this reduces rescuer fatigue and helps maintain consistent depth over prolonged resuscitation.
- Hand placement, adult: heel of one hand on the lower half of the sternum, second hand on top, fingers interlocked and lifted off the ribs.
- Infant compression technique: the two-thumb, encircling-hands technique is preferred whenever two rescuers are present (or a single rescuer whose hands can encircle the infant's chest), as it generates higher and more consistent pressures than the two-finger technique — the two-finger technique is no longer recommended. When encircling is not possible, the heel of one hand is an acceptable alternative.
- CPR feedback devices are now recommended both for live coaching during actual resuscitation and for use during CPR training, for both healthcare professionals and lay rescuers, given consistent evidence that feedback improves rate and depth compliance.
- Rescuer switching: rotate the compressor every 2 minutes (or sooner if fatigued) to prevent decay in compression quality, timing the switch to coincide with a rhythm/pulse check to avoid an additional pause.
- Mechanical (automated) chest-compression devices are not recommended for routine use but may be considered in specific situations where high-quality manual compressions are difficult or dangerous to sustain (for example, during transport or in a hazardous environment), provided deployment does not create additional interruption.

5 BLS Sequence: Adult, Child, and Infant

The core BLS sequence — check responsiveness, activate the emergency response system, check for breathing and a pulse (≤10 seconds), begin compressions, attach the AED as soon as it is available — is shared across all ages, but specific technical parameters differ by body size and the more common underlying cause of arrest (primary cardiac in adults; frequently hypoxic/respiratory in infants and children). A child is defined as age 1 year to the onset of puberty; an infant is under 1 year, excluding the newly born.

Comparison table

Table 1.1 BLS technique comparison — adult, child, and infant

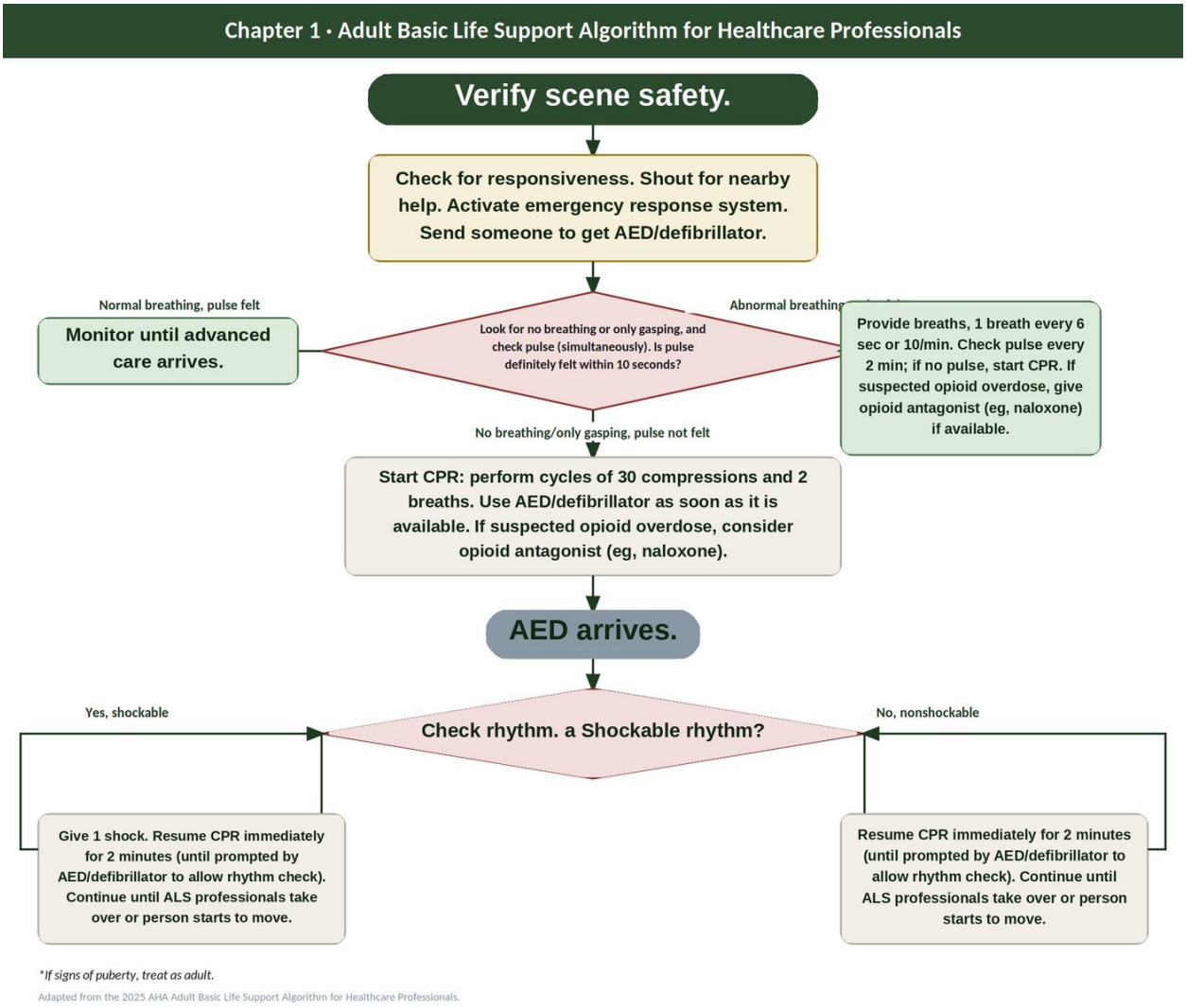
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Parameter	Adult	Child (1 yr–puberty)	Infant (<1 yr, excludes newborn)
Pulse check site	Carotid or femoral, ≤10 sec	Carotid or femoral, ≤10 sec	Brachial or femoral, ≤10 sec
Compression location	Lower half of sternum, two hands	Lower half of sternum, one or two hands	Just below the nipple line, center of chest
Compression technique (2 rescuers)	Two hands stacked	One or two hands	Two-thumb encircling-hands
Compression technique (1 rescuer)	Two hands stacked	One or two hands	Heel of one hand (encircling if hands allow)
Compression depth	≥2 in (5 cm), not >2.4 in (6 cm)	≈2 in (5 cm); ≥1/3 AP diameter	≈1.5 in (4 cm); ≥1/3 AP diameter
Compression rate	100-120/min	100-120/min	100-120/min
Ratio, single rescuer	30:2	30:2	30:2
Ratio, two rescuers (no advanced airway)	30:2	15:2	15:2
Ventilation rate with advanced airway	1 breath/6 sec (10/min)	1 breath every 2-3 sec (20-30/min)	1 breath every 2-3 sec (20-30/min)
AED pads	Adult pads	Pediatric attenuated pads/dose if available, otherwise adult	Pediatric attenuated pads/dose preferred; use adult pads if unavailable rather than withholding a shock

Rates and ratios per the 2025 AHA Guidelines for CPR and ECC; compression depth ceiling applies specifically to adults.

Figure 1.0. Adapted from the 2025 AHA Adult Basic Life Support Algorithm for Healthcare Professionals.

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6 AED Use and Rhythm Recognition

An automated external defibrillator (AED) should be applied and turned on as soon as it arrives at the patient's side, without interrupting compressions any longer than necessary. The device analyzes the rhythm automatically and advises 'shock' or 'no shock' — rescuers do not need to interpret the rhythm themselves to use an AED safely, which is precisely why public-access defibrillation programs have been effective with minimally trained lay rescuers. Manual defibrillators used by trained providers require the operator to recognize shockable versus non-shockable rhythms directly from the monitor.

Table 1.2 Cardiac arrest rhythms and shock advisory

Rhythm	ECG appearance	Shockable?	Immediate priority
Ventricular fibrillation (VF)	Chaotic, irregular, no discernible QRS complexes	Yes	Defibrillate immediately, then resume CPR
Pulseless ventricular tachycardia (pVT)	Wide, regular, rapid complexes, no palpable pulse	Yes	Defibrillate immediately, then resume CPR
Asystole	Flat line — confirm in a	No	CPR, epinephrine ASAP,

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Rhythm	ECG appearance	Shockable?	Immediate priority
	second lead and check gain/lead connections before declaring		search for reversible cause
Pulseless electrical activity (PEA)	Organized electrical rhythm present but no palpable pulse	No	CPR, epinephrine ASAP, aggressively search for reversible cause

Practice Pearls

A rhythm check should take no more than a brief pause — resume compressions the instant the rhythm/pulse determination is made; do not stop compressions to 'watch' the monitor.

Confirm asystole is real, not artifact: check lead connections, increase gain, and confirm in a second lead before concluding no shock is indicated.

PEA is not one rhythm but a category — the QRS morphology and rate on the monitor can offer clues to the underlying reversible cause (a narrow, fast PEA suggests a mechanical/obstructive cause such as tamponade or tension pneumothorax; a wide, slow PEA suggests a metabolic or toxic cause).

An AED will not shock a non-shockable rhythm even if activated — it is safe to apply and analyze in any pulseless, unresponsive patient.

7 Adult Foreign-Body Airway Obstruction (Choking)

Management of adult choking is now framed explicitly as a cyclic sequence rather than a fixed number of maneuvers, continued until the object is expelled or the victim becomes unresponsive. Mild obstruction, where the victim can still cough forcefully or speak, should not be interrupted with back blows or thrusts — forceful coughing is the most effective way to clear a partial obstruction and rescuer maneuvers should be reserved for severe obstruction.

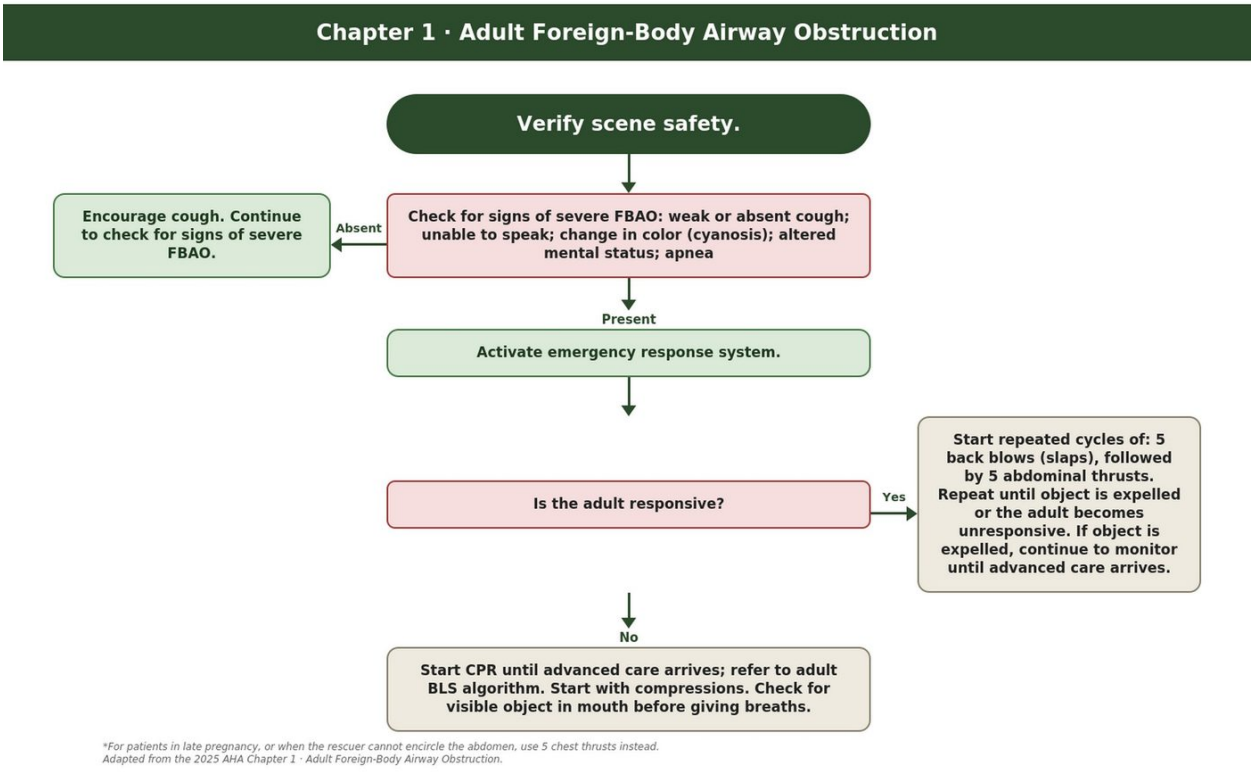
Table 1.3 Adult foreign-body airway obstruction management

Presentation	Recognition	Management
Mild obstruction	Forceful cough, can speak or make sounds, some air movement	Encourage continued coughing; stay with the victim and monitor closely; do not intervene unless it progresses
Severe obstruction, conscious	Universal choking sign (hands at throat), cannot speak, cough, or breathe, or a weak/silent cough	Cyclic sequence of 5 back blows followed by 5 abdominal thrusts, repeated until the object is expelled or the victim becomes unresponsive
Pregnant or markedly obese victim	Abdominal thrusts not feasible or ineffective	Substitute chest thrusts for abdominal thrusts within the same cyclic sequence
Victim becomes unresponsive	Collapses during obstruction	Lower to the floor, activate EMS, begin CPR starting with

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Presentation	Recognition	Management
		compressions; open the mouth before each set of ventilations and remove a visible object — do not perform a blind finger sweep
Infant	Universal choking sign or ineffective cough with poor air exchange	Cyclic sequence of 5 back blows alternating with 5 chest thrusts (no abdominal thrusts in infants)

Adapted from the 2025 AHA Adult Foreign-Body Airway Obstruction Algorithm.



8 ACLS Adult Cardiac Arrest Algorithm

The ACLS cardiac arrest algorithm branches immediately on first rhythm assessment into a shockable pathway (VF/pVT) and a non-shockable pathway (asystole/PEA), and providers must be able to move fluidly between the two if the rhythm changes during resuscitation. Throughout both pathways, CPR continues in 2-minute cycles, IV or IO access is established without interrupting compressions, an advanced airway (endotracheal tube or supraglottic airway — both are acceptable, and continuous waveform capnography should be used to confirm placement and to help monitor CPR quality) is placed when feasible, and the team systematically works through the H's and T's.

ALGORITHM — ADULT CARDIAC ARREST ALGORITHM (2025 AHA)

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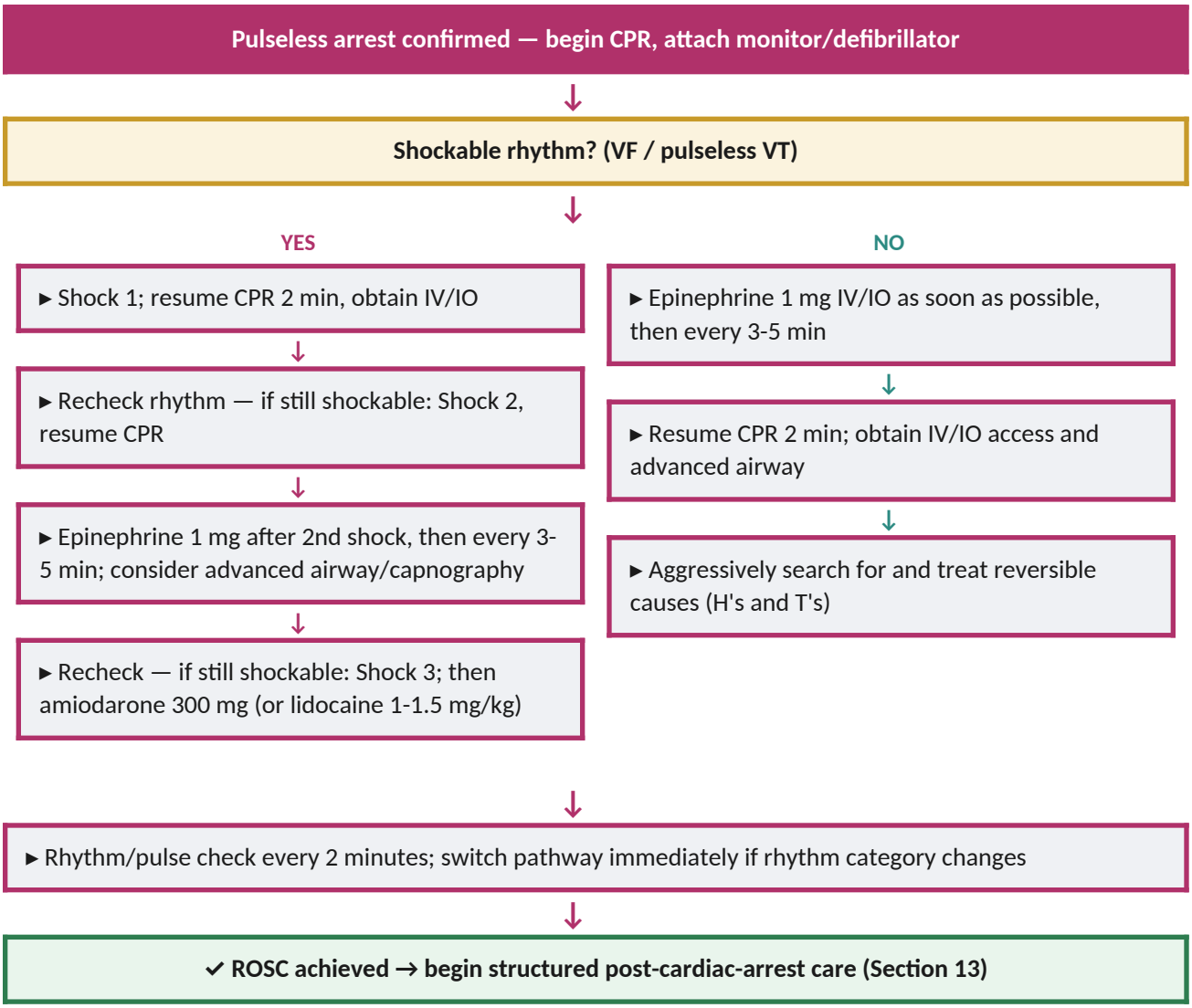
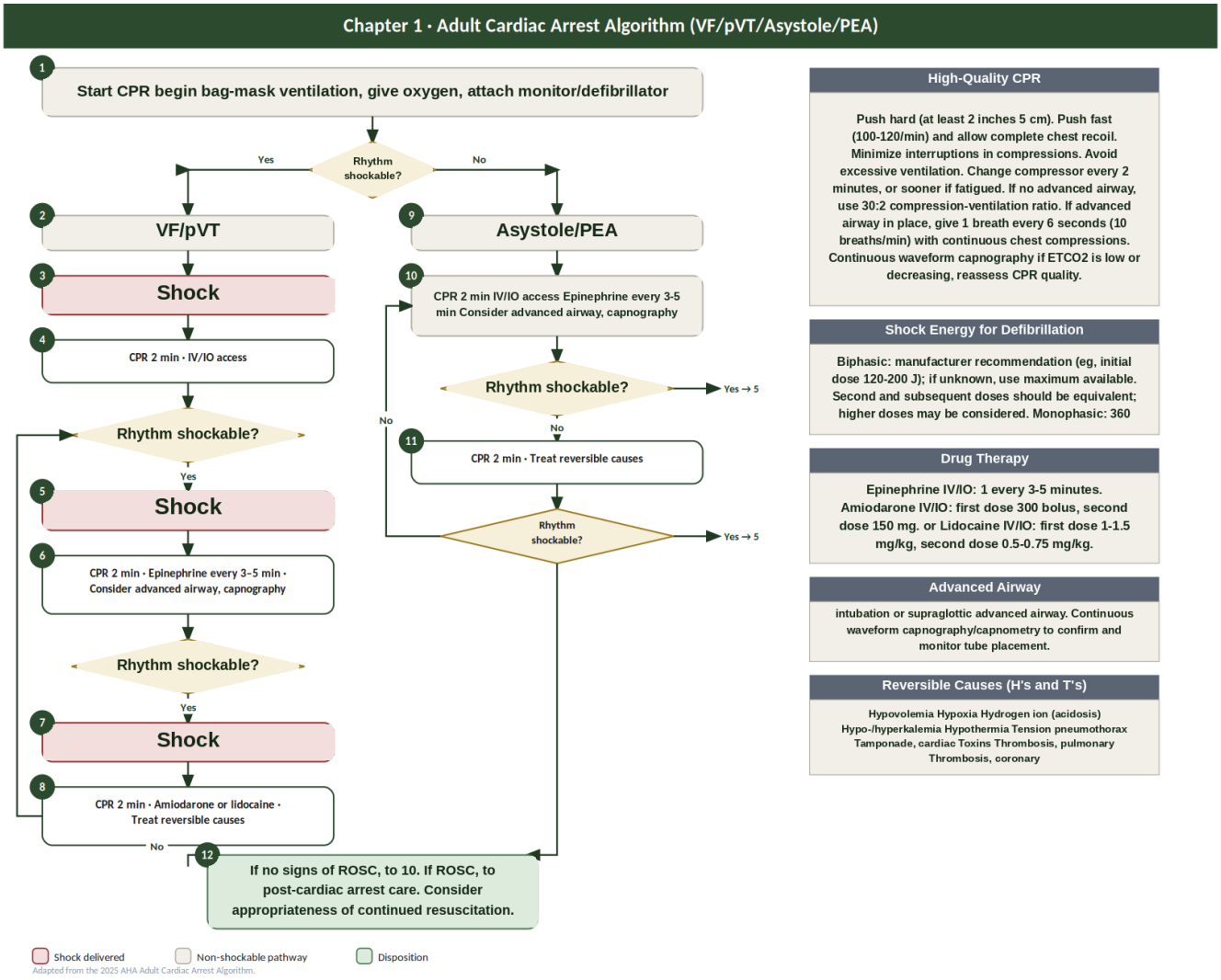


Figure 1.1. Adapted from the 2025 AHA Adult Cardiac Arrest Algorithm; consult the full official algorithm for concurrent considerations not shown here.

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Shockable pathway (VF / pulseless VT)

Deliver the first shock as soon as a shockable rhythm is confirmed. Resume CPR immediately for a full 2-minute cycle while establishing IV/IO access, then reassess the rhythm. If still shockable, deliver a second shock and resume CPR. After the second shock, begin epinephrine 1 mg IV/IO — this is the point in the algorithm at which the vasopressor is introduced for a shockable rhythm — and repeat every 3 to 5 minutes for the remainder of the arrest; consider placing an advanced airway and capnography at this stage as well. Reassess the rhythm; if still shockable, deliver a third shock and resume CPR. After the third shock, administer the first dose of an antiarrhythmic — amiodarone 300 mg IV/IO (second dose 150 mg if VF/pVT persists or recurs) or lidocaine 1-1.5 mg/kg IV/IO (second dose 0.5-0.75 mg/kg) as an accepted alternative — and continue to actively search for and treat a reversible cause. Continue cycling shock, 2 minutes of CPR, and rhythm/pulse check, with epinephrine every 3-5 minutes, until ROSC or a decision to stop resuscitation.

Non-shockable pathway (asystole / PEA)

Give epinephrine 1 mg IV/IO as early as possible — there is no waiting period analogous to the shockable pathway. Continue high-quality CPR in 2-minute cycles, repeat epinephrine every 3-5 minutes, establish IV/IO access and an advanced airway, and aggressively search for and treat reversible causes, since asystole and PEA are far more often driven by an identifiable, correctable process than VF/pVT. Reassess the rhythm at each cycle break; if it becomes shockable, move immediately into the shockable pathway with a shock.

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Decision Point — Shockable or Not?

Every rhythm/pulse check is a fork: shockable (VF/pVT) drives toward the next shock plus antiarrhythmic; non-shockable (asystole/PEA) drives toward earlier epinephrine plus an aggressive hunt for a reversible cause.

Never delay the next scheduled shock or drug dose to debate the rhythm at length — assign the rhythm interpretation to one team member and keep compressions running through the discussion.

A change in rhythm category at any point (shockable to non-shockable or vice versa) immediately redirects the team down the corresponding pathway; the 2-minute CPR/reassessment cycle itself never stops.

Return of spontaneous circulation is signaled by a palpable pulse, an abrupt sustained rise in end-tidal CO2, or, if an arterial line is in place, visible arterial waveform pulsatility. Once ROSC is achieved, transition immediately into structured post-cardiac-arrest care (Section 13).

9 Key Resuscitation Drugs and Doses

Only a small number of drugs have a well-established role in cardiac arrest itself; most other pharmacologic interventions are reserved for specific reversible causes or the peri-arrest bradycardia/tachycardia algorithms. Vasopressin is no longer recommended as a substitute for or adjunct to epinephrine, having shown no outcome advantage in trials, and routine sodium bicarbonate or calcium administration is reserved for specific indications (severe acidosis, hyperkalemia, calcium-channel-blocker toxicity) rather than given empirically.

Table 1.4 Key resuscitation drugs, doses, and timing

Drug	Indication	Dose	Route	Timing / repeat
Epinephrine (1 mg/10 mL, 1:10,000)	Cardiac arrest, all rhythms	1 mg	IV/IO	Non-shockable: as early as possible. Shockable: after the 2nd shock. Repeat every 3-5 min throughout.
Amiodarone	Refractory VF/pVT	300 mg first dose; 150 mg second dose	IV/IO	After the 3rd shock; second dose if VF/pVT persists or recurs
Lidocaine	Refractory VF/pVT (alternative to amiodarone)	1-1.5 mg/kg first dose; 0.5-0.75 mg/kg second dose	IV/IO	After the 3rd shock; repeat every 5-10 min, max 3 mg/kg total
Atropine	Symptomatic bradycardia (not used in arrest/asystole)	1 mg	IV	Repeat every 3-5 min; maximum total dose 3 mg
Adenosine	Regular narrow-complex (formerly	6 mg first dose; 12 mg second dose	Rapid IV push with immediate saline	Repeat 12 mg once if no conversion

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Drug	Indication	Dose	Route	Timing / repeat
	'SVT') tachycardia		flush	after 1-2 min
Dopamine	Bradycardia refractory to atropine	5-20 mcg/kg/min infusion	IV	Titrate to response; taper slowly
Epinephrine infusion	Bradycardia refractory to atropine; post-arrest hypotension	2-10 mcg/min infusion	IV	Titrate to response
Naloxone	Suspected opioid-associated emergency or arrest	0.4-2 mg IV/IM, or 4 mg intranasal	IV/IM/intranasal	Repeat every 2-3 min as needed; give alongside, not instead of, standard CPR/ventilation and ACLS

Doses per the 2025 AHA Guidelines for CPR and ECC and current ACLS Provider Manual; confirm local formulary and pediatric weight-based dosing separately.

10 Defibrillation and Cardioversion Energies

Unsynchronized shocks (defibrillation) are used for VF, pulseless VT, and polymorphic VT, because these rhythms have no reliably identifiable R wave for the defibrillator to synchronize against and any delay searching for one only prolongs the pause in compressions. Synchronized cardioversion, timed to the R wave to avoid shocking during the vulnerable T wave, is used for organized rhythms with a pulse that are hemodynamically unstable: monomorphic VT, regular narrow-complex tachycardia, atrial fibrillation, and atrial flutter.

Table 1.5 Defibrillation and cardioversion energies by rhythm

Rhythm	Shock type	Initial energy	Notes
VF / pulseless VT	Unsynchronized (defibrillation)	Device/manufacturer-specific; if unknown, use the maximum available energy	Escalate to maximum energy on subsequent shocks if the device allows; never delay a shock to select energy
Polymorphic VT (no pulse, or unstable and irregular)	Unsynchronized (treated as defibrillation)	Same as VF/pVT	Synchronization circuitry may fail to lock onto an irregular waveform — do not attempt to synchronize
Monomorphic VT, unstable	Synchronized cardioversion	100 J	Sedate before cardioversion whenever feasible
Narrow-complex tachycardia, regular, unstable (formerly 'SVT')	Synchronized cardioversion	100 J	Attempt vagal maneuvers/adenosine first if the patient is stable enough to allow a brief trial

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Rhythm	Shock type	Initial energy	Notes
Atrial fibrillation, unstable	Synchronized cardioversion	200 J	Higher initial energy reflects the larger irregular atrial mass being cardioverted
Atrial flutter, unstable	Synchronized cardioversion	200 J	Often converts at lower energies clinically, but 200 J is the recommended initial setting

Energies per the 2025 AHA Electrical Cardioversion and Adult Cardiac Arrest algorithms. Routine double-sequential (dual) defibrillation for refractory VF/pVT after 3 or more shocks has not been shown to be beneficial and is not recommended for routine use.

Myth vs Fact

Myth: The term 'SVT' (supraventricular tachycardia) is still the preferred term for a stable, regular, narrow-complex tachycardia.

Fact: The 2025 algorithms now use the broader term 'narrow-complex tachycardia' for regular narrow-complex rhythms, reflecting that the acute management (vagal maneuvers, adenosine, 100 J synchronized cardioversion if unstable) is driven by the QRS width and regularity rather than by pinning down the exact supraventricular mechanism in real time.

Myth: Stacking multiple defibrillators on a patient in refractory VF ('double sequential defibrillation') is now standard practice for shock-refractory arrest.

Fact: The evidence remains insufficient — the 2025 guidelines explicitly state the usefulness of vector-change or double sequential defibrillation after 3 or more consecutive shocks has not been established, and it should not be adopted as routine practice outside a research protocol.

11 Reversible Causes: The H's and T's

Cardiac arrest that is refractory to standard shocks and drugs, and virtually all PEA and asystole arrests, warrants a systematic search through the ten classic reversible causes. These are best worked through out loud as a checklist during the 2-minute CPR cycles rather than recalled under pressure.

Table 1.6 The H's and T's of reversible cardiac arrest causes

Category	Cause	Clinical clue	Treatment
H	Hypovolemia	Flat neck veins, history of hemorrhage, trauma, GI bleed	Rapid volume/blood product resuscitation, control the source of loss
H	Hypoxia	Cyanosis, airway obstruction, difficult ventilation	Secure the airway, ventilate with high-flow oxygen
H	Hydrogen ion (acidosis)	Known renal failure, DKA, prolonged arrest	Optimize ventilation; consider sodium bicarbonate if severe/refractory

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Category	Cause	Clinical clue	Treatment
H	Hypo-/hyperkalemia	Dialysis history, peaked T waves, widened QRS, missed dialysis	Calcium chloride, insulin/glucose, bicarbonate (hyperkalemia); potassium replacement (hypokalemia)
H	Hypothermia	Cold exposure, submersion, low-reading core thermometer	Active internal/external rewarming; consider extended resuscitation
T	Tension pneumothorax	Unilateral absent breath sounds, jugular venous distension, tracheal deviation	Immediate needle or finger thoracostomy
T	Tamponade, cardiac	JVD, muffled heart sounds, PEA after trauma or central line placement	Emergent pericardiocentesis
T	Toxins	History of ingestion/overdose, track marks, medication bottles at scene	Specific antidote when available (e.g., naloxone), supportive/decontamination care
T	Thrombosis, pulmonary (massive PE)	Sudden collapse, known DVT/PE risk factors or prior symptoms	Consider fibrinolytics, embolectomy, or ECPR
T	Thrombosis, coronary (acute MI)	Anginal history, ischemic ECG changes	Emergent coronary reperfusion (PCI or fibrinolytics) once ROSC achieved

12 Bradycardia and Tachycardia Algorithms

Peri-arrest bradyarrhythmias and tachyarrhythmias follow their own algorithms distinct from the pulseless arrest algorithm, because the patient still has a pulse and perfusion, and the priority is determining whether the patient is hemodynamically stable before choosing a pharmacologic versus electrical strategy.

Table 1.7 Adult bradycardia-with-a-pulse algorithm

Step	Assessment / action	Detail
1	Identify bradycardia	Heart rate typically under 50/min in the context of a bradyarrhythmia
2	Assess for instability	Hypotension, altered mental status, signs of shock, ongoing chest discomfort, acute heart failure
3	If stable	Monitor and observe; identify and treat the underlying cause

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Step	Assessment / action	Detail
4	If unstable — first line	Atropine 1 mg IV, repeat every 3-5 min, maximum total dose 3 mg
5	If atropine ineffective or unlikely to work (e.g., high-degree AV block, transplanted heart)	Transcutaneous pacing and/or dopamine infusion 5-20 mcg/kg/min and/or epinephrine infusion 2-10 mcg/min
6	Persistent instability	Expert consultation; consider transvenous pacing

Figure 1.2. Adapted from the 2025 AHA Adult Bradycardia With a Pulse Algorithm.

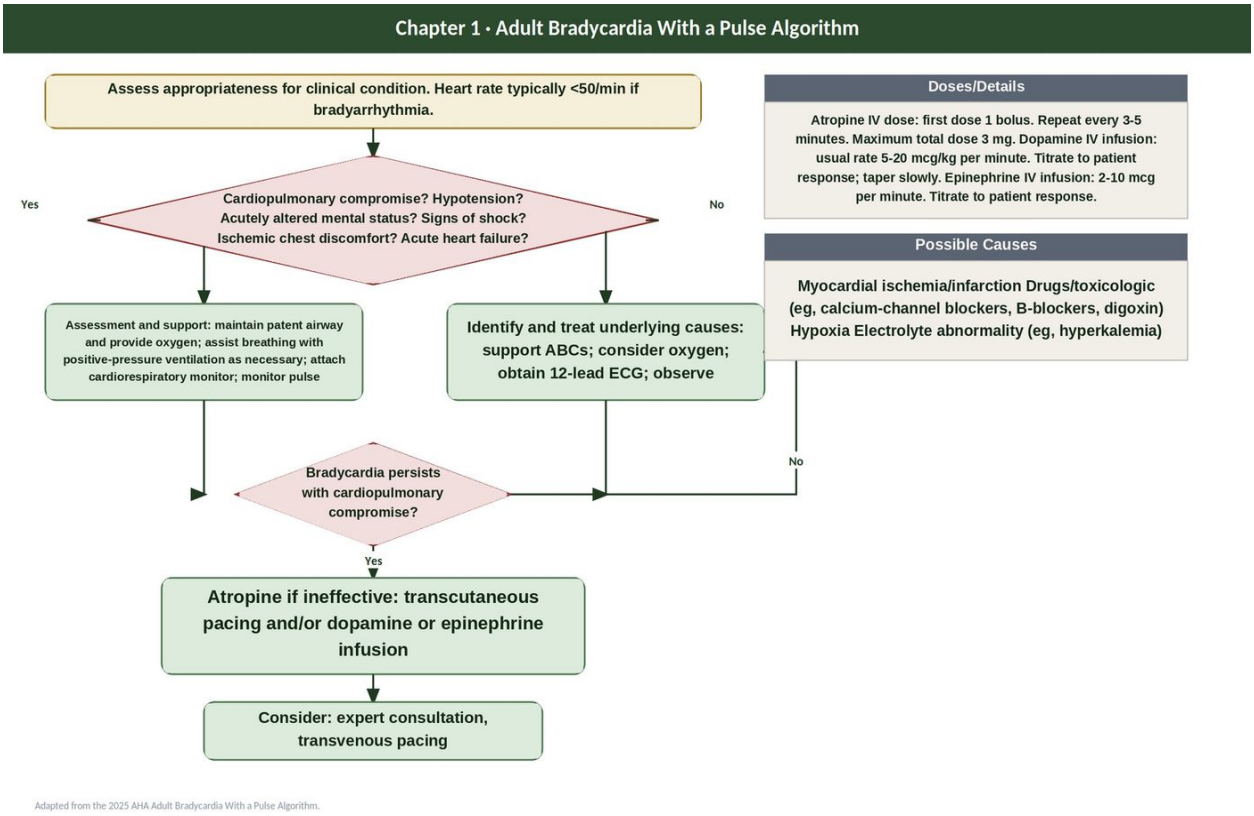


Table 1.8 Adult tachycardia-with-a-pulse algorithm summary

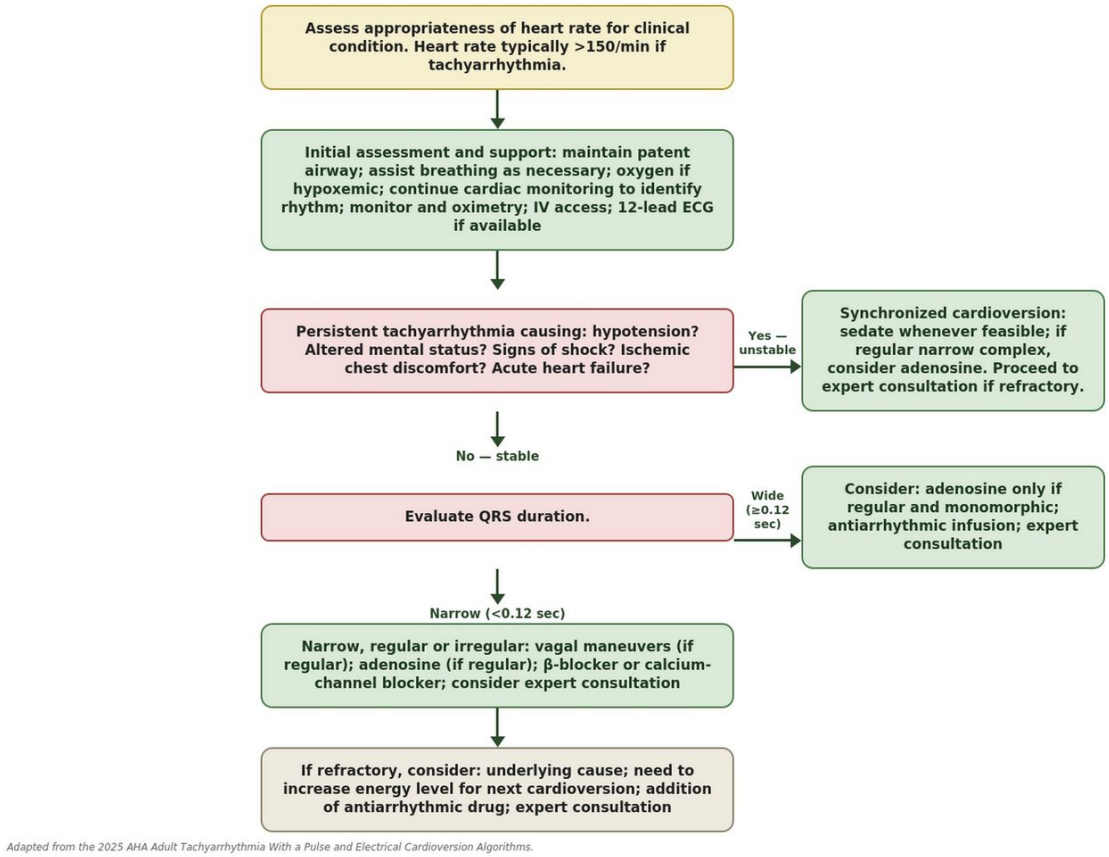
Presentation	QRS width	Regularity	First-line management
Unstable (any QRS width)	Narrow or wide	Regular or irregular	Immediate synchronized cardioversion at the energy in Table 1.5; manage polymorphic VT as unsynchronized defibrillation
Stable	Narrow	Regular	Vagal maneuvers, then adenosine 6 mg rapid IVP, followed by 12 mg if no conversion

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Presentation	QRS width	Regularity	First-line management
Stable	Narrow	Irregular (e.g., atrial fibrillation/flutter)	Rate control (beta-blocker or calcium-channel blocker), assess need for anticoagulation, expert consultation
Stable	Wide	Regular (monomorphic VT)	Antiarrhythmic infusion (amiodarone, procainamide, or sotalol) or expert consultation; treat as VT unless proven otherwise
Stable	Wide	Irregular (polymorphic VT/torsades)	Correct electrolytes and give magnesium; avoid AV-nodal blocking agents if pre-excited atrial fibrillation is suspected; expert consultation

When in doubt about the mechanism of a wide-complex tachycardia, treat it as ventricular in origin — this is the safer default assumption.

Chapter 1 · Adult Tachyarrhythmia With a Pulse Algorithm



Adapted from the 2025 AHA Adult Tachyarrhythmia With a Pulse and Electrical Cardioversion Algorithms.

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Figure 1.3. Adapted from the 2025 AHA Adult Tachyarrhythmia With a Pulse and Electrical Cardioversion Algorithms.

13 Post-Cardiac-Arrest Care

Achieving ROSC is not the endpoint of resuscitation — roughly two-thirds of early in-hospital deaths after ROSC are attributable to neurological injury, and structured post-arrest care bundles measurably improve survival with good neurological outcome. The core pillars are temperature control, avoidance of hypotension and hypoxia/hyperoxia, ventilation targets, and early identification of a treatable coronary cause.

Post-ROSC Monitoring Checklist

12-lead ECG immediately, with emergent coronary angiography if STEMI or high suspicion of an acute coronary occlusion.

Continuous temperature monitoring with active temperature control device from the earliest feasible point after ROSC.

Continuous arterial blood pressure monitoring (arterial line preferred) with a vasopressor titrated to maintain the MAP target.

Continuous pulse oximetry and, where available, arterial blood gas monitoring to keep oxygenation and ventilation within target range — avoid both hypoxia and hyperoxia.

Frequent neurologic checks and multimodal neuroprognostication (clinical exam, EEG, imaging, biomarkers) deferred until at least 72 hours post-arrest in patients treated with temperature control, since early findings are unreliable.

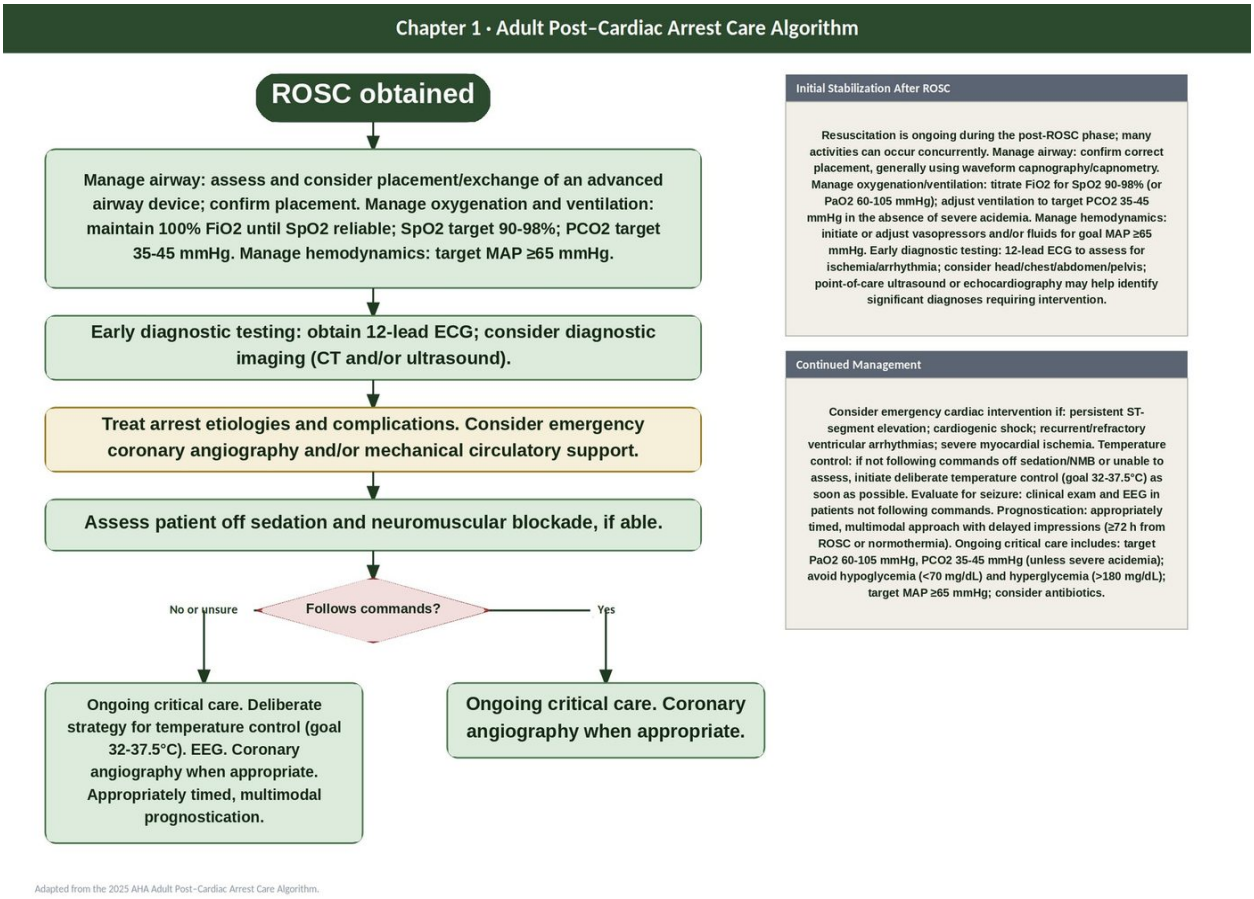
Glucose monitoring — avoid hypoglycemia and treat significant hyperglycemia per local protocol.

Table 1.9 Post-cardiac-arrest care targets

Parameter	Target	Notes
Temperature control	32-37.5°C, maintained at least 36 hours in patients who remain unresponsive to verbal commands	Actively prevent fever for at least 72 hours after arrest
Mean arterial pressure (MAP)	At least 65 mmHg (minimum floor)	Avoid hypotension; use vasopressors as needed
Oxygen saturation (SpO2)	90-98%	Avoid both hypoxia and hyperoxia
PaO2	60-105 mmHg	Corresponds to the SpO2 target range
PaCO2	35-45 mmHg	Avoid hyper- and hypocapnia; both worsen cerebral perfusion
Coronary evaluation	Immediate 12-lead ECG; emergent angiography if STEMI or strong suspicion of acute coronary occlusion	Applies regardless of neurologic status at presentation

Figure 1.4. Adapted from the 2025 AHA Adult Post–Cardiac Arrest Care Algorithm.

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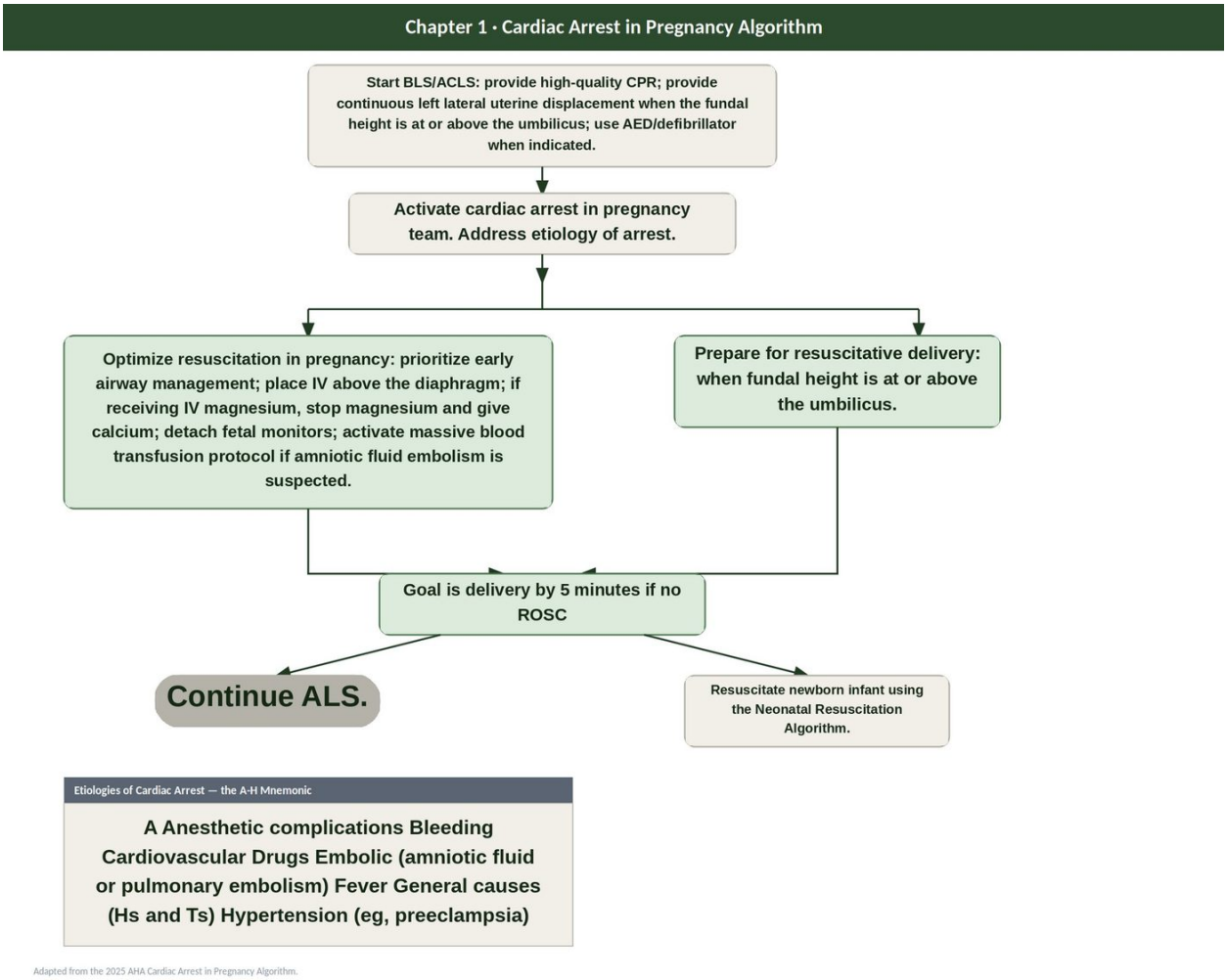
14 Special Circumstances

Three situations modify the standard adult algorithm enough to warrant specific mention: pregnancy, suspected opioid-associated arrest, and hypothermic arrest.

- Pregnancy: perform standard high-quality CPR with manual left uterine displacement to relieve aortocaval compression; standard drug doses and defibrillation energies are unchanged. If there is no ROSC with standard resuscitation, resuscitative (perimortem) cesarean delivery should be prepared for early, with a goal of completing delivery within 5 minutes of arrest onset to improve both maternal and fetal outcomes; ECPR is a reasonable consideration in peripartum arrest unresponsive to standard measures. If amniotic fluid embolism is suspected, activate a massive transfusion protocol with a balanced transfusion strategy.

Adapted from the 2025 AHA Cardiac Arrest in Pregnancy Algorithm.

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- Opioid-associated emergency/arrest: naloxone is now integrated directly into the BLS algorithm for a suspected opioid-associated emergency in a patient who is breathing but unresponsive — give naloxone while continuing to support the airway and ventilation. In confirmed cardiac arrest, standard CPR and ACLS take priority; naloxone may reasonably be given alongside resuscitation but should never delay or substitute for compressions, ventilation, and defibrillation when indicated. Patients treated for opioid overdose should be given a take-home naloxone kit and instruction on its use at discharge.
- Hypothermic arrest: continue standard CPR and follow the standard algorithm while actively rewarming; a patient in severe hypothermia (core temperature under 28°C) may tolerate a prolonged resuscitation, and extracorporeal life support (ECLS) rewarming is a reasonable option in cardiac arrest due to hypothermia, guided where possible by validated prognostication scores.

Red Flags

An unresponsive, apneic or gasping patient with pinpoint pupils, track marks, or drug paraphernalia at the scene should trigger naloxone administration in parallel with — never instead of — standard BLS/ACLS.

A pregnant patient in cardiac arrest past roughly 20 weeks gestation (fundus at or above the umbilicus) needs immediate manual left uterine displacement and early preparation for resuscitative cesarean delivery — do not wait for a 'stable' moment that will not come.

A cold, bradycardic or asystolic patient recovered from cold water or extreme environmental exposure

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should not be pronounced dead based on standard criteria until adequately rewarmed — 'no one is dead until warm and dead' remains a guiding principle in confirmed accidental hypothermia.

15 Worked Clinical Case

Case 1.1 — Witnessed collapse on a general medical ward

Presentation: A 62-year-old man with a history of coronary artery disease is found by a nurse collapsed at the bedside, unresponsive, with agonal gasping. The nurse cannot palpate a pulse within 10 seconds and immediately activates the hospital rapid-response/code system while beginning chest compressions.

Initial resuscitation: The code team arrives with the crash cart within 2 minutes. The monitor shows coarse ventricular fibrillation. A shock is delivered immediately at the device's recommended energy, and CPR resumes without pause for rhythm or pulse checking. IV access is obtained during the 2-minute cycle. Rhythm check at 2 minutes still shows VF; a second shock is delivered and CPR resumes.

Escalation: After the second shock, epinephrine 1 mg IV is given and a supraglottic airway is placed with waveform capnography confirming correct placement and providing ongoing feedback on compression quality. At the next rhythm check VF persists; a third shock is delivered, followed immediately by amiodarone 300 mg IV. The team runs through the H's and T's out loud: no evidence of hypoxia, hypovolemia, tension pneumothorax, or tamponade; the working diagnosis is thrombosis, coronary, given his cardiac history.

Outcome: After the fourth shock and a further 2-minute CPR cycle, the monitor shows an organized narrow-complex rhythm and a strong carotid pulse is palpated — ROSC is declared roughly 9 minutes after collapse. Post-ROSC care is initiated immediately: a 12-lead ECG shows anterior ST elevation, and the patient is taken emergently for coronary angiography, where a proximal LAD occlusion is opened with a stent. Temperature control is initiated at 36°C in the cardiac ICU and maintained for 36 hours, with MAP kept above 65 mmHg on a low-dose norepinephrine infusion and SpO2 maintained at 94-98%. He is extubated on hospital day 3 with no focal neurologic deficit and is discharged home on day 8.

Teaching point: This case illustrates the shockable pathway executed correctly — early defibrillation, epinephrine introduced after the second shock, an antiarrhythmic after the third, uninterrupted high-quality CPR throughout, and a seamless transition into post-arrest care that identified and treated the underlying coronary thrombosis. The short time to ROSC and immediate reperfusion were the two largest contributors to his favorable neurological outcome.

16 Chapter Quick-Reference Summary

The table below consolidates the highest-yield numbers from this chapter for rapid pre-shift or pre-exam review.

Table 1.10 Chapter 1 quick-reference summary

Parameter	Value
Compression rate (all ages)	100-120/min
Compression depth, adult	2-2.4 in (5-6 cm)
Compression-to-ventilation ratio, adult (1 or 2	30:2

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Parameter	Value
rescuers)	
Ventilation rate with advanced airway, adult	1 breath/6 sec (10/min)
Epinephrine (arrest)	1 mg IV/IO every 3-5 min
Amiodarone (refractory VF/pVT)	300 mg, then 150 mg
Lidocaine (refractory VF/pVT, alternative)	1-1.5 mg/kg, then 0.5-0.75 mg/kg
Atropine (bradycardia)	1 mg IV every 3-5 min, max 3 mg
Adenosine (narrow-complex tachycardia)	6 mg, then 12 mg, rapid IVP
VF / pulseless VT defibrillation	Unsynchronized, device-max energy if unknown
Monomorphic VT / regular narrow-complex tachycardia cardioversion	100 J synchronized
Atrial fibrillation / atrial flutter cardioversion	200 J synchronized
Post-arrest temperature control	32-37.5°C, maintained ≥36 h
Post-arrest MAP target	≥65 mmHg
Post-arrest SpO2 target	90-98%

On a Busy Shift

Tape a laminated copy of Table 1.10 inside the crash cart lid — under stress, recalling the exact epinephrine timing relative to shock number is exactly the kind of detail that gets confused, and a glance saves a debate mid-code.

Assign the H's and T's search explicitly to one team member by name at the start of any PEA/asystole arrest so it actually gets worked through systematically rather than left to whoever remembers.

17 Acute Coronary Syndrome Algorithm

Chest pain or other symptoms suggestive of acute coronary syndrome (ACS) trigger a 12-lead ECG within 10 minutes of first contact, since the ECG pattern - ST-elevation myocardial infarction (STEMI) versus non-ST-elevation ACS (NSTEMI or unstable angina) - determines the entire treatment pathway. This section reflects the 2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes, which replaced the older separate STEMI and NSTEMI guidelines.

Immediate management (both pathways)

Give aspirin 162-325 mg chewed (non-enteric-coated) - the single intervention with the strongest mortality benefit - obtain IV access, and start continuous cardiac monitoring. Supplemental oxygen is given only for oxygen saturation below 90%, respiratory distress, or other high-risk features of hypoxemia; routine oxygen in a normoxic patient is no longer recommended, since trials have raised concern that it may increase coronary vascular resistance and myocardial injury. Sublingual nitroglycerin (0.3-0.4 mg every 5 minutes, up to 3 doses) relieves ischemic pain, but is withheld if the patient has used a phosphodiesterase-5 inhibitor (sildenafil, tadalafil, vardenafil) within the preceding 24-48 hours, since the combination can cause profound,

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refractory hypotension. Morphine is reserved as a second-line agent for pain unrelieved by nitrates, since observational data associate it with delayed absorption of oral antiplatelet therapy.

STEMI pathway

ST elevation on the presenting ECG activates the cardiac catheterization laboratory immediately, targeting a cath time under 90 minutes with a radial (wrist) rather than femoral approach, which the 2025 guideline now emphasizes to reduce bleeding, vascular complications, and mortality. If primary PCI cannot be delivered within the target time (for example, a non-PCI-capable facility with a long transfer distance), fibrinolytic therapy is given instead, targeting a door-to-needle time of 30 minutes or less.

NSTE-ACS pathway (NSTEMI / unstable angina)

Without ST elevation, a high-sensitivity troponin is drawn immediately and repeated per protocol to confirm or exclude myocardial infarction, alongside risk stratification for both ischemic and bleeding risk. Patients at intermediate or high ischemic risk are managed with an invasive strategy (angiography with intent to revascularize) during the same admission; patients at low risk may be managed with a more selective invasive approach guided by further risk stratification.

Antithrombotic therapy and revascularization

Ticagrelor or prasugrel is now preferred over clopidogrel as the P2Y12 inhibitor in both STEMI and NSTEMI patients undergoing PCI, with clopidogrel reserved for patients in whom the newer agents are contraindicated, not tolerated, or unavailable. Unfractionated heparin is a standard anticoagulant choice for both pathways; bivalirudin is an alternative in STEMI shown to reduce bleeding and mortality compared with heparin, while fondaparinib is not recommended during PCI due to a risk of catheter thrombosis. A strategy of complete revascularization is recommended for both STEMI and NSTE-ACS when multivessel disease is present, and intravascular imaging is recommended to guide PCI in complex coronary lesions. Dual antiplatelet therapy (aspirin plus a P2Y12 inhibitor) is continued for at least 12 months by default in patients without a high bleeding risk, with several shortened or modified strategies available for patients who are at higher bleeding risk.

Secondary prevention

Before discharge: a beta-blocker (continued long-term if the ejection fraction is reduced), an ACE inhibitor or ARB for reduced ejection fraction, heart failure, hypertension, or diabetes, high-intensity statin therapy for all ACS patients (with ezetimibe as an add-on or alternative for statin intolerance), a fasting lipid panel 4-8 weeks after starting or adjusting lipid-lowering therapy, and referral to cardiac rehabilitation.

18 Suspected Stroke Algorithm

A sudden focal neurologic deficit is screened with a validated tool - BE-FAST (Balance, Eyes, Face, Arm, Speech, Time) extends the original FAST mnemonic to capture posterior circulation strokes that FAST alone can miss. This section reflects the 2026 AHA/ASA Guideline for the Early Management of Patients With Acute Ischemic Stroke, which replaced the 2018 guideline and its 2019 update.

The single most important data point to establish is the time last known well, since it anchors every subsequent treatment-window decision. Check a bedside glucose early, since hypoglycemia can mimic a focal deficit and is instantly reversible. Rapid transport with prehospital notification to a stroke-capable center shortens door-to-treatment time; the guideline endorses direct transport to the closest endovascular-thrombectomy-capable hospital over the nearest thrombolytic-capable facility where the local system of care

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supports it, and endorses mobile stroke units where available. On arrival, non-contrast CT is targeted within 25 minutes to distinguish ischemic from hemorrhagic stroke, since this determines whether thrombolysis is even a consideration.

If hemorrhage is present

Thrombolysis is contraindicated. Manage as intracerebral hemorrhage (Chapter 7, Section 8): blood pressure control, reversal of anticoagulation if applicable, and neurosurgical consultation.

If no hemorrhage (ischemic stroke)

Patients within the 4.5-hour thrombolytic window without exclusion criteria receive intravenous alteplase or tenecteplase - the 2026 guideline endorses either agent as non-inferior, expanding on alteplase-only prior recommendations - targeting a door-to-needle time of 60 minutes or less. Treatment is recommended for eligible patients with a disabling deficit regardless of the numerical severity (NIHSS) score, rather than withholding thrombolysis from milder-appearing presentations. Every patient, whether or not they receive thrombolysis, is evaluated for large-vessel occlusion and thrombectomy eligibility; when both thrombolysis and thrombectomy are indicated, they are pursued in parallel rather than delaying one for the other, and thrombectomy eligibility windows have been broadened compared to earlier guidelines.

Supportive care

Avoid intensive glucose control - a tight target range of 80-130 mg/dL is no longer recommended, since it increases the risk of severe hypoglycemia without improving outcome - and avoid hyperthermia, which worsens neurologic injury. Blood pressure targets differ sharply by stroke type and by whether reperfusion therapy was given, so should be set according to current stroke-specific parameters rather than general critical care defaults. Admit to a dedicated stroke unit or neurocritical care setting for the closely spaced neurologic checks that detect early deterioration.

This chapter is based on the American Heart Association 2025 Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care (Circulation, 2025), including the unified Chain of Survival, Adult Basic Life Support, Adult Advanced Life Support, and associated 2025 AHA algorithms (Adult Cardiac Arrest, Electrical Cardioversion, Bradycardia, and Tachycardia), together with the current AHA BLS and ACLS Provider Manuals. It is intended as an educational reference and is not a substitute for a certified BLS/ACLS provider course, hands-on skills verification, or official AHA course completion.

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LIFE SUPPORT COURSES · CLINICAL COMPANION MANUAL

CHAPTER 2

PALS

Pediatric Advanced Life Support

1 Clinical Snapshot

Pediatric Advanced Life Support (PALS) governs the recognition and management of the seriously ill or injured infant and child, from the compensated-shock toddler in triage to the asystolic neonate wheeled back from the playground. Unlike adults, children rarely arrest from a primary cardiac event — the dominant final common pathway is progressive respiratory failure or shock that goes unrecognized or undertreated until the heart stops. This makes early recognition, not defibrillation, the single highest-yield skill in this chapter. Outcomes after in-hospital pediatric arrest are meaningfully better than after out-of-hospital arrest, which is precisely why PALS invests so heavily in the assessment tools that catch deterioration before the arrest call goes out.

This chapter reflects the 2025 American Heart Association (AHA) and American Academy of Pediatrics (AAP) Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, which introduced a Universal Chain of Survival shared across adult and pediatric, in-hospital and out-of-hospital settings, eliminated the two-finger infant compression technique, and refined diastolic blood pressure targets during CPR and post-arrest temperature management. Numbers throughout are given as taught in the current provider course; always confirm against your institution's current algorithms.

Why this matters · Typical patient · Where you meet it

Why it matters: Pediatric arrest is uncommon compared with adult arrest, which means individual clinicians get little repetition — deliberate, protocolized recognition and dosing is what closes that experience gap and prevents a rare event from becoming a poorly run one.

Typical patient: An infant or child with an evolving respiratory or circulatory problem — bronchiolitis with silent chest, gastroenteritis with decompensating shock, status epilepticus, sepsis, drowning, or trauma — who deteriorates over minutes to hours if the warning signs are missed.

Common settings: Emergency departments, pediatric and general inpatient wards, PICUs, general operating rooms, prehospital/EMS response, and any adult-oriented facility that occasionally receives a child.

2 Learning Objectives

By the end of this chapter you should be able to:

- Apply the Pediatric Assessment Triangle and a systematic ABCDE examination to identify a sick or deteriorating child before cardiorespiratory arrest occurs.
- Deliver high-quality pediatric CPR with correct rate, depth, hand placement, and compression-to-ventilation ratio for infants and children, using the current single-rescuer and two-rescuer techniques.
- Navigate the pediatric cardiac arrest algorithm, distinguishing shockable (VF/pulseless VT) from non-shockable (asystole/PEA) pathways and sequencing epinephrine, shocks, and antiarrhythmics correctly.

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- Calculate weight-based doses for epinephrine, amiodarone, lidocaine, adenosine, and atropine, and select correct defibrillation and cardioversion energies in J/kg.
- Differentiate stable from unstable bradycardia and tachycardia, and narrow-complex from wide-complex tachycardia, and select the correct management pathway for each.
- Recognize compensated versus decompensated shock and dose fluid resuscitation appropriately by etiology.
- Manage foreign-body airway obstruction in infants and children, applying the current back-blow/thrust sequences.
- Set appropriate post-resuscitation targets for oxygenation, blood pressure, and temperature to protect the post-arrest brain.

3 The Pediatric Chain of Survival

Previous guideline cycles maintained separate Chains of Survival for adult and pediatric, and for in-hospital versus out-of-hospital arrest — four parallel frameworks that clinicians had to remember and apply correctly under stress. The 2025 AHA/AAP guidelines replaced these with a single Universal Chain of Survival that applies across age and setting, on the evidence that the underlying sequence of actions (recognize early, act fast, escalate systematically) is the same regardless of who the patient is or where the arrest happens.

Table 2.1 The Universal Chain of Survival (2025)

Link	Core action	Pediatric-specific emphasis
1. Recognition & activation	Identify cardiac arrest or peri-arrest deterioration and activate the emergency response system immediately	Because most pediatric arrests are asphyxial, recognition of respiratory failure and shock — not just pulselessness — is the critical early trigger
2. High-quality CPR	Immediate, minimally interrupted, correctly rated and correctly deep chest compressions	Depth and hand position differ from adult CPR (see Section 5); rescue breaths matter more given the respiratory etiology of most arrests
3. Defibrillation	Rapid rhythm check and shock delivery for VF/pulseless VT	Shockable rhythms are less common in children than in adults, but still mandate immediate defibrillation when present
4. Advanced resuscitation	Advanced airway management, IV/IO access, rhythm-specific drug therapy	Weight-based dosing throughout; early epinephrine is emphasized for non-shockable rhythms
5. Post-arrest care	Structured stabilization after return of spontaneous circulation (ROSC)	Targeted temperature management, avoidance of hyperoxia/hypercapnia, and hemodynamic support tailored to age-based norms
6. Recovery	Rehabilitation, neuroprognostication, and family/psychosocial support	Formal recommendations for predicting neurologic outcome after pediatric arrest were added

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Link	Core action	Pediatric-specific emphasis
		for the first time in 2025

Source: 2025 AHA/AAP Guidelines for CPR and ECC, Part 4 (Systems of Care) and Part 6 (Pediatric Basic Life Support).

In-hospital cardiac arrest (IHCA) and out-of-hospital cardiac arrest (OHCA) still differ operationally — IHCA is typically witnessed, monitored, and reached faster by a resuscitation team, while OHCA depends on bystander recognition and CPR quality before EMS arrival — but both now run through the same six-link framework rather than a bespoke pediatric chain.

4 Recognizing the Sick or Deteriorating Child

The Pediatric Assessment Triangle

The Pediatric Assessment Triangle (PAT) is a 15-30 second, hands-off, across-the-room assessment performed before touching the patient. It generates an instant physiologic impression — well-appearing, or in respiratory distress, respiratory failure, shock, or a life-threatening central nervous system/metabolic problem — that determines how urgently the subsequent hands-on examination must proceed.

Table 2.2 The Pediatric Assessment Triangle

Component	What you are assessing	Abnormal findings
Appearance	Tone, Interactiveness, Consolability, Look/gaze, Speech or cry ("TICLS")	Limp, unable to be consoled, no eye contact, weak or absent cry, does not interact with caregiver or examiner
Work of breathing	Abnormal airway sounds, abnormal positioning, retractions, nasal flaring, head bobbing	Stridor, grunting, tripod positioning, severe retractions, apnea
Circulation to skin	Color and perfusion of skin and mucous membranes	Pallor, mottling, cyanosis

Any one abnormal side of the triangle mandates a rapid hands-on ABCDE assessment; two or more abnormal sides suggest impending or actual cardiorespiratory failure.

Systematic ABCDE Assessment

Once the PAT flags a concern, move to a structured, hands-on primary survey. Each step is addressed and stabilized before moving to the next.

Table 2.3 ABCDE Primary Survey in the Pediatric Patient

Step	Key elements	Immediate interventions if abnormal
Airway	Patency, positioning, secretions, stridor, foreign body	Airway repositioning, suction, airway adjuncts, FBAO maneuvers (Section 12)
Breathing	Rate, effort, air entry, chest rise, SpO2, auscultation	Supplemental oxygen, bag-mask ventilation, treat bronchospasm/pneumothorax
Circulation	Heart rate, pulses (central vs peripheral), capillary refill, blood	IV/IO access, fluid bolus, vasoactive support, hemorrhage control

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Step	Key elements	Immediate interventions if abnormal
	pressure, skin temperature/color, urine output	
Disability	AVPU or GCS, pupils, point-of-care glucose, focal deficits	Treat hypoglycemia, seizures; protect airway if obtunded
Exposure	Full skin exam, temperature, signs of trauma or rash, weight (or Broselow length)	Treat hyper/hypothermia, identify occult hemorrhage or non-accidental trauma

Red Flags for Impending Arrest

Bradycardia in a sick infant or child is a pre-arrest rhythm, not a benign finding — it almost always reflects hypoxia and demands immediate airway and oxygenation intervention.

A silent chest in a child with known severe respiratory distress is more ominous than audible wheeze — it may mean too little air movement to generate sound.

Normal blood pressure does not exclude shock. Children compensate with tachycardia and vasoconstriction until very late; hypotension is a pre-arrest sign, not an early one.

5 High-Quality Pediatric CPR — Technical Parameters

Compression quality is the single largest modifiable determinant of survival in cardiac arrest, and the 2025 guidelines sharpened several pediatric-specific technical points, most notably eliminating the two-finger technique for infant compressions.

Table 2.4 Compression Technique by Age Group

Age group	Rate	Depth	Hand position/technique	Compression-to-ventilation ratio (no advanced airway)
Infant (<1 year)	100-120/min	At least 1/3 AP chest diameter (~4 cm)	2-thumb encircling-hands technique preferred (2 rescuers); 1-hand heel-of-hand technique acceptable for a lone rescuer who cannot achieve adequate depth with thumbs. Two-finger technique is no longer taught.	30:2 single rescuer; 15:2 with 2 rescuers
Child, pre-pubertal (1 year to puberty)	100-120/min	At least 1/3 AP chest diameter (~5 cm)	1-hand (small child) or 2-hand heel-of-hand technique on	30:2 single rescuer; 15:2 with 2 rescuers

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Age group	Rate	Depth	Hand position/technique	Compression-to-ventilation ratio (no advanced airway)
			the lower half of the sternum	
Adolescent, post-pubertal	100-120/min	At least 5 cm, not exceeding 6 cm	2-hand technique, adult positioning	30:2 regardless of rescuer number (adult ratio applies)

With an advanced airway in place, compressions are continuous (no pauses for breaths) at 100-120/min, with ventilations delivered at 20-30 breaths/min (approximately 1 breath every 2-3 seconds) for infants and children — a substantially higher rate than the adult post-intubation ventilation rate, reflecting children's higher metabolic demand.

Myth vs Fact — Infant Compression Technique

Myth: The two-finger technique is still an acceptable single-rescuer option for infant CPR because it frees the rescuer's other hand to support the airway.

Fact: The 2025 AHA/AAP update eliminated the two-finger technique entirely because it produces shallower compressions and more rescuer fatigue than the alternatives. A lone rescuer now uses either the 2-thumb encircling-hands technique or, if depth cannot be achieved that way, the 1-hand heel-of-hand technique.

Compression Quality Non-Negotiables

Rate 100-120/min for every pediatric age group — do not slow down for a smaller patient.

Push hard enough to depress at least one-third the AP chest diameter (~4 cm infant, ~5 cm child) and allow full chest recoil between compressions.

Minimize interruptions — keep any pause in compressions under 10 seconds, including for rhythm checks and pulse checks.

Rotate the compressor every 2 minutes (or sooner) to prevent fatigue-related decay in compression quality, even if the rescuer does not feel tired.

6 Pediatric BLS Sequence

The pediatric basic life support sequence follows the same C-A-B (compressions-airway-breathing) logic as adult BLS, with modifications that reflect smaller anatomy and the predominantly respiratory etiology of pediatric arrest.

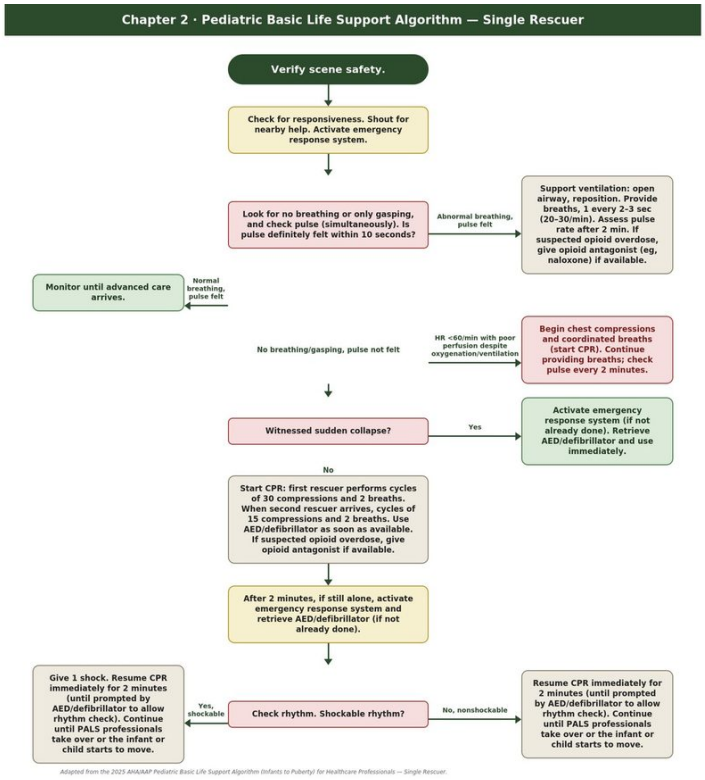
- Confirm scene safety, then check responsiveness and simultaneously look for breathing and assess for a pulse (brachial in infants, carotid or femoral in children) for no more than 10 seconds.
- For a lone healthcare-provider rescuer who witnesses a sudden collapse, activate the emergency response system and retrieve a defibrillator/AED before starting CPR, as in an adult (sudden witnessed collapse is more likely to be a primary shockable arrhythmia).
- For an unwitnessed pediatric collapse, or when working alone in the more typical asphyxial-arrest scenario, give 2 minutes of CPR first, then activate the emergency response system and retrieve the defibrillator ("phone fast," not "phone first").

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- If no pulse, or pulse <60/min with signs of poor perfusion despite adequate oxygenation and ventilation, begin chest compressions immediately.
- Open the airway with a head-tilt/chin-lift (or jaw-thrust if trauma is suspected) and deliver rescue breaths with visible chest rise, avoiding hyperventilation.
- As soon as a second rescuer and an AED/defibrillator arrive, attach pads (using pediatric attenuator pads/dose for infants and children under about 25 kg or 8 years old when available) and follow the device's shock/no-shock prompts without interrupting compressions any longer than necessary.

> **Busy-Shift Tip**
>
> If you are the lone rescuer for a collapsed child on a general ward, do not leave the bedside to find help before starting CPR. Call out or use the call bell/code button while beginning compressions — get 2 minutes of CPR running before anyone leaves the room to activate the full team, unless the collapse was sudden and witnessed.

Figure 2.0. Adapted from the 2025 AHA/AAP Pediatric Basic Life Support Algorithm (Infants to Puberty) for Healthcare Professionals — Single Rescuer.



7 Pediatric Cardiac Arrest Algorithm

Once pulseless arrest is confirmed, management branches on the rhythm. Shockable rhythms (ventricular fibrillation and pulseless ventricular tachycardia) are managed with a shock-drug-shock cycle; non-shockable rhythms (asystole and pulseless electrical activity) are managed with early, repeated epinephrine and aggressive search for reversible causes. High-quality CPR with minimal interruption underlies both pathways throughout.

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ALGORITHM — PEDIATRIC CARDIAC ARREST ALGORITHM

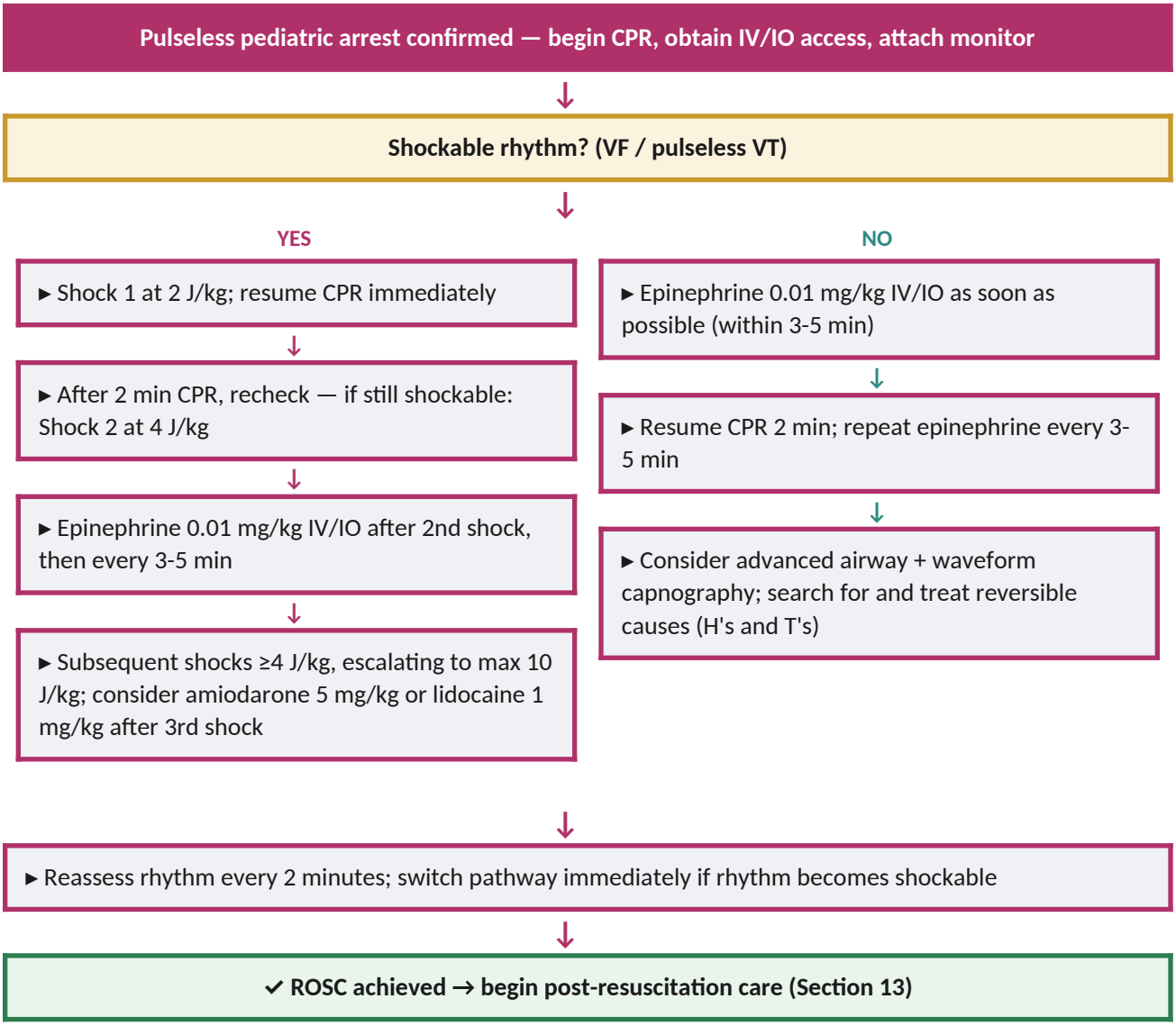
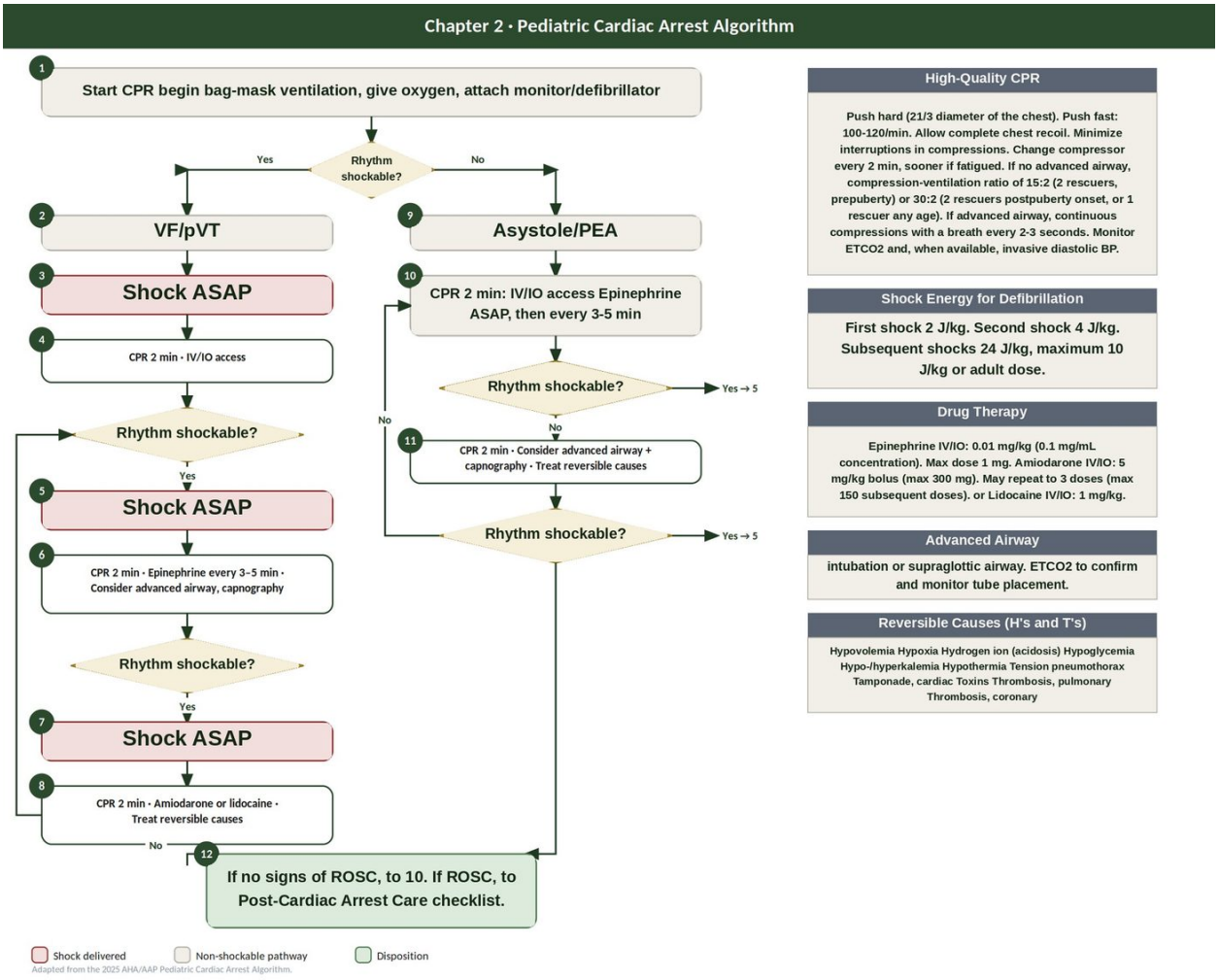


Figure 2.1. Adapted from the 2025 AHA/AAP Pediatric Cardiac Arrest Algorithm; see Table 2.5 for the corresponding step table.

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Decision Point — Shockable or Not?

Rhythm check every 2 minutes, pausing compressions for the shortest time possible. VF or pulseless VT on the monitor: shock immediately, then resume CPR for 2 minutes before the next rhythm check. Asystole or an organized rhythm without a palpable pulse (PEA): do not shock — resume CPR immediately and give epinephrine as soon as possible, ideally within the first 3-5 minutes of the arrest, then every 3-5 minutes thereafter.

Table 2.5 Pediatric Cardiac Arrest Algorithm — Key Steps

Step	Non-shockable (asystole/PEA)	Shockable (VF/pulseless VT)
1	Start/continue CPR; obtain IV/IO access	Start/continue CPR; obtain IV/IO access
2	Give epinephrine 0.01 mg/kg IV/IO as soon as possible (within 3-5 min)	Deliver first shock at 2 J/kg; resume CPR immediately
3	Resume CPR for 2 min; repeat epinephrine every 3-5 min	After 2 min CPR, recheck rhythm; if still shockable, give second shock at 4 J/kg
4	Consider advanced airway and	Give epinephrine 0.01 mg/kg IV/IO

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Step	Non-shockable (asystole/PEA)	Shockable (VF/pulseless VT)
	waveform capnography; search for and treat reversible causes (Hs and Ts)	after the second shock, then every 3-5 min; consider amiodarone 5 mg/kg or lidocaine 1 mg/kg after the third shock
5	Reassess rhythm every 2 min; switch pathway immediately if rhythm becomes shockable	Subsequent shocks at ≥4 J/kg, escalating as needed to a maximum of 10 J/kg or the adult maximum dose, whichever is lower; continue searching for reversible causes

Reversible causes (Hs and Ts): Hypovolemia, Hypoxia, Hydrogen ion (acidosis), Hypo-/Hyperkalemia, Hypothermia, Hypoglycemia; Tension pneumothorax, Tamponade (cardiac), Toxins, Thrombosis (pulmonary/coronary).

8 Weight-Based Drug Dosing

Every resuscitation drug in pediatrics is dosed by weight, so an accurate or well-estimated weight must be obtained in seconds, not minutes. A length-based resuscitation tape (e.g., Broselow) that cross-references length to weight and pre-calculated doses is the preferred method when a stated weight is unavailable or unreliable; verbal parental report of weight is acceptable when the child is known to the family and reasonably recent.

Table 2.6 Weight-Based Resuscitation Drug Dosing

Drug	Indication	Dose	Maximum single dose	Notes
Epinephrine (IV/IO)	Cardiac arrest (all rhythms); symptomatic bradycardia	0.01 mg/kg of 0.1 mg/mL (1:10,000) concentration	1 mg	Repeat every 3-5 min during arrest
Epinephrine (ET, if no IV/IO access)	Cardiac arrest, airway already secured	0.1 mg/kg of 1 mg/mL (1:1,000) concentration	2.5 mg	Ten-fold higher dose than IV/IO; obtain IV/IO access as soon as possible and switch routes
Amiodarone	Shock-refractory VF/pulseless VT	5 mg/kg IV/IO bolus	300 mg per dose	May repeat up to 2 additional times during arrest (max ~15 mg/kg/day)
Lidocaine	Shock-refractory VF/pulseless VT (alternative to amiodarone)	1 mg/kg IV/IO loading dose	Per weight, no fixed cap in arrest dosing	Follow with infusion 20-50 mcg/kg/min if ROSC achieved
Adenosine	Stable or unstable SVT (first-line)	0.1 mg/kg rapid IV/IO push, followed immediately by rapid saline flush	6 mg (first dose)	Second dose 0.2 mg/kg, max 12 mg, if first dose ineffective

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Drug	Indication	Dose	Maximum single dose	Notes
Atropine	Symptomatic bradycardia from increased vagal tone or primary AV block; pre-medication for certain intubations	0.02 mg/kg IV/IO	0.5 mg (child), 1 mg (adolescent)	Minimum single dose 0.1 mg regardless of calculated weight-based dose; may repeat once

Confirm concentrations before drawing up epinephrine — 1:10,000 (IV/IO) and 1:1,000 (ET/IM) are not interchangeable and a tenfold dosing error is a recognized, serious medication error in pediatric resuscitation.

9 Defibrillation and Cardioversion Energy Dosing

Energy is dosed in joules per kilogram (J/kg) and escalates with repeated attempts. Pediatric attenuator pads/cables reduce the delivered energy for AEDs used on infants and small children and should be used whenever available for patients under approximately 25 kg or 8 years of age; if unavailable, use standard adult pads/energy rather than withholding defibrillation.

Table 2.7 Defibrillation and Synchronized Cardioversion Energy Dosing

Indication	First attempt	Subsequent attempts	Maximum
Defibrillation (VF/pulseless VT)	2 J/kg	4 J/kg for the second shock; ≥4 J/kg thereafter, escalating as needed	10 J/kg or the adult maximum single dose, whichever is lower
Synchronized cardioversion (unstable SVT or unstable ventricular tachycardia with a pulse)	0.5-1 J/kg	Increase to 2 J/kg if the first attempt is unsuccessful	2 J/kg (may reasonably increase further per expert/toxicology-cardiology consultation in refractory cases)

Always sedate/analgese an unstable-but-conscious patient before synchronized cardioversion if doing so will not meaningfully delay treatment.

10 Bradycardia and Tachycardia Algorithms

Arrhythmia management in children hinges on two questions asked in sequence: is the patient stable or unstable (adequate versus inadequate perfusion, mental status, and blood pressure), and — for tachycardia — is the QRS narrow or wide? Bradycardia in a child is treated aggressively because it is so often a pre-arrest hypoxic rhythm rather than a primary conduction problem.

Table 2.8 Pediatric Bradycardia Algorithm

Clinical picture	First-line action	If cardiopulmonary compromise persists	Drug therapy
HR <60/min with poor perfusion, despite adequate oxygenation and ventilation	Start chest compressions	Continue CPR; identify and treat underlying cause (hypoxia is the most common)	Epinephrine 0.01 mg/kg IV/IO every 3-5 min; atropine 0.02 mg/kg if increased vagal tone or primary AV block is suspected

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Clinical picture	First-line action	If cardiopulmonary compromise persists	Drug therapy
Bradycardia with adequate perfusion	Support airway/breathing; give oxygen; obtain monitor/12-lead and IV/IO access	Treat reversible causes (hypoxia, hypothermia, heart block, toxin/drug effect, increased ICP)	Consider transcutaneous or transvenous pacing for symptomatic bradycardia due to complete heart block or sinus node dysfunction unresponsive to medications

Table 2.9 Pediatric Tachycardia Algorithm

QRS width	Stable	Unstable (poor perfusion, hypotension, altered mental status)
Narrow (≤0.09 s) — usually SVT	Attempt vagal maneuvers (ice to face in infants, Valsalva in older children) while preparing adenosine; give adenosine 0.1 mg/kg (max 6 mg), then 0.2 mg/kg (max 12 mg) if needed	Synchronized cardioversion 0.5-1 J/kg (increase to 2 J/kg if unsuccessful); may attempt adenosine first only if IV access is immediately available and it will not delay cardioversion
Wide (>0.09 s) — consider VT until proven otherwise	Expert consultation; consider adenosine only if rhythm is regular and monomorphic (may represent SVT with aberrancy); consider amiodarone or procainamide for confirmed VT	Synchronized cardioversion 0.5-1 J/kg (increase to 2 J/kg if unsuccessful); treat as pulseless arrest and defibrillate if the patient loses a pulse at any point

Practice Pearls — Arrhythmias

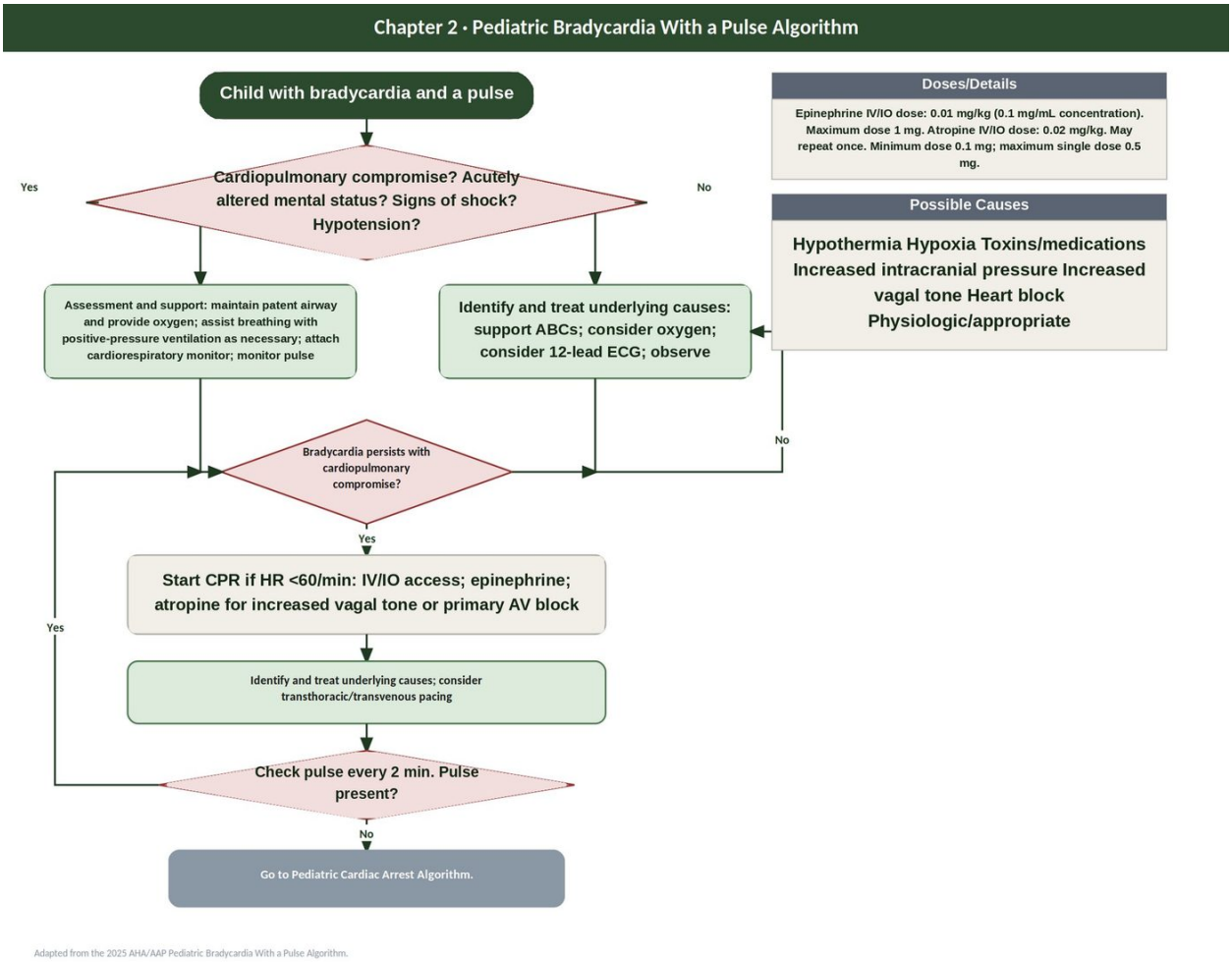
Adenosine has an extremely short half-life; push it as close to the IV/IO hub as possible, immediately followed by a rapid saline flush, ideally through the largest, most proximal vein available.

A narrow-complex tachycardia with a rate that varies beat-to-beat and P waves visible before every QRS is far more likely to be sinus tachycardia from fever, pain, or hypovolemia than SVT — treat the underlying cause, not the rate, in that scenario.

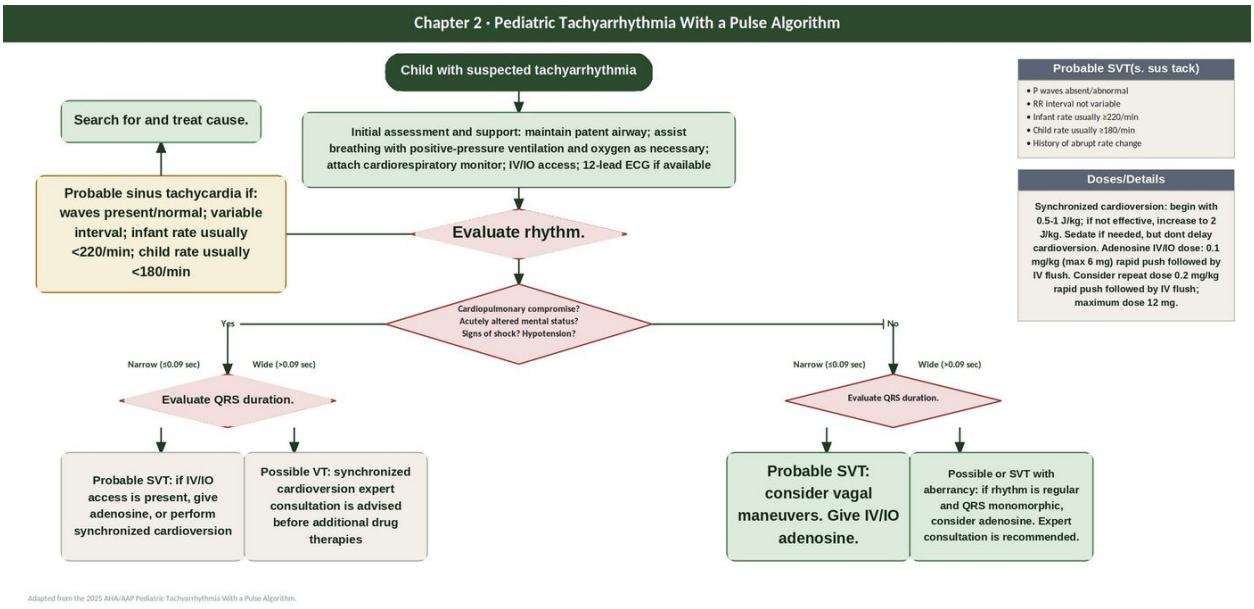
Do not delay synchronized cardioversion in an unstable child to place additional lines or complete a full history — instability defines the urgency.

Adapted from the 2025 AHA/AAP Pediatric Bradycardia With a Pulse Algorithm.

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Adapted from the 2025 AHA/AAP Pediatric Tachyarrhythmia With a Pulse Algorithm.



11 Shock Recognition and Fluid Resuscitation

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Shock is a state of inadequate tissue perfusion to meet metabolic demand. Because children compensate for falling perfusion far more effectively than adults — through tachycardia and peripheral vasoconstriction — blood pressure is preserved until late, and a normal blood pressure must never be used alone to exclude shock.

Table 2.10 Compensated vs Decompensated Shock and Fluid Management

Feature	Compensated shock	Decompensated shock
Blood pressure	Normal for age	Hypotensive for age — a late and ominous finding
Heart rate	Tachycardic	Tachycardic, may become bradycardic pre-arrest
Capillary refill	Delayed (>2 seconds)	Markedly delayed or absent
Mental status	Normal to mildly irritable/anxious	Lethargic, obtunded, unresponsive
Peripheral pulses	Present but may be weak/thready distally	Weak or absent peripherally, weak centrally
Initial fluid bolus	10-20 mL/kg isotonic crystalloid (balanced solution or normal saline) over 5-20 min; reassess after every bolus	Same initial bolus, given as rapidly as the clinical picture and access allow; reassess after every bolus and escalate to vasoactive support if shock persists after 40-60 mL/kg
Caution	Use smaller, more frequent boluses (10 mL/kg) with reassessment for fluid overload if cardiogenic shock, myocarditis, or severe malnutrition is suspected	Same caution applies; do not withhold fluid in hypovolemic/septic shock for fear of overload, but reassess after every bolus for hepatomegaly, rales, and worsening work of breathing

Reassess perfusion, mental status, and signs of fluid overload after every bolus — fluid resuscitation is titrated to response, not given as a single fixed volume.

Red Flags for Decompensated Shock

Any measured hypotension for age in a child previously thought to be only mildly unwell — treat as decompensated shock and escalate immediately.

New lethargy or inability to recognize a caregiver in a child with tachycardia and poor perfusion.

Mottled or gray skin color, especially with cool extremities extending proximally toward the trunk.

12 Pediatric Foreign-Body Airway Obstruction

Choking management differs by age because of infants' fragile abdominal organs and the mechanics of expelling an object from a small airway. The 2025 guidelines added back blows to the child/adult choking sequence, which previously relied on abdominal thrusts alone.

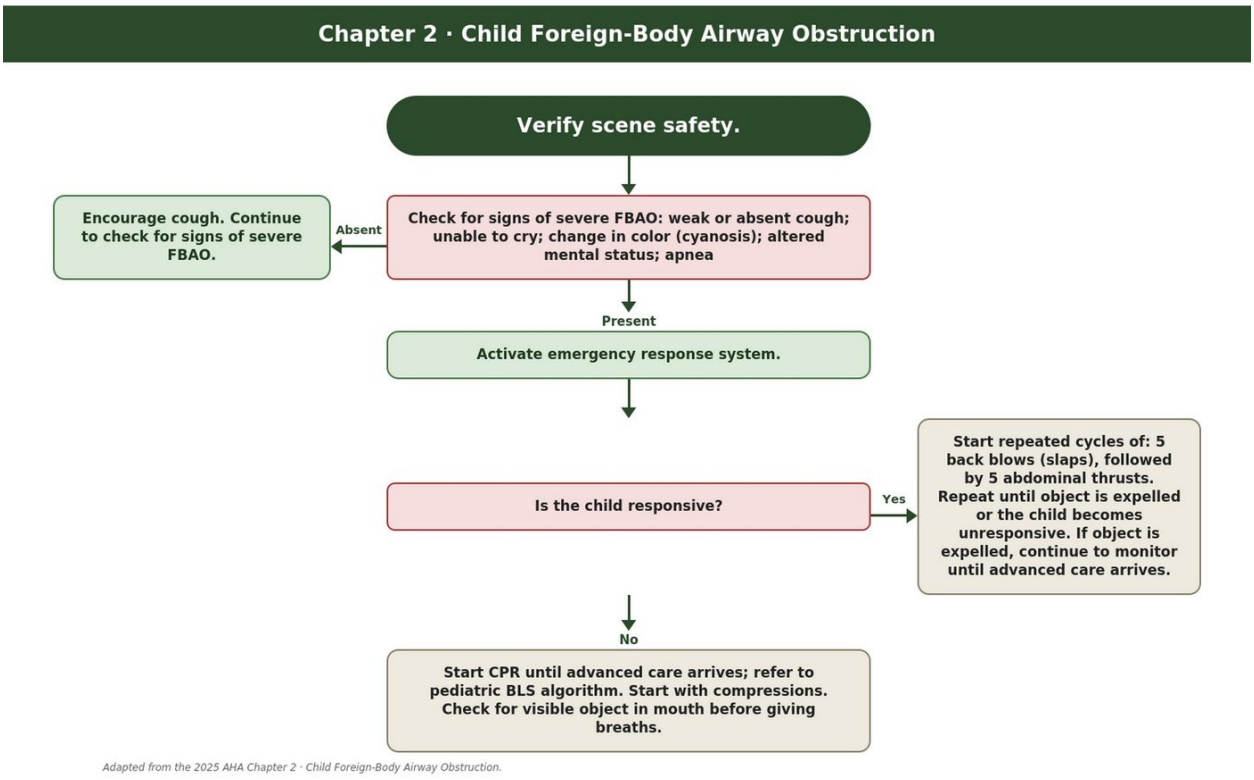
Table 2.11 Foreign-Body Airway Obstruction Management

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Age group	Conscious/responsive obstruction	If the patient becomes unresponsive
Infant (<1 year)	Alternate cycles of 5 back blows (with infant prone, head lower than trunk, supported on rescuer's forearm) and 5 chest thrusts (infant supine, two fingers on lower sternum)	Begin CPR; before each set of breaths, open the mouth and remove a visible object only if one can be seen (no blind finger sweeps)
Child (≥1 year) and adult	Alternate cycles of 5 back blows (between the shoulder blades) and 5 abdominal thrusts (Heimlich maneuver, fist above the umbilicus)	Begin CPR; before each set of breaths, open the mouth and remove a visible object only if one can be seen (no blind finger sweeps)

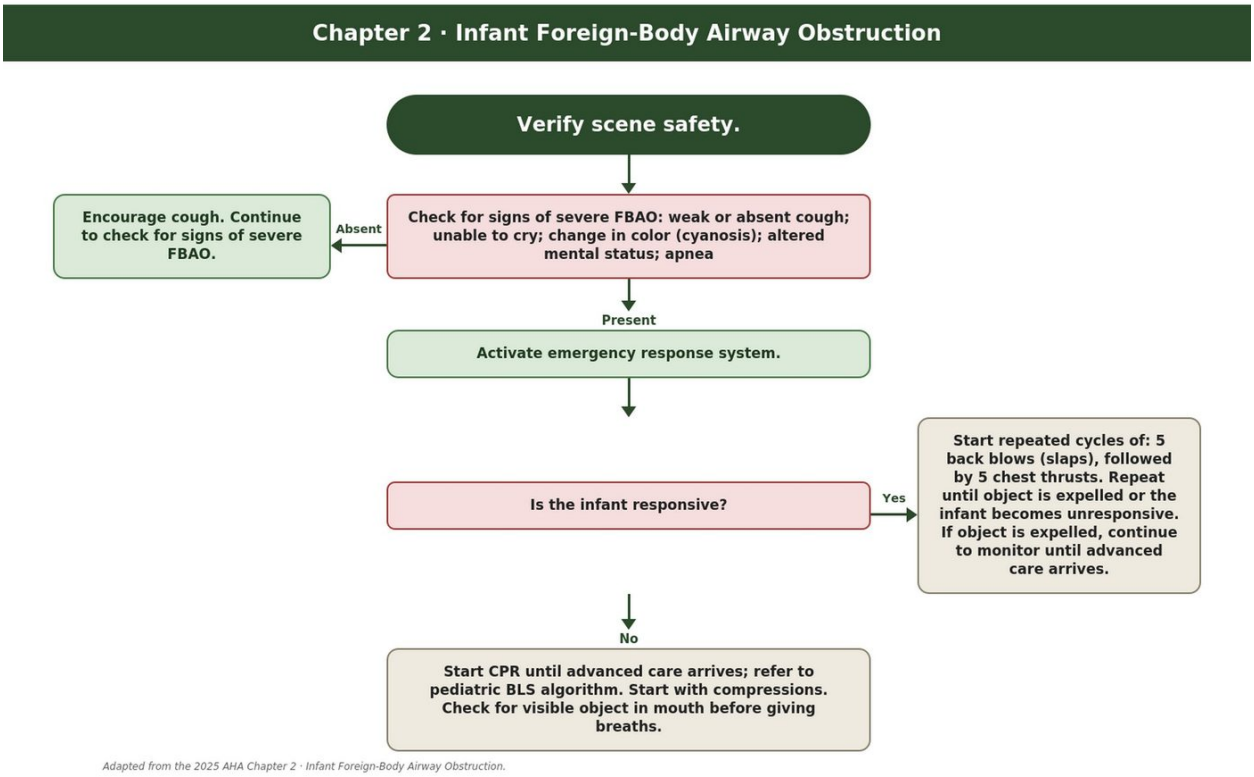
Abdominal thrusts are not used in infants under 1 year because of the risk of solid-organ injury. In visibly pregnant or very obese patients, use chest thrusts instead of abdominal thrusts regardless of age.

Adapted from the 2025 AHA/AAP Child Foreign-Body Airway Obstruction Algorithm.



Adapted from the 2025 AHA/AAP Infant Foreign-Body Airway Obstruction Algorithm.

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13 Post-Resuscitation Care

Care in the minutes to hours after ROSC determines much of the eventual neurologic outcome. The guiding principle is to avoid every secondary insult — hypoxia, hyperoxia, hypotension, hypercapnia, hypoglycemia, and fever — while the brain and myocardium recover from the arrest.

Table 2.12 Post-ROSC Targets

Parameter	Target	Rationale/notes
Oxygenation	Titrate FiO2 to SpO2 94-99%	Avoid both hypoxia and unnecessary hyperoxia; wean supplemental oxygen once the target range is reliably achieved
Ventilation	Normocapnia for the patient's baseline	Avoid routine hyperventilation, which reduces cerebral blood flow
Blood pressure	Maintain systolic BP and MAP above the 5th percentile for age; use fluids and vasoactive infusions as needed	Hypotension after ROSC is associated with worse neurologic outcome
Diastolic blood pressure during CPR (if arterial line in place)	≥25 mmHg in infants; ≥30 mmHg in children	Used intra-arrest to titrate compression quality/vasopressor timing, not a post-ROSC target per se, but reflects the same perfusion-pressure principle

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Parameter	Target	Rationale/notes
Temperature	Continuous temperature monitoring; target 32-37.5°C for at least 36 hours in comatose patients; actively avoid and treat fever (temperature >37.5°C) in all post-arrest patients	Hyperthermia worsens neurologic injury after arrest; targeted temperature management is preferred over unmanaged normothermia in comatose survivors
Glucose	Monitor closely; treat hypoglycemia promptly and avoid marked hyperglycemia	Both extremes are associated with worse outcomes
Neurologic monitoring	EEG monitoring for seizures where available; formal neuroprognostication deferred to a defined multimodal assessment, not made acutely	2025 guidelines added the first formal recommendations for predicting neurologic outcome after pediatric arrest

Post-ROSC Monitoring Checklist

Continuous cardiac and SpO2 monitoring with a low threshold to add continuous or serial blood pressure monitoring, ideally arterial line if the child remains unstable.

Continuous core temperature monitoring — do not rely on intermittent spot checks, which will miss a rising trend toward fever.

Serial neurologic exams and point-of-care glucose until the patient is stable and metabolically normalized.

12-lead ECG and directed workup for the arrest etiology (echocardiogram, chest imaging, toxicology, electrolytes) once the patient is stabilized enough to tolerate it.

Early, structured communication with family, including realistic framing of prognosis once available, and early PICU/critical-care involvement.

14 Worked Clinical Case

Case 2.1 — Septic Shock Progressing to Pulseless Electrical Activity

Presentation: A 7-month-old, weight approximately 8 kg, is brought to the emergency department with 2 days of fever and decreased oral intake. On arrival the Pediatric Assessment Triangle is abnormal in all three components: the infant is lethargic and difficult to console, has moderate work of breathing, and has mottled, cool skin to the knees. Heart rate is 190/min, capillary refill is 4 seconds, and blood pressure is 78/50 mmHg (within the low-normal range for age).

Management: The team recognizes compensated shock despite the 'normal' blood pressure, given the tachycardia, delayed capillary refill, and mottling. IV/IO access is obtained; a 20 mL/kg (160 mL) isotonic crystalloid bolus is given over 15 minutes and blood cultures plus empiric broad-spectrum antibiotics are administered promptly. After the first bolus, perfusion has not improved and the infant is now less responsive; a second 20 mL/kg bolus is given while a vasoactive infusion is prepared. Ten minutes later the infant becomes unresponsive and apneic. A pulse check reveals no palpable pulse; the monitor shows

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an organized narrow-complex rhythm at 60/min — pulseless electrical activity. CPR is started immediately (2-thumb encircling-hands technique, rate 100-120/min, depth ~4 cm, 15:2 with two rescuers), and epinephrine 0.08 mg (0.01 mg/kg for 8 kg) IV/IO is given within the first 3 minutes of the arrest and repeated every 3-5 minutes. Bag-mask ventilation is optimized and reversible causes are systematically checked; profound hypovolemia from septic shock is identified as the leading cause, and fluid resuscitation continues alongside compressions.

Outcome/teaching point: ROSC is achieved after 8 minutes of CPR and 2 doses of epinephrine. Post-ROSC care targets SpO2 94-99%, normocapnia, blood pressure support with a norepinephrine infusion, continuous temperature monitoring with active fever avoidance, and PICU admission. The case illustrates the central PALS lesson: this arrest was predictable from the moment the Pediatric Assessment Triangle was abnormal, and a normal-appearing blood pressure early on nearly masked decompensating shock. Aggressive, reassessed fluid resuscitation and early antibiotics were the interventions most likely to have prevented the arrest; once arrest occurred, early recognition of the non-shockable rhythm and prompt, correctly dosed epinephrine were what enabled ROSC.

15 Chapter Quick-Reference Summary

The table below consolidates the numbers a resuscitation team needs at the bedside in the first minutes of a pediatric emergency.

Table 2.13 PALS Quick-Reference Summary

Parameter	Infant (<1 year)	Child (1 year-puberty)	Post-pubertal/ adolescent
Compression rate	100-120/min	100-120/min	100-120/min
Compression depth	~4 cm (1/3 AP diameter)	~5 cm (1/3 AP diameter)	≥5 cm, not exceeding 6 cm
Compression technique	2-thumb encircling-hands (preferred) or 1-hand heel-of-hand; 2-finger technique no longer taught	1-hand or 2-hand heel-of-hand	2-hand, adult technique
Compression:ventilation ratio (no advanced airway)	30:2 (1 rescuer); 15:2 (2 rescuers)	30:2 (1 rescuer); 15:2 (2 rescuers)	30:2 regardless of rescuer number
Ventilation rate with advanced airway	20-30/min (continuous compressions)	20-30/min (continuous compressions)	10/min (adult rate applies)
Epinephrine (arrest/bradycardia)	0.01 mg/kg IV/IO (max 1 mg), every 3-5 min	0.01 mg/kg IV/IO (max 1 mg), every 3-5 min	0.01 mg/kg IV/IO (max 1 mg), every 3-5 min
Defibrillation energy	2 J/kg → 4 J/kg → ≥4 J/kg (max 10 J/kg or adult dose)	2 J/kg → 4 J/kg → ≥4 J/kg (max 10 J/kg or adult dose)	2 J/kg → 4 J/kg → ≥4 J/kg (max 10 J/kg or adult dose)
Synchronized cardioversion	0.5-1 J/kg, then 2 J/kg	0.5-1 J/kg, then 2 J/kg	0.5-1 J/kg, then 2 J/kg
Adenosine	0.1 mg/kg (max 6 mg),	0.1 mg/kg (max 6 mg),	0.1 mg/kg (max 6 mg),

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Parameter	Infant (<1 year)	Child (1 year-puberty)	Post-pubertal/ adolescent
	then 0.2 mg/kg (max 12 mg)	then 0.2 mg/kg (max 12 mg)	then 0.2 mg/kg (max 12 mg)
Fluid bolus (shock)	10-20 mL/kg isotonic crystalloid, reassess and repeat	10-20 mL/kg isotonic crystalloid, reassess and repeat	10-20 mL/kg isotonic crystalloid, reassess and repeat
DBP target during CPR (arterial line)	≥25 mmHg	≥30 mmHg	≥30 mmHg
Post-ROSC temperature	32-37.5°C target; avoid/treat fever >37.5°C	32-37.5°C target; avoid/treat fever >37.5°C	32-37.5°C target; avoid/treat fever >37.5°C
Post-ROSC SpO2 target	94-99%	94-99%	94-99%

All doses assume standard concentrations and should be cross-checked against a weight-based dosing reference (e.g., a length-based resuscitation tape) at the bedside.

Content in this chapter is based on the 2025 American Heart Association and American Academy of Pediatrics Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care (Circulation), including Part 6: Pediatric Basic Life Support and Part 8: Pediatric Advanced Life Support. This material is an educational reference only and is not a substitute for enrollment in, or certification through, an official AHA/AAP PALS provider course; always follow your institution's current resuscitation policies and the manufacturer guidance for your specific defibrillator/monitor.

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LIFE SUPPORT COURSES · CLINICAL COMPANION MANUAL

CHAPTER 3

ATLS

Advanced Trauma Life Support

1 Clinical Snapshot

Traumatic injury remains the leading cause of death for people ages 1 to 44 in the United States and a top-five cause of death across all ages worldwide. Advanced Trauma Life Support (ATLS) provides the internationally standardized, sequential approach that lets any clinician - regardless of specialty - identify and treat immediately life-threatening injuries in the order most likely to kill the patient first. The American College of Surgeons Committee on Trauma (ACS-COT) released the 11th edition of the ATLS course in the fall of 2025, and it is the current reference standard used throughout this chapter.

ATLS is not a diagnostic exercise. It is a resuscitation algorithm: identify a threat, treat it immediately, then move to the next step. The same primary survey sequence applies whether the patient arrives after a low-speed fall or a high-energy multi-system crash, which is precisely why it is teachable, repeatable, and auditable across trauma systems of every resource level.

Why this matters - Typical patient - Where you meet it

Why it matters: Preventable trauma death is concentrated in the first hour after injury, driven overwhelmingly by uncontrolled hemorrhage and airway loss. A disciplined primary survey catches these threats before they become unrecoverable.

Typical patient: Blunt polytrauma from motor vehicle collisions and falls dominates volume in most systems; penetrating trauma (gunshot, stab) makes up a smaller but disproportionately time-critical share requiring immediate hemorrhage control and, at times, resuscitative thoracotomy.

Common settings: Emergency department trauma bays, prehospital/EMS response, military and austere care, rural facilities awaiting transfer, and any inpatient unit where a patient acutely deteriorates after injury.

2 Learning Objectives

- Apply the ATLS xABCDE primary survey sequence to identify and treat immediately life-threatening injuries in priority order.
- Recognize field triage and trauma team activation criteria and describe the rationale for full versus partial activation.
- Select airway adjuncts and definitive airway techniques appropriate to the clinical scenario, including indications for surgical airway.
- Classify hemorrhagic shock by class (I-IV) and match initial fluid/blood product strategy to each class.
- Describe damage control resuscitation principles: permissive hypotension, balanced massive transfusion ratios, tranexamic acid timing and dosing, and avoidance of the lethal triad.
- Perform and interpret the FAST exam and identify appropriate adjuncts to the primary survey.

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- Conduct a systematic secondary survey and AMPLE history, and recognize tension pneumothorax, cardiac tamponade, flail chest, traumatic brain injury, and spinal trauma requiring immediate intervention.

3 Preparation, Triage, and Trauma Team Activation

Trauma outcomes are determined before the patient arrives. Preparation includes pre-arrival notification, assignment of team roles (team leader, airway, circulation/procedures, scribe, nursing), donning of personal protective equipment, warmed fluids and blood products staged, and confirmation that imaging, laboratory, and operating room resources are alerted for anticipated needs.

Field triage uses physiologic, anatomic, mechanism-of-injury, and special-consideration criteria (aligned with CDC Guidelines for Field Triage of Injured Patients) to identify patients who need transport to the highest level of trauma care available. Trauma team activation criteria mirror these categories and determine whether a full team (surgeon, anesthesia, multiple nurses) or a limited/partial team responds.

Table 3.1 Field Triage and Trauma Team Activation Criteria

Category	Example Criteria	Typical Response
Physiologic	GCS <14; systolic BP <90 mmHg; respiratory rate <10 or >29/min (<20 in infant); need for ventilatory support	Full trauma team activation
Anatomic	Penetrating injury to head, neck, torso, or proximal extremities; flail chest; two or more proximal long-bone fractures; crushed, degloved, mangled, or pulseless extremity; amputation proximal to wrist/ankle; pelvic fractures; open or depressed skull fracture; paralysis	Full trauma team activation
Mechanism of injury	High-risk auto crash (intrusion, ejection, death of occupant); falls from height (adults >6 m, children >3 m or 2-3x height); motorcycle crash >20 mph; auto vs. pedestrian/cyclist	Full or partial activation per local protocol
Special considerations	Age >65 years; anticoagulant/bleeding disorder; burns with associated trauma; pregnancy >20 weeks; EMS provider judgment; end-stage renal disease on dialysis	Partial activation; lower threshold for full activation

Adapted from CDC Guidelines for Field Triage of Injured Patients and ACS-COT resource criteria for trauma center designation.

4 Primary Survey - The xABCDE Approach

The ATLS 11th edition (2025) formally revised the primary survey mnemonic from ABCDE to xABCDE, elevating control of exsanguinating external hemorrhage - visible arterial spurting, pooling blood, or

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traumatic amputation - to the first priority step in select patients, ahead of airway. This change brings civilian trauma care into alignment with battlefield-derived <C>ABC / MARCH doctrine, which recognizes that a patient can die of external exsanguination faster than of airway compromise. When no exsanguinating external bleeding is present, the sequence proceeds through airway first as in prior editions.

ALGORITHM — PRIMARY SURVEY — THE XABCDE SEQUENCE

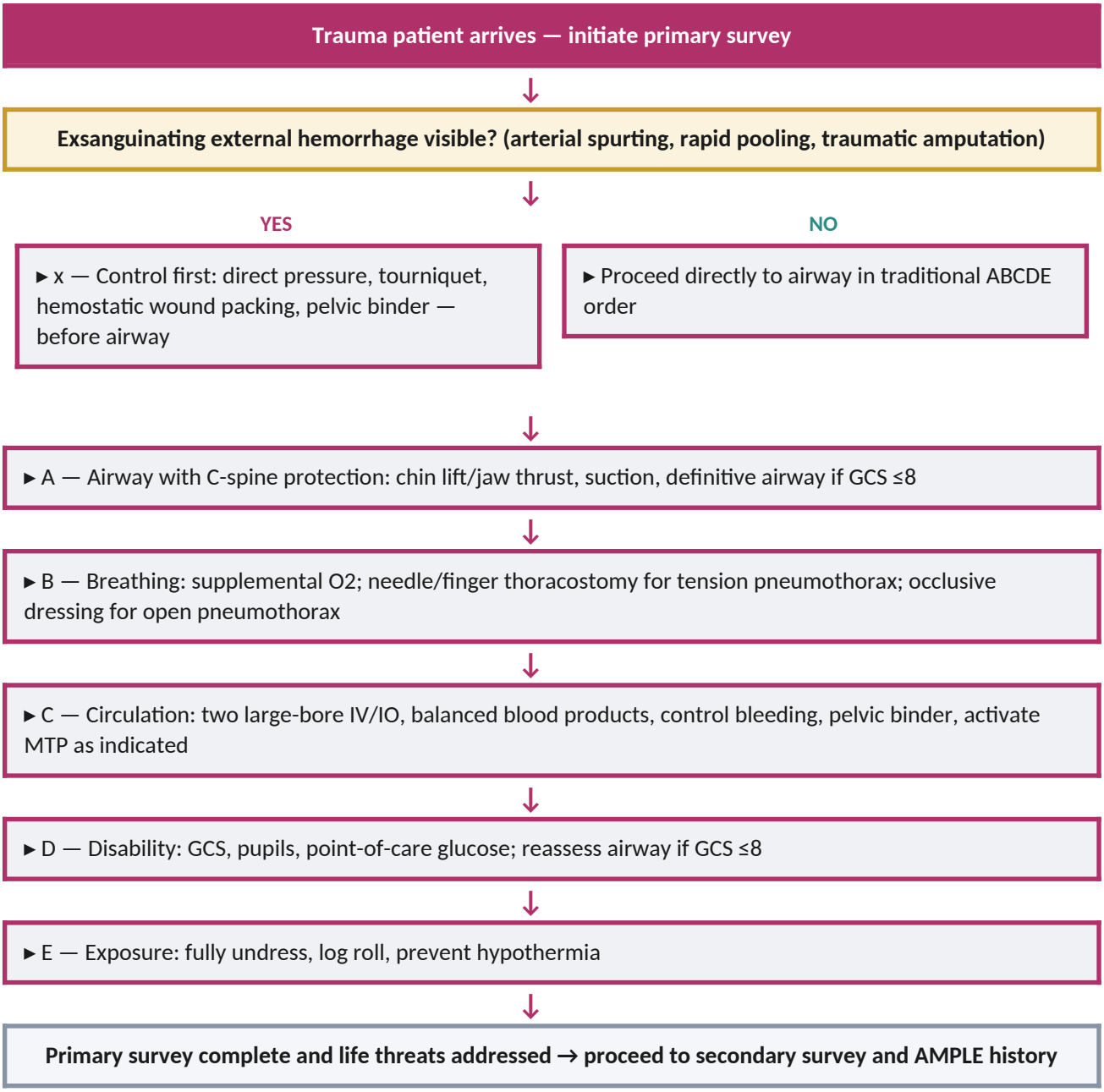


Figure 3.1. Reflects the ATLS 11th-edition (2025) xABCDE sequence; see Table 3.2 for assessment findings and interventions by step.

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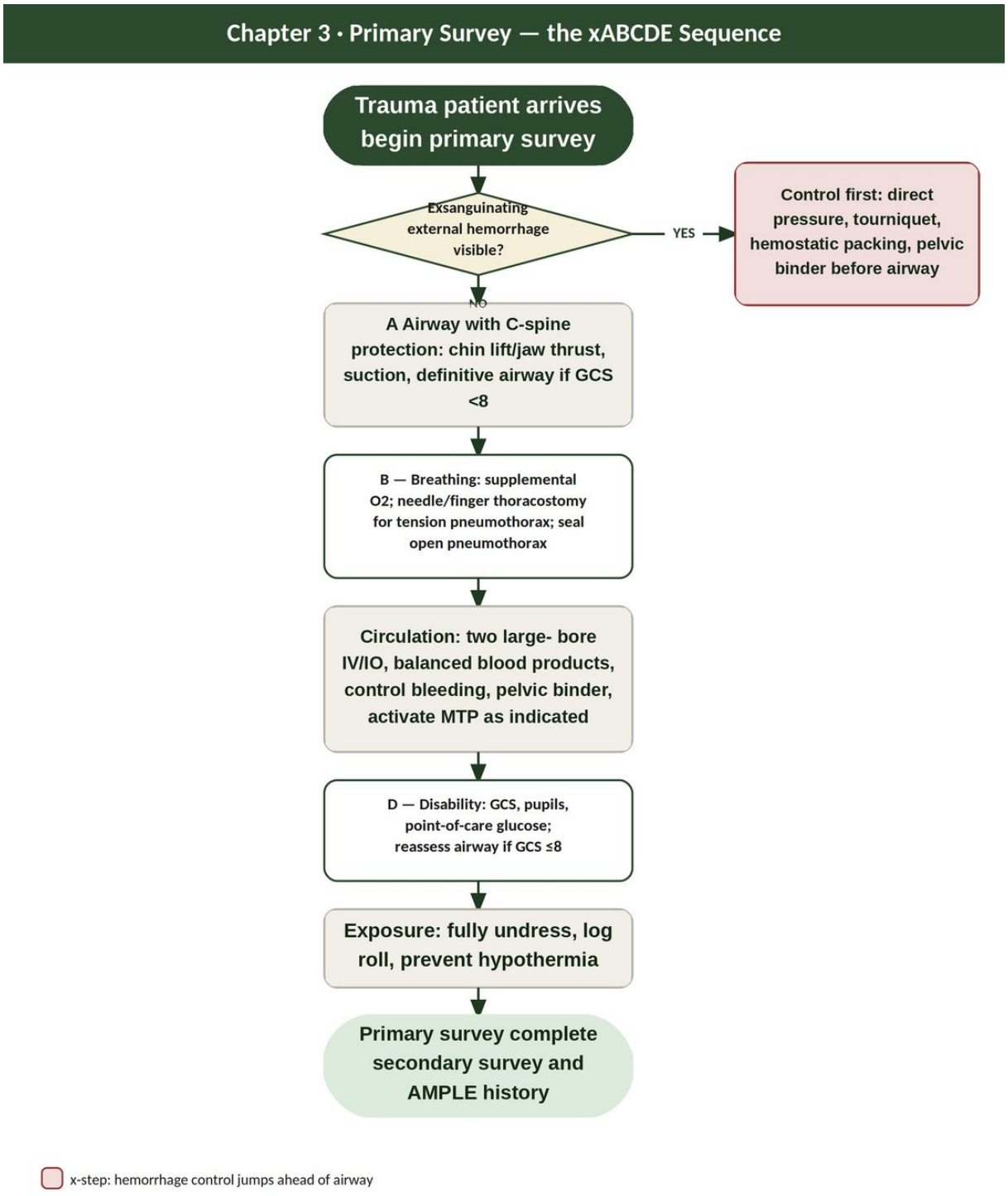


Table 3.2 Primary Survey: Assessment and Immediate Intervention by Step

Step	Assessment Focus	Immediate Intervention
x - Exsanguinating hemorrhage	Visible life-threatening external bleeding: arterial spurting, rapidly pooling blood, traumatic amputation	Direct pressure, tourniquet, wound packing with hemostatic gauze, pelvic binder - performed before airway management

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Step	Assessment Focus	Immediate Intervention
A - Airway with C-spine protection	Patency, stridor, secretions/blood, facial or laryngeal trauma, GCS <=8	Chin lift/jaw thrust, suction, oro-/nasopharyngeal airway, definitive airway (endotracheal tube or surgical airway) with manual in-line cervical stabilization
B - Breathing and ventilation	Respiratory rate and effort, symmetry, breath sounds, tracheal position, SpO2	Supplemental oxygen; needle or finger thoracostomy for tension pneumothorax; occlusive dressing for open pneumothorax; assisted ventilation
C - Circulation with hemorrhage control	Pulse rate/quality, skin color and temperature, capillary refill, blood pressure, source of external or internal bleeding	Two large-bore IV/IO lines, balanced blood product resuscitation, control external bleeding, pelvic binder, activate massive transfusion protocol as indicated
D - Disability (neurologic status)	Glasgow Coma Scale, pupillary size and reactivity, lateralizing signs, point-of-care glucose	Reassess airway if GCS <=8; treat hypoglycemia; avoid hypoxia and hypotension that worsen secondary brain injury
E - Exposure and environmental control	Complete undressing, log roll to inspect back, identify hidden injuries	Prevent hypothermia with warm blankets and warmed fluids while maintaining patient dignity and privacy

Source: ACS-COT Advanced Trauma Life Support, 11th edition (2025).

When does hemorrhage control jump ahead of airway?

Apply direct pressure or a tourniquet to visible exsanguinating hemorrhage before completing the airway assessment only when the bleeding is immediately life-threatening and external (arterial spurting, amputation, rapidly expanding pool).

If the patient has no visible exsanguinating bleeding, proceed through airway first as in the traditional sequence - the x-step does not replace airway priority, it precedes it only for this specific threat.

Reassess constantly: a tourniquet applied during the x-step does not end circulation management - definitive hemorrhage control (surgery, embolization, MTP) still follows in the C step.

5 Airway Management - Decision Points and Adjuncts

Indications for a definitive airway include apnea, inability to protect the airway (classically GCS <=8), inadequate oxygenation or ventilation despite basic adjuncts, impending airway compromise from an expanding neck hematoma or inhalation injury, and severe combativeness that precludes safe evaluation. ATLS 11 places new emphasis on correcting hemodynamic instability before intubation: hypotensive trauma patients are at high risk of peri-intubation cardiovascular collapse and arrest, so volume/blood resuscitation and vasopressor support should be optimized in parallel with airway preparation rather than rushing to rapid

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sequence induction in a patient who is already in shock. Video laryngoscopy is now the preferred first-attempt device where available, given higher first-pass success compared with direct laryngoscopy.

Table 3.3 Airway Adjuncts and Difficult-Airway Red Flags

Adjunct or Sign	Clinical Use	Key Notes
Jaw thrust / chin lift	First-line maneuver to relieve tongue-based obstruction	Use jaw thrust in suspected C-spine injury to avoid neck extension
Oropharyngeal airway	Unconscious patient without a gag reflex	Contraindicated if gag reflex present - risk of vomiting/aspiration
Nasopharyngeal airway	Semi-conscious patient with intact gag	Avoid with suspected basilar skull or midface fracture
Supraglottic device	Bridge to definitive airway when intubation is delayed or fails	Not a definitive airway - does not protect against aspiration
Video laryngoscopy	Preferred first-attempt intubation method	Higher first-pass success; keep direct laryngoscopy skills current for equipment failure
Surgical cricothyroidotomy	Cannot-intubate, cannot-oxygenate scenario	Definitive rescue airway; do not delay once CICO is declared

Cannot Intubate, Cannot Oxygenate

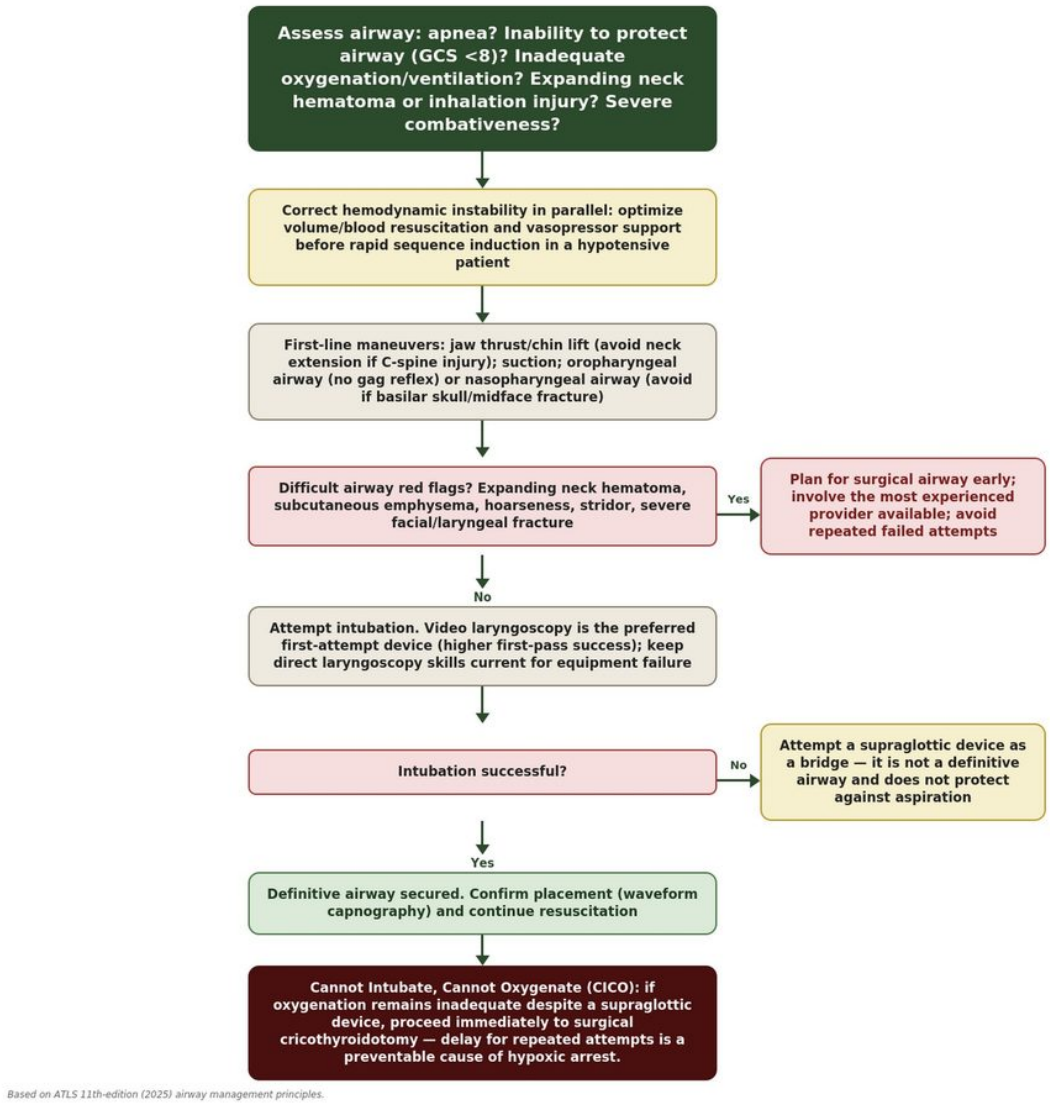
Expanding neck hematoma, subcutaneous emphysema, hoarseness, stridor, or severe facial/laryngeal fracture predict a difficult airway - plan for a surgical airway early rather than repeated failed attempts.

Once CICO is declared, proceed immediately to surgical cricothyroidotomy. Delay for repeated laryngoscopy attempts is a preventable cause of hypoxic arrest.

Based on ATLS 11th-edition (2025) airway management principles.

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Chapter 3 · Trauma Airway Management Decision Pathway



Based on ATLS 11th-edition (2025) airway management principles.

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6 Breathing and Ventilation - Immediate Chest Threats

A small set of chest injuries kill within minutes and must be actively excluded during the B step rather than waiting for imaging.

- Tension pneumothorax: hypotension, distended neck veins, tracheal deviation away from the affected side, absent breath sounds, hyperresonance - decompress immediately with needle or finger thoracostomy, then place a chest tube.
- Open pneumothorax: audible air movement through a chest wall wound - apply a three-sided occlusive dressing to create a one-way valve, then place a chest tube remote from the wound.
- Massive hemothorax: >1500 mL blood on initial chest tube output or ongoing output >200 mL/hr - resuscitate with blood products, consider autotransfusion, and involve surgery early as a thoracotomy trigger.
- Flail chest with pulmonary contusion: paradoxical chest wall motion from three or more contiguous ribs fractured in two or more places - the underlying contusion, not the flail segment itself, usually drives respiratory failure; manage with aggressive analgesia and pulmonary toilet.

7 Circulation and Hemorrhage Control

Hemorrhage is the leading cause of preventable death after injury. Control is pursued in parallel with resuscitation, not after it: external bleeding is addressed with direct methods, while internal or occult hemorrhage (chest, abdomen, pelvis, long bones, retroperitoneum) is presumed in any hypotensive trauma patient without an obvious external source and triggers rapid activation of surgical, interventional radiology, and massive transfusion resources.

Table 3.4 Hemorrhage Control by Bleeding Source

Bleeding Source	First-Line Control	Escalation
Extremity, arterial or venous	Direct pressure and hemostatic gauze packing	Windlass tourniquet placed 5-7 cm proximal to the wound; record application time
Junctional (groin, axilla, neck base)	Wound packing with direct pressure	Junctional tourniquet device where standard limb tourniquets cannot be applied
Pelvic fracture	Commercial pelvic binder positioned at the level of the greater trochanters	Angioembolization, preperitoneal pelvic packing, or REBOA (resuscitative endovascular balloon occlusion of the aorta)
Intra-abdominal (solid organ, mesenteric)	FAST-directed assessment, permissive hypotension en route to control	Damage control laparotomy
Long-bone fracture	Splinting or traction splint	Operative fixation once hemodynamically stabilized

Tourniquet discipline on a busy trauma bay

Write the application time directly on the tourniquet (skin marker or tape) the moment it is placed - this

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single habit prevents unnecessary limb ischemia and informs the surgical team's urgency.

Do not loosen or remove a prehospital tourniquet in the trauma bay to 'check' bleeding unless a surgeon is present and prepared to convert to definitive control - reperfusion of an uncontrolled source can precipitate rapid decompensation.

8 Disability and Exposure

The Glasgow Coma Scale (GCS) scores eye opening (4 points), verbal response (5 points), and motor response (6 points) for a total of 3 to 15. Severe traumatic brain injury is GCS <=8, moderate is 9-12, and mild is 13-15. Pupillary asymmetry, a fixed and dilated pupil, or lateralizing motor weakness suggests an expanding intracranial mass lesion requiring urgent neurosurgical evaluation and imaging. Check point-of-care glucose in any altered patient - hypoglycemia is a rapidly reversible mimic of head injury.

Exposure requires complete removal of clothing and a log roll to inspect the back, buttocks, and perineum for wounds that are otherwise missed. Exposure must be paired immediately with environmental control: a hypothermic trauma patient develops worsening coagulopathy that compounds ongoing hemorrhage, so warmed blankets, a warmed room, and warmed IV fluids/blood products are not optional comfort measures - they are part of hemorrhage control.

Disability and temperature - key numbers

GCS severe: 3-8 GCS moderate: 9-12 GCS mild: 13-15

Pupils: assess size, symmetry, and reactivity to light bilaterally before any sedating or paralytic agent is given whenever feasible

Hypothermia threshold: core temperature <35 degC significantly worsens coagulopathy and mortality in the actively bleeding trauma patient

9 Damage Control Resuscitation

Damage control resuscitation (DCR) is the coordinated strategy of limiting crystalloid, restoring blood volume with balanced blood products in ratios approximating whole blood, controlling hemorrhage as early as possible (including abbreviated 'damage control' surgery rather than definitive repair in the unstable patient), and actively preventing the lethal triad of hypothermia, acidosis, and coagulopathy - three processes that feed one another and, left unchecked, become irreversible.

Permissive hypotension accepts a lower-than-normal blood pressure target until the hemorrhage source is controlled, on the principle that aggressive normalization of blood pressure before surgical or angiographic control can dislodge early clot and accelerate blood loss. This target is deliberately abandoned in traumatic brain injury, where adequate cerebral perfusion pressure takes priority over permissive hypotension.

Table 3.5 Damage Control Resuscitation Parameters

Parameter	Target / Dose	Key Evidence or Note
Permissive hypotension (no TBI)	Systolic BP 80-90 mmHg, or a palpable radial pulse, until hemorrhage source is controlled	Avoids clot disruption from over-resuscitation; abandon once bleeding is surgically controlled

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Parameter	Target / Dose	Key Evidence or Note
Blood pressure target with concurrent TBI	Systolic BP >=100-110 mmHg (age-adjusted)	Cerebral perfusion pressure takes priority over permissive hypotension when TBI is present
Massive transfusion ratio	Packed red cells : plasma : platelets approximately 1:1:1, or low-titer O-negative whole blood where available	PROPPR randomized trial; balanced ratios reduce early death from hemorrhage versus crystalloid-heavy resuscitation
Tranexamic acid (TXA), general trauma hemorrhage	1 g IV bolus over 10 minutes, then 1 g infusion over 8 hours, given within 3 hours of injury	CRASH-2 trial; no mortality benefit and possible harm to give TXA beyond the 3-hour window
Tranexamic acid in traumatic brain injury	1 g bolus + 1 g infusion regimen; some protocols use a single 2 g bolus	CRASH-3 trial; benefit concentrated in mild-to-moderate TBI given early - confirm local protocol
Crystalloid volume	Minimize; roughly 1 L or less before transitioning to blood products in hemorrhagic shock	Excess crystalloid dilutes clotting factors and worsens coagulopathy and edema
Lethal triad targets	Core temp >35-36 degC, pH >7.2, ionized calcium normalized	Active warming and calcium replacement with each massive transfusion cycle; untreated triad is self-perpetuating

TXA dosing and timing per CRASH-2 (2010) and CRASH-3 (2019) trial protocols; MTP ratio per PROPPR (2015).

Crystalloid boluses in hemorrhagic shock

Myth: Every hypotensive trauma patient should receive a rapid 1-2 liter crystalloid bolus before blood products arrive.

Fact: Current damage control resuscitation minimizes crystalloid and moves directly to balanced blood products (or whole blood) as soon as they are available, because large-volume crystalloid dilutes clotting factors, worsens acidosis, and is an independent predictor of coagulopathy and death in major hemorrhage.

10 Hemorrhagic Shock Classification

The Class I-IV hemorrhagic shock classification estimates blood loss from vital signs and mental status to guide the intensity of initial fluid and blood product management. It remains a teaching framework rather than a precise measurement: compensatory mechanisms in young, fit patients or medications such as beta blockers can mask the expected tachycardic response, so clinical judgment and serial reassessment (including lactate and base deficit trends) should always accompany the table.

Table 3.6 Hemorrhagic Shock Classification (Class I-IV)

Parameter	Class I	Class II	Class III	Class IV
Blood loss (%)	<15%	15-30%	30-40%	>40%
Blood loss, 70 kg adult (mL)	<750	750-1500	1500-2000	>2000

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Parameter	Class I	Class II	Class III	Class IV
Heart rate (bpm)	<100	100-120	120-140	>140
Blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate (breaths/min)	14-20	20-30	30-40	>35
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Initial fluid/blood management	Crystalloid	Crystalloid; consider blood products	Blood products - consider MTP activation	Immediate massive transfusion protocol activation

Adapted from ACS-COT ATLS shock classification. Compensated physiology in young or athletic patients can mask Class III-IV losses; treat the trend, not a single reading.

Practice Pearls - shock class recognition

A young, healthy patient can maintain a near-normal blood pressure into Class III shock through peripheral vasoconstriction - narrowing pulse pressure and rising respiratory rate are earlier warning signs than falling systolic BP.

Patients on beta blockers or with a paced rhythm may not mount the expected tachycardia - do not let a 'normal' heart rate reassure you in a patient with a mechanism and exam consistent with major hemorrhage.

Serial lactate and base deficit trends often reveal ongoing occult shock before vital signs fully declare it.

11 FAST Exam and Adjuncts to the Primary Survey

The Focused Assessment with Sonography for Trauma (FAST) exam is a rapid bedside ultrasound performed during or immediately after the primary survey to detect free fluid (presumed blood) in the pericardial and peritoneal spaces. The extended FAST (eFAST) adds bilateral thoracic windows to detect pneumothorax and hemothorax. FAST is fast and repeatable but operator-dependent and cannot reliably exclude retroperitoneal or hollow viscus injury; a negative FAST in a persistently unstable patient does not exclude significant hemorrhage.

Table 3.7 FAST/eFAST Windows

Window	View Obtained	Positive Finding
Pericardial (subxiphoid)	Heart and pericardial sac	Pericardial effusion suggesting tamponade
Perihepatic (right upper quadrant)	Hepatorenal recess (Morison's pouch)	Free fluid - most sensitive abdominal window
Perisplenic (left upper quadrant)	Splenorenal recess	Free fluid
Pelvic (suprapubic)	Retrovesical / rectouterine pouch	Free fluid
Thoracic (eFAST extension)	Bilateral pleural line, lung sliding	Loss of lung sliding (pneumothorax) or effusion (hemothorax)

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Adjuncts to the primary survey - checklist

Chest radiograph and pelvis radiograph in the unstable blunt trauma patient

eFAST for pericardial fluid, intraperitoneal fluid, and pneumothorax/hemothorax

Type and crossmatch, hemoglobin, lactate, base deficit, coagulation studies, viscoelastic testing (TEG/ROTEM) where available

Urinary catheter and gastric tube unless contraindicated (e.g., suspected urethral injury, midface fracture)

Continuous cardiac monitoring, pulse oximetry, and end-tidal CO2 if intubated

12 Secondary Survey and AMPLE History

The secondary survey is a systematic head-to-toe examination performed only after the primary survey is complete and resuscitation is underway, with reassessment of the primary survey at any sign of deterioration. It combines a focused history (the AMPLE mnemonic) with a full physical exam, including a log roll to inspect the back and a rectal exam when indicated, to identify injuries that do not present as immediate threats to life but still require timely diagnosis and treatment.

Table 3.8 AMPLE History and Head-to-Toe Secondary Survey

Element	What to Assess
A - Allergies	Drug, environmental, and contrast allergies
M - Medications	Current medications, especially anticoagulants and antiplatelets
P - Past illness / Pregnancy	Comorbidities, prior surgeries, pregnancy status
L - Last meal	Timing of last oral intake, relevant to aspiration risk and anesthesia planning
E - Events / Environment	Mechanism of injury, hazardous exposures, events surrounding the injury
Head-to-toe regions	Scalp/skull, face and orbits, neck (after C-spine clearance criteria met), chest wall, abdomen and pelvis, perineum/rectum/genitourinary, all four extremities, and back/spine via log roll

13 Specific Injury Recognition and Management

A limited set of injuries recur across trauma presentations and require pattern recognition during the primary and secondary survey rather than waiting for definitive imaging. ATLS 11 also shifts spinal motion restriction from routine full immobilization of every trauma patient to a selective, criteria-based approach (using validated decision rules such as NEXUS or the Canadian C-Spine Rule), reserving rigid collars and backboards for patients who meet specific risk criteria rather than applying them universally.

Table 3.9 Specific Injury Recognition and Immediate Management

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Injury	Key Findings	Immediate Management
Tension pneumothorax	Hypotension, distended neck veins, tracheal deviation, absent unilateral breath sounds, hyperresonance to percussion	Immediate needle or finger thoracostomy at the 4th/5th intercostal space, anterior to mid-axillary line, followed by chest tube placement
Cardiac tamponade	Beck's triad (hypotension, distended neck veins, muffled heart sounds), pulsus paradoxus, positive pericardial FAST	Pericardiocentesis or resuscitative thoracotomy if in extremis; definitive surgical repair
Flail chest / pulmonary contusion	Paradoxical chest wall motion; three or more contiguous ribs fractured in two or more places; progressive hypoxia	Aggressive analgesia (consider regional nerve block), pulmonary toilet, selective intubation and positive-pressure ventilation for respiratory failure
Traumatic brain injury	Declining GCS, unequal or nonreactive pupils, Cushing's triad (hypertension, bradycardia, irregular respirations) signaling herniation	Maintain SBP >=100-110 mmHg, target PaCO2 35-40 mmHg (avoid prophylactic hyperventilation unless actively herniating), elevate head of bed 30 degrees, urgent CT and neurosurgical consult, hypertonic saline or mannitol if herniation is evident
Spinal trauma	Focal neurologic deficit, spinal tenderness or step-off, priapism, neurogenic shock (hypotension with bradycardia and warm extremities rather than compensatory tachycardia)	Selective spinal motion restriction per validated criteria, maintain MAP >=85 mmHg to support spinal cord perfusion, avoid prolonged rigid backboard time

Cushing's triad - do not wait for imaging

Hypertension with widening pulse pressure, bradycardia, and irregular respirations together signal impending brain herniation.

Treat immediately: elevate the head of bed, ensure adequate sedation/analgesia, correct hypoxia and hypercapnia, consider hyperosmolar therapy, and mobilize neurosurgery in parallel with - not after - imaging.

14 Worked Clinical Case

Case 3.1 - High-speed motorcycle collision with pelvic and femur fracture

Presentation: A 28-year-old male is brought in by EMS after a motorcycle-versus-car collision at approximately 50 mph. He is awake, GCS 14, HR 132, BP 82/50, RR 24, with an obviously deformed left thigh and pelvic instability on prehospital exam. There is no visible exsanguinating external hemorrhage.

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Full trauma team activation occurs based on mechanism and physiologic criteria.

Management: Primary survey proceeds xABCDE: no exsanguinating bleed identified in the x step; airway is patent and protected; breathing is symmetric with clear breath sounds bilaterally; circulation reveals tachycardia and hypotension consistent with Class III shock, prompting immediate pelvic binder placement, a traction splint to the femur, two large-bore IVs, and activation of the massive transfusion protocol at a 1:1:1 ratio. Tranexamic acid 1 g IV bolus is given at 45 minutes post-injury, well within the 3-hour window, followed by the 1 g infusion. Permissive hypotension is maintained at a target systolic BP of 85 mmHg while blood products are administered en route to the operating room. eFAST shows free fluid in Morison's pouch. Disability exam is non-focal; exposure reveals no additional external wounds, and warmed blankets and fluids are applied throughout to prevent hypothermia. The patient proceeds to a damage control laparotomy with splenic packing and external fixation of the pelvis and femur rather than definitive fixation.

Outcome/teaching point: The patient is transferred to the ICU hemodynamically stable after correction of coagulopathy and rewarming, for planned definitive orthopedic fixation once physiologically optimized. This case illustrates classic damage control resuscitation: early recognition of Class III shock from a source that was never directly visualized externally, prompt balanced transfusion and TXA within the evidence-based window, permissive hypotension until source control, and staged (not definitive) surgery to avoid completing the lethal triad.

15 Chapter Quick-Reference Summary

The following table consolidates the thresholds, ratios, and doses most frequently needed at the bedside during trauma resuscitation.

Table 3.10 ATLS Quick-Reference Summary

Item	Key Value
Primary survey sequence (ATLS 11)	xABCDE - exsanguinating external hemorrhage controlled first when present, then airway/C-spine, breathing, circulation, disability, exposure
TXA dose and window	1 g IV bolus over 10 min, then 1 g infusion over 8 h, within 3 hours of injury (CRASH-2 general trauma; CRASH-3 TBI)
Massive transfusion ratio	Packed red cells : plasma : platelets approximately 1:1:1, or low-titer O-negative whole blood (PROPPR)
Permissive hypotension target (non-TBI)	Systolic BP 80-90 mmHg, or palpable radial pulse, until hemorrhage source is controlled
Blood pressure target with concurrent TBI	Systolic BP >=100-110 mmHg (age-adjusted) to preserve cerebral perfusion
GCS categories	Severe 3-8, Moderate 9-12, Mild 13-15
Shock Class III/IV threshold	Class III >=30% blood loss (~1500 mL); Class IV >40% (~2000 mL) - triggers immediate MTP activation
Tension pneumothorax decompression site	4th/5th intercostal space, anterior to mid-axillary line (chest-tube line); 2nd ICS midclavicular line as an

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Item	Key Value
	alternative
Lethal triad thresholds	Core temperature <35-36 degC, pH <7.2, and coagulopathy compound one another - correct all three simultaneously

16 Traumatic Brain Injury (ATLS Framework)

Primary brain injury is the structural damage sustained at the moment of impact and cannot be reversed once it has occurred; the emergency department's job is preventing secondary brain injury - the additional damage from hypoxia, hypotension, hyperthermia, hypo- or hyperglycemia, and rising intracranial pressure that compounds the primary insult over the following hours.

Severity classification and initial assessment

The Glasgow Coma Scale (GCS) stratifies TBI as mild (13-15), moderate (9-12), or severe (8 or below); a GCS of 8 or less is the threshold for definitive airway management. Serial GCS scoring matters more than any single value, since a downward trend signals expanding intracranial injury before other signs appear. Pupillary size, symmetry, and reactivity are checked and repeated at the same interval as GCS.

Preventing secondary injury

Maintain oxygen saturation and avoid any period of hypoxia; maintain systolic blood pressure adequate for cerebral perfusion, since a single episode of hypotension after TBI is independently associated with worse outcomes; avoid hyperventilation except as a brief, targeted bridge for overt herniation signs, since routine hyperventilation lowers PaCO2, causes cerebral vasoconstriction, and can worsen ischemia; and treat fever and hyperglycemia promptly. Elevate the head of the bed when the spine allows it, and keep a cervical collar from being tight enough to impede jugular venous outflow.

Herniation and surgical decision-making

A new pupillary asymmetry, a GCS drop of 2 or more points, posturing, Cushing's triad (hypertension, bradycardia, irregular respirations), or sudden apnea are signs of impending herniation requiring immediate action - see Chapter 7, Section 6 for the ENLS-guided herniation-rescue measures (hyperosmolar therapy, brief hyperventilation) and emergent neurosurgical consultation for evacuation of a mass lesion where one is present. CT imaging of the head is obtained early in any TBI with loss of consciousness, amnesia, a GCS below 15, a focal deficit, or a high-risk mechanism, and repeated if the clinical exam changes even when the first scan was unremarkable.

17 Burn Management

Burns are assessed by depth and extent, both of which drive the fluid resuscitation and disposition decision. Superficial (first-degree) burns involve the epidermis only. Partial-thickness (second-degree) burns extend into the dermis and blister. Full-thickness (third-degree) burns destroy the full dermis and appear white, leathery, or charred - often painless at the burn itself from destroyed nerve endings, though adjacent partial-thickness burn is usually present and very painful.

Airway first

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Facial burns, singed nasal hair or eyebrows, soot in the mouth or sputum, hoarseness, or a closed-space fire history all raise concern for inhalation injury, where airway swelling can progress over hours to complete obstruction; a low threshold for early, controlled intubation is safer than waiting for obvious respiratory distress to develop.

Extent and fluid resuscitation

Extent is estimated with the rule of nines (each arm 9%, each leg 18%, anterior and posterior trunk 18% each, head and neck 9%, genitalia 1%, adjusted for the relatively larger pediatric head and smaller legs) or, for smaller burns, the patient's own palm including fingers representing approximately 1% of body surface area. Fluid resuscitation using the modified (ATLS) Parkland formula is indicated for burns of 20% total body surface area (TBSA) or more in adults, or 10% or more in children (second- and third-degree burns only, not first-degree).

Table 3.2 Modified Parkland Formula for Burn Fluid Resuscitation

Parameter	Value
Total 24-h crystalloid volume	2-4 mL x weight (kg) x %TBSA burned (lactated Ringer's)
First half of volume	Given over the first 8 hours from the time of injury (not arrival)
Second half of volume	Given over the following 16 hours
Pediatric adjustment	Add maintenance fluid with dextrose; Parkland alone may cause hypoglycemia in children
Inhalation injury adjustment	May require 30-40% more fluid than calculated
Titration target	Urine output 0.5-1 mL/kg/h (adults); 1 mL/kg/h (children)

Time zero is the time of burn injury, not hospital arrival; treat the calculated volume as a starting estimate, titrated to urine output and clinical exam.

Local wound care and special burn types

Stop the burning process first (remove smoldering clothing and jewelry; brush off dry chemical powder before flushing with water, since some chemicals react with water), then cover the burn with a dry, clean dressing - avoid ice, which worsens tissue injury and hypothermia in larger burns. Electrical burns carry a high risk of cardiac arrhythmia and deep tissue injury far exceeding what the skin surface suggests, and warrant cardiac monitoring and evaluation for compartment syndrome and myoglobinuria along the current's path.

Burn center referral criteria

Refer to a dedicated burn center: any full-thickness burn regardless of size; partial- or full-thickness burns over 10% TBSA; burns involving the face, hands, feet, genitalia, perineum, or major joints; inhalation injury; significant chemical or electrical injury including lightning; and burns in patients with comorbidities or concomitant trauma that would complicate management.

This chapter is based on the American College of Surgeons Committee on Trauma Advanced Trauma Life Support (ATLS) Student Course Manual, 11th edition (2025), together with the CRASH-2 (2010) and CRASH-3 (2019) tranexamic acid trials and the PROPPR (2015) massive transfusion ratio trial. It is an educational reference summary only and is not a substitute for completion and verification of an official ATLS provider course or for institutional trauma protocols, which should always take precedence.

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LIFE SUPPORT COURSES · CLINICAL COMPANION MANUAL

CHAPTER 4

ADLS

Advanced Disaster Life Support

1 Clinical Snapshot

Why this matters · Typical patient · Where you meet it

Why it matters: A single mass-casualty incident (MCI) can generate more patients in minutes than a busy emergency department sees in a day. Clinicians who have never trained for disaster response default to routine, one-patient-at-a-time medicine — a mindset that fails, and costs lives, when resources are suddenly outstripped by demand.

Typical scenario: A charter bus rolls on the interstate with 40 passengers; a chemical tanker ruptures near a school; a category-4 hurricane knocks out power to three hospitals; a novel respiratory virus fills every ICU bed in the region simultaneously.

Common settings: Mass-casualty incidents (transportation crashes, building collapses, active-shooter/explosive events), natural disasters (hurricanes, earthquakes, floods, wildfires), CBRNE releases (chemical, biological, radiological, nuclear, explosive), and public-health emergencies/pandemics that produce sustained surge rather than a single-day spike.

Advanced Disaster Life Support (ADLS) is the intermediate-to-advanced tier of the National Disaster Life Support Foundation (NDLSF) course suite, which also includes Basic Disaster Life Support (BDLS), Core Disaster Life Support (CDLS), and Pediatric Disaster Life Support (PDLS). ADLS is built for physicians, nurses, physician assistants, EMTs, paramedics, pharmacists, and other allied health professionals who need working knowledge of mass-casualty triage, hospital surge operations, incident command, and CBRNE response — not just individual patient resuscitation. Successful completion of BDLS is a standard prerequisite.

The core reframe this chapter teaches is the shift from individual-patient medicine, where every resource is committed to one patient, to population-based medicine, where the objective is the greatest good for the greatest number given finite resources. That shift underlies triage, surge capacity, and crisis standards of care alike, and it is the single hardest cognitive adjustment for clinicians trained exclusively in one-to-one care.

- Mass-casualty incidents: multi-vehicle crashes, building or structural collapse, active-shooter and explosive events, transportation disasters (rail, aviation)
- Natural disasters: hurricanes and tropical storms, earthquakes, tornadoes, wildfires, floods, extreme heat and winter weather events
- CBRNE events: chemical releases, biological exposures/bioterrorism, radiological or nuclear incidents, explosive/blast events
- Pandemics and public-health emergencies: novel infectious disease surge, prolonged resource scarcity, crisis staffing and equipment shortages

2 Learning Objectives

By the end of this chapter the learner should be able to:

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- Describe the four phases of the disaster management cycle and give an example intervention for each
- Apply an all-hazards framework to classify a disaster as natural, technological, or human-made/CBRNE
- Explain the structure of the Incident Command System (ICS) under NIMS, including Command Staff and General Staff roles
- Perform mass-casualty triage using both the START and SALT algorithms and correctly assign triage tag categories
- Describe the 4 S's of surge capacity (space, staff, stuff, systems) and give hospital-level examples of each
- Recognize clinical and scene clues suggestive of chemical, biological, radiological, nuclear, or explosive (CBRNE) exposure
- Select an appropriate level of personal protective equipment (PPE) and describe basic decontamination principles
- Describe hospital disaster preparedness elements, including crisis standards of care and communication redundancy
- Identify the special needs of pediatric, elderly, disabled, and pregnant patients during disaster response
- Apply the core principles of psychological first aid to disaster survivors and responders

3 The Disaster Management Cycle

Disaster medicine organizes activity into four interlocking phases that repeat continuously rather than occurring in strict sequence. Mitigation and preparedness happen before an event and reduce its ultimate impact; response and recovery happen during and after and determine how well a community and its healthcare system absorb the shock.

Mitigation

Mitigation consists of sustained actions taken to eliminate or reduce the probability or impact of a disaster before it occurs. Examples include seismic building codes, floodplain zoning restrictions, chemical facility safety regulations, and vaccination programs that reduce the population susceptible to an outbreak.

Preparedness

Preparedness builds the capability to respond effectively when mitigation fails to prevent an event. This includes hazard vulnerability analysis, stockpiling supplies, writing and exercising emergency operations plans, training staff, and conducting tabletop and full-scale disaster drills — the exercises that make ICS assignments and mutual-aid agreements second nature rather than something read for the first time during a real event.

Response

Response is the operational phase: activating the emergency operations plan, standing up incident command, triaging and treating casualties, allocating scarce resources, and communicating with the public and partner agencies. This is the phase most of this chapter addresses in operational detail.

Recovery

Recovery restores essential services and infrastructure and supports the long-term physical and psychological healing of the affected population. Hospital recovery includes restocking, equipment repair, after-action review, and staff support for burnout and moral distress accumulated during the response.

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Practice Pearls

The four phases overlap continuously — recovery from one event is simultaneously mitigation and preparedness for the next.

After-action reports and hot-wash debriefs are not optional paperwork; they are the mechanism by which mitigation and preparedness actually improve.

A plan that has never been exercised should be assumed not to work.

4 All-Hazards Approach and Disaster Classification

An all-hazards approach means building generic response capability — command structure, triage, surge, communications — that applies across disaster types, then layering hazard-specific expertise on top. This is efficient because the operational skeleton (who is in charge, how patients are sorted, how resources scale) is nearly identical whether the trigger is a hurricane or a chemical release, even though the clinical specifics differ sharply.

Table 4.1 Disaster classification under the all-hazards framework

Category	Definition	Representative Examples
Natural	Events arising from geophysical, meteorological, or biological processes	Hurricanes, earthquakes, tornadoes, floods, wildfires, pandemics/epidemics
Technological	Events arising from failure or malfunction of human-made systems and infrastructure	Industrial chemical release, power grid failure, transportation crashes, dam or structural failure
Human-made / intentional (CBRNE)	Deliberate acts intended to cause harm, disruption, or terror	Chemical, biological, radiological, nuclear, or explosive attacks; active-shooter/mass-violence events

Many real events are hybrid — e.g., a hurricane (natural) causing a chemical plant release (technological).

Regardless of classification, every disaster response draws on the same four operational pillars covered in this chapter: command and coordination (ICS/NIMS), casualty sorting (triage), resource scaling (surge capacity), and hazard-specific protection (CBRNE recognition, PPE, and decontamination).

5 Incident Command System and NIMS

The National Incident Management System (NIMS), maintained by FEMA, provides the common framework U.S. jurisdictions use to prevent, protect against, respond to, and recover from incidents of any scale. Its centerpiece for on-scene management is the Incident Command System (ICS), a standardized, scalable organizational structure with clear reporting lines, common terminology, and a manageable span of control. Hospitals implement the same logic through the Hospital Incident Command System (HICS), which maps hospital functions (nursing, pharmacy, security, facilities) onto the standard ICS sections so that hospital and field responders speak the same organizational language.

Every incident, regardless of size, has a single Incident Commander (IC) who holds overall authority and sets incident objectives. As an incident grows, the IC can activate Command Staff (functions that support the IC directly) and General Staff (the four functional sections that carry out the work). Multiple agencies with

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jurisdiction over the same incident use Unified Command, in which representatives from each agency jointly determine objectives and priorities rather than competing for control.

Table 4.2 ICS command structure — key roles and responsibilities

Role	Reports To	Core Responsibility
Incident Commander (IC)	Elected/appointed authority	Overall command; sets objectives, approves the Incident Action Plan, ensures safety
Public Information Officer (PIO)	Incident Commander	Develops and releases accurate incident information to media and the public
Safety Officer	Incident Commander	Monitors and assesses hazardous/unsafe conditions; has authority to stop unsafe operations
Liaison Officer	Incident Commander	Point of contact for representatives of assisting and cooperating agencies
Operations Section Chief	Incident Commander	Directs all tactical actions needed to meet incident objectives (e.g., triage, treatment, decon, security)
Planning Section Chief	Incident Commander	Collects and evaluates information, tracks resources, develops the Incident Action Plan
Logistics Section Chief	Incident Commander	Provides facilities, equipment, supplies, communications, and personnel support
Finance/Administration Section Chief	Incident Commander	Tracks incident costs, procurement, timekeeping, and compensation/claims

Command Staff = PIO, Safety Officer, Liaison Officer. General Staff = the four Section Chiefs. Structure per FEMA NIMS/ICS.

Key Numbers — Command, Triage, and Surge

Optimal ICS span of control: 1 supervisor to 5 subordinates (acceptable range 3–7)

START/SALT primary triage target: roughly 30–60 seconds per patient

Retriage interval for Delayed (yellow) patients awaiting transport: every 10–15 minutes

Conventional hospital 72-hour self-sufficiency expectation for utilities, water, and supplies (Joint Commission emergency management standard)

Crisis standards of care activation: declared when demand for critical resources (ventilators, ICU beds, blood products) exceeds supply despite contingency measures

6 Mass-Casualty Triage — START and SALT

Triage sorts casualties by clinical urgency so that limited personnel and transport resources reach the patients who benefit most, most quickly. In an MCI, triage is a continuous, iterative process — not a single tag

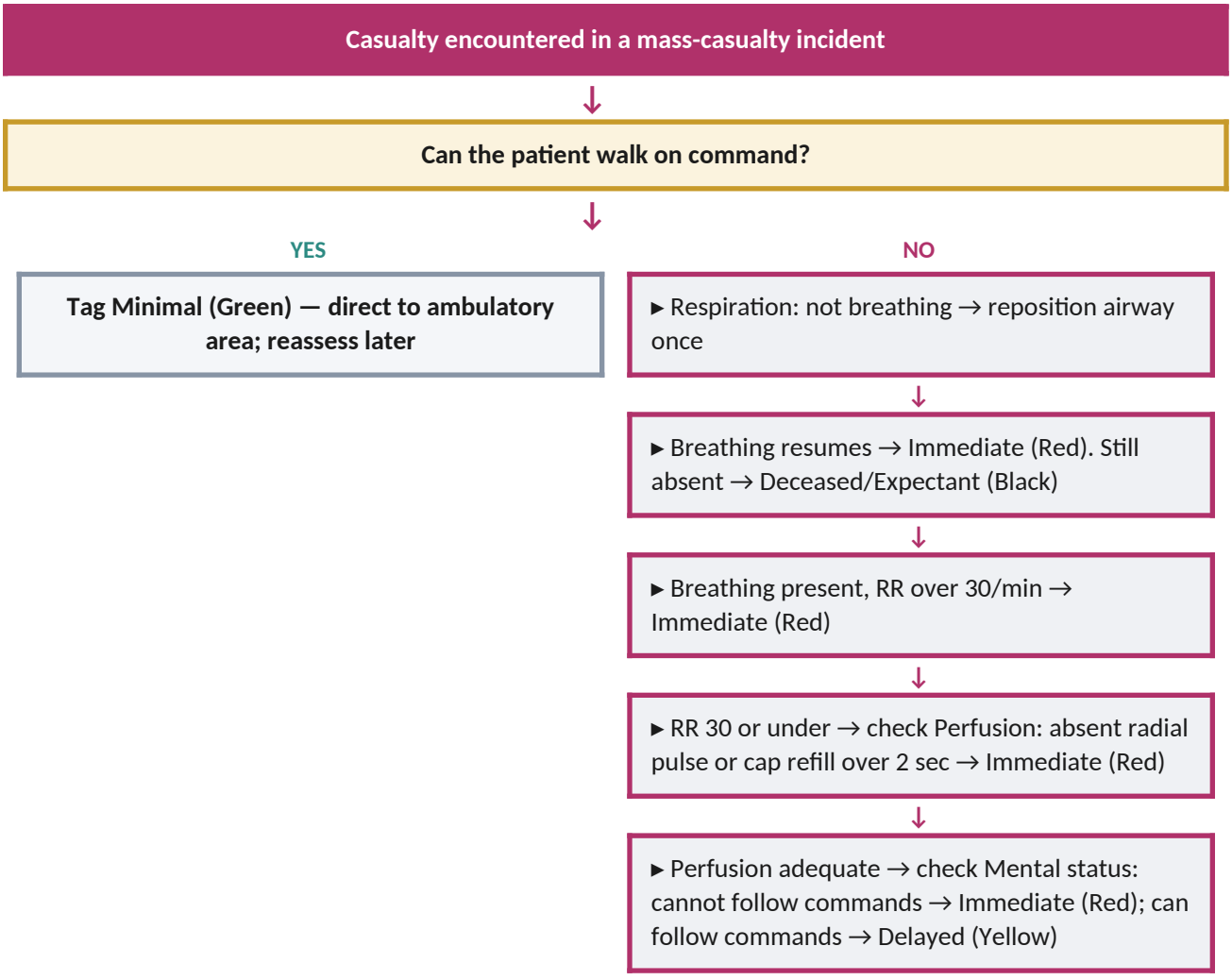
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applied once — performed first in the field (primary triage), then repeated at treatment areas and transport points (secondary and ongoing triage) as patients' conditions evolve. Two algorithms dominate U.S. practice: START, the long-standing EMS field standard, and SALT, the federally recommended unifying framework.

START (Simple Triage And Rapid Treatment)

START begins by asking all victims who are able to walk to move to a designated area; these patients are tagged Minimal (green) and reassessed later. Remaining patients are assessed one at a time using the RPM sequence — Respiration, Perfusion, Mental status. If a patient is not breathing, the airway is repositioned once; if breathing resumes, the patient is tagged Immediate (red); if not, the patient is tagged Deceased/Expectant (black). If breathing is present at a rate over 30/minute, the patient is Immediate. For patients breathing normally, perfusion is checked by radial pulse or capillary refill; an absent radial pulse or capillary refill over 2 seconds yields an Immediate tag. Patients with adequate perfusion are checked for mental status by simple command (e.g., squeeze my hand); inability to follow commands yields an Immediate tag; ability to follow commands yields a Delayed (yellow) tag. The entire assessment is designed to take under a minute per patient, and only life-saving interventions — opening an airway, controlling major hemorrhage — are performed during this pass.

ALGORITHM — START MASS-CASUALTY TRIAGE

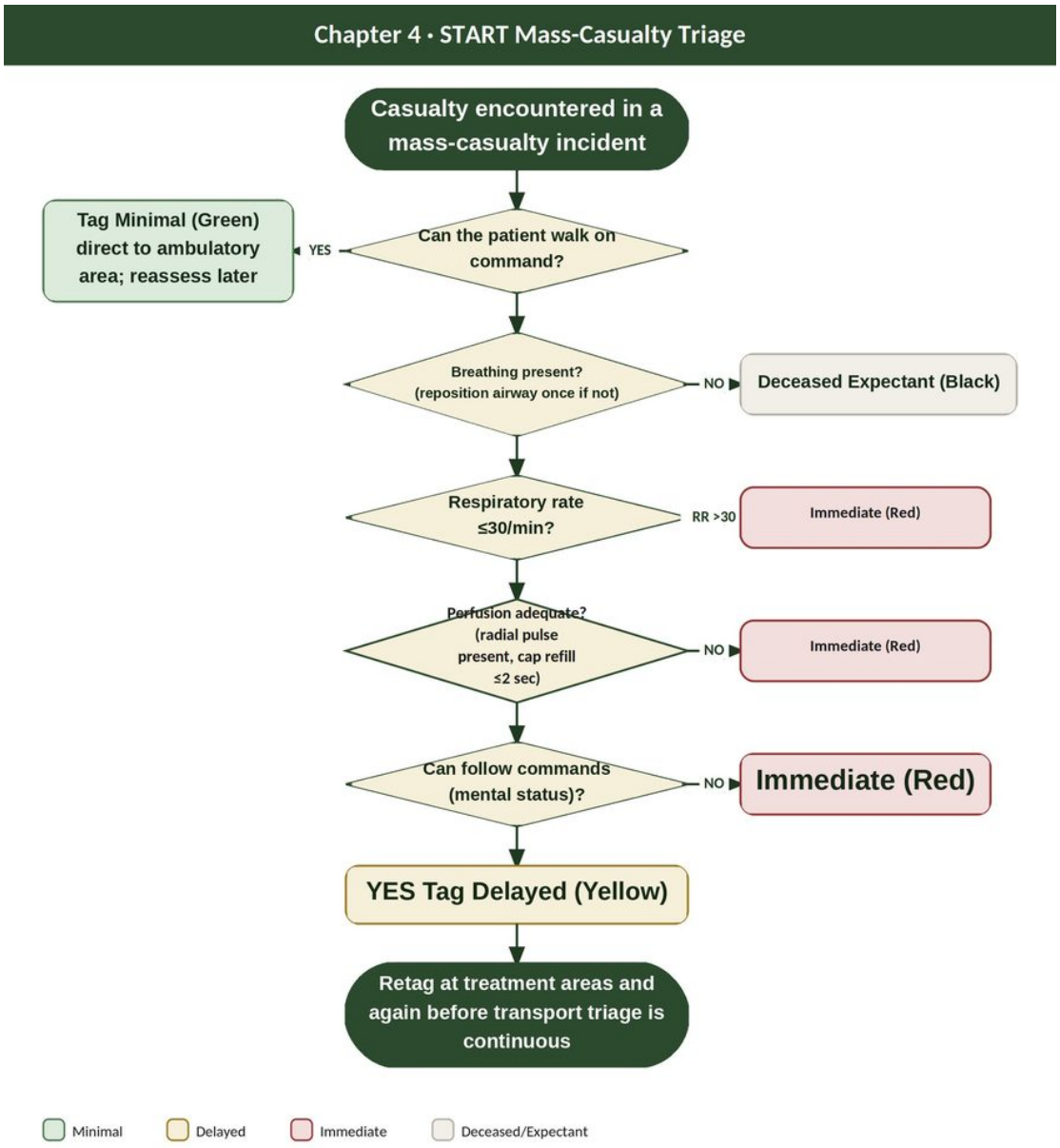


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Figure 4.1. The START (Simple Triage And Rapid Treatment) algorithm; see Table 4.4 for a comparison with SALT.

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SALT (Sort, Assess, Lifesaving Interventions, Treatment/Transport)

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SALT is the mass-casualty triage framework endorsed for national use because it standardizes terminology across EMS, hospital, and federal partners and explicitly incorporates brief lifesaving interventions into the sorting step. The first step, global sorting, calls out simple voice commands to the entire scene: those who walk toward the responder's voice are sorted Minimal; those who wave or make purposeful movement in response are sorted Delayed; those who do not move, or who have an obvious immediately life-threatening injury, are assessed first as possible Immediate or Expectant. The second step, individual assessment, checks for and treats obvious life threats on the spot — controlling major hemorrhage, opening the airway, decompressing a tension pneumothorax, sealing an open chest wound, or administering an antidote — before assigning a final category of Immediate, Expectant, Delayed, Minimal, or Dead based on whether the patient is breathing, has a pulse, is in respiratory distress or uncontrolled hemorrhage, and is likely to survive given available resources.

Table 4.3 Triage tag categories

Category	Tag Color	Clinical Criteria	Priority / Disposition
Immediate	Red	Life-threatening problem (airway, breathing, circulation/hemorrhage) that is survivable with prompt intervention	Treated and transported first
Delayed	Yellow	Significant injury but stable enough to tolerate a delay in care without immediate threat to life	Treated and transported after Immediate patients; reassessed frequently
Minimal	Green	Walking wounded; minor injuries not requiring urgent intervention	Directed to a self-care/ambulatory area; transported last
Expectant	Black / Gray	Injuries incompatible with survival given the resources realistically available, or no signs of life after airway repositioning	Comfort care provided; resources reallocated to salvageable patients; reassessed if resources allow

START and SALT use the same four color-coded outcome categories; SALT adds an explicit Dead category for victims with no signs of life and no response to any intervention.

Table 4.4 START versus SALT — key differences

Feature	START	SALT
First step	Ambulatory patients self-move (walk) → Minimal	Global voice sort: walk / wave-purposeful movement / still
Core assessment	Respiration – Perfusion – Mental status (RPM)	Individual assessment with brief lifesaving interventions performed during sorting
Interventions during triage	Airway repositioning and major hemorrhage control only	Airway, hemorrhage control, chest seal, needle decompression, antidote as indicated
Endpoint categories	Immediate, Delayed, Minimal, Expectant/Deceased	Immediate, Expectant, Delayed, Minimal, Dead

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Feature	START	SALT
Adoption	Long-standing EMS field standard in many U.S. systems	Federally recommended (CDC/ASPR) unifying algorithm for cross-agency consistency

Decision Point — Can the Patient Walk or Purposefully Move?

Yes, walks on command: Tag Minimal (green); direct to ambulatory area; reassess later.

Waves or makes purposeful movement, cannot walk: Proceed to individual assessment; likely Delayed pending RPM/injury findings.

No movement or obvious catastrophic injury: Assess first — highest likelihood of Immediate or Expectant category; check airway and major hemorrhage immediately.

Myth vs Fact — Mass-Casualty Triage

Myth: A triage tag, once applied, is permanent for that patient.

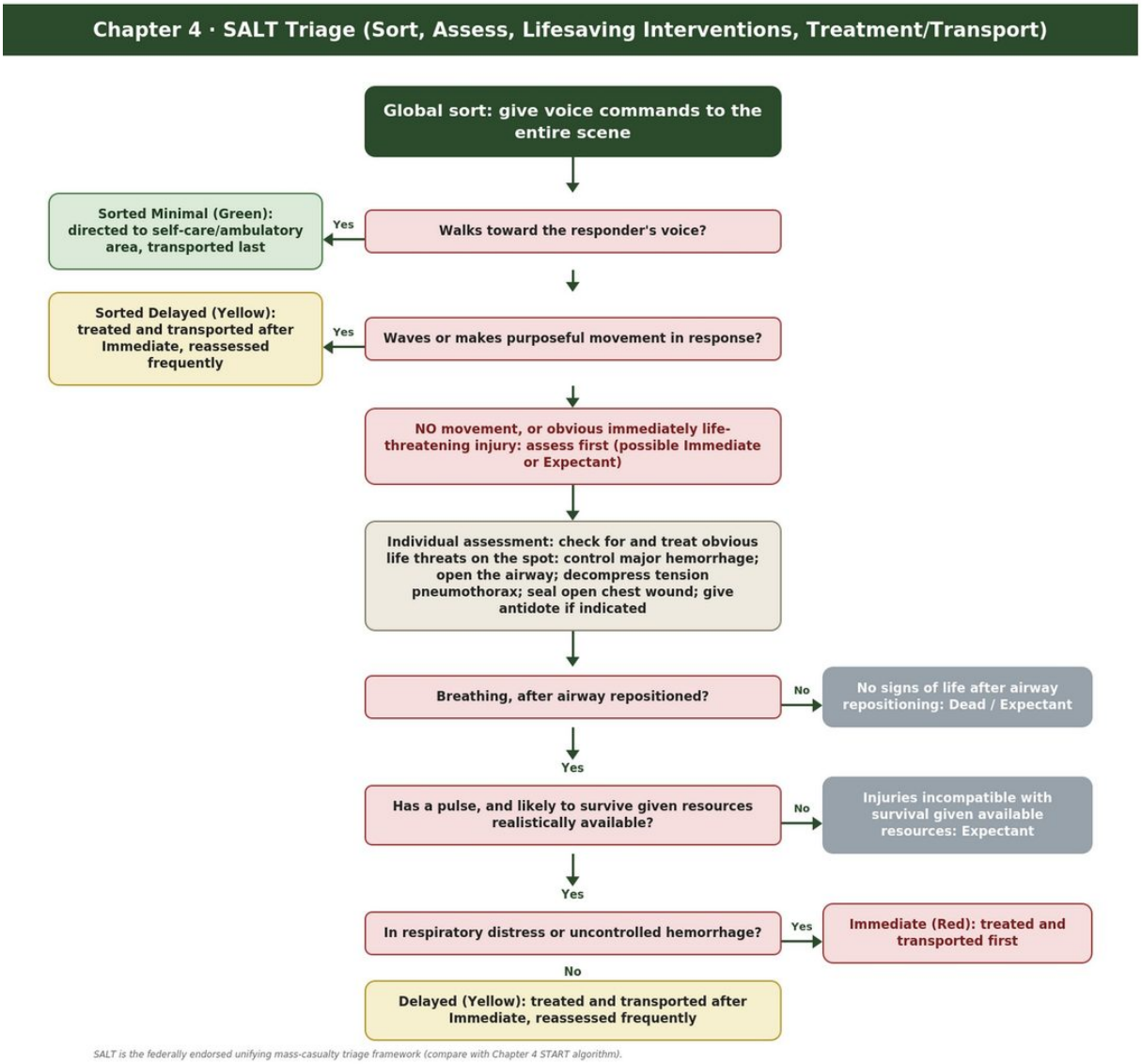
Fact: Triage is dynamic. Patients are reassessed at treatment areas and again before transport; a patient can move up or down in category as their condition changes.

Myth: Every casualty should be transported to the closest hospital.

Fact: Transport should be distributed across regional receiving facilities per the incident's medical branch/transport officer to prevent overwhelming the nearest single ED — a phenomenon that has crippled trauma centers in real MCIs.

SALT is the federally endorsed unifying mass-casualty triage framework.

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7 Surge Capacity — The 4 S's

Surge capacity is a healthcare system's ability to expand rapidly beyond normal operations to meet an extraordinary and sustained demand for care. It is conventionally organized around four resource domains —

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space, staff, stuff, and systems — each of which must scale together, since a surplus in one domain cannot substitute for a deficit in another (extra beds are useless without staff to run them).

Table 4.5 Surge capacity — the 4 S's

Domain	Definition	Hospital-Level Examples
Space	Physical locations where care can be delivered	Converting PACU/endoscopy/lobby areas to patient care bays; using hallway beds; alternate care sites (tents, convention centers)
Staff	Available and appropriately credentialed personnel	Callback and mutual-aid staffing plans, cross-training, extending shifts, activating the Medical Reserve Corps or credentialed volunteers
Stuff	Equipment, supplies, medications, and consumables	Stockpiled ventilators, PPE, IV fluids, blood products, pharmaceuticals; cache and vendor-managed inventory agreements
Systems	Command, coordination, information, and policy structures that make the other three usable	Hospital Incident Command activation, crisis standards of care policies, EHR/communication redundancy, mutual-aid agreements

Surge planning distinguishes three intensity tiers: conventional capacity (usual space/staff/supplies, minor adaptation), contingency capacity (functionally equivalent care using adapted spaces, staff, or supplies — e.g., one nurse supervising more patients than usual), and crisis capacity (resources are so constrained that care delivered is no longer consistent with usual standards, requiring formal crisis standards of care). This tiering is expanded in Section 12.

8 CBRNE Threats Overview

CBRNE — chemical, biological, radiological, nuclear, and explosive — describes the categories of agents and mechanisms most associated with mass-casualty terrorism and high-consequence industrial or transportation accidents. Recognizing the pattern of illness or injury early, before a laboratory confirms the agent, is often the single most important clinical action, because it drives immediate decisions about scene safety, PPE, and decontamination before more responders and patients are exposed.

Table 4.6 CBRNE overview — agents, recognition, and initial management

Category	Key Agents / Examples	Recognition Clues	Initial Management / Decon Approach
Chemical	Nerve agents (sarin, VX), vesicants (sulfur mustard), pulmonary/choking agents (chlorine, phosgene), cyanide	Sudden collapse of multiple people in one area; unusual odor; dead insects, birds, or vegetation nearby; miosis, excess secretions,	Remove from source, hasty decontamination (disrobe, dry then water/soap decon), specific antidotes where available

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Category	Key Agents / Examples	Recognition Clues	Initial Management / Decon Approach
		seizures (SLUDGE toxidrome)	(atropine/pralidoxime for nerve agents, hydroxocobalamin for cyanide), airway/ventilatory support
Biological	Anthrax, plague, smallpox, botulinum toxin, viral hemorrhagic fevers, novel respiratory pathogens	Delayed onset (days), unusual clustering of illness or an atypical disease for the region/season, unexplained animal die-off (zoonotic agents)	Isolation/cohorting per transmission route, appropriate contact/droplet/airborne precautions, post-exposure prophylaxis or antitoxin where available, urgent public-health notification
Radiological	Radiological dispersal device ("dirty bomb"), lost/stolen industrial or medical source, contaminated scene	Radiation detector/dosimeter alarm, unexplained localized burns, incident occurring near a known radioactive source	Time-distance-shielding to limit exposure, external decontamination (removing clothing eliminates most surface contamination), radiation survey before and after decon, potassium iodide only if radioiodine exposure confirmed
Nuclear	Improvised nuclear device or state/military nuclear detonation	Massive blast plus thermal injury plus radiation exposure over a wide area; widespread structural destruction	Shelter-in-place versus evacuation decision based on fallout modeling, combined mass trauma and radiation triage, prioritized decontamination and burn/blast management
Explosive (conventional)	Improvised explosive devices, secondary/tertiary devices, industrial explosions	Characteristic blast injury pattern: primary (blast lung, tympanic membrane rupture), secondary (penetrating fragment wounds), tertiary (blunt trauma from being thrown/structural collapse), quaternary (burns, crush, inhalation)	Scene safety first (assume secondary devices), hemorrhage control, evaluate for blast lung and TM rupture, staged evacuation once scene is cleared by law enforcement/EOD

Sources: CDC emergency preparedness and response guidance; WHO CBRN guidance.

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Red Flags Suggesting a CBRNE Event

Multiple patients with the same unusual symptom cluster arriving from one location

Unexplained odors (bitter almond, garlic, fresh-cut grass, bleach) at the scene

Dead animals, insects, or wilted vegetation with no clear environmental cause

Casualties collapsing in a pattern radiating outward from a point source

A second explosion or device discovered after responders arrive (secondary-device risk)

Any single event should raise suspicion; the combination of several is highly specific for an intentional release

9 Decontamination and Personal Protective Equipment

Decontamination removes or neutralizes contaminants before they cause ongoing harm to the patient or spread to responders and receiving facilities — a failure of decontamination is the single most common way a hospital emergency department becomes secondarily contaminated and forced to close during a chemical incident. The core principle is simple: disrobing alone removes roughly 80–90 percent of surface contamination for most chemical agents, and this step should never be skipped even when full wet decontamination is unavailable.

Decontamination generally proceeds through zones: the Hot Zone (contaminated, restricted to appropriately protected personnel), the Warm Zone (decontamination corridor, reduced PPE), and the Cold Zone (clean, support and command functions, minimal PPE). Gross (hasty) decontamination — removing clothing and rinsing with water — is performed as quickly as possible for ambulatory and non-ambulatory casualties alike, followed by technical/secondary decontamination (soap-and-water wash, sequenced head-to-toe) when time and resources allow. Selection of PPE for responders and decontamination teams is matched to the hazard and the zone.

Table 4.7 PPE levels A–D

Level	Respiratory Protection	Skin / Suit Protection	Typical Use Scenario
Level A	Pressure-demand, positive-pressure SCBA (self-contained breathing apparatus)	Fully encapsulating, vapor-tight chemical-resistant suit with sealed seams, integrated gloves and boots	Highest known or unknown hazard, IDLH atmosphere, unidentified agent, hot-zone entry for unknown chemical release
Level B	Pressure-demand SCBA or supplied-air respirator, worn outside the suit	Non-encapsulating, chemical-resistant splash suit; protects against liquid splash but not full vapor exposure	Highest respiratory protection needed with lesser skin/vapor threat; known liquid hazard; initial hot-zone rescue
Level C	Air-purifying respirator (APR) or powered air-purifying respirator (PAPR) with agent-appropriate cartridge	Chemical-resistant coverall/splash suit	Agent identified, oxygen adequate (≥19.5 percent), concentration below IDLH; hospital decontamination teams commonly use this level

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Level	Respiratory Protection	Skin / Suit Protection	Typical Use Scenario
Level D	None (or standard surgical mask only)	Standard work uniform/scrubs; minimal skin protection	Cold-zone support, command, and administrative functions where no hazardous exposure is expected

Per OSHA HAZWOPER and CBRN PPE ensembles; hospital decon teams most commonly operate in Level C.

Quick Tip — Hasty Decontamination

Remove and bag all clothing and jewelry before any other decon step — this single action clears most surface contamination.

Do not delay life-threatening airway or hemorrhage interventions to complete full decon; perform them concurrently in the warm zone with appropriate PPE.

Protect wounds and IV sites with waterproof covering before water decontamination to prevent driving contaminant into the wound.

10 Hospital Disaster Preparedness

Hospitals prepare for disasters through an emergency operations plan built on the Hospital Incident Command System (HICS), which activates the same command-and-general-staff structure used in the field so that hospital leadership and outside responders coordinate seamlessly. Preparedness spans four operational pillars: surge protocols (rapid bed, staff, and supply expansion per Section 7), redundant communication systems (satellite phones, amateur radio, runners, and regional resource-tracking systems that function when cellular and internet networks fail or are overloaded), resource allocation frameworks (triage of scarce resources such as ventilators or blood products according to pre-established, ethically vetted criteria), and crisis standards of care.

Crisis Standards of Care

Crisis standards of care describe a formal, legally recognized shift in the level of care a system delivers when resource scarcity is so severe that usual standards cannot be maintained despite conventional and contingency adaptations. Activation typically requires a declaration from state or local health authority, and it substitutes population-outcome-focused allocation (often guided by a triage committee applying transparent, pre-established criteria) for the usual first-come, individual-patient-focused allocation. The goal of crisis standards of care is not to abandon ethical care but to make necessarily hard allocation decisions consistently, transparently, and outside the bedside clinician's sole discretion — protecting both patients and providers from ad hoc rationing under stress.

Table 4.8 Capacity tiers and crisis standards of care

Tier	Trigger	Resource Allocation Approach
Conventional	Usual demand; resources adequate with minor adaptation	Usual individual-patient standard of care
Contingency	Sustained above-normal demand	Functionally equivalent care using adapted space, staff, or supplies (e.g., altered nurse-to-patient ratios)

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Tier	Trigger	Resource Allocation Approach
Crisis	Demand exceeds available resources despite contingency measures	Population-outcome-focused allocation under a formal, transparent triage protocol/committee; usual standards cannot be fully maintained

Hospital Surge Monitoring Checklist

Real-time bed and ICU capacity across all units

Staff availability, callback status, and fatigue/burnout indicators

PPE and critical-supply burn rate versus stockpile

Decontamination corridor throughput and PPE readiness

Communication system status (primary and backup channels)

Blood product and pharmaceutical inventory versus projected demand

Mortuary/surge capacity and family reunification center status

11 Special Populations in Disasters

Standard disaster protocols are calibrated to a healthy adult, and applying them unmodified to pediatric, elderly, disabled, and pregnant patients systematically under-triages or under-serves them. Disaster planning that fails to account for these groups predictably fails them first — historically, children, older adults, and people with disabilities have been disproportionately harmed in disasters precisely because generic plans assumed an able-bodied adult population.

Table 4.9 Special populations — key disaster considerations

Population	Key Considerations
Pediatric	Adult triage tools misclassify children; use a pediatric-adapted algorithm (e.g., JumpSTART); weight-based medication dosing; higher risk of hypothermia, dehydration, and rapid decompensation; prioritize rapid family reunification and avoid separating children from caregivers when possible
Elderly	Comorbidities and polypharmacy complicate assessment and dosing; altered cognitive or sensory baseline can be mistaken for acute injury (or mask one); mobility limitations slow evacuation; higher risk of rapid decompensation and of being left behind in evacuation
Disabled / functional needs	Communication barriers (hearing, vision, cognitive, language); dependence on durable medical equipment (wheelchairs, ventilators, dialysis) and power for that equipment; service animals should be kept with their handler when feasible; evacuation plans must include

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	accessible transport and routes
Pregnant	Effectively two patients — maternal and fetal status both matter; position in left lateral tilt in later pregnancy to avoid aortocaval compression; consider Rh status before any transfusion; weigh radiation, medication, and vaccine exposure risk against maternal benefit; anticipate obstetric surge needs (preterm labor, delivery) in shelters and alternate care sites

12 Psychological First Aid

Psychological first aid (PFA) is an evidence-informed approach to supporting disaster survivors and responders in the immediate aftermath of an event — it is not clinical mental health treatment, and it does not require a mental health license to deliver. Its aims are to promote safety, calm, connectedness, self- and community efficacy, and hope, while avoiding interventions known to be ineffective or harmful, such as mandatory single-session debriefing that forces detailed recounting of the traumatic event.

- Make contact and engage: approach calmly, introduce yourself, ask permission, and respect the person's pace
- Ensure safety and comfort: address immediate physical needs (warmth, food, water, a quiet space) before deeper conversation
- Stabilize: for an overwhelmed or disoriented person, use grounding techniques and calm, simple language
- Gather information and help prioritize needs: ask what is most pressing right now rather than assuming
- Offer practical assistance: help with concrete needs — reconnecting with family, finding shelter, understanding next steps
- Connect with social supports: reunite the person with family, friends, or community resources whenever possible
- Provide information on coping and normal stress reactions: most acute stress responses are normal and self-limited
- Link with collaborative services: refer for ongoing mental health support when distress is severe or persistent

Responders themselves are not exempt from these principles. Disaster medicine sustains staff through mandatory rest rotations, peer-support programs, and explicit permission to step back — a responder who is not looking after their own basic needs cannot reliably look after anyone else's.

13 Worked Case Example

Case 4.1 — Charter Bus Collision, 22 Casualties

Scenario: A charter bus carrying 34 passengers rolls on a rural interstate at dusk. First arriving EMS units find the bus on its side, roughly 22 patients with injuries ranging from minor lacerations to entrapment, and a scene with no working streetlights and limited initial resources.

Response: The first paramedic on scene assumes Incident Command and immediately requests additional

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ambulances, a medical branch, and law enforcement for scene safety, then begins START triage while awaiting arrival of a designated Triage Officer. Ambulatory patients are directed to walk to a marked collection point and tagged green. Non-ambulatory patients are assessed by RPM: two patients with absent radial pulses and altered mental status are tagged red; a patient breathing at 34/min after airway repositioning is tagged red; several patients with normal RPM findings but obvious fractures are tagged yellow; one patient found apneic with no return of breathing after airway repositioning is tagged black. As additional units arrive, Incident Command expands to include a Triage Officer, Treatment Officer, and Transport Officer, and a Staging Area is designated a quarter mile from the wreck to prevent ambulance gridlock. Regional dispatch distributes red-tag patients across three trauma centers rather than sending all of them to the closest hospital, per the transport officer's load-balancing decision.

Outcome/teaching point: All red-tag patients are transported within 40 minutes; yellow-tag patients are re-triaged at 15-minute intervals while awaiting transport, and one yellow patient is upgraded to red after developing worsening respiratory distress. The case illustrates rapid establishment of command, disciplined RPM-based triage under time pressure, expansion of the ICS structure as resources arrive, continuous re-triage rather than a one-time tag, and deliberate transport distribution to avoid overwhelming a single receiving facility.

14 Chapter Summary — Quick Reference

The tables below consolidate the highest-yield reference points from this chapter for rapid recall during an actual event.

Table 4.10 ADLS quick-reference summary

Domain	Element	One-Line Definition
Triage	Immediate (Red)	Life-threatening but survivable with prompt intervention; treated/transported first
Triage	Delayed (Yellow)	Serious but stable; can tolerate a delay in care; reassessed every 10–15 minutes
Triage	Minimal (Green)	Walking wounded; minor injury; self-care area; transported last
Triage	Expectant (Black/Gray)	Not survivable given available resources, or no signs of life; comfort care
ICS	Incident Commander	Overall authority; sets objectives and approves the Incident Action Plan
ICS	Operations Section	Executes tactical actions — triage, treatment, decon, security
ICS	Planning Section	Tracks resources and situational information; builds the Incident Action Plan
ICS	Logistics Section	Supplies facilities, equipment,

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Domain	Element	One-Line Definition
		personnel, and communications support
ICS	Finance/Administration Section	Tracks cost, procurement, timekeeping, and claims
PPE	Level A	SCBA plus fully encapsulating vapor-tight suit; unknown/highest hazard
PPE	Level B	SCBA plus splash-protective (non-encapsulating) suit; known liquid hazard
PPE	Level C	APR/PAPR plus chemical-resistant coverall; identified agent, adequate oxygen
PPE	Level D	Standard work clothing; no expected hazardous exposure
Surge	4 S's	Space, Staff, Stuff, Systems — all four must scale together

13 Crisis Standards of Care and Ethical Resource Allocation

Conventional and contingency care preserve usual standards of care through substitution and conservation of resources. When demand overwhelms even contingency measures, a jurisdiction may formally transition to crisis standards of care (CSC): a documented, government- or institution-sanctioned shift in the level of care that can be delivered, intended to do the greatest good for the greatest number rather than the maximum for each individual patient. This transition is a formal declaration, not an informal bedside decision, and should trigger activation of a predetermined triage framework rather than ad hoc rationing by individual clinicians.

Principles of scarce-resource allocation

Allocation frameworks developed before a crisis (rather than improvised during one) consistently emphasize: transparency in the criteria used; consistency in applying the same criteria across all patients rather than case-by-case judgment; use of objective, validated physiologic scoring rather than perceived social worth or quality of life; and separation of the triage decision-maker from the treating clinician where possible, so the bedside team can remain focused on the individual patient in front of them rather than on population-level trade-offs.

Explicitly excluded from allocation criteria in essentially all published frameworks: age alone, disability status, race, ethnicity, insurance or ability to pay, and perceived social worth or "value" to society. Reliance on these factors is both ethically unsound and, in many jurisdictions, unlawful.

Triage teams and documentation

A triage officer or triage committee - not the bedside provider - applies the pre-established framework and documents the reasoning for each allocation decision. This structure protects both patients (consistent application of criteria) and staff (moral distress is reduced when a resource-allocation decision is institutional

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rather than individually authored under duress). Reassessment at defined intervals allows resources to be reallocated as a patient's trajectory becomes clearer, rather than treating the initial triage decision as permanent.

Legal and moral protections for responders

Most jurisdictions extend some form of liability protection to providers and facilities operating under a formally declared crisis-standards-of-care period, provided care is delivered in good faith and consistent with the declared framework; know whether and how this protection is activated in your own system before it is needed. Institutions should also anticipate the psychological toll of crisis triage on staff and build in structured debriefing and mental health support, since moral injury from resource-allocation decisions made under crisis conditions is a recognized occupational risk in disaster response.

This chapter synthesizes content consistent with the National Disaster Life Support Foundation (NDLSF) Advanced Disaster Life Support (ADLS) curriculum, FEMA National Incident Management System (NIMS)/Incident Command System guidance, CDC and federal (ASPR/REMM) SALT and START mass-casualty triage guidance, and OSHA HAZWOPER/CBRN PPE standards. It is an educational reference only and is not a substitute for completion of an accredited ADLS/BDLS course or official course certification.

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LIFE SUPPORT COURSES · CLINICAL COMPANION MANUAL

CHAPTER 5

NRP / NALS

Neonatal Resuscitation Program

1 Clinical Snapshot

Roughly ten percent of newborns need some assistance to begin breathing at birth, and about one percent need extensive resuscitation including chest compressions or medications. Because these events are individually rare at any single delivery but collectively common across a busy service, every birth attendant must be ready to act within seconds. The Neonatal Resuscitation Program (NRP), developed jointly by the American Academy of Pediatrics (AAP) and the American Heart Association (AHA), is the standardized framework used in the United States and widely internationally to prepare, evaluate, and resuscitate the newly born infant in the first minutes after delivery. The program's algorithm is built around a repeating cycle of rapid evaluation and response, escalating from warmth and stimulation, through positive-pressure ventilation, to chest compressions and medications, with continuous reassessment driving each next step.

The typical patient is not a single stereotype but a spectrum: the term infant who is simply slow to transition and responds to stimulation and drying; the preterm infant who needs help establishing a stable airway and gentle initial ventilation; and the truly depressed, apneic, bradycardic newborn who requires the full sequence of positive-pressure ventilation, coordinated chest compressions, and epinephrine. NRP skills are exercised in the delivery room and operating room at every hospital birth, in neonatal intensive care unit (NICU) admissions and transports, and in out-of-hospital and community birth settings where the margin for error is narrower and additional personnel may be delayed.

Why this matters · Typical patient · Where you meet it

Why it matters: Untreated perinatal transition failure is a leading cause of neonatal death and hypoxic-ischemic brain injury worldwide; the first "Golden Minute" after birth determines much of the subsequent trajectory.

Typical patient: A newborn who is apneic, gasping, floppy, or bradycardic in the first seconds to minutes of life, spanning term infants with acute depression to extremely preterm infants needing respiratory support from the first breath.

Common settings: Hospital labor and delivery/operating rooms, NICU admission and stabilization, neonatal transport teams, and community or out-of-hospital birth attended by EMS, midwives, or family physicians.

2 Learning Objectives

By the end of this chapter the reader should be able to:

- Identify antenatal and intrapartum risk factors that should trigger an expanded resuscitation team and equipment check before delivery.
- Perform the rapid initial evaluation and correctly sequence the initial steps of warming, positioning, clearing the airway if needed, drying, and stimulating.

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- State current evidence-based practice for umbilical cord management, including deferred cord clamping and the limited role of cord milking.
- Describe indications, technique, and corrective (MR SOPA) steps for positive-pressure ventilation, and recognize when to escalate to an advanced airway.
- Perform coordinated chest compressions using the 3:1 compression-to-ventilation ratio and two-thumb encircling technique.
- State the dose, route, and timing of epinephrine and volume expanders in neonatal resuscitation, and describe emergency vascular access options.
- Apply the targeted preductal oxygen saturation table to titrate supplemental oxygen during resuscitation.
- Describe current management of meconium-stained amniotic fluid, including when tracheal suctioning is and is not indicated.
- Recognize special scenarios (extreme prematurity, congenital diaphragmatic hernia) and the framework for considering discontinuation of resuscitation.
- Assign an Apgar score accurately and understand its role (and limitations) in guiding resuscitation.

3 Anticipation and Preparation

Most neonatal resuscitations can be anticipated. A structured review of antenatal and intrapartum risk factors, performed before every delivery, identifies the roughly one in ten births that will need more than routine care and allows the team to mobilize personnel and equipment before the infant is born rather than after. When risk factors are present, at least one person whose only responsibility is the newborn — and who is skilled in initiating resuscitation including positive-pressure ventilation — should be present, with a fully trained resuscitation team immediately available for higher-risk deliveries such as suspected extreme prematurity or major fetal anomaly.

Table 5.1 Selected antenatal and intrapartum risk factors warranting an expanded resuscitation team

Category	Examples
Antenatal	Multiple gestation; preterm (<37 weeks) or post-term (>41 weeks) gestation; known fetal anomaly or hydrops; maternal diabetes, hypertension, or substance use; oligohydramnios/polyhydramnios; no prenatal care
Intrapartum	Category II/III fetal heart rate tracing; chorioamnionitis/maternal fever; placental abruption or placenta previa with bleeding; prolapsed cord or cord avulsion; shoulder dystocia; operative or instrumented delivery; general anesthesia; meconium-stained amniotic fluid
Maternal	Maternal cardiac arrest or hemodynamic instability; opioid administration close to delivery; maternal hemorrhage

Any single risk factor may not require a full team, but multiple concurrent factors, or an unpredictable event during labor, should prompt escalation before delivery.

Equipment failure or unavailability at the moment of need is a preventable cause of poor outcome. A standardized pre-delivery equipment check, performed at the start of every shift and again immediately

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before high-risk deliveries, confirms that the radiant warmer is on and pre-heated, that suction is functioning, and that airway, ventilation, and medication supplies are complete and within their expiration dates.

Table 5.2 Delivery room equipment and supply checklist (abbreviated)

Category	Key items
Thermal management	Pre-warmed radiant warmer, warmed towels/blankets, polyethylene wrap or bag (for infants <32 weeks), chemical warming mattress, cap, servo temperature probe, room temperature 23–25 °C for very preterm deliveries
Airway/suction	Bulb syringe, suction catheters (8F–10F), meconium aspirator, appropriately sized face masks (term and preterm), laryngoscope with straight blades (00–1), endotracheal tubes (2.5–4.0 mm), stylet, CO2 detector, laryngeal mask airway
Ventilation	T-piece resuscitator or self-inflating bag with manometer, blended oxygen/air source with flowmeter, pulse oximeter with neonatal probe
Circulation/medications	Umbilical catheterization tray, 3.5F/5F umbilical catheters, intraosseous needle, epinephrine 1:10,000, normal saline for flush and volume expansion, syringes, three-way stopcocks, ECG leads/cardiac monitor
Documentation/team	Apgar timer/clock, code sheet, warmed identification bands, dedicated roles assigned before delivery

Practice Pearls

Every delivery, not just high-risk ones, should have at least one person capable of initiating newborn resuscitation immediately available and with no other competing duties.

A brief pre-delivery team briefing — anticipated gestational age, identified risk factors, assigned roles, and equipment confirmation — takes under two minutes and measurably improves team performance.

Warm the room and the warmer before the infant arrives; hypothermia on admission to the NICU is independently associated with increased mortality.

4 The NRP Algorithm Overview

The NRP algorithm is organized as a repeating loop: evaluate, decide, act, then re-evaluate. Three questions are asked immediately after birth — term gestation, good tone, and breathing or crying — to decide whether the infant can stay with the mother for routine care or needs to move to the warmer for further evaluation. From that point, heart rate and respiratory effort (assessed primarily by auscultation and pulse oximetry, with ECG monitoring added early once resuscitation beyond the initial steps is needed) drive every subsequent decision, with roughly 30- to 60-second blocks allotted to each intervention before reassessment. The algorithm diagram below shows the full decision branches; Table 5.3 that follows condenses the same sequence into a step-by-step reference for quick lookup.

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ALGORITHM — THE NRP ALGORITHM

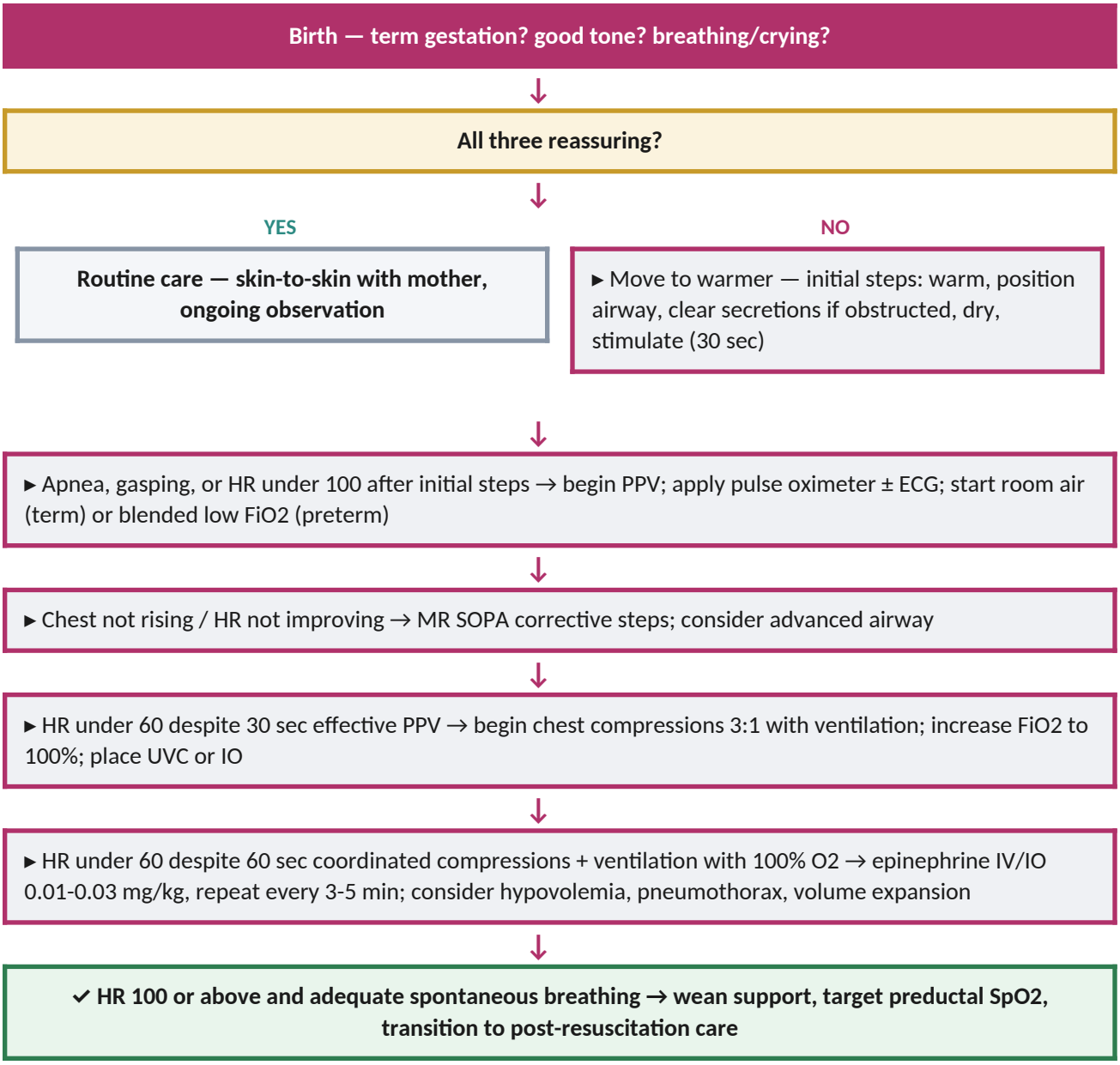


Figure 5.1. Adapted from the AAP/AHA NRP algorithm; see Table 5.3 for the corresponding step table and consult the official course diagram for full concurrent considerations.

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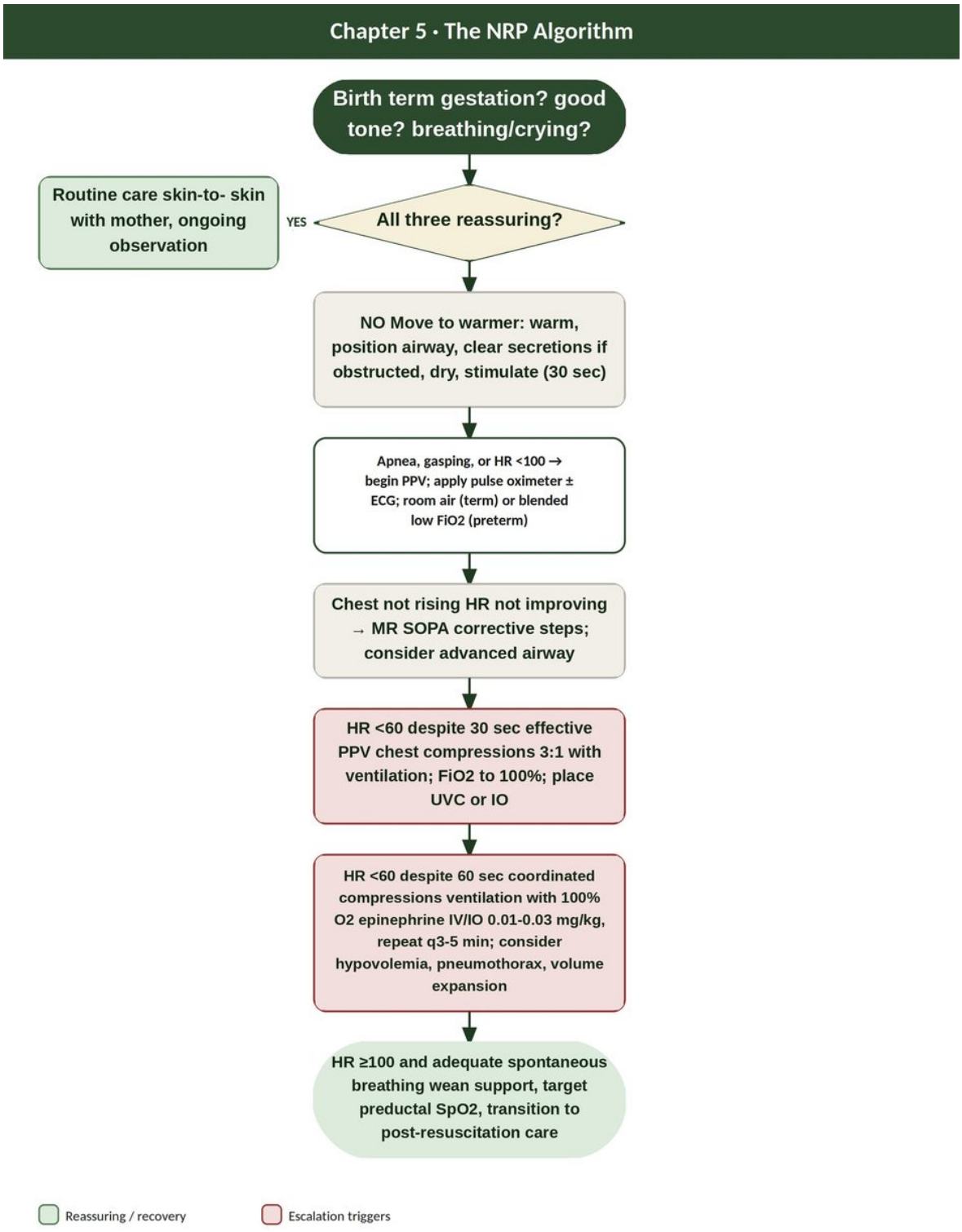


Table 5.3 NRP algorithm — structured step-by-step flow

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Step	Rapid evaluation / trigger	Action	Reassess after
0	Before delivery	Antenatal risk assessment, equipment check, team briefing, assign roles, plan cord management	—
1	At birth: term gestation? good tone? breathing/crying?	If all yes: routine care, skin-to-skin with mother, ongoing observation. If any no: move to warmer	Immediate
2	At warmer	Initial steps: warm, position airway, clear secretions only if obstructed, dry (except very preterm), stimulate	30 seconds
3	Apnea, gasping, or HR <100 bpm after initial steps	Begin positive-pressure ventilation (PPV); apply pulse oximeter ± ECG leads; start with room air (term) or blended low FiO2 (preterm)	15 seconds, then every 30 seconds
4	Chest not rising / HR not improving with PPV	Perform MR SOPA corrective steps; consider advanced airway (ETT or laryngeal mask)	30–60 seconds
5	HR <60 bpm despite 30 seconds of effective PPV (via advanced airway if possible)	Begin chest compressions coordinated 3:1 with ventilation; increase FiO2 to 100%; place UVC or IO	60 seconds
6	HR <60 bpm despite 60 seconds of coordinated compressions + ventilation with 100% oxygen	Administer epinephrine IV/IO (repeat every 3–5 minutes); consider hypovolemia, pneumothorax, and volume expansion	Continuous, reassess HR every 60 seconds
7	HR ≥100 bpm and adequate spontaneous breathing	Wean support, target SpO2 per minute-of-life table, transition to post-resuscitation/routine care	Ongoing

Adapted in condensed table form from the AAP/AHA NRP algorithm; consult the official flowchart for full decision branches and concurrent considerations.

Key Numbers at a Glance

Initial PPV rate: 40–60 breaths/min (acceptable range roughly 30–60/min)

Initial peak inspiratory pressure: 20–25 cmH2O (term), lower for preterm; PEEP 5 cmH2O

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Chest compressions begin: HR <60 bpm despite 30 sec of effective PPV

Compression:ventilation ratio: 3:1 (90 compressions + 30 breaths = 120 events/min)

Epinephrine: IV/IO 0.01–0.03 mg/kg of 1:10,000 solution, repeat every 3–5 min

Deferred cord clamping: at least 60 seconds for infants not requiring immediate resuscitation

5 Initial Steps and Thermoregulation

The initial steps — providing warmth, positioning the airway, clearing secretions only if they are obstructing, drying, and stimulating — are performed within the first 30 seconds after an infant is moved to the warmer and accomplish two goals simultaneously: they are often sufficient to trigger spontaneous respiration, and they prevent the compounding harm of hypothermia. The airway is positioned in a neutral or slightly extended "sniffing" position; a rolled towel under the shoulders can help maintain this position, particularly in an infant with a prominent occiput. Routine deep oropharyngeal or nasopharyngeal suctioning is no longer performed for every newborn — it can trigger vagally mediated bradycardia and provides no benefit for infants with clear secretions — and suctioning is now reserved for infants with visible airway obstruction or those unable to establish effective ventilation.

Tactile stimulation (rubbing the back or flicking the soles of the feet, briefly and no more than twice) is appropriate for an infant who is not breathing adequately after drying and positioning. Stimulation should not be prolonged or repeated if there is no response — a non-responsive infant needs PPV, not further stimulation.

Thermoregulation, including for the preterm infant

Admission hypothermia (temperature below 36.5 °C) is common, preventable, and independently associated with increased neonatal mortality and morbidity, including intraventricular hemorrhage, respiratory distress, hypoglycemia, and late-onset sepsis. The target is a normal admission temperature between 36.5 °C and 37.5 °C, achieved through a bundle of interventions rather than any single step: a pre-warmed radiant warmer, an ambient delivery/operating room temperature of at least 23–25 °C for anticipated very preterm birth, prompt drying and removal of wet linens for term and moderately preterm infants, a cap, and — critically for infants born before about 32 weeks' gestation — placing the undried infant's torso and limbs directly into a polyethylene wrap or bag immediately after birth without drying first, combined with an exothermic (chemical) warming mattress and, where available, servo-controlled temperature monitoring. Hyperthermia should be avoided as well; it carries its own risks and warming measures should be titrated, not maximized indiscriminately.

Busy-Shift Tip

For an anticipated preterm delivery, turn the warmer temperature up and the room thermostat up well before the birth — thermal preparation cannot be rushed retroactively once the infant is out.

Keep the polyethylene bag and cap within arm's reach at every preterm delivery setup, not just those already flagged extremely preterm; gestational age estimates are sometimes wrong.

6 Umbilical Cord Management

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Current guidance favors deferred (delayed) cord clamping for at least 60 seconds after birth for both term and preterm infants who do not require immediate resuscitation, allowing placental transfusion that improves iron stores, reduces the incidence of intraventricular hemorrhage in preterm infants, and improves transitional hemodynamics. The infant can typically be dried, stimulated, and even receive initial evaluation while the cord remains intact and unclamped, at or below the level of the placenta or on the mother's abdomen/chest. For a non-vigorous infant who appears to need resuscitation, current recommendations still favor attempting to accomplish the initial steps and even PPV with an intact cord when feasible ("resuscitation with intact cord"), but clamping should not be delayed if it would meaningfully delay needed resuscitative interventions or if the clinical situation (e.g., maternal hemorrhage, need to separate for CPR) does not allow it.

Umbilical cord milking — stripping placental blood toward the infant over a few seconds before clamping — is not a substitute for deferred clamping and carries a distinct risk profile. It may be considered as an alternative when deferred clamping is not feasible in infants born between approximately 28 and 36+6 weeks' gestation, but it is not recommended in infants born before 28 weeks' gestation, where rapid volume shifts have been associated with an increased risk of severe intraventricular hemorrhage.

Myth vs Fact — Cord Management

Myth: A non-vigorous or depressed infant should always be clamped and cut immediately to start resuscitation on the warmer.

Fact: When feasible without meaningfully delaying resuscitative care, initial steps and even PPV can be started with the cord intact; clamping is deferred to preserve placental transfusion, and only expedited when the clinical situation genuinely requires separation.

Myth: Cord milking is a safe, faster alternative to delayed clamping for all preterm infants.

Fact: Milking is reasonable only for infants roughly 28–36+6 weeks when deferred clamping is not possible, and it is avoided below 28 weeks because of an increased risk of severe intraventricular hemorrhage.

7 Positive-Pressure Ventilation (PPV)

Establishing effective ventilation is the single most important and most effective action in neonatal resuscitation — the vast majority of newborns who need help respond to PPV alone, without ever requiring compressions or medications. PPV is indicated for an infant who is apneic or gasping, or whose heart rate remains below 100 bpm after the initial steps, and should begin within the first 60 seconds of life ("the Golden Minute") when indicated. A T-piece resuscitator is generally preferred over a self-inflating bag when compressed gas is available because it delivers more consistent pressures and allows sustained PEEP, but a self-inflating bag remains an essential and effective backup, particularly where compressed gas is unavailable.

Initial settings are a peak inspiratory pressure (PIP) of approximately 20–25 cmH2O for a term infant (occasionally higher if the chest fails to rise and corrective steps have been exhausted), positive end-expiratory pressure (PEEP) of 5 cmH2O, and a ventilation rate in the range of 40–60 breaths per minute ("breathe-two-three, breathe-two-three"), with contemporary guidance accepting a somewhat broader 30–60/min range. Preterm infants typically start with lower initial pressures and lower initial oxygen concentrations, titrated to chest rise and the targeted SpO2 table rather than to a fixed number. The single best indicator that PPV is effective is a rising heart rate; visible, symmetric chest rise without excessive rise (which risks volutrauma and pneumothorax) is the secondary indicator.

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When ventilation is not working: MR SOPA

If the heart rate does not improve and the chest is not moving with PPV, do not simply continue unchanged ventilation or jump immediately to intubation — work through the standardized corrective steps, summarized by the mnemonic MR SOPA, checking after each pair of steps for chest movement.

Table 5.4 MR SOPA ventilation corrective steps

Letter	Step	What to check/do
M	Mask adjustment	Reapply the mask to achieve a firm seal around the mouth and nose; reposition fingers/hand to eliminate leak
R	Reposition the airway	Adjust head to a neutral/slightly extended sniffing position; consider a shoulder roll
S	Suction mouth and nose	Clear secretions or blood with a bulb syringe or suction catheter if visibly obstructing the airway
O	Open the mouth	Open the infant's mouth slightly and lift the jaw forward (jaw thrust) to relieve obstruction behind the tongue
P	Pressure increase	Increase PIP in small increments (e.g., by 5–10 cmH2O) if chest still not moving after M-R-S-O
A	Alternative airway	Place an endotracheal tube or laryngeal mask airway if chest rise remains inadequate after all prior steps

Reassess for chest movement and heart rate response after each cluster of steps (MR, then SOPA) rather than waiting until all six are attempted.

Decision Point — Escalating the Airway

If MR SOPA corrective steps fail to produce adequate chest rise and heart rate response with a face mask, move promptly to an advanced airway rather than persisting with an ineffective mask technique.

A supraglottic (laryngeal mask) airway is an acceptable — and in some centers preferred initial — alternative to endotracheal intubation for infants born at 34 weeks' gestation or later, particularly when intubation is difficult or delayed.

For infants below approximately 34 weeks or when a laryngeal mask does not fit or function, endotracheal intubation remains the advanced airway of choice; confirm placement with an exhaled CO2 detector and clinical signs.

8 Chest Compressions

Chest compressions are indicated when the heart rate remains below 60 bpm after at least 30 seconds of PPV that is judged to be effectively moving the chest — ideally delivered via an advanced airway, since ineffective

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ventilation is the most common reason compressions fail to improve heart rate. Before starting compressions, confirm and optimize ventilation (MR SOPA, consider intubation) and increase the delivered oxygen concentration to 100%; compressions superimposed on ineffective ventilation will not improve oxygen delivery.

The two-thumb encircling-hands technique is preferred over the two-finger technique because it generates higher and more consistent peak systolic and coronary perfusion pressures and is less fatiguing for the compressor, allowing the thumbs to be placed side-by-side or overlapping on the lower third of the sternum (just below an imaginary line connecting the nipples), with the fingers encircling the torso and supporting the back. Compression depth is approximately one-third of the anterior-posterior chest diameter, with full chest recoil between compressions. Compressions and ventilation are coordinated, never simultaneous, at a ratio of 3 compressions to 1 breath, delivered as "one-and-two-and-three-and-breathe-and," achieving approximately 90 compressions and 30 breaths (120 total events) per minute. A second provider should attach ECG leads if not already placed, as auscultation and pulse oximetry become unreliable during compressions; heart rate is reassessed after 60 seconds of coordinated compressions and ventilation without interrupting compressions to check more frequently than that.

Red Flags During Compressions

Heart rate not improving after 60 seconds of well-coordinated 3:1 compressions and 100% oxygen PPV via a secured advanced airway — proceed to epinephrine and obtain emergency vascular access if not already in place.

Absent breath sounds on one side, a shifted apical impulse, or sudden deterioration during PPV/compressions should raise suspicion for pneumothorax; consider needle decompression.

Pallor, poor perfusion, weak pulses, or a history consistent with acute blood loss (e.g., placental abruption, vasa previa, fetomaternal hemorrhage) despite adequate ventilation and compressions should prompt early consideration of volume expansion.

9 Medications

Fewer than 0.2% of newborns require medications during resuscitation, but when the heart rate remains below 60 bpm despite 60 seconds of coordinated chest compressions and effective PPV with 100% oxygen, epinephrine is indicated and should not be delayed. The intravenous (umbilical venous catheter) or intraosseous route is strongly preferred over the endotracheal route because absorption from the lung is slow and unreliable; the endotracheal route, at a substantially higher dose, is reserved for the brief interval while vascular access is being obtained, and IV/IO dosing should follow as soon as access is achieved.

Table 5.6 Neonatal resuscitation drug and fluid doses

Agent	Route	Dose	Notes
Epinephrine (1:10,000 = 0.1 mg/mL)	IV or IO	0.01–0.03 mg/kg (0.1–0.3 mL/kg), flush with 3 mL normal saline	Preferred route; may repeat every 3–5 minutes if HR remains <60 bpm
Epinephrine (1:10,000 = 0.1 mg/mL)	Endotracheal	0.05–0.1 mg/kg (0.5–1 mL/kg)	Only while establishing IV/IO access; switch to IV/IO dosing as soon as access obtained; do not use the higher dose IV

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Agent	Route	Dose	Notes
Normal saline (volume expander)	IV or IO	10 mL/kg over 5–10 minutes	For suspected hypovolemia/acute blood loss with poor response to resuscitation; may repeat once
O-negative uncrossmatched (or type-specific) blood	IV or IO	10 mL/kg over 5–10 minutes	Preferred over crystalloid when acute significant blood loss is suspected or confirmed
Dextrose 10% in water	IV	2 mL/kg, then infusion	Not a resuscitation medication per se, but indicated post-resuscitation for confirmed or high-risk hypoglycemia

Naloxone is no longer recommended during acute neonatal resuscitation; respiratory depression from maternal opioids is managed with PPV, as naloxone does not address the underlying apnea and carries risks including precipitated withdrawal/seizures in an opioid-exposed infant.

Monitoring Checklist During Escalating Resuscitation

Pulse oximeter probe on the right hand or wrist (preductal) applied as soon as PPV is anticipated or begun.

ECG leads applied promptly once compressions are anticipated — auscultation and pulse oximetry both become unreliable during chest compressions and low-flow states.

Confirm advanced airway placement (if used) with an exhaled CO2 detector, not clinical impression alone.

Reassess heart rate approximately every 60 seconds during compressions/medications; avoid over-frequent interruptions that reduce coronary perfusion.

10 Vascular Access

Emergency umbilical venous catheterization (UVC) is the standard route for medications and volume during neonatal resuscitation because the umbilical vein is large, accessible, and requires no needle stick in a small, often hypothermic and hypotensive infant. After sterile (or expedited clean, in a true emergency) preparation, the cord is cut flat approximately 1–2 cm from the skin, the single thin-walled umbilical vein (typically at the 12 o'clock position, larger and more oval than the two thicker-walled arteries) is cannulated with a saline-flushed 3.5F (very preterm) or 5F umbilical catheter, and the catheter is advanced only until free blood return is obtained — typically 2–4 cm in a term infant for emergency "low-lying" placement — rather than threaded to full central position, which can be confirmed and adjusted later by radiograph once the emergency has passed.

Intraosseous (IO) access is now formally recognized as an acceptable alternative to UVC when umbilical access is not readily available, technically difficult, or has failed, and should not be viewed as a last resort reserved only after prolonged UVC attempts. A neonatal or pediatric IO needle is placed in the proximal tibia

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(flat anteromedial surface, 1–2 cm below and medial to the tibial tuberosity), and all resuscitation medications and fluids can be given by this route at the same doses as IV/IO.

Table 5.7 Emergency vascular access — UVC versus IO

Feature	Umbilical venous catheter (UVC)	Intraosseous (IO)
Typical scenario	First-line for delivery room resuscitation with intact/fresh cord	Alternative when UVC unavailable, difficult, or failed; also used after cord is no longer accessible
Site	Umbilical vein, cut cord stump	Proximal tibia (anteromedial, below tibial tuberosity)
Key steps	Sterile prep, cut cord flat, identify thin-walled vein, cannulate, advance to free blood return, secure	Stabilize leg, identify landmark, insert IO needle perpendicular to bone until "give"/loss of resistance, confirm marrow return or free flow, secure
Advantages	Large, familiar vessel; no bone penetration; can be left for ongoing NICU care	Fast to place; usable in infants of any size; not dependent on cord anatomy
Cautions	Requires cord stump access; risk of arterial cannulation, infection, thrombosis if left long-term	Risk of extravasation, growth plate injury, compartment syndrome if misplaced; typically removed once UVC or peripheral IV secured

11 Targeted Preductal Oxygen Saturation

Healthy newborns do not reach adult-range oxygen saturations immediately at birth; saturations climb gradually over roughly the first ten minutes as fetal-to-neonatal circulatory transition completes. Resuscitation is guided by preductal SpO2 (measured with the probe on the right hand or wrist) referenced against a minute-of-life target range rather than a single fixed number, avoiding both hypoxia and the harms of hyperoxia (which is particularly important in preterm infants, where excess oxygen is linked to retinopathy of prematurity and other oxidative injury).

Table 5.5 Targeted preductal SpO2 by minute of life

Minute of life	Target preductal SpO2
1 minute	60%–65%
2 minutes	65%–70%
3 minutes	70%–75%
4 minutes	75%–80%
5 minutes	80%–85%
10 minutes	85%–95%

Supplemental oxygen is titrated up or down to keep the infant within this range; a pulse oximeter typically takes 60–90 seconds to acquire a reliable signal, so do not delay indicated PPV while waiting for the reading.

Initial oxygen concentration is now stratified by gestational age rather than uniformly starting at 21% or 100%. Term and late preterm infants start on room air (21%); moderately preterm infants start on a modest

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blend; and the most immature infants may start with a higher initial concentration, titrated promptly to the target table using the pulse oximeter, rather than fixed rigidly at any single starting point.

Table 5.8 Initial FiO2 by gestational age at start of PPV

Gestational age	Suggested starting FiO2	Titration
≥35 weeks (term/late preterm)	21% (room air)	Increase if SpO2 remains below target despite effective ventilation
30–34+6 weeks (moderate preterm)	21%–30%	Titrate to targeted SpO2 table
<30 weeks (very/extremely preterm)	30%–100%, per local protocol	Wean promptly toward target once saturations rise; avoid sustained hyperoxia

Local unit protocols and blender availability may specify a single institutional starting point within these ranges; always titrate by pulse oximetry rather than leaving FiO2 fixed.

12 Meconium-Stained Amniotic Fluid

Meconium-stained amniotic fluid (MSAF) complicates roughly 10% of deliveries and can lead to meconium aspiration syndrome. Management has changed substantially over the past decade. Routine intrapartum oropharyngeal and nasopharyngeal suctioning on the perineum before delivery of the shoulders is not recommended — it has not been shown to reduce meconium aspiration syndrome and offers no benefit. After birth, the historical distinction between a "vigorous" and "non-vigorous" infant with MSAF, which previously dictated routine direct tracheal suctioning via endotracheal tube for non-vigorous infants, has been superseded: routine tracheal intubation and suctioning for meconium is no longer recommended even for a depressed, non-vigorous infant, because trials showed no reduction in meconium aspiration syndrome and intubation attempts can meaningfully delay initiation of PPV, worsening outcomes.

Current practice is to bring every MSAF infant promptly to the warmer, begin the standard initial steps, and proceed to PPV if the infant is apneic, gasping, or bradycardic — exactly as for any other newborn. Airway suctioning (oral, then nasal, with a bulb syringe or catheter) is reserved for infants with visible obstruction of the airway or those in whom PPV is not moving the chest despite MR SOPA corrective steps, in which case direct laryngoscopy with suctioning under direct vision (and, if particulate meconium is seen obstructing the trachea, endotracheal suctioning) is reasonable as part of the corrective sequence rather than as a routine first step.

Table 5.9 Meconium-stained amniotic fluid — current management approach

Infant status at birth	Action
Vigorous or non-vigorous, any tone	Do NOT perform routine intrapartum or routine post-birth tracheal suctioning; proceed directly to standard initial steps at the warmer
Breathing/crying, good tone, HR ≥100	Routine care; observe closely for evolving respiratory distress over the following hours
Apneic/gasping or HR <100 after initial steps	Begin PPV promptly, exactly as for any depressed newborn; do not delay ventilation to attempt tracheal suctioning
Visible airway obstruction, or chest not moving with PPV despite MR SOPA	Laryngoscopy with suctioning under direct vision; endotracheal suctioning if particulate meconium

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Infant status at birth	Action
	obstructs the airway, then resume PPV without delay

13 Special Considerations

Extreme prematurity

For infants at the borderline of viability (roughly 22–24 weeks' gestation), decisions about whether to initiate resuscitation are individualized, made ideally through antenatal counseling with the family and a shared understanding of local outcome data, and should be reflected in the pre-delivery team briefing so the delivery room team is not making that decision for the first time as the infant is being born. When resuscitation is undertaken, gentle initial PPV with lower starting pressures, meticulous thermal management (polyethylene wrap, cap, warmed transport), and cautious oxygen titration to avoid hyperoxia are all emphasized given the extreme fragility of the immature lung and germinal matrix.

Congenital diaphragmatic hernia

When congenital diaphragmatic hernia (CDH) is suspected or known antenatally, face-mask PPV should be avoided if possible because insufflating the herniated bowel further compromises already hypoplastic lungs; early endotracheal intubation is preferred if respiratory support is needed. An orogastric tube should be placed early and left to suction/gravity drainage to decompress the stomach and reduce mass effect on the thorax. Ventilation pressures should be kept as low as effective, accepting a higher PaCO2 (permissive hypercapnia) where feasible, given the severe underlying pulmonary hypoplasia and risk of pneumothorax in the contralateral lung.

Other anomalies affecting the airway

- Suspected choanal atresia: an oral airway or McGovern nipple may be needed since these infants are obligate nasal breathers in significant distress.
- Robin sequence/micrognathia: prone or lateral positioning and airway adjuncts (nasopharyngeal airway or laryngeal mask) may be needed before or instead of laryngoscopy.
- Suspected tracheoesophageal fistula/esophageal atresia: avoid prolonged bag-mask ventilation if feasible (risk of gastric distension); a replogle tube to low suction and early surgical consultation are indicated.

Considering discontinuation of resuscitation

When a newborn has no detectable heart rate despite all steps of resuscitation performed correctly and completely — effective ventilation confirmed via an advanced airway, adequately coordinated chest compressions, and appropriately dosed epinephrine given by an adequate route — it is reasonable to discuss discontinuing resuscitative efforts with the team at approximately 20 minutes of asystole. This is a substantial change from older, shorter time thresholds and reflects evidence that some infants without a detectable heart rate at 10 minutes have gone on to survive, sometimes without severe disability, when resuscitation was technically adequate throughout. The decision should be individualized — considering the presumed etiology, gestational age, resource setting, and quality of resuscitation actually delivered — and, wherever possible, should involve real-time communication with the family and clear team debriefing afterward regardless of outcome.

Decision Point — Should We Stop?

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Confirm technical adequacy first: is the airway actually patent and secured, is the chest genuinely moving with ventilation, are compressions correctly performed, has epinephrine been given by an adequate route and repeated appropriately?

If resuscitation has been technically adequate throughout and there is still no detectable heart rate by approximately 20 minutes, it is reasonable for the team to discuss stopping.

This is a team and, where feasible, family discussion — not a single provider's unilateral call — and should be followed by a structured debrief.

14 Apgar Scoring

The Apgar score, assigned at 1 and 5 minutes of life (and every 5 minutes thereafter if the 5-minute score is below 7, up to 20 minutes), is a standardized, rapidly communicable summary of the newborn's condition and response to resuscitation. It is a useful shorthand for documentation and communication, but it is not used to decide whether or when to initiate resuscitation — resuscitative steps are driven by real-time evaluation of tone, respiratory effort, and heart rate as they occur, not by waiting for a 1-minute score to be tallied.

Table 5.10 Apgar scoring system

Sign	0 points	1 point	2 points
Appearance (color)	Blue/pale all over	Body pink, extremities blue (acrocyanosis)	Completely pink
Pulse (heart rate)	Absent	<100 bpm	≥100 bpm
Grimace (reflex irritability)	No response to stimulation	Grimace/weak cry with stimulation	Cry or active withdrawal with stimulation
Activity (tone)	Limp	Some flexion of extremities	Active motion, well flexed
Respiration	Absent	Slow, irregular, weak cry	Good, strong cry

Score each category 0–2 and sum for a total of 0–10; when resuscitative interventions are in progress at the time of scoring (e.g., PPV, oxygen, compressions), the specific interventions in place should be documented alongside the score, as some expanded Apgar reporting forms require.

15 Worked Clinical Case

Case 5.1 — The Depressed Term Newborn

Presentation: A 39-week gestation infant is delivered by urgent cesarean section for a category III fetal heart rate tracing after a prolonged second stage. At delivery the infant is limp, apneic, and cyanotic. Amniotic fluid was clear. The pre-briefed team moves the infant to the warmer while beginning a 60-second countdown.

Management: Initial steps are performed: the infant is dried, positioned with a shoulder roll, and stimulated — no respiratory effort develops and the tone remains poor. At 30 seconds of life the team auscultates a heart rate of 70 bpm and begins PPV via T-piece resuscitator on room air, with the pulse oximeter probe placed on the right hand. At 45 seconds the chest is not visibly rising; MR SOPA corrective steps are performed in sequence — the mask is reapplied with a tighter seal and the head repositioned,

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which produces visible chest rise on reassessment. By one minute of life the heart rate has risen to 130 bpm with spontaneous, though still weak, respiratory effort; PPV is continued and gradually weaned as the infant's own respiratory effort strengthens over the next two minutes, with FiO2 titrated to keep preductal SpO2 within the minute-of-life target range. ECG leads had been placed as a precaution when compressions seemed possible but were never needed.

Outcome/teaching point: The infant is breathing comfortably in room air by 5 minutes of life with an Apgar score of 3 at one minute and 8 at five minutes, and is admitted to the newborn nursery for routine observation. This case illustrates the central teaching point of NRP: effective PPV, promptly initiated and corrected using MR SOPA when the first attempt does not work, resolves the overwhelming majority of neonatal resuscitations without ever requiring chest compressions or medications — the skill that matters most is recognizing early that ventilation is not working and fixing it fast, rather than escalating prematurely to advanced interventions or persisting with an ineffective technique.

16 Chapter-End Quick Reference

The table below consolidates the highest-yield numbers from this chapter for rapid bedside or pre-shift review.

Table 5.11 NRP quick-reference summary

Parameter	Value
Deferred cord clamping	At least 60 seconds if infant does not require immediate resuscitation
Cord milking	Consider 28–36+6 weeks only if DCC not feasible; avoid <28 weeks
Initial PPV rate	40–60 breaths/min (acceptable range ~30–60/min)
Initial PIP / PEEP	~20–25 cmH2O / 5 cmH2O (term); lower initial pressures for preterm
MR SOPA	Mask adjustment, Reposition airway, Suction, Open mouth, Pressure increase, Alternative airway
Start chest compressions	HR <60 bpm despite 30 seconds of effective PPV
Compression:ventilation ratio	3:1 (90 compressions + 30 breaths = 120 events/min), two-thumb encircling technique
Epinephrine (IV/IO)	0.01–0.03 mg/kg of 1:10,000, repeat q3–5 min; ET dose 0.05–0.1 mg/kg only while obtaining access
Volume expander	10 mL/kg normal saline or O-negative blood over 5–10 min for suspected hypovolemia
Targeted preductal SpO2	1 min 60–65%; 2 min 65–70%; 3 min 70–75%; 4 min 75–80%; 5 min 80–85%; 10 min 85–95%
MSAF management	No routine intrapartum or tracheal suctioning; proceed to standard initial steps/PPV as for any newborn
Consider discontinuing resuscitation	~20 minutes of no detectable heart rate despite

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Parameter	Value
	technically adequate resuscitation, individualized

Final Practice Pearls

The single highest-yield action in nearly every neonatal resuscitation is prompt, effective positive-pressure ventilation — most infants who need help need only this.

When PPV appears ineffective, work the MR SOPA sequence methodically rather than escalating pressure or jumping to intubation reflexively.

Confirm ventilation is truly effective (ideally via a secured advanced airway) before attributing a persistently low heart rate to a cardiac cause requiring compressions or epinephrine.

Anticipation, a pre-delivery team briefing, and a completed equipment check prevent the majority of delays that turn a routine transition into an emergency.

Content in this chapter reflects the AAP/AHA Neonatal Resuscitation Program (NRP) framework and the 2020 AHA Guidelines for CPR and ECC — Part 5: Neonatal Resuscitation, as subsequently modified by the 2023 AHA/AAP Focused Update on Neonatal Resuscitation and the 2025 AHA/AAP neonatal resuscitation guideline update (deferred cord clamping duration, stratified initial oxygen by gestational age, expanded role of intraosseous access, earlier supraglottic airway use, and non-routine airway suctioning including in meconium-stained fluid). This chapter is an educational reference summary only, is not exhaustive, and is not a substitute for completing an official, currently accredited NRP provider course and hands-on skills verification; always follow your institution's current protocols and the official AAP/AHA course materials in effect at the time of clinical care.

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CHAPTER 6

AHLS

Advanced Hazmat Life Support

1 Clinical Snapshot

Why this matters · Typical patient · Where you meet it

Why it matters: A hazmat patient arrives contaminated, symptomatic, and time-critical, and the clinician must diagnose a toxidrome and begin antidotal therapy while simultaneously protecting the receiving team, the department, and the facility from secondary contamination. Getting the sequence wrong (treating before decontaminating, or delaying antidotes to complete a perfect decon) can convert one casualty into a dozen and cost a life that speed would have saved.

Typical scenario: An industrial release of a volatile chemical (chlorine, ammonia, an organophosphate pesticide) or a transportation incident (tanker rollover, rail derailment) produces one or more symptomatic patients who self-transport or arrive by EMS still contaminated, sometimes before the department has been warned.

Common settings: Industrial and agricultural facilities, transportation corridors (rail, highway, pipeline), laboratories, water/wastewater treatment plants, and — for the deliberate-release subset — mass-gathering or transportation-hub terrorism using chemical, biological, radiological, or nuclear (CBRN) agents.

Advanced Hazmat Life Support (AHLS) content, developed through the University of Arizona and the AHLS International Foundation, trains clinicians to recognize toxic syndromes quickly, apply the correct level of personal protection, sequence decontamination ahead of definitive treatment, and dose a small set of high-stakes antidotes correctly under pressure. This chapter distills that framework into a bedside- and dispatch-usable reference; it does not replace certified AHLS provider training.

Most hazmat events a hospital sees are small and chemical rather than radiological or biological, but the rare large-scale or deliberate event is the one that overwhelms an unprepared emergency department. The clinical skills are the same at every scale: recognize the pattern, protect yourself and the facility, decontaminate, and treat.

2 Learning Objectives

- Describe the general approach to a contaminated patient, including the primacy of scene safety, PPE selection, and decontamination before hands-on treatment.
- Match PPE levels A through D to the corresponding hazard scenario and respiratory/skin protection provided.
- Outline the decontamination zones (hot/warm/cold) and the steps of gross, technical, and field decontamination.
- Recognize the five classic toxidromes (cholinergic, anticholinergic, sympathomimetic, sedative-hypnotic/opioid, serotonergic) from signs and symptoms alone.

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- Dose atropine, pralidoxime, and benzodiazepines correctly for nerve agent and organophosphate/carbamate poisoning, titrating to clinical endpoint.
- Select and dose cyanide antidotes (hydroxocobalamin, sodium thiosulfate, nitrites) appropriately, including when nitrites are contraindicated.
- Identify additional key antidotes for opioid, methemoglobinemia, hydrofluoric acid, and heavy-metal exposures.
- Apply basic radiological triage principles, including contamination versus irradiation and age-based potassium iodide dosing.
- Describe initial recognition and management of anthrax, botulism, and chemical asphyxiant/pulmonary irritant exposures.
- Outline hospital hazmat receiving protocol basics to protect staff and the facility during a contaminated-patient surge.

3 General Approach to the Hazmat Patient

Scene Safety and PPE Before Patient Contact

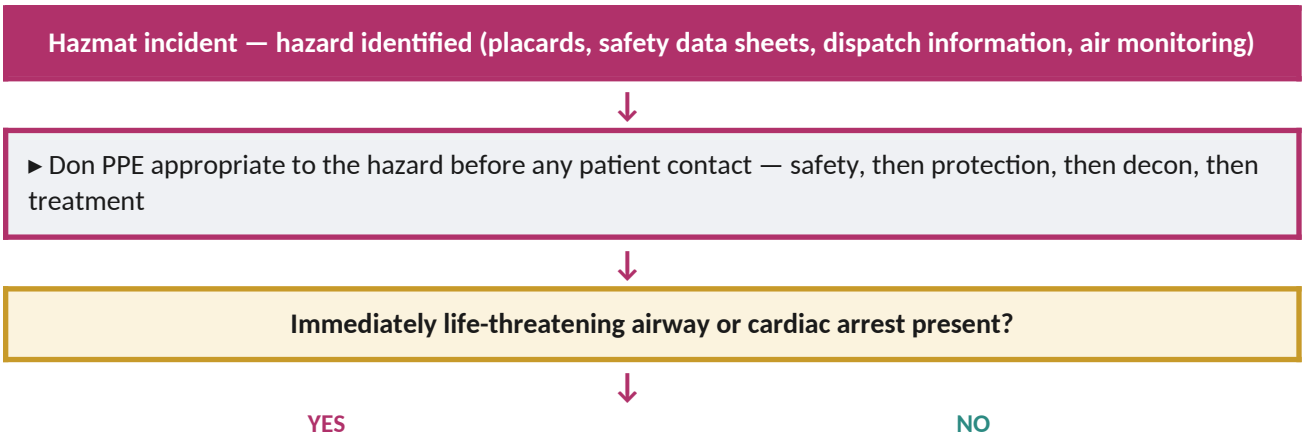
The single most important principle in hazmat medicine is sequence: safety first, then protection, then decontamination, then treatment. No responder or clinician approaches a contaminated patient without first identifying the hazard (placards, safety data sheets, dispatch information, or air monitoring) and donning PPE appropriate to that hazard. An untrained or unprotected rescuer who runs toward a downed victim routinely becomes the second patient — this is the most common preventable death in hazmat response.

Once protected, responders establish zones, remove patients from the source, and begin gross decontamination before any hands-on assessment or treatment beyond immediately life-saving airway maneuvers performed in appropriate PPE.

Decontamination Before Treatment

With rare exceptions, decontamination precedes treatment. Contaminated skin, hair, and clothing continue to off-gas vapor and transfer liquid contact hazard to every surface and person they touch, including IV lines, monitoring equipment, and the ambulance or emergency department itself. Treating an undecontaminated patient converts the treatment area into a hot zone and can incapacitate the treating team.

ALGORITHM — GENERAL APPROACH TO THE HAZMAT PATIENT



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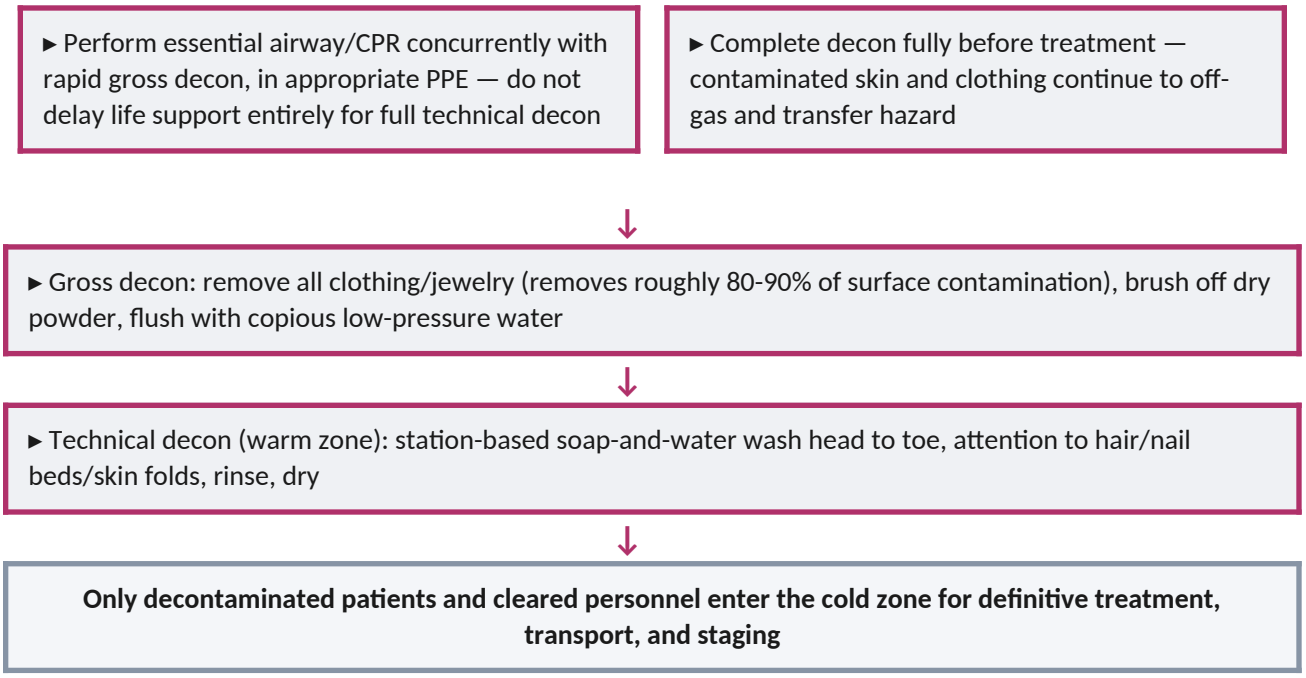
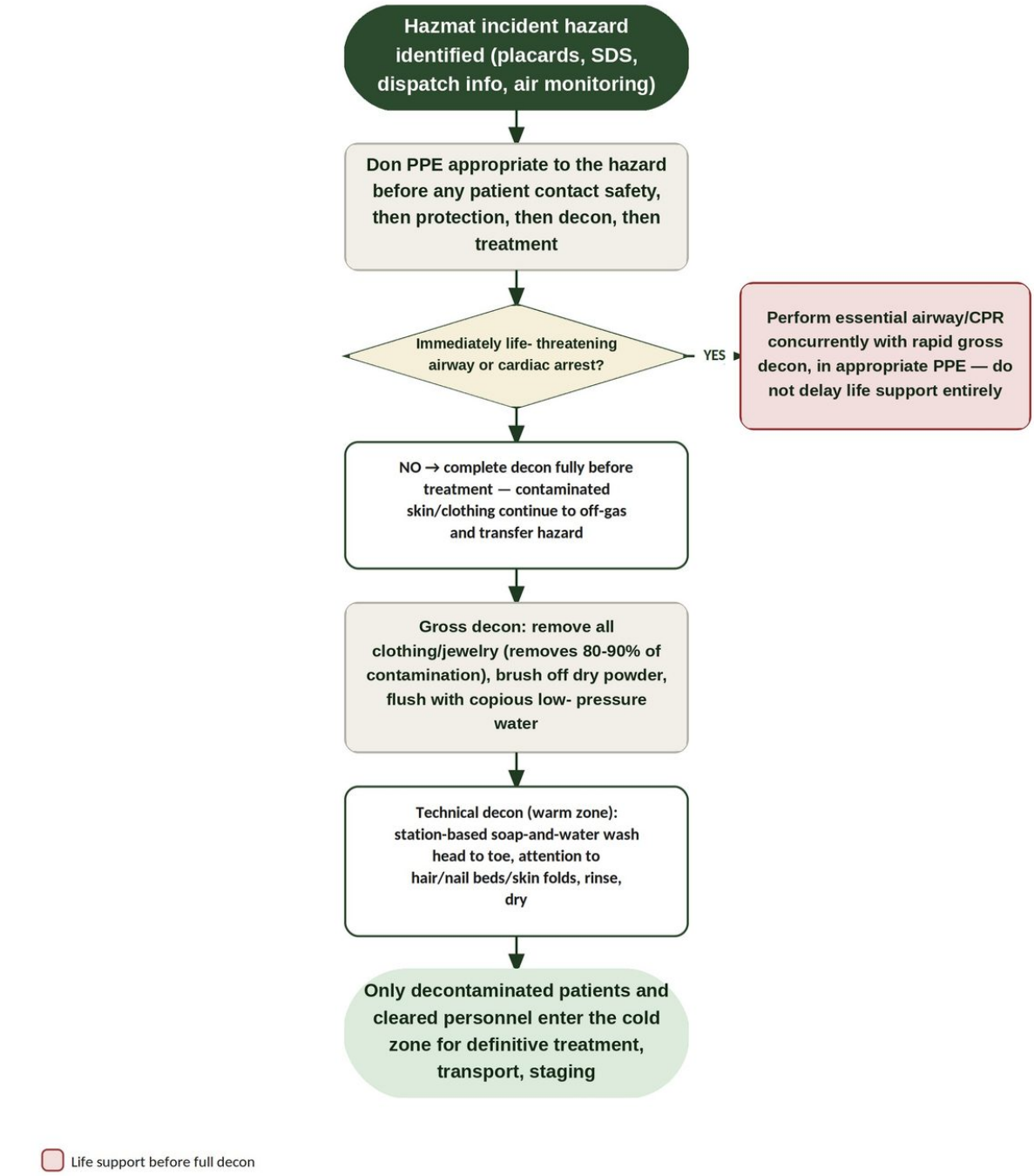


Figure 6.1. General hazmat decontamination sequence; see Table 6.2 for zone-by-zone PPE and activities.

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Chapter 6 · General Approach to the Hazmat Patient



Secondary Contamination Is a Real and Recurring Failure Mode

Emergency department staff have been sickened — sometimes requiring their own decontamination and hospitalization — after receiving a contaminated patient who bypassed pre-hospital decon.

Organophosphate pesticides, nerve agents, and many industrial liquids readily cross gloves and gowns not rated for chemical splash; standard exam gloves are not chemical PPE.

If a patient arrives contaminated and unannounced, the correct response is to redirect them to a

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designated outdoor decon area before ED entry, not to receive them in a standard treatment bay.

When Decon Can Be Deferred

For an immediately life-threatening airway or cardiac arrest in a contaminated patient, rescuers in appropriate PPE perform essential airway/CPR interventions concurrently with rapid gross decontamination rather than delaying life support entirely for a complete technical decon.

Dry powders and solid contaminants are brushed off before any water is applied — water can activate or spread some powdered agents (notably some reactive metals and certain nerve agent formulations).

Once the patient is stable enough to tolerate movement, full technical decon is completed before transport into a clean treatment area whenever feasible.

4 Personal Protective Equipment

PPE selection follows the OSHA/EPA four-level classification (A through D), driven by the identity of the hazard, its concentration relative to IDLH (Immediately Dangerous to Life or Health), and whether the primary risk is inhalational, dermal, or both. Higher letter does not mean higher protection — Level A is the most protective and Level D the least.

Table 6.1 PPE levels and use scenarios

Level	Respiratory Protection	Skin/Body Protection	Typical Use Scenario
A	Positive-pressure SCBA or supplied-air respirator worn inside the suit	Fully encapsulated, vapor/liquid/gas-tight chemical-resistant suit	Highest known or suspected hazard; unidentified substance, high-toxicity vapor/gas, or agent with significant skin-absorption risk (e.g., unknown nerve agent release)
B	Positive-pressure SCBA or supplied-air respirator	Chemical-resistant clothing (non-encapsulated, taped seams); highest respiratory protection but lesser skin protection than Level A	Substance identified, inhalation is the dominant hazard, oxygen-deficient or IDLH atmosphere present (e.g., initial rescue entry)
C	Air-purifying respirator (APR) or powered air-purifying respirator (PAPR) with agent-appropriate cartridge	Chemical-resistant splash suit	Contaminant identified and measured below IDLH, adequate ambient oxygen, correct cartridge available (e.g., decon-line personnel, known liquid splash hazard)
D	Standard work clothing; no respiratory protection	Coveralls or normal work attire; minimal skin	No inhalation or significant skin hazard

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Level	Respiratory Protection	Skin/Body Protection	Typical Use Scenario
		protection	identified; cold-zone/support functions only

Adapted from OSHA HAZWOPER (29 CFR 1910.120) PPE classification as applied in AHLS course materials.

5 Decontamination Principles

Decontamination Zones

Response scenes and hospital receiving areas are organized into three concentric zones so that contamination is progressively reduced before a patient reaches definitive, unprotected care.

Table 6.2 Decontamination zones and activities

Zone	Alternate Name	PPE Required	Activities
Hot Zone	Exclusion / red zone	Level A or B, per hazard assessment	Source control, rescue of victims, initiation of gross decontamination
Warm Zone	Contamination reduction / yellow zone	Level B or C	Technical decon line, PPE doffing, patient disrobing and rinse, equipment decon
Cold Zone	Support zone / green zone	Level D	Command post, triage, definitive treatment, transport, staging — only decontaminated patients and cleared personnel enter

Gross, Technical, and Field Decontamination

- Gross decontamination: rapid removal of the bulk of contamination — brush off dry/powder material, remove all clothing and jewelry (this alone removes an estimated 80-90 percent of surface contamination for most agents), then flush with copious low-pressure water.
- Technical decontamination: a structured, station-based process — soap-and-water wash from head to toe, attention to hair, nail beds, and skin folds, rinse, and dry — typically performed in the warm zone under trained decon-team supervision.
- Field (emergency) decontamination: an abbreviated version used for mass-casualty or immediately life-threatening presentations — disrobe and flush with water, accepting a less thorough wash in exchange for speed when large numbers of patients or unstable physiology demand it.

Quick Tip for the Receiving Team

Water alone, applied for at least 2-3 minutes with full disrobing, removes the great majority of contamination for most industrial chemicals and biological/radiological particulates — do not withhold decon while waiting for a perfect chemical-specific protocol.

Warm water is more comfortable and better tolerated than cold, improving compliance and

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completeness of decon, but cold water is acceptable and should never be a reason to delay.

Eyes get irrigated first and longest — ocular exposure to corrosive or irritant agents causes rapid, sometimes irreversible injury.

6 Toxidrome Recognition

Before a specific agent is identified, the physical exam pattern — the toxidrome — usually points directly to the correct antidote class. Learning five patterns cold lets a clinician begin correct treatment within seconds of laying eyes on a patient.

Table 6.3 Toxidrome recognition

Toxidrome	Key Signs and Symptoms	Typical Causative Agents
Cholinergic	SLUDGE/DUMBELS pattern — salivation, lacrimation, urination, diarrhea/defecation, GI cramping, emesis; plus miosis, bronchorrhea, bronchospasm, bradycardia, fasciculations, weakness, seizures, altered mental status	Nerve agents (sarin, VX, tabun, soman), organophosphate and carbamate pesticides
Anticholinergic	'Hot as a hare, blind as a bat, dry as a bone, red as a beet, mad as a hatter' — mydriasis, dry flushed skin, urinary retention, tachycardia, hyperthermia, delirium	Atropine/scopolamine overdose, jimsonweed and other anticholinergic plants, diphenhydramine, tricyclic antidepressants
Sympathomimetic	Tachycardia, hypertension, hyperthermia, diaphoresis (moist skin — distinguishes from anticholinergic), mydriasis, agitation, seizures	Cocaine, amphetamines/methamphetamine, synthetic cathinones
Sedative-hypnotic / Opioid	CNS and respiratory depression, hyporeflexia, hypotension, bradypnea; miosis characteristic of opioids, variable pupils with other sedative-hypnotics	Opioids (fentanyl, heroin), benzodiazepines, barbiturates, GHB
Serotonergic	Triad of altered mental status, autonomic instability (hyperthermia, tachycardia), and neuromuscular findings — clonus, hyperreflexia, tremor, lower-extremity predominant	Serotonergic drug combinations (SSRI plus MAOI, tramadol), MDMA, serotonergic overdose

Practice Pearls

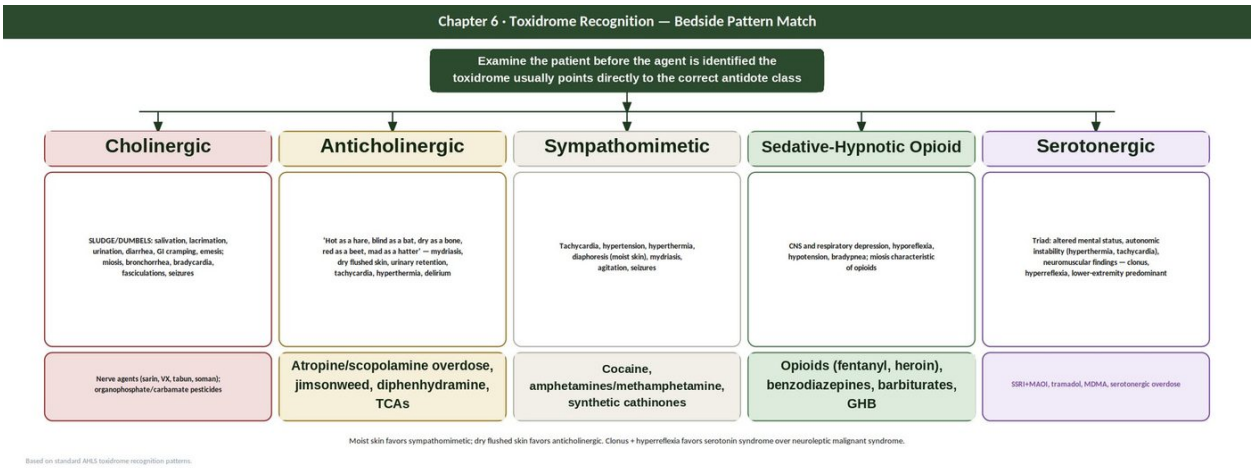
Moist skin favors sympathomimetic toxicity; dry, flushed skin favors anticholinergic — this single exam finding often separates two otherwise similar-looking agitated, tachycardic patients.

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Miosis with copious secretions in an occupational or mass-casualty setting is cholinergic poisoning until proven otherwise — do not wait for confirmatory testing to give atropine.

Clonus and lower-extremity hyperreflexia distinguish serotonin syndrome from neuroleptic malignant syndrome, which instead shows lead-pipe rigidity and bradyreflexia.

Based on standard AHLS toxidrome recognition patterns.



7 Nerve Agents and Organophosphate/Carbamate Poisoning

Nerve agents (sarin, VX, tabun, soman, and novel agents such as the Novichok series) and organophosphate/carbamate pesticides inhibit acetylcholinesterase, allowing acetylcholine to accumulate at muscarinic and nicotinic synapses and in the CNS. Muscarinic excess produces the SLUDGE/DUMBELS picture plus bronchorrhea, bronchospasm, and bradycardia — the classic 'killer B's' (bronchorrhea, bronchospasm, bradycardia) that cause death by asphyxiation rather than cardiac collapse. Nicotinic excess produces fasciculations, weakness, and eventually flaccid paralysis; CNS involvement produces agitation, seizures, and coma.

Treatment is threefold and time-critical: atropine to reverse muscarinic effects, pralidoxime to reactivate acetylcholinesterase before the enzyme-agent bond 'ages' into permanence, and benzodiazepines to control or pre-empt seizures. All three are given without waiting for laboratory confirmation in a patient with a compatible exposure history and exam.

Table 6.4 Nerve agent / organophosphate antidote dosing

Antidote	Adult Dose	Pediatric Dose	Notes
Atropine	Mild-moderate: 1-2 mg IV/IM. Severe: 2-6 mg IV/IM initial; repeat/double every 5-10 min until secretions dry and bronchospasm resolves; up to 50 mg may be required in the first 24 hours	0.05 mg/kg IV/IM (minimum single dose 0.1 mg to avoid paradoxical bradycardia); repeat every 10-20 min	Titrate to drying of bronchial secretions and resolution of bronchospasm/bradycardia — NOT to pupil size; there is no fixed ceiling dose in severe poisoning
Pralidoxime (2-PAM)	1-2 g IV in 100 mL saline	25-50 mg/kg IV over 15-	Give in addition to, not

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Antidote	Adult Dose	Pediatric Dose	Notes
	over 15-30 min (or 30 mg/kg load); may repeat in 1 hour or begin infusion ~8 mg/kg/hr	30 min, max 2 g per dose	instead of, atropine; most effective when started within minutes to hours, before irreversible enzyme 'aging' occurs
Benzodiazepine (seizure control/prophylaxis)	Diazepam 5-10 mg IV/IM or midazolam 5-10 mg IM; repeat per seizure protocol	Diazepam 0.2-0.5 mg/kg IV/IM; midazolam 0.2 mg/kg IM	Give for any seizure activity, or empirically in severe exposure even without observed seizure; military autoinjectors deliver a fixed 10 mg IM dose

Doses adapted from CHEMM (chemm.hhs.gov) Medical Countermeasures Database and standard toxicology references; confirm against current local protocol and product labeling.

Critical Numbers to Remember

Atropine severe adult initial dose: 2-6 mg IV/IM, doubling every 5-10 minutes to effect.

Pralidoxime adult dose: 1-2 g IV over 15-30 minutes (or 30 mg/kg load).

Pediatric atropine minimum single dose: 0.1 mg, to prevent paradoxical bradycardia.

Endpoint for atropine titration: dry secretions and resolved bronchospasm, not mydriasis.

Myth vs Fact

Myth: Atropine dosing should stop once the pupils dilate.

Fact: Pupil size is an unreliable endpoint. Titrate atropine to drying of secretions and resolution of bronchospasm/bradycardia; mydriasis may lag or never fully occur despite adequate reversal.

Myth: Pralidoxime is optional if atropine is on board.

Fact: Atropine treats only the muscarinic effects; it does nothing for nicotinic weakness/paralysis or reactivates the enzyme. Pralidoxime is a separate, complementary therapy and should be given as early as possible.

8 Cyanide Poisoning

Cyanide inhibits cytochrome c oxidase, blocking oxidative phosphorylation and forcing cells into anaerobic metabolism regardless of arterial oxygen content. Sources include industrial processes (electroplating, mining, plastics manufacturing), combustion of synthetic materials in structure fires (smoke inhalation is a leading real-world cause), and deliberate release. The classic 'bitter almond' odor is genetics-dependent (roughly 40 percent of people cannot detect it) and must never be relied upon to rule out exposure. Presentation ranges from headache and anxiety to seizures, cardiovascular collapse, and severe lactic acidosis with an elevated anion gap out of proportion to other findings; venous blood that appears unusually oxygenated (arterialized) reflects impaired oxygen extraction at the tissue level.

Table 6.5 Cyanide antidote dosing

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Antidote	Adult Dose	Pediatric Dose	Notes
Hydroxocobalamin	5 g IV over 15 min; may repeat once (5 g) for a total of 10 g in severe poisoning	70 mg/kg IV (max 5 g) over 15 min	Preferred first-line agent, especially in smoke-inhalation/combined carbon-monoxide exposure; safe with concurrent CO poisoning; causes transient red discoloration of skin/urine and interferes with some colorimetric labs
Sodium Thiosulfate	12.5 g (50 mL of 25% solution) IV over 10-20 min	1 mL/kg of 25% solution (max 50 mL) IV	Safe adjunct given with or after hydroxocobalamin; supplies sulfur substrate for rhodanese-mediated conversion of cyanide to thiocyanate for renal excretion
Amyl Nitrite / Sodium Nitrite	Amyl nitrite by inhalation while establishing IV access; sodium nitrite 300 mg (10 mL of 3% solution) IV over 5-20 min	Sodium nitrite 0.2 mL/kg (max 10 mL) of 3% solution, dose-adjusted for hemoglobin/anemia	AVOID in smoke-inhalation or suspected combined CO exposure — nitrite-induced methemoglobinemia further reduces oxygen-carrying capacity; largely supplanted by hydroxocobalamin where it is stocked

Doses adapted from the CHEMM Cyanide Antidotes page (chemm.hhs.gov) and current US antidote product labeling (Cyanokit, Nithiodote).

Do Not Give Nitrites After Smoke Inhalation

Fire victims commonly have combined carbon monoxide and cyanide poisoning. Nitrites deliberately induce methemoglobinemia to bind free cyanide, but methemoglobin cannot carry oxygen — stacking this on top of an already-reduced oxygen-carrying capacity from carboxyhemoglobin can be fatal.

Hydroxocobalamin does not affect hemoglobin oxygen-carrying capacity and is the preferred cyanide antidote whenever combined smoke-inhalation exposure is possible or confirmed.

9 Other Key Antidotes

Beyond nerve agents and cyanide, a small additional set of antidotes covers most of the remaining high-yield hazmat presentations: opioid overdose, toxin-induced methemoglobinemia, hydrofluoric acid exposure, acute heavy-metal poisoning, and severe anticholinergic delirium.

Table 6.6 Other key hazmat antidotes

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Exposure/Toxidrome	Antidote	Adult Dose	Notes
Opioid toxicity	Naloxone	0.4-2 mg IV/IM/IN; titrate to adequate respiration, not full arousal; repeat or infuse for long-acting agents	Short half-life relative to many opioids and fentanyl analogs — observe closely for re-sedation after apparent reversal
Methemoglobinemia (nitrites, aniline dyes, some local anesthetics)	Methylene blue	1-2 mg/kg IV over 5 min; may repeat in 1 hour if methemoglobin remains above 30 percent or patient remains symptomatic	Contraindicated/use extreme caution in G6PD deficiency — can precipitate hemolysis; ineffective, and potentially harmful, in sulfhemoglobinemia
Hydrofluoric acid (dermal/inhalation)	Calcium gluconate	Topical: 2.5% gel massaged into affected skin. Systemic hypocalcemia: 10 mL of 10% IV calcium gluconate, repeat per ionized calcium and cardiac monitoring	Fluoride ion binds calcium and magnesium, causing liquefactive tissue necrosis and life-threatening hypocalcemia/hyperkalemia/dysrhythmia; treat systemic toxicity aggressively for burns over ~5 percent BSA or concentrated (greater than 50%) exposures
Acute severe heavy-metal poisoning (arsenic, mercury, lead)	Dimercaprol (BAL) or DMSA/succimer	Dimercaprol 3-5 mg/kg deep IM every 4 hours, specialist-guided	Reserve for symptomatic acute poisoning; coordinate dosing and duration with poison control/medical toxicology
Severe anticholinergic delirium (selected cases)	Physostigmine	0.5-2 mg IV slow push, specialist-guided, with cardiac monitoring	Reserve for pure, confirmed anticholinergic toxicity with dangerous agitation; avoid if any suspicion of tricyclic antidepressant overdose or cardiac conduction abnormality

10 Chemical Asphyxiants and Pulmonary Irritant Agents

Industrial and transportation releases frequently involve gases that either displace/block oxygen delivery (chemical asphyxiants) or injure the airway and alveoli directly (pulmonary irritants). Distinguishing a highly water-soluble agent (immediate upper-airway injury) from a poorly water-soluble one (delayed, deep-lung injury) predicts both the timing of symptoms and the required period of observation.

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Table 6.7 Chemical asphyxiants and pulmonary irritant agents

Agent	Mechanism / Onset	Recognition	Management
Chlorine	Reacts with mucosal moisture to form hydrochloric/hypochlorous acid; predominantly upper-airway irritant at low-moderate exposure	Immediate eye and airway irritation, cough, dyspnea, characteristic pungent odor; pulmonary edema with severe exposure	Remove from source, humidified oxygen, bronchodilators; observe for delayed pulmonary edema; no specific antidote
Phosgene	Low water solubility allows deep lung penetration with minimal early warning; slow hydrolysis delays injury	Often minimal early symptoms (may be asymptomatic 1-24 hours), then abrupt-onset non-cardiogenic pulmonary edema	Mandatory minimum 24-hour observation after significant exposure even if asymptomatic; supportive care, oxygen, avoid exertion; no specific antidote
Ammonia	Highly water-soluble — immediate reaction with mucous membranes	Severe immediate eye/nose/throat pain, cough, laryngospasm, corneal injury	Copious irrigation, early airway management for supraglottic edema, supportive care
Hydrogen Sulfide	Inhibits cytochrome oxidase like cyanide; causes sudden 'knockdown' at high concentration	Rotten-egg odor at low concentration but olfactory fatigue makes odor unreliable at high concentration; headache, sudden collapse or apnea	Remove to fresh air, 100 percent oxygen; hydroxocobalamin/nitrites used off-label in severe cases; hyperbaric oxygen considered at some centers
Carbon Monoxide	Binds hemoglobin with far greater affinity than oxygen and shifts the oxyhemoglobin dissociation curve leftward	Headache, nausea, confusion, syncope; cherry-red skin is a late and unreliable sign; standard pulse oximetry reads falsely normal	100 percent high-flow oxygen; consider hyperbaric oxygen for loss of consciousness, COHb greater than 25 percent, pregnancy, or cardiac/neurologic findings

Delayed Pulmonary Edema Observation Checklist

Any significant phosgene (or other low-solubility irritant) exposure gets a minimum 24-hour monitored observation period regardless of how well the patient looks at presentation.

Watch for: new dyspnea, falling SpO2, tachypnea, rales — these can appear abruptly hours after an asymptomatic interval.

Chest radiograph and serial exam at intervals through the observation period; discharge only after the full observation window has passed without deterioration.

11 Radiological and Nuclear Exposure Basics

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Contamination vs Irradiation

Irradiation means exposure to radiation energy without the patient becoming radioactive — an irradiated patient poses no hazard to caregivers and needs no decontamination. Contamination means radioactive material (dust, liquid, particulate) is actually on or in the patient's body, hair, clothing, or wounds — this patient can transfer material to staff and surfaces and requires decontamination, generally using the same water-and-disrobing technique as chemical decon. A patient can be irradiated, contaminated, both, or neither, and the distinction changes what protective steps the receiving team must take, not whether the patient needs treatment.

Triage principles: treat immediately life-threatening trauma and medical emergencies first, exactly as in any other mass-casualty event — radiation injury (acute radiation syndrome) evolves over hours to weeks and is rarely the most time-critical problem in the first hour. For combined-injury patients (trauma plus radiation contamination), stabilize and treat the trauma; decontaminate as soon as feasible without delaying life-saving intervention.

Table 6.8 Potassium iodide (KI) dosing by age group

Age Group	Predicted Thyroid Dose Threshold	KI Dose	Administration
Adults over 40 years	5 Gy (500 cGy) or greater	130 mg	1 tablet (130 mg) or 2 tablets (65 mg)
Adults 18-40 years	0.1 Gy (10 cGy) or greater	130 mg	1 tablet (130 mg) or 2 tablets (65 mg)
Pregnant or lactating women	0.05 Gy (5 cGy) or greater	130 mg	1 tablet (130 mg) or 2 tablets (65 mg)
Adolescents 12-18 years (under 70 kg)	0.05 Gy (5 cGy) or greater	65 mg	1/2 of a 130 mg tablet, or 1 of a 65 mg tablet
Children 3-12 years	0.05 Gy (5 cGy) or greater	65 mg	1/2 of a 130 mg tablet, or 1 of a 65 mg tablet
Children 1 month-3 years	0.05 Gy (5 cGy) or greater	32 mg	Oral solution 0.5 mL (65 mg/mL)
Infants birth-1 month	0.05 Gy (5 cGy) or greater	16 mg	Oral solution 0.25 mL (65 mg/mL)

Adapted from FDA guidance and REMM (remm.hhs.gov). Adolescents at or above 70 kg (154 lb) receive the full adult 130 mg dose regardless of age. KI protects only the thyroid from radioiodine uptake — it provides no protection against other radionuclides or external irradiation, and is administered based on predicted internal thyroid exposure, not simple proximity to an incident.

Quick Tip for Radiological Incidents

Life-threatening injuries always take priority over decontamination and over KI administration — treat the trauma, then decontaminate, then dose KI as directed by public health/radiation safety guidance.

A radiation survey meter, not clinical judgment, confirms when a patient is adequately decontaminated — external decon is complete when counts return to near-background.

KI must be given before or promptly after radioiodine exposure to be effective; it does not reverse existing thyroid uptake and provides no benefit once uptake is complete.

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12 Biological Agent Basics

Biological agents of concern typically present after an incubation period rather than immediately, so recognition depends on a cluster of unusual presentations or a known exposure history rather than a single dramatic bedside sign.

- Anthrax (Bacillus anthracis): inhalational form presents with a brief flu-like prodrome followed by abrupt respiratory failure and a characteristic widened mediastinum on chest imaging from hemorrhagic mediastinitis; cutaneous form produces a painless black eschar with surrounding edema. Treat with combination antimicrobial therapy (e.g., ciprofloxacin or doxycycline plus a second agent) per current CDC guidance, plus antitoxin for systemic disease, and report immediately to public health.
- Botulinum toxin: produces a descending, symmetric flaccid paralysis with prominent bulbar findings (diplopia, dysarthria, dysphagia) and normal mentation/sensation; respiratory failure from diaphragmatic paralysis is the life threat. Treatment is supportive (often prolonged mechanical ventilation) plus antitoxin administered as early as possible — antitoxin halts progression but does not reverse existing paralysis.
- Plague, smallpox, and other agents: suspect a deliberate biological release when multiple patients present with an unusual illness cluster, an uncommon pathogen for the region, or a rapidly progressive respiratory illness with high mortality; isolate per appropriate precautions and notify public health and infection control immediately — early recognition and reporting is the single highest-yield action for biological incidents.

13 Chemical Asphyxiants and Pulmonary Agents in Hospital Receiving

Hospital Hazmat Receiving Protocol Basics

Every receiving hospital should have a written hazmat plan that designates a decontamination area outside (or in a dedicated, separately ventilated space adjacent to) the main department, stocks appropriate PPE for staff, and defines who may authorize a patient's entry into clean treatment space.

- Decon before ED entry: contaminated patients — whether self-presenting or brought by EMS — are directed to the outdoor/dedicated decon area and processed there before crossing into the main department, regardless of how the patient arrived.
- Staff protection: decon-team staff wear PPE appropriate to the suspected hazard (typically Level C with PAPR for most industrial chemical scenarios); receiving clinical staff who will only handle already-decontaminated patients typically require standard precautions plus a lower level of chemical protection per facility policy.
- Facility protection: dedicated ventilation, contained runoff/wastewater capture from decon showers, and a controlled single point of entry prevent contamination of the building's air handling and water systems.
- Notification: early two-way communication between the scene, EMS, and the receiving hospital hazmat/incident command allows the facility to activate its plan and staff the decon area before contaminated patients arrive.

An Undecontaminated Walk-In Is a Department-Wide Event

A single contaminated self-transporting patient who reaches the main triage desk or waiting room before being recognized can require evacuation and decontamination of the entire affected area and every person who had contact with the patient.

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Front-desk and triage staff should be trained to recognize red-flag histories (chemical odor, industrial exposure, multiple simultaneously ill coworkers) and redirect to the outdoor decon area before the patient enters the building.

14 Worked Clinical Case

Case 6.1 — Organophosphate Pesticide Exposure in an Agricultural Worker

Presentation: A 45-year-old agricultural worker is found unresponsive in a field after a backpack-sprayer malfunction; coworkers report a chemical odor and that the worker vomited before collapsing. EMS finds the patient supine with profuse oral secretions, diaphoresis, miosis, fasciculations of the extremities, heart rate 48, respiratory rate 8 with audible wheeze and rhonchi, and GCS 9.

Management: The responding crew dons Level C PPE (PAPR plus chemical-resistant splash suit — SCBA is not required once the agent is identified as a liquid organophosphate insecticide in an outdoor setting with adequate ambient oxygen) before approaching. The patient is moved to the warm zone, all clothing is fully removed, and skin is irrigated with copious low-pressure water and mild soap (gross and technical decon) before any hands-on treatment beyond airway positioning. Once decontaminated, the patient is moved to the cold-zone treatment area: airway secured with suction and bag-mask ventilation, atropine 2 mg IV given and doubled every 5 minutes (4 mg, then 8 mg) until secretions dry and bronchospasm resolves, for a cumulative dose of 14 mg over roughly 20 minutes; pralidoxime 2 g IV is infused over 20 minutes; a generalized seizure en route is treated with midazolam 5 mg IM.

Outcome/teaching point: Secretions dry, wheeze resolves, and heart rate rises to 88 with improving mentation; the patient is transported to the hospital's pre-designated decontaminated-patient entrance and admitted to the ICU, recovering fully over several days. Teaching point: had the crew treated before decontaminating, liquid organophosphate would have been transferred to their gloves, the ambulance interior, and potentially the receiving department, risking secondary poisoning of responders and staff. The large cumulative atropine dose was expected and appropriate — dosing was titrated to the clinical endpoint of dry secretions and resolved bronchospasm/bradycardia, not to an arbitrary ceiling or to pupil size.

15 Chapter Quick-Reference Summary

The table below consolidates the highest-yield antidote and first-action information from this chapter into a single scan-and-act reference for the most common hazmat presentations.

Table 6.9 Quick-reference summary: key antidotes and first actions by category

Category	Agent Example	First-Line Antidote/Action	Adult Starting Dose
Nerve agent / organophosphate	Sarin, VX, malathion, chlorpyrifos	Atropine + Pralidoxime + Benzodiazepine	Atropine 2-6 mg IV/IM, doubling q5-10min; 2-PAM 1-2 g IV over 15-30 min; diazepam 5-10 mg IV/IM for seizures
Cyanide	Hydrogen cyanide, smoke	Hydroxocobalamin (+	Hydroxocobalamin 5 g IV

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Category	Agent Example	First-Line Antidote/Action	Adult Starting Dose
	inhalation, industrial cyanide salts	sodium thiosulfate)	over 15 min, may repeat to 10 g total
Opioid	Fentanyl, heroin, prescription opioids	Naloxone	0.4-2 mg IV/IM/IN, titrate to adequate respiration
Methemoglobinemia	Nitrites, aniline dyes, some local anesthetics	Methylene blue	1-2 mg/kg IV over 5 min (avoid in G6PD deficiency)
Hydrofluoric acid	Rust remover, glass etching, industrial HF	Calcium gluconate	Topical 2.5% gel; 10 mL 10% IV for systemic hypocalcemia
Radiological (internal thyroid)	Radioiodine from reactor release or fallout	Potassium iodide (KI)	130 mg PO for adults 18-40 yr; see Table 6.8 for full age-based dosing
Chemical asphyxiant	Hydrogen sulfide, carbon monoxide	100 percent oxygen (+/- hydroxocobalamin for H2S)	High-flow oxygen immediately; hyperbaric O2 if severe
Pulmonary irritant (delayed)	Phosgene	Observation + supportive care	Minimum 24-hour monitored observation even if asymptomatic

Final Practice Pearls

Protect the responder first: no PPE, no approach — an unprotected rescuer is the most preventable hazmat casualty.

Decon before treatment, except for immediately life-threatening airway/cardiac interventions performed in appropriate PPE.

Recognize the toxidrome before you know the agent name — treatment for cholinergic, cyanide, and opioid poisoning can start on exam findings alone.

Titrate antidotes to clinical endpoint (dry secretions, adequate respiration, resolved hypocalcemia) rather than to a fixed maximum dose.

16 Vesicant (Blister) Agents

Vesicant agents cause severe blistering of the skin, eyes, and airway and remain a chemical warfare and terrorism concern because they are comparatively simple to produce and persist in the environment. The three classic vesicants - sulfur mustard, lewisite, and phosgene oxime - are distinguished chiefly by the timing of pain after exposure, which is the single most useful bedside clue to which agent is involved.

Table 6.4 Vesicant Agent Recognition

Agent	Odor / onset	Key features
Sulfur mustard (HD)	Garlic, mustard, or horseradish-like	Erythema progressing to blistering; airway

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Agent	Odor / onset	Key features
	odor; delayed pain, sometimes not until hours after exposure	and eye involvement; bone-marrow suppression (lymphopenia, neutropenia) over the following 1-2 weeks; known carcinogen
Lewisite (L)	Geranium-like odor; immediate pain on contact, distinguishing it from mustard	Faster acting than mustard (blistering within hours); an arsenical - can cause systemic arsenic toxicity in addition to local injury; a specific antidote exists (see below)
Phosgene oxime (CX)	Sharp, irritating odor; immediate pain	Technically a corrosive/urticant rather than a true vesicant, but classified with this group; produces a distinctive wheal-like skin lesion rather than a fluid-filled blister

Decontamination and management

As with all chemical exposures, decontamination is the single highest-yield intervention and is time-critical: skin decontamination within 2 minutes is ideal, and decontamination performed even 15-20 minutes after exposure can still reduce the eventual size of blistering. Remove all clothing, irrigate copiously, and protect responders with appropriate PPE before approaching a contaminated patient (Section 4). Treatment beyond decontamination is supportive for mustard and phosgene oxime - there is no specific antidote - and focuses on burn-style wound care for blistered skin, ocular irrigation and ophthalmology consultation for eye involvement, and airway support for inhalational injury. Patients exposed to mustard should have a baseline complete blood count with differential and repeat testing over the following two weeks to catch delayed bone-marrow suppression.

Lewisite is the exception: its arsenical structure responds to a specific antidote, British anti-lewisite (BAL, dimercaprol), which chelates the arsenic and can reduce both local and systemic toxicity when given promptly. Ocular lewisite exposure is a particular emergency - corneal damage and permanent vision loss can result if decontamination is delayed beyond about one minute.

Sources consulted: Advanced Hazmat Life Support (AHLS) course materials, University of Arizona/AHLS International Foundation (ahls.org); American College of Medical Toxicology (ACMT); CDC/ATSDR toxic syndrome and biological agent guidance (cdc.gov); CHEMM — Chemical Hazards Emergency Medical Management, US HHS/ASPR (chemm.hhs.gov), including its Medical Countermeasures Database entries for atropine, pralidoxime, obidoxime, sodium thiosulfate, and cyanide antidotes; REMM — Radiation Emergency Medical Management, US HHS/ASPR (remm.hhs.gov) and FDA guidance on potassium iodide dosing; NCBI/StatPearls reviews of nerve agent and pralidoxime pharmacology. This chapter is an educational quick-reference only and is not a substitute for completing official AHLS provider certification, consulting current product labeling/institutional protocols, or contacting a regional poison control center (in the US, 1-800-222-1222) or medical toxicologist for real-time patient management.

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CHAPTER 7

ENLS

Emergency Neurological Life Support

1 Clinical Snapshot

Emergency Neurological Life Support (ENLS) is the Neurocritical Care Society's structured approach to the first hour of a neurological emergency, the interval during which a clinician's actions most reliably change outcome. Unlike a single disease-specific guideline, ENLS is a set of parallel protocols, one per emergency, each built around a short checklist of time-critical actions that a bedside clinician can execute before a specialist arrives. The philosophy is identical to BLS/ACLS for cardiac arrest: reduce a complex, high-stakes problem to an ordered sequence that any trained provider can run under pressure.

The stakes are unusually high in neurological emergencies because neurons that die from delayed reperfusion, uncontrolled seizure, or unrecognized herniation do not regenerate. Minutes of delay in thrombolysis, benzodiazepine administration, or hyperosmolar therapy translate directly into infarcted tissue, permanent neuronal injury, or death. ENLS protocols therefore emphasize recognition scales, explicit time windows, and weight-based dosing tables that remove ambiguity from decisions that must be made in minutes, not hours.

Why this matters · Typical patient · Where you meet it

Why it matters: Time-dependent neurological injury is largely irreversible; the difference between a full recovery and permanent disability or death is frequently measured in single-digit minutes to a few hours.

Typical patient: Ranges from a 68-year-old with sudden right-sided weakness and slurred speech, to a 34-year-old found seizing at home, to a comatose trauma patient with a blown pupil, to a postpartum woman with the 'worst headache of my life.'

Common settings: Emergency department, prehospital/EMS transport, neuro-ICU, general ICU, stroke unit, and inter-facility transfer coordination between community hospitals and comprehensive stroke/neurosurgical centers.

2 Learning Objectives

By the end of this chapter the learner should be able to:

- Apply a structured first-hour checklist to any suspected neurological emergency, regardless of final diagnosis.
- State the time windows for IV thrombolysis and mechanical thrombectomy in acute ischemic stroke and identify absolute contraindications to thrombolysis.
- Execute the stepwise status epilepticus algorithm, including weight-based benzodiazepine and second-line antiseizure medication dosing.
- Recognize the clinical signs of rising intracranial pressure and herniation and initiate hyperosmolar therapy correctly.
- Classify traumatic brain injury severity by GCS and identify indications for neurosurgical consultation and ICP monitoring.

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- Recognize subarachnoid hemorrhage, control blood pressure appropriately before the aneurysm is secured, and initiate vasospasm prophylaxis.
- Describe immobilization principles and time-critical decompression concepts in acute spinal cord compression.
- Identify red flags of neuromuscular respiratory failure in Guillain-Barré syndrome and myasthenic crisis.
- Apply a systematic approach to coma, including the GCS and reversible causes that must be excluded before assuming a poor prognosis.
- State the empiric antimicrobial and antiviral approach to suspected bacterial meningitis and herpes encephalitis and the target time to first dose.

3 The ENLS Approach — The First Hour

Every ENLS protocol shares a common architecture: rapid recognition using a validated scale or sign pattern, an ABC-first stabilization sequence, a small set of time-critical interventions, and a structured communication handoff to the team that will deliver definitive care (interventional neuroradiology, neurosurgery, neuro-ICU). This shared first-hour skeleton is what allows a generalist, an emergency physician, a prehospital provider, or a floor nurse to begin appropriate treatment before a neurologist or neurosurgeon is physically present.

- Airway, breathing, circulation, and finger-stick glucose — treat hypoglycemia immediately, as it is a fully reversible stroke and seizure mimic.
- A rapid, focused neurological examination: level of consciousness (GCS), pupils, gross motor asymmetry, and a validated deficit scale (NIHSS for stroke, GCS for TBI/coma).
- Establish the single most important time anchor for the presentation — last known well, seizure onset, or time of injury — because every downstream time window is calculated from it.
- Identify and treat any immediately reversible, life-threatening process (herniation, refractory seizure, airway compromise) before pursuing definitive diagnostic imaging.
- Communicate a structured handoff (SBAR-style) to the receiving specialist team with the time anchor, exam findings, relevant labs/imaging, and interventions already given.

First-Hour Neuro-Checklist (any presentation)

Vital signs and pulse oximetry, target SpO2 ≥94%

Finger-stick glucose — treat if <60 mg/dL

IV access (18-gauge peripheral preferred for stroke codes)

GCS and pupillary exam documented with a timestamp

Time anchor established (onset, last known well, or time of injury)

CBC with platelets, basic metabolic panel, PT/INR, PTT; beta-hCG in women of childbearing age

12-lead ECG

Non-contrast CT head as the first-line study for most acute presentations

4 Acute Ischemic Stroke

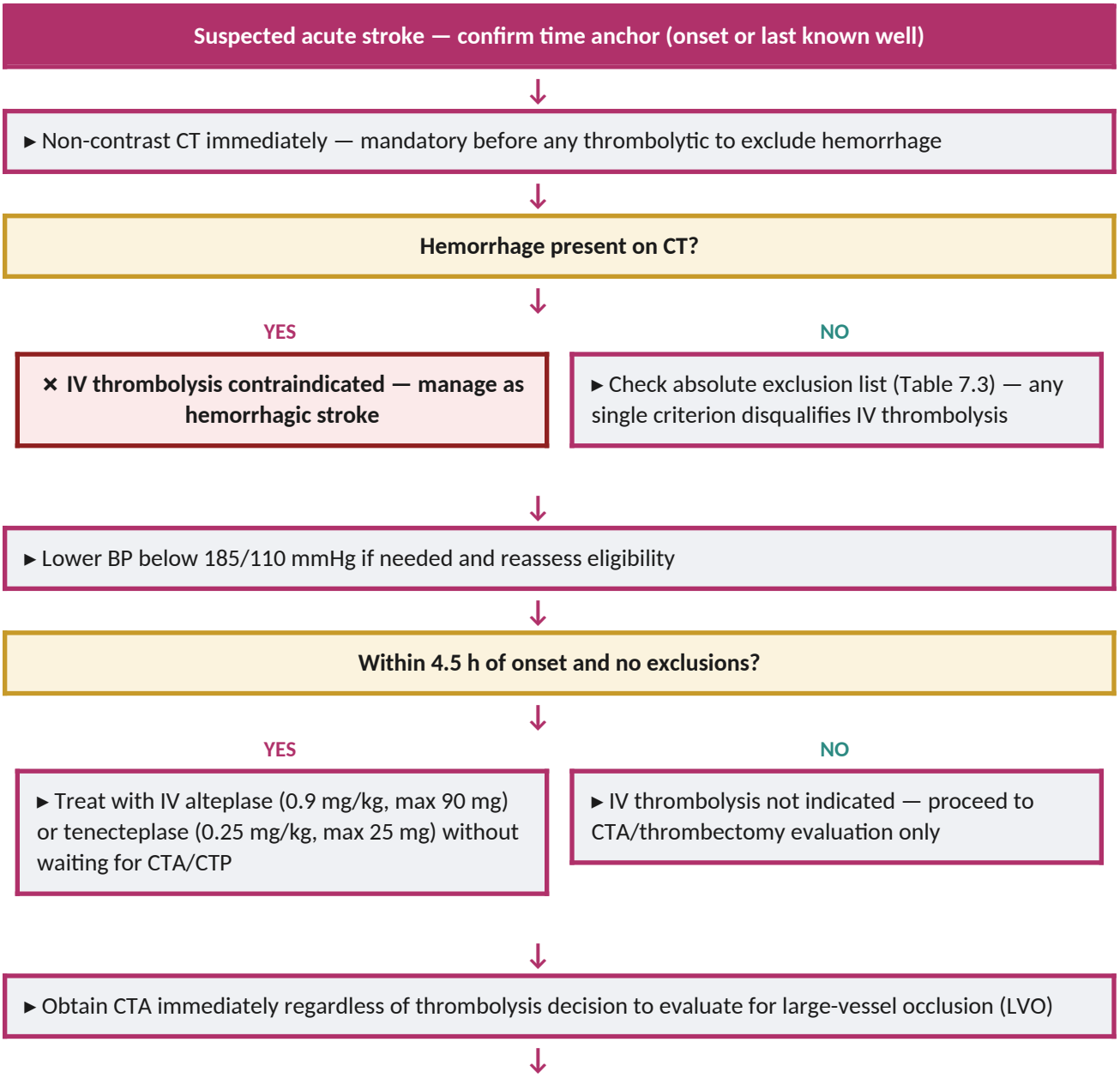
Recognition begins prehospital with a screening tool such as BE-FAST (Balance, Eyes, Face, Arm, Speech, Time) and is quantified on arrival with the NIH Stroke Scale (NIHSS); an NIHSS greater than 6 raises suspicion for a large-vessel occlusion (LVO) and should prompt CT angiography alongside the non-contrast CT. The

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ASPECTS score (Alberta Stroke Program Early CT Score) is applied to the non-contrast CT to estimate the extent of early ischemic change; a score of 6 or higher is generally required for endovascular candidacy. Non-contrast CT is obtained first in essentially every pathway because it reliably excludes hemorrhage, the one finding that changes everything about subsequent management.

Once hemorrhage is excluded and a time anchor is established, eligibility for intravenous thrombolysis is assessed against the time windows and exclusion criteria below. Alteplase remains the only IV thrombolytic with full US FDA approval; tenecteplase, given as a single bolus, is endorsed by AHA/ASA guidelines as a reasonable and increasingly preferred alternative because of its ease of administration and comparable efficacy. Mechanical thrombectomy is pursued in parallel for any suspected LVO and is not contingent on IV thrombolysis being given — patients should receive thrombolysis if eligible and be transferred for thrombectomy simultaneously rather than sequentially ('drip and ship, not drip then ship').

ALGORITHM — THROMBOLYSIS DECISION PATHWAY — ACUTE ISCHEMIC STROKE



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✓ LVO present within window → activate mechanical thrombectomy in parallel, not sequentially

Figure 7.1. Adapted from AHA/ASA acute ischemic stroke reperfusion guidance; see Tables 7.1-7.3 for full dosing, BP targets, and exclusion criteria.

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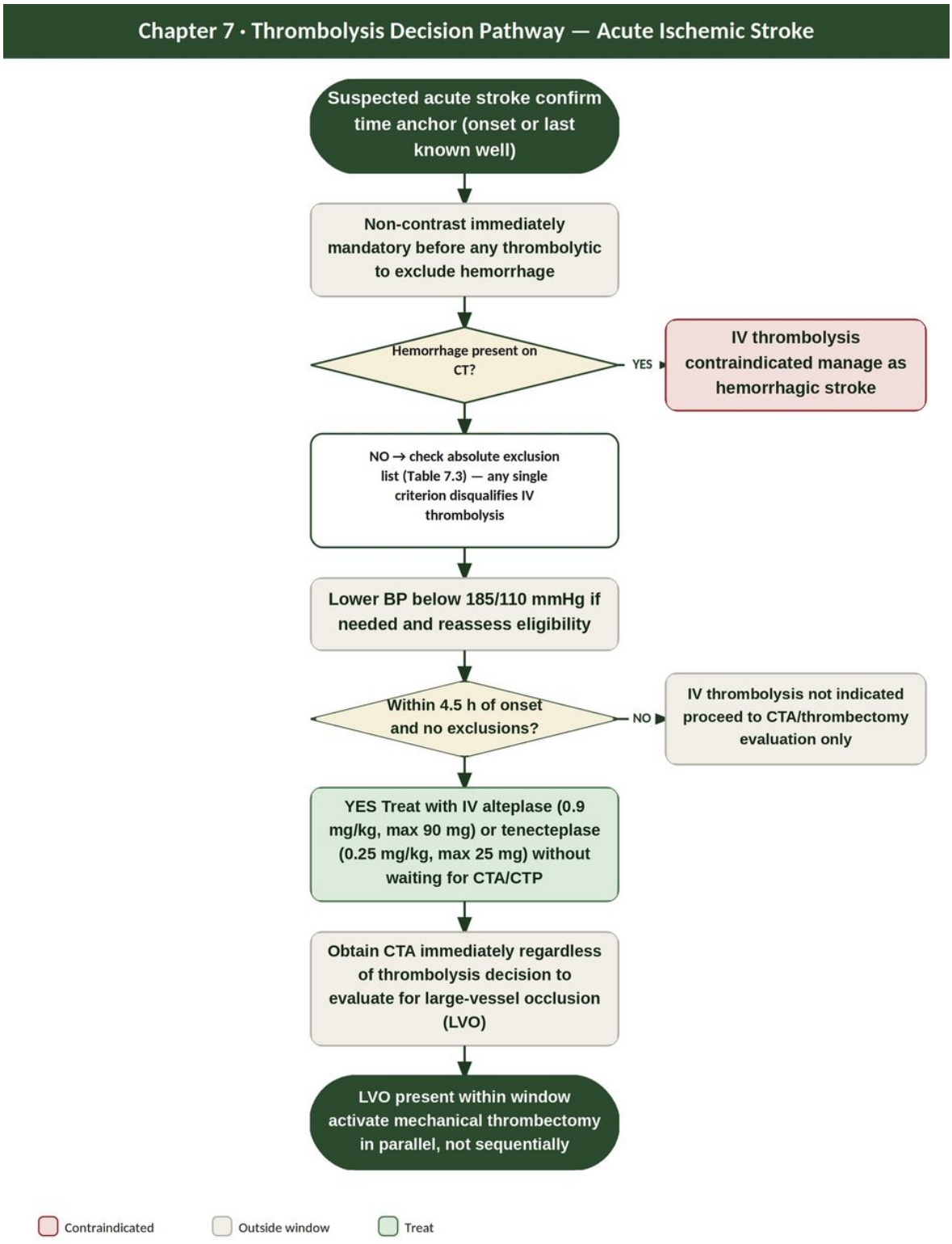


Table 7.1 Acute Ischemic Stroke — Reperfusion Time Windows and Dosing

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Therapy	Time Window	Dose	Key Notes
IV alteplase — standard window	Onset <3 h	0.9 mg/kg IV, max 90 mg; 10% as bolus over 1 min, remainder over 60 min	Only FDA-approved IV thrombolytic in the US
IV alteplase — extended window	3–4.5 h from onset	Same dosing as above	AHA/ASA-endorsed, not FDA-labeled; exclude age >80, NIHSS >25, prior stroke + diabetes, or oral anticoagulant use
IV tenecteplase	Onset <4.5 h (reasonable alternative to alteplase)	0.25 mg/kg IV single bolus, max 25 mg	Single bolus simplifies transfer logistics; increasingly favored where LVO/thrombectomy transfer is anticipated
Wake-up / unclear onset	Last known well 4.5–24 h	Alteplase per weight if imaging-selected	Requires MRI DWI-positive/FLAIR-negative mismatch or CT-perfusion core-penumbra mismatch; involve stroke neurology before treating
Mechanical thrombectomy — early window	0–6 h from last known well	N/A (endovascular)	LVO of proximal M1 MCA, intracranial ICA, or basilar artery; ASPECTS ≥6
Mechanical thrombectomy — extended window	6–24 h from last known well	N/A (endovascular)	Requires CT perfusion or DWI-MRI mismatch imaging (DAWN/DEFUSE 3 criteria) to confirm salvageable penumbra

Times are calculated from onset or last known well, whichever is later established. A 2026 comprehensive AHA/ASA acute ischemic stroke guideline update further broadens imaging-based eligibility for extended-window therapy; confirm local protocol and current guideline version before treating a patient outside the standard 3–4.5 h window.

Table 7.2 Acute Ischemic Stroke — Blood Pressure Targets

Clinical Scenario	BP Target	First-Line Agents
Before IV thrombolysis	Systolic <185 mmHg and diastolic <110 mmHg	Labetalol 10–20 mg IV q10min (double to max 300 mg) + infusion; nicardipine 5 mg/h, titrate by 2.5 mg/h q5–15min, max 15 mg/h; clevidipine 1–2 mg/h, double q90sec, max 32 mg/h
During and for 24 h after thrombolysis	Maintain <180/105 mmHg	Same titratable IV agents as above
Not receiving thrombolysis / thrombectomy	Permissive hypertension, treat only if >220/120 mmHg	Cautious, gradual reduction; avoid abrupt drops that can extend the ischemic penumbra

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Clinical Scenario	BP Target	First-Line Agents
TIA	Permissive, not to exceed 220/120 mmHg	Lower gradually over 24–48 h once stroke is excluded

Table 7.3 Acute Ischemic Stroke — IV Thrombolysis Contraindications

Category	Exclusion Criteria
Absolute	Symptoms suggestive of subarachnoid hemorrhage; major head trauma or intracranial/intraspinal surgery within 3 months; non-compressible arterial puncture or lumbar puncture within 7 days; history of intracranial neoplasm, aneurysm, or AVM; active bleeding or acute fracture on exam; INR >1.7 or therapeutic anticoagulation; direct thrombin/factor Xa inhibitor use within 48 h (unless normal specific coagulation assays); therapeutic heparin within 48 h with elevated aPTT; platelet count <100,000/mm3
Relative (weigh risk vs. benefit)	Pregnancy; seizure at onset with postictal neurological impairment; major surgery or trauma within 14 days; GI or urinary hemorrhage within 21 days; myocardial infarction within 3 months; NIHSS >25 (use caution); treatment-dose low-molecular-weight heparin within 24 h (prophylactic dose acceptable)

Adapted from ENLS Acute Ischemic Stroke Protocol v6.0, Neurocritical Care Society.

Decision Point: Is This Patient a Thrombolysis Candidate?

1. Confirm time anchor (onset or last known well) and calculate elapsed time.
2. Non-contrast CT excludes hemorrhage — mandatory before any thrombolytic.
3. Check the absolute exclusion list (Table 7.3); any single absolute criterion disqualifies IV thrombolysis regardless of time window.
4. Lower BP below 185/110 mmHg if needed and reassess eligibility.
5. If within 4.5 h and no exclusions: treat with alteplase or tenecteplase without waiting for CTA/CTP results.
6. Regardless of thrombolysis decision, obtain CTA immediately to evaluate for LVO and activate the thrombectomy pathway in parallel.

5 Status Epilepticus

Status epilepticus (SE) is defined operationally as 5 or more minutes of continuous convulsive activity, or two or more discrete seizures within a 5-minute window without return to baseline between them. This 5-minute operational definition (rather than the classic 30-minute definition) reflects the recognition that seizures rarely stop spontaneously after 5 minutes and that earlier treatment produces better seizure control and outcomes. A patient who seizes and then fails to regain consciousness may be in non-convulsive status epilepticus and typically requires continuous EEG (cEEG) to confirm the diagnosis.

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Management follows a strict, time-anchored, stepwise algorithm: emergent initial benzodiazepine therapy, followed by urgent control therapy with a second-line antiseizure medication if seizures persist, followed by refractory management with continuous-infusion anesthetic agents and airway control if seizures still persist. Under-dosing of the initial benzodiazepine is one of the most common and consequential errors in SE management — clinicians frequently give too little, too late.

Table 7.4 Status Epilepticus — Stepwise Management Algorithm

Stage	Onset Timing	Adult Dosing	Pediatric Dosing	Key Notes
Emergent initial therapy (benzodiazepine, IV access)	0–5 min	Lorazepam 0.1 mg/kg IV (max 4 mg) over 2 min; repeat once at 3–5 min if seizures continue	Lorazepam 0.1 mg/kg IV, max 4 mg; repeat once at 3–5 min	Diazepam or clonazepam IV are acceptable alternatives
Emergent initial therapy (no IV access)	0–5 min	Midazolam 10 mg IM	Intranasal midazolam 0.2 mg/kg, max 10 mg	Alternatives: rectal diazepam 20 mg (adult), buccal or intranasal midazolam
Urgent control therapy (second-line ASM)	10–20 min after adequate benzodiazepine	Choose one: fosphenytoin 20 mg/kg IV up to 150 mg/min (max 1500 mg); valproic acid 40 mg/kg IV over 10 min (max 3000 mg); levetiracetam 60 mg/kg IV (max 4500 mg)	Same weight-based agents; phenobarbital 40 mg/kg IV (max 1000 mg) is a pediatric alternative	No single agent shown clearly superior; select based on comorbidities (avoid phenytoin/fosphenytoin in cardiac conduction disease, avoid valproate in hepatic disease or known mitochondrial disorder)
Refractory SE	Seizures persist despite emergent + urgent control therapy	Intubate; midazolam infusion (load 0.2 mg/kg over 2–5 min, repeat q5min to max 2 mg/kg, then 0.05–2 mg/kg/h) or propofol (load 1–2 mg/kg over 3–5 min, repeat q3–5min to max 10 mg/kg total, then 30–200 mcg/kg/min)	Phenobarbital 20 mg/kg IV at 1 mg/kg/min, or midazolam 0.1–0.2 mg/kg load then 0.1–1 mg/kg/h infusion	Continuous EEG monitoring is mandatory; titrate to seizure suppression or burst suppression
Super-refractory SE	Seizures continue or recur ≥24 h despite	Continue/escalate anesthetic infusion	Same principles, weight-based	Involve neurocritical care;

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Stage	Onset Timing	Adult Dosing	Pediatric Dosing	Key Notes
	anesthetic infusion	targeting burst suppression; consider ketamine infusion, inhaled anesthetics, immunotherapy if autoimmune etiology suspected		evaluate for autoimmune/paraneoplastic encephalitis and consider ketogenic diet or electroconvulsive therapy in refractory cases

Adapted from ENLS Status Epilepticus Protocol v6.0, Neurocritical Care Society.

Practice Pearls — Status Epilepticus

Under-dosing the first benzodiazepine is the single most common preventable error; dose by actual weight and do not round down.

Check a bedside glucose in every seizing patient — hypoglycemia is rapidly reversible and easily missed under time pressure.

A seizure that has clinically stopped but the patient remains unresponsive at 10 minutes should raise suspicion for non-convulsive SE — obtain EEG rather than assuming a prolonged postictal state.

Do not delay the second-line agent while waiting to see if a second benzodiazepine dose 'might still work' — the algorithm is time-triggered, not response-triggered.

Red Flags Requiring Immediate Airway Control

Oxygen desaturation or inadequate respiratory effort during or after benzodiazepine administration

Seizures ongoing at the 20-minute mark despite first- and second-line therapy

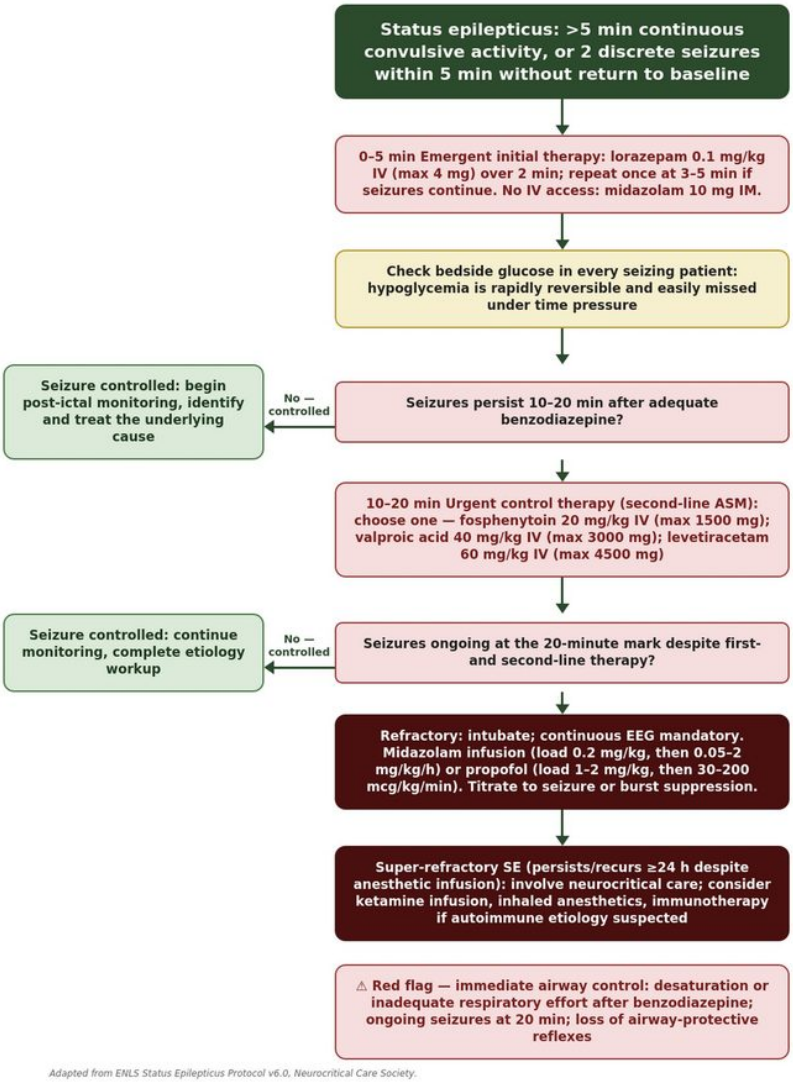
Loss of airway-protective reflexes with ongoing convulsive activity

Escalating doses of benzodiazepines given without effect (increases risk of respiratory depression without seizure control)

Adapted from ENLS Status Epilepticus Protocol v6.0, Neurocritical Care Society.

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Chapter 7 · Status Epilepticus — Stepwise Management



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6 Elevated Intracranial Pressure and Cerebral Herniation

Sustained elevation of intracranial pressure (ICP) above 22 mmHg for more than 5 minutes defines intracranial hypertension requiring treatment. Early symptoms are nonspecific — headache, nausea and vomiting, and a decline in mental status — while pupillary asymmetry is a more concerning and specific sign. Cushing's triad (hypertension, bradycardia, and irregular respirations) reflects brainstem compression from herniation but is an uncommon and often late finding; clinicians should not wait for the full triad before acting on other signs.

Herniation syndromes differ by anatomic location. Uncal (lateral transtentorial) herniation classically presents with acute decline in consciousness plus an ipsilateral fixed and dilated pupil (compression of cranial nerve III) and contralateral hemiparesis (corticospinal tract compression). Subfalcine herniation involves the medial frontal lobe shifting under the falx and may cause contralateral leg weakness from anterior cerebral artery compression. Tonsillar herniation, from posterior fossa or global mass effect, compresses the medulla and can cause sudden apnea and cardiovascular collapse — a true neurological code requiring immediate action, not diagnostic delay.

Signs of Impending or Active Herniation

New or worsening pupillary asymmetry, especially a unilateral fixed, dilated pupil

Acute decline in GCS of 2 or more points

New extensor (decerebrate) or flexor (decorticate) posturing

Cushing's triad — hypertension, bradycardia, irregular respirations

Sudden apnea or respiratory pattern change in a patient with known mass effect

Table 7.5 Hyperosmolar Therapy for Elevated ICP

Agent	Typical Dose	Access	Key Cautions
Mannitol	0.5–1 g/kg IV bolus over 5–15 min; may repeat every 4–6 h	Peripheral IV acceptable	Causes osmotic diuresis and can precipitate hypotension/hypovolemia; monitor serum osmolality and osmolar gap; use caution in renal impairment
Hypertonic saline 3%	250–500 mL (or 2–5 mL/kg) IV bolus over 10–20 min; may repeat or run as continuous infusion	Peripheral IV acceptable for short-term/lower concentration use	Keep serum sodium <160 mEq/L; correct/avoid over-rapid correction to reduce osmotic demyelination risk; monitor for volume overload
Hypertonic saline 23.4%	30 mL IV bolus over approximately 10 min	Central line required	Reserved for acute herniation rescue; large osmotic bolus can cause transient hypotension; requires central access which may delay

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Agent	Typical Dose	Access	Key Cautions
			administration

Concentrations ≥7.5% require central venous access per ENLS Intracranial Hypertension and Herniation Protocol.

- Head of bed elevated above 30 degrees with the neck kept midline to optimize venous outflow.
- Adequate analgesia and sedation (propofol is favored where hemodynamics allow, as it also reduces cerebral metabolic demand).
- Brief hyperventilation to a PaCO2 of 30–35 mmHg is a temporizing bridge for impending herniation only, limited to under 2 hours, because prolonged hyperventilation risks cerebral vasoconstriction and secondary ischemia.
- Target normothermia (36–37.4°C); mild therapeutic hypothermia (32–34°C) is reserved for advanced, refractory tiers of care.
- CSF diversion via external ventricular drain for obstructive hydrocephalus.
- Avoid hyponatremia, hypotension, hypoxia, hyperthermia, and noxious/painful stimuli, all of which independently worsen ICP or secondary brain injury.

ICP and CPP Key Numbers

Intracranial hypertension: sustained ICP >22 mmHg for >5 minutes

Cerebral perfusion pressure (CPP) is supported with vasopressors as needed to maintain adequate cerebral perfusion once ICP is being actively managed

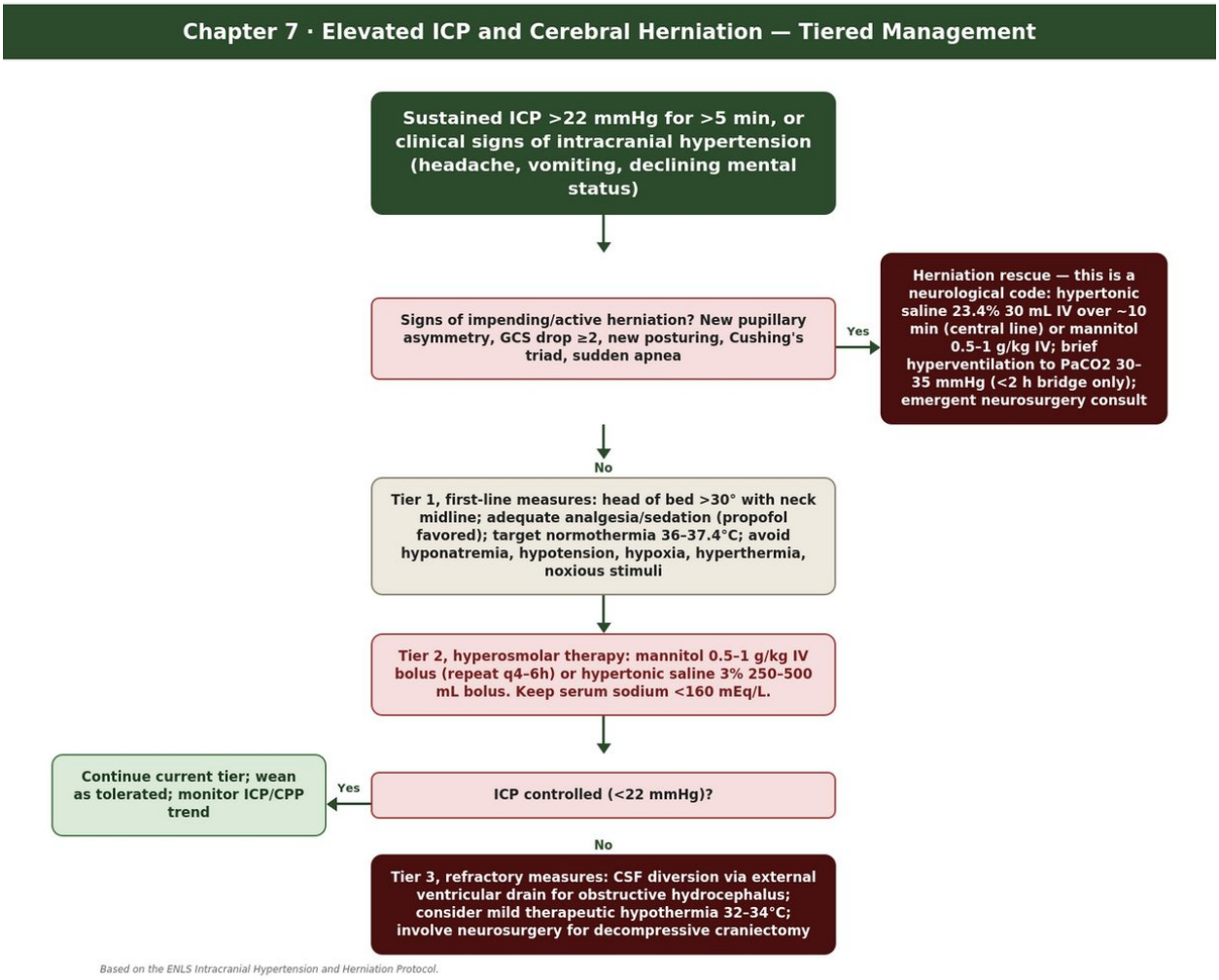
Therapeutic hyperventilation target PaCO2 30–35 mmHg, limited to <2 hours

Target normothermia 36–37.4°C; hypothermia tier 32–34°C

Serum sodium ceiling with hypertonic saline: <160 mEq/L

Based on the ENLS Intracranial Hypertension and Herniation Protocol.

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7 Traumatic Brain Injury

TBI severity is classified by the post-resuscitation Glasgow Coma Scale, obtained after airway, breathing, and circulation have been stabilized and any sedating or paralytic agents accounted for. This classification drives triage, imaging urgency, and monitoring decisions across every trauma system.

Table 7.6 Glasgow Coma Scale Components

Component	Response	Score
Eye opening	Spontaneous / To voice / To pain /	4 / 3 / 2 / 1

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Component	Response	Score
	None	
Verbal response	Oriented / Confused / Inappropriate words / Incomprehensible sounds / None	5 / 4 / 3 / 2 / 1
Motor response	Obeys commands / Localizes pain / Withdraws from pain / Abnormal flexion (decorticate) / Extension (decerebrate) / None	6 / 5 / 4 / 3 / 2 / 1

Total GCS = Eye + Verbal + Motor, range 3–15.

Table 7.7 TBI Severity Classification and Initial Management

Severity	GCS	Typical Features	Management Priorities
Mild	13–15	Brief or no loss of consciousness, may have normal CT	Observation, CT per validated head-injury decision rule, safe discharge planning if imaging normal and exam reliable
Moderate	9–12	Lethargy, may follow commands inconsistently	CT head, admit for serial neuro checks, low threshold for neurosurgical consultation
Severe	3–8	Unable to follow commands, may require airway protection	Secure airway, CT head, mandatory neurosurgical consultation, consider ICP monitoring per criteria below

- Maintain SpO2 ≥94% and target end-tidal CO2 35–38 mmHg; avoid prophylactic hyperventilation unless active herniation is present.
- Hemodynamic targets to avoid secondary injury: systolic BP ≥110 mmHg for ages 15–49 and over 70, and ≥100 mmHg for ages 50–69.
- Correct coagulopathy expeditiously within the first hour — check PT/PTT, platelets, and fibrinogen, and reverse anticoagulants immediately when identified.
- Tranexamic acid (TXA) may benefit select mild-to-moderate TBI when given within 3 hours of injury; discuss with trauma/neurosurgery per local protocol.
- Seizure prophylaxis with levetiracetam (preferred) can be considered for up to 7 days in moderate-to-severe TBI to reduce early post-traumatic seizures.
- ICP monitoring is indicated for GCS ≤8 with an abnormal head CT, or GCS ≤8 with a normal CT plus two or more of: age >40, motor posturing, or systolic BP <90 mmHg.

Busy-Shift Tip — TBI

Neurosurgical consultation is warranted for every moderate-to-severe TBI and for any mild TBI with a

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skull fracture, unexplained neurological deficit, cervical spine injury, or signs of CSF leak (rhinorrhea/otorrhea) — do not wait for deterioration to call.

8 Subarachnoid Hemorrhage

Aneurysmal subarachnoid hemorrhage (SAH) classically presents as an abrupt, severe headache that the patient describes as qualitatively different from any prior headache — 'the worst headache of my life' — often reaching maximal intensity within seconds (thunderclap headache). Roughly half of patients have a normal neurological exam at presentation, and headaches sometimes respond partially to standard analgesics or improve spontaneously, both of which can falsely reassure an unwary clinician. Any thunderclap headache warrants non-contrast CT, and if CT is negative but suspicion remains, lumbar puncture or CT angiography to exclude SAH.

Table 7.8 SAH Clinical Grading Scales

Grade	Hunt-Hess Criteria	WFNS (GCS-Based)
1	Asymptomatic or mild headache, slight nuchal rigidity	GCS 15, no motor deficit
2	Moderate-to-severe headache, nuchal rigidity, no deficit other than cranial nerve palsy	GCS 13–14, no motor deficit
3	Drowsiness, confusion, or mild focal deficit	GCS 13–14, with motor deficit
4	Stupor, moderate-to-severe hemiparesis	GCS 7–12, with or without motor deficit
5	Coma, decerebrate posturing	GCS 3–6, with or without motor deficit

- Before the aneurysm is secured, treat severe hypertension with short-acting, titratable agents (nicardipine or clevidipine) to a systolic target below 160 mmHg; avoid nitroprusside and nitroglycerin, both of which can raise ICP.
- Rebleeding prevention: strict bed rest, pain and anxiety control with short-acting IV agents, avoidance of Valsalva/straining, and stool softeners.
- Definitive aneurysm securing (endovascular coiling or surgical clipping) should occur as soon as feasible, ideally within the first 24 hours, since rebleeding risk is highest in this early window.
- Start nimodipine 60 mg orally or via feeding tube every 4 hours for 21 days to reduce delayed cerebral ischemia from vasospasm; if hypotension limits tolerance, reduce to 30 mg every 2 hours rather than stopping the drug.
- Monitor for vasospasm with serial clinical exams and transcranial Doppler ultrasonography, typically peaking days 4–14 after hemorrhage; manage symptomatic vasospasm with euvolemia and induced hypertension once the aneurysm is secured.

Myth vs. Fact — Vasospasm Management

Myth: Aggressive 'Triple-H therapy' (hypertension, hypervolemia, hemodilution) should be started

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prophylactically in every SAH patient to prevent vasospasm.

Fact: Current practice favors maintaining euvolemia (not hypervolemia) in all patients, reserving induced hypertension for patients who develop symptomatic vasospasm after the aneurysm is secured; routine prophylactic hypervolemia and hemodilution increase complications without proven benefit.

9 Spinal Cord Injury and Acute Spinal Compression

Acute spinal cord injury (traumatic) and non-traumatic acute spinal cord compression (epidural abscess, malignant cord compression, hematoma) share a common urgency: neurological deficits from cord compression can become permanent within hours, and both recognition and immobilization must happen quickly. Assess and document a motor and sensory level, distinguish spinal shock (transient loss of all cord function below the lesion, including reflexes) from neurogenic shock (loss of sympathetic tone causing hypotension with paradoxical bradycardia), and maintain a high index of suspicion for cord compromise in any trauma patient with midline spinal tenderness, focal weakness, a sensory level, or bowel/bladder dysfunction.

- Immobilize the spine with a rigid collar and appropriate transport precautions, log-rolling for all position changes; avoid prolonged rigid immobilization once imaging clears the spine, since extended collar use itself causes morbidity (pressure injury, aspiration risk).
- Maintain mean arterial pressure at 85–90 mmHg for 5–7 days after acute traumatic spinal cord injury to support cord perfusion, using vasopressors as needed once hypovolemia is excluded.
- High-dose methylprednisolone (the NASCIS protocol) is no longer routinely recommended — current guidelines regard the evidence for benefit as weak and outweighed by infection and GI bleeding risk; if used at all, it should follow local/regional protocol and shared decision-making rather than reflexive administration.
- For non-traumatic compression (epidural abscess or malignant cord compression), obtain emergent MRI of the entire spine and involve neurosurgery/spine surgery immediately — decompression within hours, not the next day, is associated with better neurological recovery.
- For malignant cord compression, start dexamethasone promptly while arranging emergent radiation oncology or surgical evaluation.

Decision Point: Non-Traumatic Cord Compression

New back pain with focal weakness, a sensory level, or new bowel/bladder dysfunction = emergent whole-spine MRI, not outpatient workup.

Fever plus back pain plus a neurological deficit = presume epidural abscess until proven otherwise; obtain blood cultures and start empiric antibiotics after (not instead of) urgent imaging and neurosurgical contact.

Known malignancy with new back pain and any neurological change = presume cord compression, start dexamethasone, and obtain emergent MRI same-day.

10 Non-Traumatic Weakness: Guillain-Barré Syndrome and Myasthenic Crisis

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Guillain-Barré syndrome (GBS) typically presents as ascending, symmetric limb weakness with areflexia, often following a preceding infection, and can progress to respiratory failure over hours to days; autonomic instability (labile blood pressure, arrhythmia) is common and can be life-threatening. The critical bedside task is not diagnosis alone but serial respiratory monitoring — trending forced vital capacity (FVC) and negative inspiratory force (NIF) — because clinical deterioration in GBS can be abrupt and a patient who looks stable can decompensate quickly. Treatment is IVIG or plasma exchange, started as early as possible; corticosteroids are not effective in GBS and should not be used.

Myasthenic crisis is an acute worsening of myasthenia gravis causing respiratory failure, often precipitated by infection, surgery, tapering of immunosuppression, or certain medications (aminoglycosides, fluoroquinolones, magnesium, and some beta-blockers can all worsen neuromuscular transmission and should be avoided). As with GBS, serial FVC/NIF trending — rather than pulse oximetry alone, which can remain normal until very late — is the key bedside monitoring tool, since oxygenation is often preserved until ventilatory failure is imminent. Treatment is IVIG or plasma exchange; a distinguishing pitfall is that high-dose corticosteroids can transiently worsen myasthenic weakness in the first 5–10 days and should be started cautiously, ideally with the patient already in a monitored setting.

- Trend serial FVC and NIF (not pulse oximetry alone) in any patient with suspected neuromuscular respiratory failure; a rapidly falling FVC (below roughly 15–20 mL/kg, or NIF less negative than -20 to -25 cmH2O) signals impending ventilatory failure and should prompt early, elective intubation.
- Avoid depolarizing paralytics (succinylcholine) in GBS and myasthenia due to unpredictable hyperkalemic response or resistance/sensitivity to neuromuscular blockade.

11 Coma and Altered Mental Status

Coma is a state of unresponsiveness to external stimuli that cannot be explained by sleep, sedation, or a primary psychiatric process, and it always demands a systematic search for a cause before any prognosis is offered. The approach is: stabilize ABCs, obtain a rapid finger-stick glucose, perform a focused neurological exam (GCS, pupils, brainstem reflexes, motor asymmetry), and pursue a broad but structured differential that separates structural (mass lesion, hemorrhage, herniation, large stroke) from diffuse/metabolic-toxic causes (hypoglycemia, hypoxia, electrolyte derangement, uremia, hepatic encephalopathy, toxin/overdose, hypothermia, and postictal states).

- A widely used mnemonic for reversible causes is AEIOU-TIPS: Alcohol/toxins, Epilepsy, Insulin (hypo/hyperglycemia), Oxygen (hypoxia), Uremia, Trauma, Infection, Psychiatric, Stroke/Structural — every letter should be actively excluded before assuming an irreversible cause.
- Naloxone and dextrose are reasonable empiric therapies in undifferentiated coma with a plausible history, and thiamine should be given before or with dextrose in patients at risk of Wernicke encephalopathy (alcohol use, malnutrition) to avoid precipitating or worsening the condition.
- A non-contrast CT head is obtained early in essentially every undifferentiated coma to exclude a structural or hemorrhagic cause requiring emergent intervention.
- Do not anchor on a single presumed cause (e.g., 'found down, probably intoxicated') until reversible and structural causes have been actively excluded — this is one of the most common sources of missed diagnoses in altered mental status.

12 Meningitis and Encephalitis

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Suspected bacterial meningitis or herpes simplex encephalitis is a true emergency in which the priority is empiric antimicrobial and antiviral therapy within one hour of presentation — imaging and lumbar puncture should never delay the first dose of antibiotics when bacterial meningitis is suspected. Blood cultures should be drawn immediately before antibiotics if this does not cause delay; if CT is required before lumbar puncture (altered mental status, focal deficit, papilledema, immunocompromise, or seizure), give empiric antibiotics and, when indicated, dexamethasone before the CT rather than waiting.

Table 7.9 Empiric Therapy for Suspected Meningitis/Encephalitis

Population / Risk Factor	Empiric Regimen	Notes
Immunocompetent adult	Vancomycin + ceftriaxone	Give dexamethasone with or just before the first antibiotic dose to reduce neurological complications of pneumococcal meningitis
Age ≥50, alcoholism, or immunocompromised	Vancomycin + ceftriaxone + ampicillin	Ampicillin added for Listeria monocytogenes coverage
Post-neurosurgery, penetrating trauma, or CSF shunt	Vancomycin + cefepime (or meropenem)	Covers hospital-acquired and Pseudomonas/staphylococcal pathogens
Suspected HSV encephalitis (any age with focal deficit, seizure, or temporal lobe findings)	Add acyclovir 10 mg/kg IV every 8 hours	Start empirically pending CSF HSV PCR; untreated HSV encephalitis carries very high morbidity/mortality

Regimens should be adjusted for local resistance patterns and confirmed against institutional infectious disease guidance.

13 Worked Clinical Case

Case 7.1 — Sudden Right-Sided Weakness and Slurred Speech

Presentation: A 71-year-old woman is brought to the ED by EMS after her husband found her slumped and unable to speak clearly at 2:10 PM; he had spoken with her normally at 1:40 PM. Prehospital BE-FAST screen is positive for facial droop, arm drift, and slurred speech. On arrival vital signs are BP 176/94, HR 88, SpO2 97% on room air, glucose 118 mg/dL. NIHSS is 14 (facial palsy, right arm and leg plegia, dysarthria, mild neglect).

Management: Stroke code is activated immediately. Non-contrast CT at 2:35 PM shows no hemorrhage and ASPECTS of 9; CTA shows a left M1 MCA occlusion. Last known well is 1:40 PM, so she is 55 minutes from onset, well within the standard alteplase window and the thrombectomy 0–6 h window. No absolute exclusion criteria are present. IV alteplase 0.9 mg/kg (bolus plus 60-minute infusion) is started at 2:42 PM (door-to-needle 32 minutes) while she is simultaneously transferred to interventional neuroradiology for mechanical thrombectomy. BP is kept below 180/105 mmHg with a nicardipine infusion throughout.

Outcome/teaching point: Thrombectomy achieves complete recanalization at 3:40 PM. By post-procedure day 2 her NIHSS has fallen to 3. This case illustrates the core ENLS principle for stroke: thrombolysis and thrombectomy pathways run in parallel, not sequentially — the patient was treated with alteplase and transferred for thrombectomy simultaneously rather than waiting to see if thrombolysis alone would work, and BP was actively controlled throughout to stay within the post-thrombolysis target.

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14 Chapter Summary and Quick Reference

The table below consolidates the time-critical numbers and doses covered in this chapter for rapid bedside recall. It is not a substitute for the full protocols and local institutional pathways.

Table 7.10 ENLS Quick-Reference — Key Time Windows and Doses

Domain	Parameter	Value
Ischemic stroke	IV alteplase standard window	<3 h from onset, extended to 4.5 h per AHA/ASA
Ischemic stroke	IV alteplase dose	0.9 mg/kg (max 90 mg); 10% bolus, remainder over 60 min
Ischemic stroke	IV tenecteplase dose	0.25 mg/kg (max 25 mg) single bolus
Ischemic stroke	Mechanical thrombectomy window	0–6 h standard; up to 24 h with favorable perfusion/DWI imaging
Ischemic stroke	Pre-thrombolysis BP target	<185/110 mmHg
Ischemic stroke	Post-thrombolysis BP target (24 h)	<180/105 mmHg
Status epilepticus	First-line benzodiazepine (adult, IV)	Lorazepam 0.1 mg/kg IV, max 4 mg; repeat once at 3–5 min
Status epilepticus	Second-line ASM options	Fosphenytoin, valproic acid, or levetiracetam, all weight-based IV
Status epilepticus	Refractory SE onset	Seizures persisting despite emergent + urgent control therapy — intubate, start continuous infusion, begin cEEG
Elevated ICP	Treatment threshold	Sustained ICP >22 mmHg for >5 min
Elevated ICP	Mannitol dose	0.5–1 g/kg IV bolus, may repeat q4–6h
Elevated ICP	Hypertonic saline 3%	250–500 mL IV bolus; keep Na <160 mEq/L
TBI	GCS severity bands	Mild 13–15 / Moderate 9–12 / Severe 3–8
TBI	TXA window	Within 3 h of injury (select mild-moderate TBI)
SAH	Nimodipine	60 mg PO/NG q4h x 21 days (reduce to 30 mg q2h if hypotensive)
SAH	Pre-securing BP target	Systolic <160 mmHg
SAH	Time to aneurysm securing	Ideally within 24 h
Spinal cord injury	MAP target	85–90 mmHg for 5–7 days

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Domain	Parameter	Value
Meningitis/encephalitis	Time to first antibiotic/antiviral dose	Within 1 hour of presentation

15 Intracerebral Hemorrhage

Spontaneous (non-traumatic) intracerebral hemorrhage (ICH) is a stroke subtype in its own right, distinct from subarachnoid hemorrhage (Section 8) and from the ischemic stroke pathway (Section 4). This section reflects the AHA/ASA Guideline for the Management of Spontaneous Intracerebral Hemorrhage.

Blood pressure control

Lowering systolic blood pressure to a target of 130-140 mmHg is safe and reasonable in improving functional outcome in acute ICH, reflecting evidence that intensive early lowering reduces hematoma expansion. Pushing the target below roughly 130 mmHg has not shown consistent additional benefit and has raised some safety concerns in trial data, so aggressive over-correction is not the goal; achieve the target range as smoothly and stably as possible rather than with large swings.

Anticoagulation reversal

Hematoma expansion, rapid deterioration, and poor outcome are all more common in patients on anticoagulation at the time of hemorrhage, making emergency reversal a priority parallel to blood pressure control, not a sequential afterthought. Reversal agent choice depends on which anticoagulant the patient is taking (vitamin K antagonist, direct oral anticoagulant, or antiplatelet agent) and should follow your institution's specific reversal protocol; an INR of 1.5 or below is recommended before any invasive intervention such as external ventricular drain (EVD) placement.

Hematoma volume and monitoring

The ABC/2 method estimates hematoma volume at the bedside from a single CT slice: A is the largest diameter of the hemorrhage, B is the largest diameter perpendicular to A on the same image, and C is the number of CT slices on which the hemorrhage appears multiplied by slice thickness; volume in cubic centimeters is approximately (A x B x C) / 2. Serial neurologic checks and repeat imaging watch for hematoma expansion or the development of hydrocephalus, either of which can trigger the intracranial hypertension and herniation pathway in Section 6.

Systems of care

The guideline recommends organized regional systems for initial ICH care with the capacity for rapid transfer to a center with neurocritical care and neurosurgical capability when the presenting facility lacks these resources - the same systems-of-care principle that underlies stroke and trauma center networks elsewhere in this manual.

16 Temperature Control

Fever independently worsens outcome after acute brain injury - ischemic stroke, ICH, subarachnoid hemorrhage, and traumatic brain injury alike - by increasing metabolic demand in already-vulnerable tissue. Temperature control (sometimes still called targeted temperature management, TTM) in this context means aggressively maintaining normothermia, not inducing therapeutic hypothermia, which is a distinct

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intervention used in specific post-cardiac-arrest protocols (Chapter 1, Section 13) rather than routine neurocritical care.

A controlled normothermia target of 36.0-37.5°C is recommended as a therapeutic option for patients felt to be at risk of secondary brain injury from fever, with continuous temperature monitoring (or at minimum hourly checks) in any ventilated patient with ICH, subarachnoid hemorrhage, or acute ischemic stroke in a critical care setting. An automated, feedback-controlled cooling device provides more precise and reliable temperature control than intermittent antipyretics and cooling blankets alone when fever is refractory. Shivering during active temperature control is itself harmful - it raises metabolic rate and can increase intracranial pressure - and is managed with a stepwise approach (counter-warming of the hands and face, then pharmacologic treatment) rather than tolerated as an unavoidable side effect.

17 Agitation

Agitation in a patient with acute brain injury is a symptom with a differential diagnosis, not a behavior to simply sedate away: it can signal pain, hypoxia, a full bladder, alcohol or sedative withdrawal, a new intracranial event, or the underlying brain injury itself, and the underlying cause should be actively sought before or alongside treating the agitation.

A validated scale - the Richmond Agitation-Sedation Scale (RASS) is the most widely used - standardizes assessment and communicates severity across a care team far better than descriptive terms like "agitated" or "combative" alone. Non-pharmacologic measures (reorientation, reducing unnecessary noise and stimulation, involving family, correcting sensory deficits such as missing glasses or hearing aids) are first-line for mild agitation and delirium. When medication is required, agents are chosen with the underlying neurologic problem in mind: sedation should be as light as the clinical situation allows so that neurologic exams remain interpretable, since over-sedation can mask a new deficit or a deteriorating exam - the same principle that governs sedative choice during targeted temperature management in Section 16. Physical restraint is a last resort, used only when the patient is an immediate danger to themselves or the care team and de-escalation and medication have failed, and is applied per institutional protocol with regular reassessment for the least restrictive effective measure.

This chapter is adapted from the Neurocritical Care Society's Emergency Neurological Life Support (ENLS) protocols, Version 6.0, and current (2026) AHA/ASA acute ischemic stroke guideline updates, verified current as of August 2026. It is intended as an educational reference summary and is not a substitute for official ENLS course certification, institutional protocols, or the full current-version source protocols, which should be consulted directly for clinical decision-making.

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LIFE SUPPORT COURSES · CLINICAL COMPANION MANUAL

CHAPTER 8

PHTLS

Prehospital Trauma Life Support

1 Clinical Snapshot

Trauma outcomes are shaped as much in the field as in the trauma bay. PHTLS (Prehospital Trauma Life Support) is the prehospital counterpart to ATLS: it governs the assessment and care an EMS crew delivers from the moment of dispatch through handoff, when scene hazards, limited resources, extrication, and transport time all shape decision-making in ways an emergency department team does not face. The full PHTLS provider course runs two days and combines didactic teaching with hands-on skills stations and a practical evaluation; this chapter distills its core content into a single reference, covering scene management, kinematics, the MARCH primary assessment sequence, hemorrhage control, thoracic and traumatic brain injury, spinal motion restriction, abdominal, pelvic, musculoskeletal, and burn trauma, special populations, and the transport decisions that determine which patients need the shortest possible scene time.

2 Learning Objectives

- Perform a scene size-up that addresses responder safety, PPE, number of patients, and mechanism of injury before patient contact.
- Relate the kinematics of blunt, penetrating, and blast mechanisms to predictable, occult injury patterns.
- Apply the MARCH sequence (Massive hemorrhage, Airway, Respiration, Circulation, Hypothermia/Head injury) as the prehospital primary assessment.
- Select and correctly apply a tourniquet or hemostatic dressing for life-threatening external hemorrhage.
- Recognize and manage the immediately life-threatening chest injuries: tension pneumothorax, open pneumothorax, flail chest, massive hemothorax, and cardiac tamponade.
- Describe the pathophysiology of hemorrhagic and non-hemorrhagic shock and the prehospital resuscitation strategies that limit further harm.
- Distinguish primary from secondary traumatic brain injury and apply management that protects cerebral perfusion.
- Apply current, evidence-based spinal motion restriction criteria rather than routine full immobilization.
- Recognize and provide initial field management for abdominal, pelvic, musculoskeletal, and burn trauma.
- Adapt assessment and management for pediatric, geriatric, and pregnant trauma patients.
- Use the CDC field triage decision scheme to select an appropriate destination facility and construct a structured MIST handoff.

3 Scene Size-Up and Scene Safety

Scene size-up happens before patient contact and is repeated throughout the call. It answers four questions: Is the scene safe for responders? Is body substance isolation/PPE appropriate to the hazard? How many patients are there, and is additional help (extra units, fire/rescue, law enforcement, air medical) needed? What does the mechanism of injury suggest about likely injury patterns before formal assessment begins?

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An unsafe scene is not made safe by the presence of a patient. Traffic, unstable structures, hostile bystanders, downed power lines, and hazardous materials all take priority over patient contact until controlled or mitigated by the appropriate specialty resource. Extrication, when needed, is coordinated with fire/rescue; rapid extrication is reserved for patients with immediately life-threatening findings who cannot be adequately treated while still confined, while a more deliberate approach is used when the patient is stable and freeing them safely will take longer.

4 Kinematics of Trauma

Mechanism of injury (kinematics) is a field-only data point: the vehicle intrusion, fall height, or weapon type is usually far clearer at the scene than it will ever be by the time the patient reaches the hospital. Reporting it accurately lets the receiving team anticipate injuries that are not yet clinically obvious.

Blunt trauma

Blunt injury results from compression, shear, and rapid deceleration forces distributed over a broad body surface. In motor vehicle crashes, the pattern of intrusion and restraint use (seatbelt, airbag deployment) predicts specific injuries: frontal impact with an unrestrained occupant suggests head, face, and chest injury from steering wheel or windshield contact; lateral impact suggests ipsilateral chest, pelvic, and abdominal injury; and rear impact suggests cervical spine hyperextension. Falls are characterized by height and landing surface; axial loading from a fall onto the feet or buttocks transmits force up the spine and can produce calcaneal, tibial, hip, pelvic, and lumbar spine fractures far from the point of impact.

Penetrating trauma

Penetrating injury severity depends on the energy of the wounding object and the tissue it passes through. Low-velocity penetrating injury (most stab wounds) damages tissue largely along the direct path of the object. High-velocity gunshot wounds transfer far more kinetic energy and create a temporary cavity many times the bullet's diameter as tissue is stretched and compressed outward from the permanent wound track, injuring structures well away from the visible entry and exit wounds. Never assume a straight-line path between entry and exit; bullets deflect off bone and change course, and the number of wounds should always be counted and reported rather than assumed.

Blast injury

Blast injuries are classified in four groups. Primary blast injury results from the overpressure wave itself and preferentially damages air-fluid interface organs: the tympanic membranes, lungs (blast lung, which can produce diffuse alveolar hemorrhage without external chest signs), and bowel. Secondary blast injury comes from fragments and debris propelled by the explosion, producing penetrating wounds. Tertiary blast injury results from the patient's body being thrown by the blast wind, producing blunt trauma. Quaternary blast injury covers everything else related to the event - burns, inhalation injury, and crush injury from structural collapse.

5 The MARCH Algorithm — Prehospital Primary Assessment

MARCH is the field-adapted primary assessment sequence, developed from battlefield trauma care (TCCC) and adopted by PHTLS for civilian prehospital use. Like the in-hospital xABCDE sequence used elsewhere in this manual, it moves control of life-threatening external hemorrhage ahead of airway, reflecting how quickly a patient can exsanguinate from a compressible source.

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M — Massive hemorrhage

Identify and control exsanguinating external hemorrhage first, before airway. Apply a tourniquet or hemostatic dressing as described in Section 6 without waiting to complete a full exam.

A — Airway

Open and maintain the airway with manual in-line stabilization considerations in a trauma patient; jaw thrust is preferred over head-tilt-chin-lift when spinal injury cannot be excluded. Suction, insert an airway adjunct, and prepare for a surgical airway if the airway cannot be secured by other means.

R — Respiration

Expose the chest and assess for tension pneumothorax, open pneumothorax, and flail chest (see Section 8). Needle decompress a suspected tension pneumothorax and apply a vented chest seal to an open chest wound, leaving a flutter valve effect rather than a fully occlusive dressing to prevent converting an open pneumothorax into a tension pneumothorax.

C — Circulation

Reassess for additional sources of bleeding, obtain IV or IO access en route rather than delaying transport on scene, and follow a permissive-hypotension resuscitation strategy (target a systolic pressure sufficient to maintain a palpable radial pulse, roughly 80-90 mmHg, using small crystalloid boluses) in penetrating or hemorrhagic trauma without suspected traumatic brain injury. Give tranexamic acid (TXA) per local protocol - 1 g IV over 10 minutes - as early as possible and within 3 hours of injury in patients with significant hemorrhage or shock.

H — Hypothermia / Head injury

Remove wet or cold clothing, cover the patient, and use warmed fluids where available; hypothermia compounds the trauma lethal triad of hypothermia, acidosis, and coagulopathy, each of which worsens the other two. In suspected traumatic brain injury (Section 10), avoid hypoxia and hypotension, which independently worsen secondary brain injury, and avoid routine hyperventilation unless there are signs of impending herniation.

Once MARCH is complete and immediate threats are controlled, a brief secondary survey - a systematic head-to-toe exam - identifies less urgent injuries, ideally performed en route rather than extending scene time.

6 Hemorrhage Control in the Field

Uncontrolled hemorrhage remains the leading cause of preventable death after injury, which is why the public Stop the Bleed campaign and PHTLS both teach the same core skills: direct pressure, wound packing, and tourniquet application.

Tourniquets

Extremity hemorrhage that is life-threatening (spurting, pooling, or has already caused signs of shock) is controlled with a windlass-type tourniquet applied 2-3 inches proximal to the wound (never directly over a joint), tightened until bleeding stops and the distal pulse is absent, and time-stamped. A second tourniquet may be applied side-by-side, proximal to the first, if bleeding continues or if a single tourniquet cannot generate enough pressure on a large thigh. Direct pressure is not a substitute once a tourniquet is indicated - apply the tourniquet first for clearly life-threatening bleeding rather than trialing pressure alone. A

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tourniquet that has controlled hemorrhage should not be loosened or removed in the field; conversion to a pressure dressing is a hospital decision made once the wound can be properly assessed.

Wound packing and hemostatic dressings

Junctional hemorrhage (groin, axilla, neck) and non-extremity wounds not amenable to a tourniquet are managed by packing the wound directly with plain or hemostatic gauze, maintaining firm, continuous direct pressure for at least three minutes without peeking to check the wound, or with a purpose-built junctional tourniquet device where available. Packing should fill the wound cavity, not merely cover its surface.

7 Airway and Breathing Management

Field airway management is shaped by limited resources and short transport times: a definitive airway is not always necessary or achievable on scene. Basic maneuvers (jaw thrust, suction, an oropharyngeal or nasopharyngeal airway) resolve most obstructions. Extraglottic devices are the field bridge when bag-mask ventilation is inadequate and intubation is not immediately achievable; drug-assisted intubation, where within scope, is reserved for patients who cannot be oxygenated or protected by simpler means. A nasopharyngeal airway should be avoided, or used with caution, when a significant midface or suspected basilar skull fracture is present.

8 Thoracic Trauma

The chest houses the heart, great vessels, and lungs, and its injuries account for a disproportionate share of immediately preventable trauma deaths. Six chest injuries are considered immediately life-threatening and are actively sought during the R step of MARCH.

Table 8.1 Immediately Life-Threatening Chest Injuries

Injury	Key field findings	Field management
Tension pneumothorax	Respiratory distress, unilateral absent/decreased breath sounds, hypotension, distended neck veins; tracheal deviation is late and unreliable	Needle decompression (2nd ICS midclavicular line or 4th/5th ICS anterior-to-mid axillary line per local protocol)
Open pneumothorax	Visible open chest wound, air movement/sucking sound at the wound	Vented occlusive (chest-seal) dressing; do not fully occlude without a vent
Flail chest	Paradoxical chest wall movement over 2+ adjacent ribs fractured in 2+ places	High-flow oxygen; assist ventilation if respiratory failure develops; splinting/positioning for comfort
Massive hemothorax	Decreased breath sounds with dullness to percussion, signs of hemorrhagic shock	Treat as hemorrhagic shock; high-flow oxygen; rapid transport, no field drainage procedure
Cardiac tamponade	Beck's triad (muffled heart sounds, jugular venous distension, hypotension) - often incomplete in the field	High-flow oxygen; rapid transport; no field intervention corrects tamponade

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Injury	Key field findings	Field management
Traumatic asphyxia / massive chest wall injury	Facial/neck cyanosis and petechiae from severe chest compression	Support airway and breathing; treat associated injuries; rapid transport

ICS = intercostal space. Needle decompression site and technique should follow your local medical direction.

Simple rib fractures and pulmonary contusion are common, less immediately dangerous chest injuries; pain from rib fractures can itself cause shallow breathing and secondary hypoventilation, so pain control and encouraging deep breathing (splinting the area with a hand or pillow rather than rigid binding) matter even when no single fracture is life-threatening on its own.

9 Shock Pathophysiology and Prehospital Management

Shock is inadequate tissue perfusion to meet metabolic demand, and in trauma it is hemorrhagic until proven otherwise. Early compensated shock is easy to miss in the field because sympathetic-driven tachycardia and peripheral vasoconstriction can maintain a normal blood pressure despite significant blood loss, particularly in young, healthy patients - by the time hypotension appears, compensatory mechanisms are failing (see the hemorrhagic shock classification in Chapter 3, Section 10, for the in-hospital staging this feeds into). Early clues include tachycardia, delayed capillary refill, anxiety or altered mental status, and cool, pale, or diaphoretic skin, often before the systolic pressure falls at all.

Non-hemorrhagic causes of shock in trauma include tension pneumothorax and cardiac tamponade (obstructive), spinal cord injury (neurogenic, from loss of sympathetic tone below the level of injury, producing hypotension with a paradoxically normal or slow heart rate rather than tachycardia), and, less commonly in the acute trauma setting, cardiogenic causes from a cardiac contusion. Recognizing which category is in play changes the response: neurogenic shock is treated with careful fluid administration and, per protocol, a vasopressor rather than the aggressive volume resuscitation appropriate for hemorrhage.

Prehospital management favors permissive hypotension and small, titrated crystalloid boluses over aggressive fluid resuscitation, which can dilute clotting factors and dislodge early clot formation; the definitive treatment for hemorrhagic shock is blood and surgical or interventional control of bleeding, not crystalloid, so transport should never be delayed to complete fluid resuscitation on scene.

10 Traumatic Brain Injury

Primary brain injury is the structural damage that occurs at the moment of impact and cannot be reversed in the field. Secondary brain injury develops afterward from hypoxia, hypotension, hyperthermia, and hypo- or hyperglycemia, and is where prehospital care makes its biggest difference: a single episode of hypotension or hypoxia after TBI is associated with substantially worse outcomes.

Serial Glasgow Coma Scale scoring (see Chapter 3 for the full scale) is more useful than a single reading, since a worsening trend signals expanding intracranial injury. Watch for signs of impending herniation - a new pupillary asymmetry, a GCS drop of 2 or more points, posturing, Cushing's triad (hypertension, bradycardia, irregular respirations), or sudden apnea - and communicate any of these findings immediately, since they change the receiving facility's preparation and may warrant the brief hyperventilation and hyperosmolar therapy described in Chapter 7, Section 8 as an ENLS-guided herniation-rescue measure.

Field management is protective rather than curative: maintain the airway (avoiding hypoxia), support blood pressure (avoiding hypotension, and avoiding overly aggressive fluid resuscitation that could exacerbate

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intracranial bleeding), keep the head of a supine patient at a slight elevation when the spine allows it, keep the cervical collar from being excessively tight, and avoid hyperventilation except as a brief bridge for overt herniation signs. Treat every significant head injury as a potential spine injury until proven otherwise.

11 Spinal Trauma and Motion Restriction

Spinal cord injury can be worsened by unnecessary movement, but the historical practice of immobilizing every trauma patient on a rigid backboard has itself been shown to add little benefit while adding real harm - pressure injury, impaired ventilation, aspiration risk, increased pain, and increased scene time - which is why PHTLS has moved to selective spinal motion restriction (SMR).

SMR is indicated when any of the following is present:

- Altered level of consciousness (cannot reliably report pain).
- Midline spinal tenderness, step-off, or deformity on palpation.
- A focal neurologic deficit or complaint (numbness, weakness, paresthesia).
- A distracting, painful injury that could mask spinal pain.
- Intoxication by drugs or alcohol.
- A high-risk mechanism in a patient who cannot be reliably assessed.

If none of these are present and the patient is reliable and cooperative, SMR may be omitted even after a significant mechanism. When SMR is indicated, a rigid cervical collar plus securing the patient to the transport device (cot) with padding under natural contours is generally preferred to prolonged rigid backboard time; the backboard itself is best reserved for extrication and short transfers, then removed as soon as practical.

A spinal cord injury above roughly the T6 level can produce neurogenic shock (Section 9): hypotension with bradycardia or a normal heart rate rather than the tachycardia expected from hemorrhage. Do not assume a spinal-injured, hypotensive patient is simply volume-depleted without also considering a concurrent hemorrhagic source, since the two frequently coexist after a high-energy mechanism.

12 Abdominal and Pelvic Trauma

Blunt abdominal trauma (most often from motor vehicle crashes) can produce solid-organ (liver, spleen) or hollow-viscus injury with minimal external signs; a soft, non-tender abdomen on scene does not exclude significant internal hemorrhage, and unexplained shock after blunt torso trauma should always raise suspicion for intra-abdominal bleeding. Penetrating abdominal wounds are managed by covering the wound; an impaled object is stabilized in place and never removed in the field, and eviscerated bowel is covered with a moist, sterile dressing and an occlusive outer layer rather than pushed back inside.

Pelvic fractures from high-energy blunt trauma can bleed massively into the retroperitoneal space without any external wound. Do not spring or rock the pelvis to test for instability, since this can dislodge early clot; if pelvic fracture is suspected from mechanism, exam findings, or a positive field triage anatomic criterion, apply a commercial pelvic binder (or an improvised circumferential wrap) centered over the greater trochanters, and handle and transport the patient to minimize further pelvic movement.

13 Musculoskeletal Trauma

Extremity fractures and dislocations are rarely immediately life-threatening on their own but are a very common source of pain, bleeding, and, if a major artery is compromised, limb loss. Assess and document

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distal pulse, motor function, and sensation before and after any splinting. Splint a fracture in the position found if it is neurovascularly intact and the splint can accommodate it; if the limb is pulseless and deformed, a single gentle attempt at realignment with longitudinal traction is reasonable to restore perfusion before splinting, per local protocol. A femur fracture is splinted with a traction splint when there is no suspected pelvic, hip, knee, or lower-leg injury that traction would worsen; traction reduces pain and blood loss into the thigh by restoring approximate bone alignment and muscle length.

Crush syndrome develops when a limb has been compressed for a prolonged period (classically over an hour) and is then released, allowing potassium, myoglobin, and other byproducts of muscle breakdown to enter the circulation; this can cause sudden hyperkalemic cardiac arrest at the moment of release and later kidney injury. When prolonged entrapment is anticipated, coordinate with medical direction before extrication, since fluid administration prior to release, where within protocol, is part of mitigating this risk. Compartment syndrome - pain out of proportion to the injury, pain with passive stretch of the compartment's muscles, and a tensely swollen limb - cannot be corrected in the field but should be recognized and clearly communicated at handoff, since it is a time-sensitive surgical diagnosis.

14 Burn Injuries

Burns are described by depth and extent. Superficial (first-degree) burns involve the epidermis only and are painful and red without blistering. Partial-thickness (second-degree) burns extend into the dermis, blister, and are exquisitely painful. Full-thickness (third-degree) burns destroy the full dermis, appear white, leathery, or charred, and are often painless at the burn itself because nerve endings are destroyed - though surrounding partial-thickness burn is typically present and very painful.

Extent is estimated with the rule of nines (each arm 9%, each leg 18%, anterior and posterior trunk 18% each, head and neck 9%, genitalia 1%, adjusted for pediatric proportions which have a relatively larger head and smaller legs) or, for smaller or irregular burns, the patient's own palm (including fingers) representing approximately 1% of body surface area.

The airway is the first field priority in any burn with facial involvement, singed nasal hair or eyebrows, soot in the mouth or sputum, hoarseness, or a history of a closed-space fire, since thermal and inhalation injury can cause airway swelling that progresses over hours to complete obstruction; a low threshold for early transport and advanced airway evaluation is appropriate even when the patient is initially speaking normally. Stop the burning process (remove smoldering clothing and jewelry, brush off dry chemical powder before flushing with water), cover the burn with a dry, clean dressing (avoid ice, which worsens tissue injury and hypothermia in large burns), and treat electrical burns with a high index of suspicion for cardiac arrhythmia and deep tissue injury far exceeding what the skin surface suggests. Fluid resuscitation targets for major burns (such as the Parkland formula) are typically calculated and initiated at the receiving facility, but should be anticipated and communicated as part of the handoff for any burn covering a large body-surface-area.

15 Special Populations

Pediatric trauma

Children compensate for blood loss longer than adults through vigorous vasoconstriction and tachycardia, then decompensate abruptly; a normal blood pressure does not exclude significant hemorrhage, and hypotension in a child is a late and ominous sign. A length- or age-based reference should guide fluid and drug dosing en route. Children have a proportionally larger head and are more prone to airway obstruction from swelling or secretions; padding under the torso is often needed to maintain neutral spinal alignment given the head's relative size.

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Geriatric trauma

Older adults may not mount a tachycardic response to shock (rate-limiting medications, intrinsic conduction disease) and tolerate hypotension poorly even at numbers considered normal in a younger adult; a lower threshold for trauma-center transport is appropriate at age 65 and older. Anticoagulant or antiplatelet medication use significantly raises the risk of serious intracranial hemorrhage even after a low-energy fall, and this history should always be actively sought and reported.

Trauma in pregnancy

Pregnant trauma patients beyond about 20 weeks should be transported with a left lateral tilt or manual uterine displacement to relieve aortocaval compression, which otherwise reduces venous return and can cause maternal hypotension independent of blood loss. Maternal blood volume expands substantially in pregnancy, so a mother can lose a significant volume of blood while still appearing hemodynamically stable, even as the fetus is compromised; any trauma in pregnancy beyond a trivial mechanism warrants obstetric evaluation regardless of how minor the visible injury appears.

16 Scene Time, the Golden Period, and Transport Decisions

The historic “golden hour” has been reframed as the Golden Period: the principle that time to definitive hemorrhage control matters, without a rigid one-hour cutoff. In practice this means minimizing scene time for any patient meeting field trauma triage criteria - completing MARCH, applying only the interventions that cannot wait, and moving to transport, obtaining IV/IO access and continuing assessment en route rather than on scene.

17 Field Trauma Triage and Destination Decisions

The CDC Guidelines for Field Triage of Injured Patients (the same framework referenced from the hospital side in Chapter 3, Table 3.1) is applied prehospital as a stepwise decision scheme: physiologic criteria are checked first, then anatomic injury, then mechanism of injury, then special patient considerations. Meeting any criterion at a given step routes the patient to the highest level of trauma care within the local system, by ground or air, without needing to proceed to the next step.

Table 8.2 CDC Field Triage Decision Scheme (Stepwise)

Step	Criteria checked	Action if met
1. Physiologic	GCS <14; systolic BP <90 mmHg; respiratory rate <10 or >29/min (<20 in infant); need for ventilatory support	Transport to highest-level trauma center; consider air medical
2. Anatomic	Penetrating injury to head/neck/torso/proximal extremities; flail chest; two or more proximal long-bone fractures; crushed, degloved, mangled, or pulseless extremity; amputation proximal to wrist/ankle; pelvic fracture; open/depressed skull fracture; paralysis	Transport to highest-level trauma center; consider air medical
3. Mechanism	High-risk auto crash (intrusion, ejection, same-vehicle death); falls (adults >6 m, children >3 m or 2-3x height); motorcycle crash >20 mph; auto vs. pedestrian/cyclist	Transport to trauma center; contact medical control if uncertain

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Step	Criteria checked	Action if met
4. Special considerations	Age >65; anticoagulant or bleeding disorder; burns with associated trauma; pregnancy >20 weeks; end-stage renal disease on dialysis; EMS provider judgment	Consider trauma center; lower threshold to transport there

Adapted from the CDC Guidelines for Field Triage of Injured Patients.

18 Documentation and Handoff

A structured MIST report - Mechanism of injury, Injuries found, Signs (vital signs and exam findings), Treatment given - delivered on arrival gives the receiving team everything gathered in the field in a predictable, rapid format, and should be prepared during transport rather than assembled on arrival. Include times: time of injury (or last known well for mechanism-only calls), time of key interventions (tourniquet application, needle decompression, medications given), and time of any change in mental status or vital signs.

19 Worked Clinical Cases

Case 1: Motor vehicle crash with extremity hemorrhage

EMS is dispatched to a single-vehicle crash with intrusion into the driver's compartment and a prolonged extrication. On arrival, the scene is secured by fire/rescue. The driver is awake, restless, with obvious arterial bleeding from a mangled lower leg and no other externally visible injury.

MARCH: massive hemorrhage is controlled immediately with a tourniquet applied high on the thigh, tightened until the bleeding stops and the distal pulse disappears, and time-stamped. Airway is patent; the patient is talking. Respirations are unlabored with equal breath sounds. Circulation: a radial pulse is present but thready and fast; a second IV is deferred in favor of prompt extrication and transport, with access planned en route. The patient is kept warm during extrication. Given the high-risk mechanism (significant intrusion) and the mangled extremity meeting anatomic field-triage criteria, the patient is transported by the fastest available means to the highest-level trauma center, with a MIST report prepared during transport and TXA given per protocol given evident significant hemorrhage within the 3-hour window.

Case 2: Fall with chest trauma and altered mental status

EMS responds to a fall from a second-story roof (approximately 5 meters). The patient is confused, tachypneic, and has visibly paradoxical movement over the left lower chest wall.

MARCH: no external hemorrhage. Airway is maintained with a jaw thrust given the fall mechanism and altered mental status; an oropharyngeal airway is placed and tolerated, prompting reassessment of mental status as a possible airway/respiratory problem rather than isolated head injury. On exposure, the left chest shows flail segment movement with decreasing breath sounds on that side and early hypotension - findings concerning for an evolving hemothorax or pneumothorax; high-flow oxygen is applied, ventilation is assisted, and the crew prepares for needle decompression if tension physiology develops (increasing distress, worsening hypotension, distended neck veins). Given altered mental status, the patient cannot reliably report spinal pain, so SMR is applied. Given the physiologic (altered mental status) and mechanism (fall >3 m) field-triage criteria, the patient is transported emergently to the highest-level trauma center with a MIST report that specifically flags the chest findings and the fall height so the team is ready for possible tube thoracostomy on arrival.

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20 Chapter Quick-Reference Summary

- Scene safety and PPE before patient contact, every time.
- MARCH: Massive hemorrhage → Airway → Respiration → Circulation → Hypothermia/Head injury.
- Tourniquet 2-3 in. proximal to the wound, never over a joint; hemostatic gauze + 3 min direct pressure for junctional/non-extremity bleeding.
- Six immediately life-threatening chest injuries: tension pneumothorax, open pneumothorax, flail chest, massive hemothorax, cardiac tamponade, traumatic asphyxia.
- Needle-decompress a suspected tension pneumothorax on scene; vent, don't fully occlude, an open chest wound.
- Shock in trauma is hemorrhagic until proven otherwise; consider obstructive (tension pneumothorax/tamponade) and neurogenic (bradycardic hypotension) causes too.
- Protect the injured brain: avoid hypoxia and hypotension; treat any significant head injury as a possible spine injury.
- SMR is selective, not routine - based on mental status, midline tenderness, neuro deficit, distracting injury, intoxication, and reliable exam.
- Stabilize impaled objects and eviscerated bowel in place; bind a suspected pelvic fracture without springing the pelvis.
- Splint fractures in the position found when neurovascularly intact; consider traction splinting an isolated femur fracture.
- Rule of nines/palm method for burn extent; airway is the first priority in any burn with facial or inhalation involvement.
- Permissive hypotension (target a palpable radial pulse) in penetrating/hemorrhagic trauma without suspected TBI; TXA within 3 hours if indicated.
- Minimize scene time (the Golden Period); obtain access and reassess en route rather than delaying transport.
- Apply the CDC field triage decision scheme stepwise to choose destination; hand off with a structured, timed MIST report.

This chapter is adapted from PHTLS (Prehospital Trauma Life Support), 10th Edition (NAEMT, in cooperation with the American College of Surgeons Committee on Trauma), and the CDC Guidelines for Field Triage of Injured Patients. It is an educational reference summary only and is not a substitute for enrollment in, or certification through, an official PHTLS provider course, or for your EMS system's medical direction and protocols.

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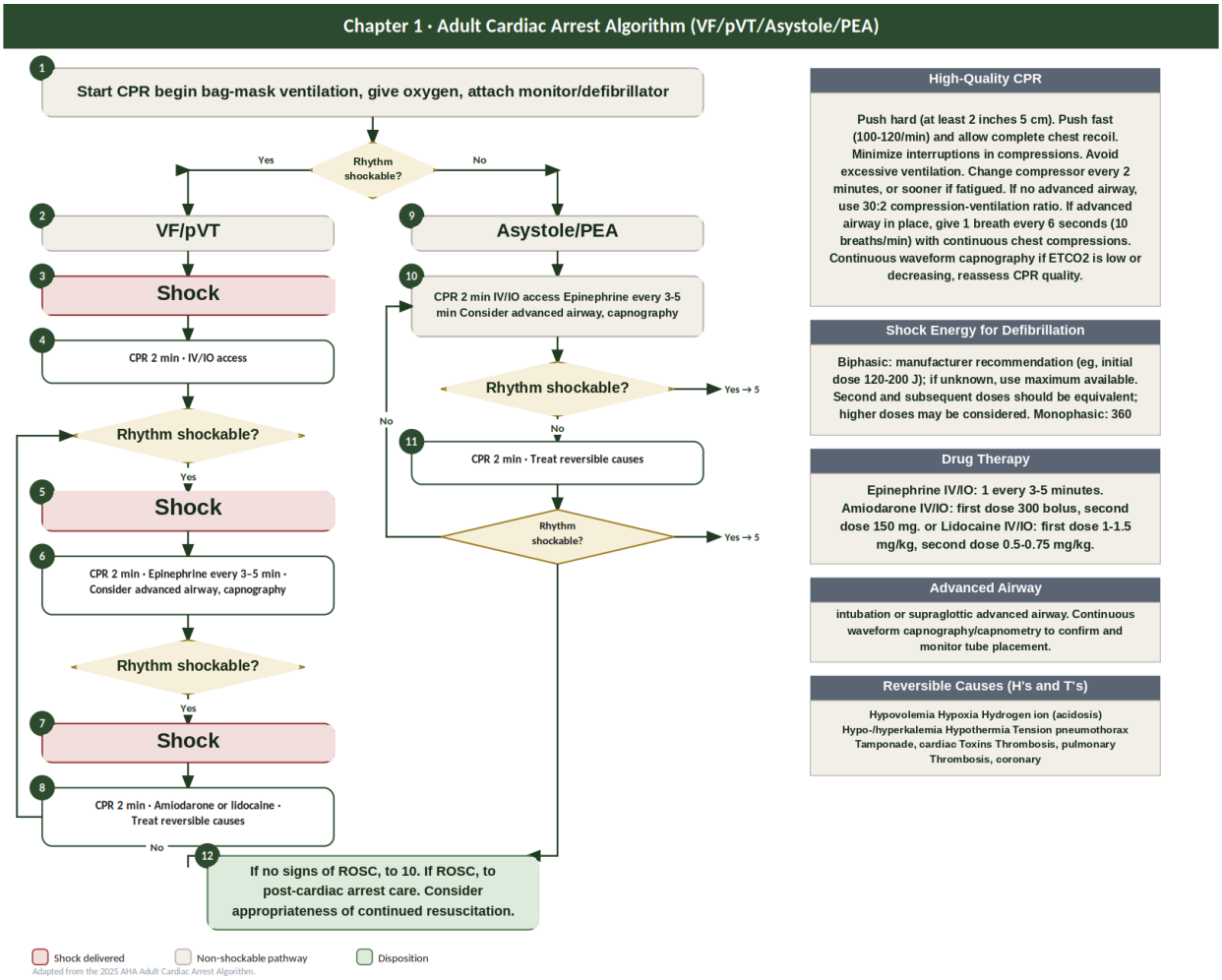
QUICK-REFERENCE APPENDIX

APPENDIX A

Quick-Reference Flowcharts

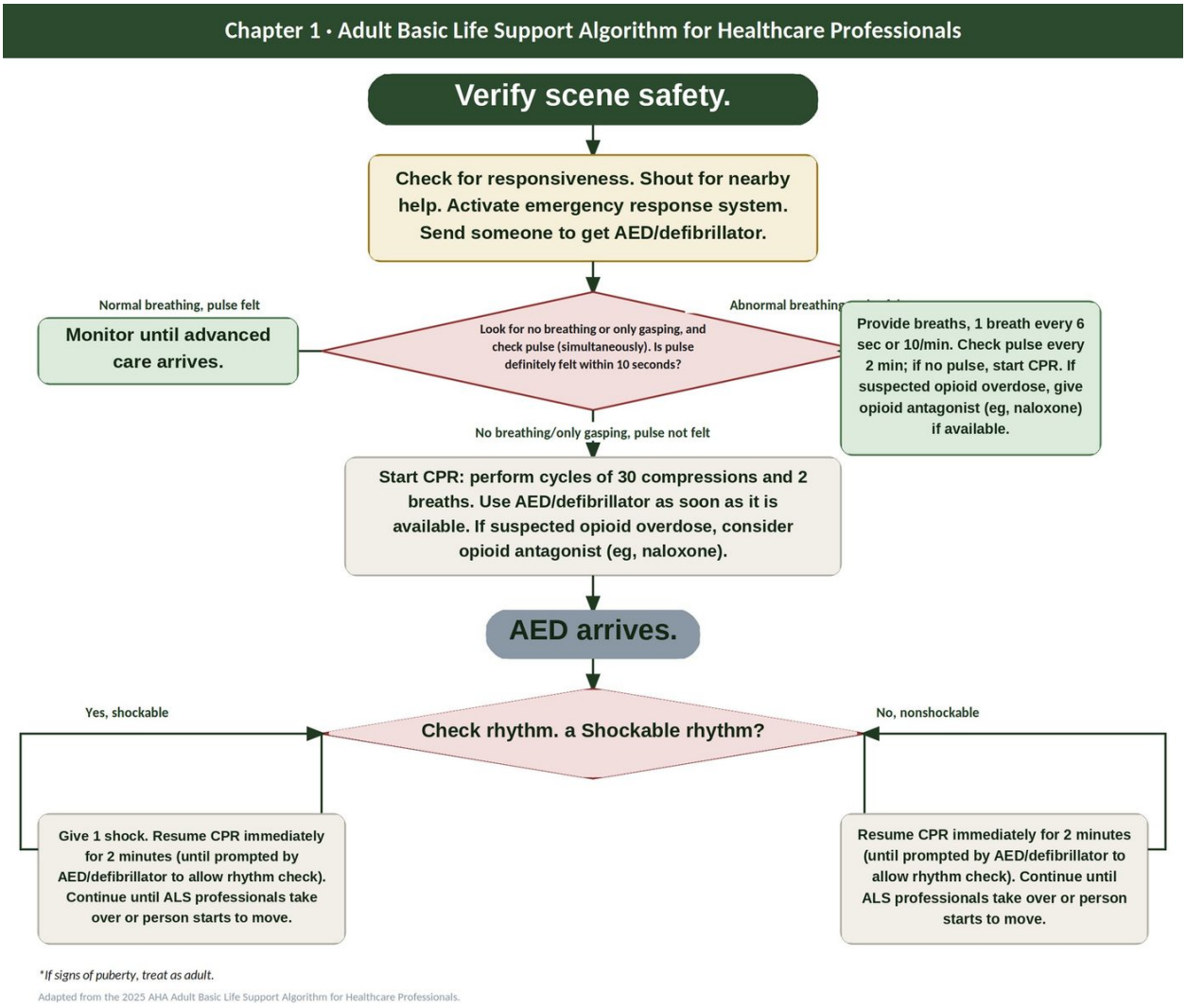
The core algorithm from each of the eight chapters, collected here for rapid bedside or pre-shift review.

Chapter 1 · BLS/ACLS — Adult Cardiac Arrest Algorithm



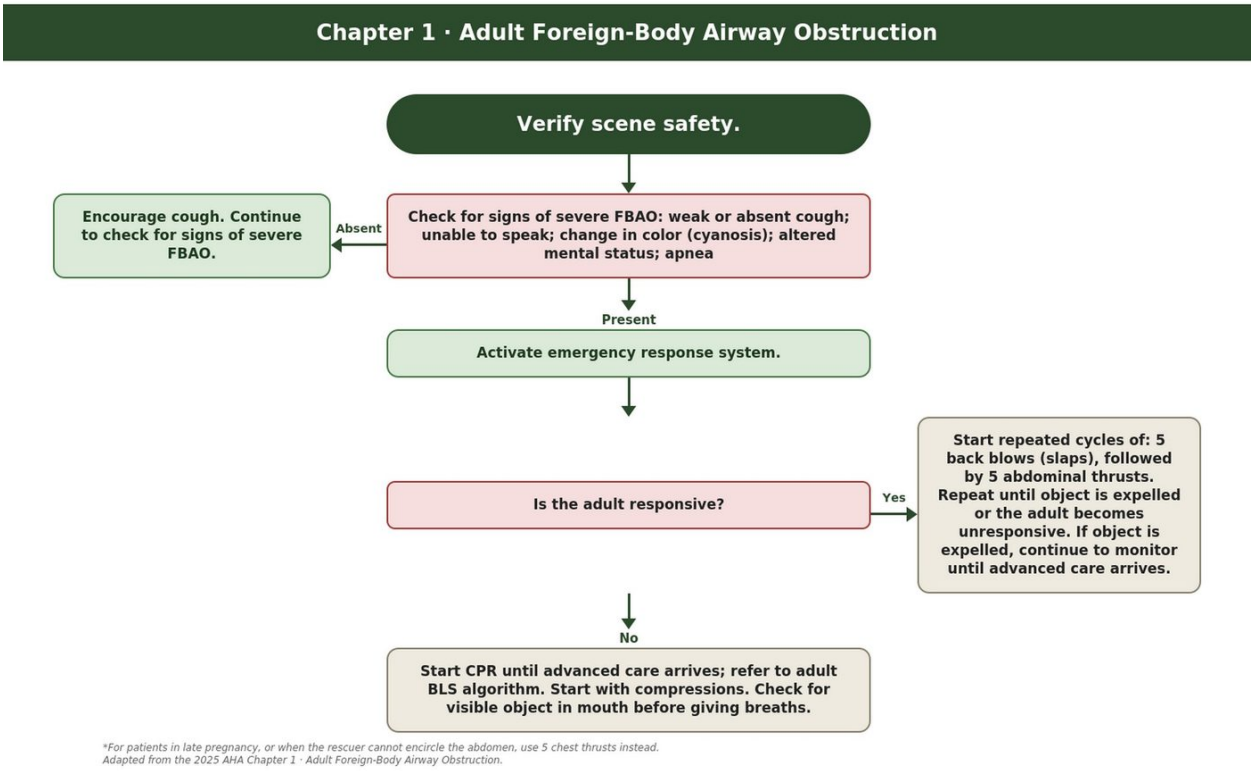
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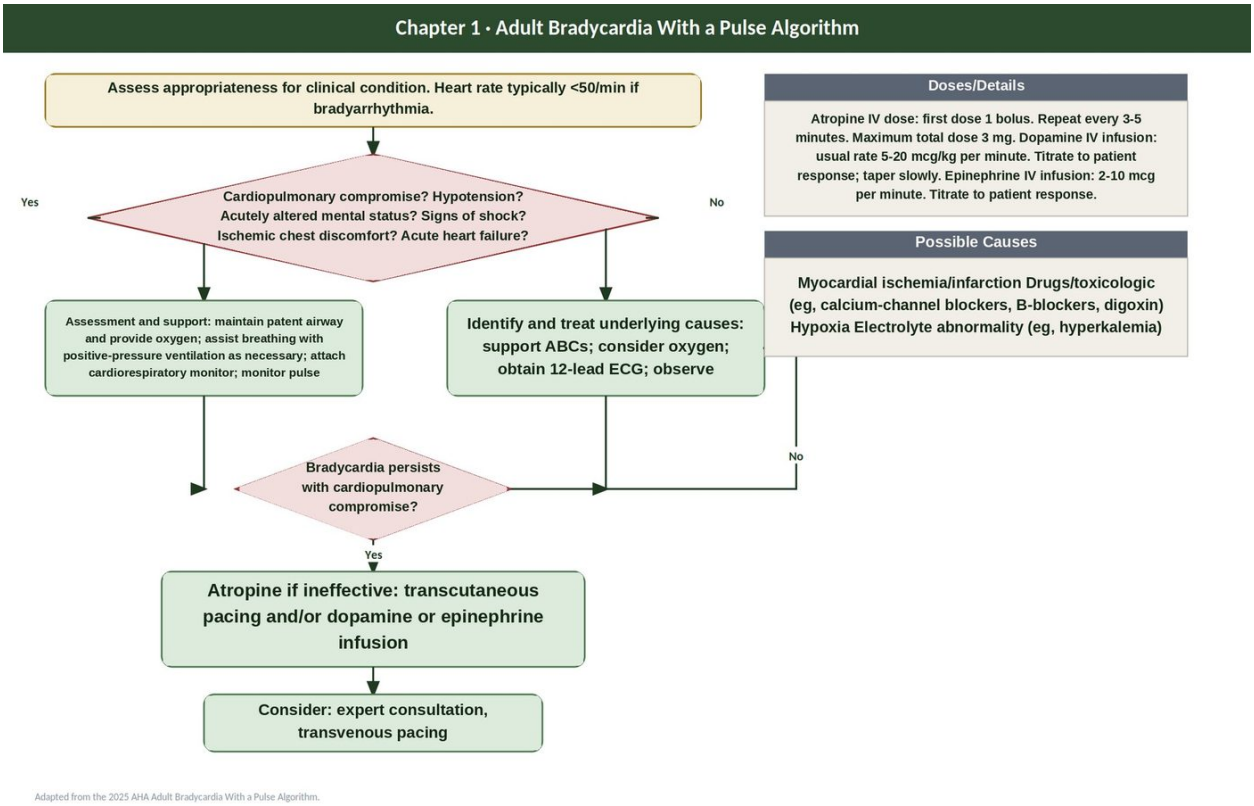


Chapter 1 · BLS/ACLS — Adult Foreign-Body Airway Obstruction

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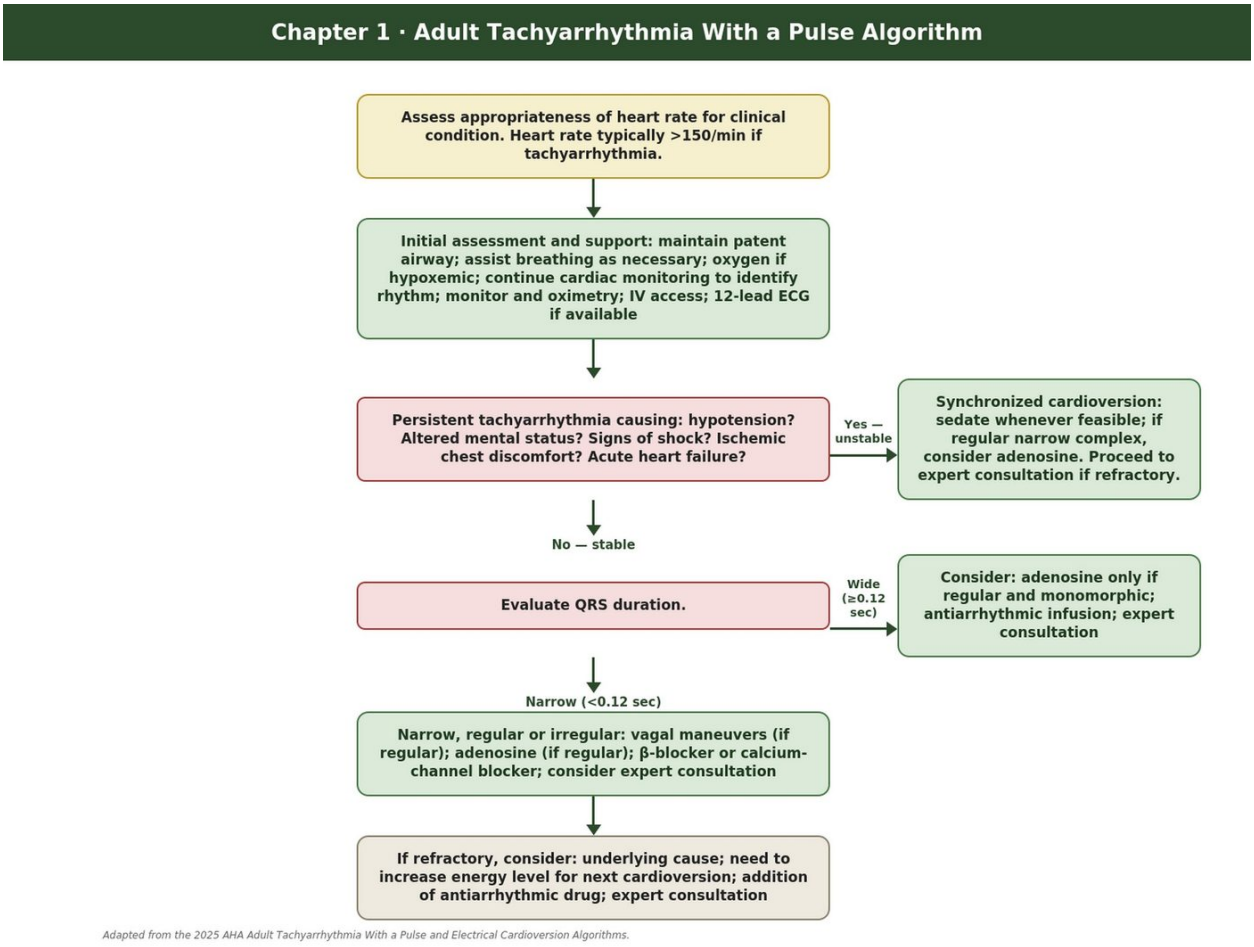


Chapter 1 · BLS/ACLS — Adult Bradycardia With a Pulse Algorithm



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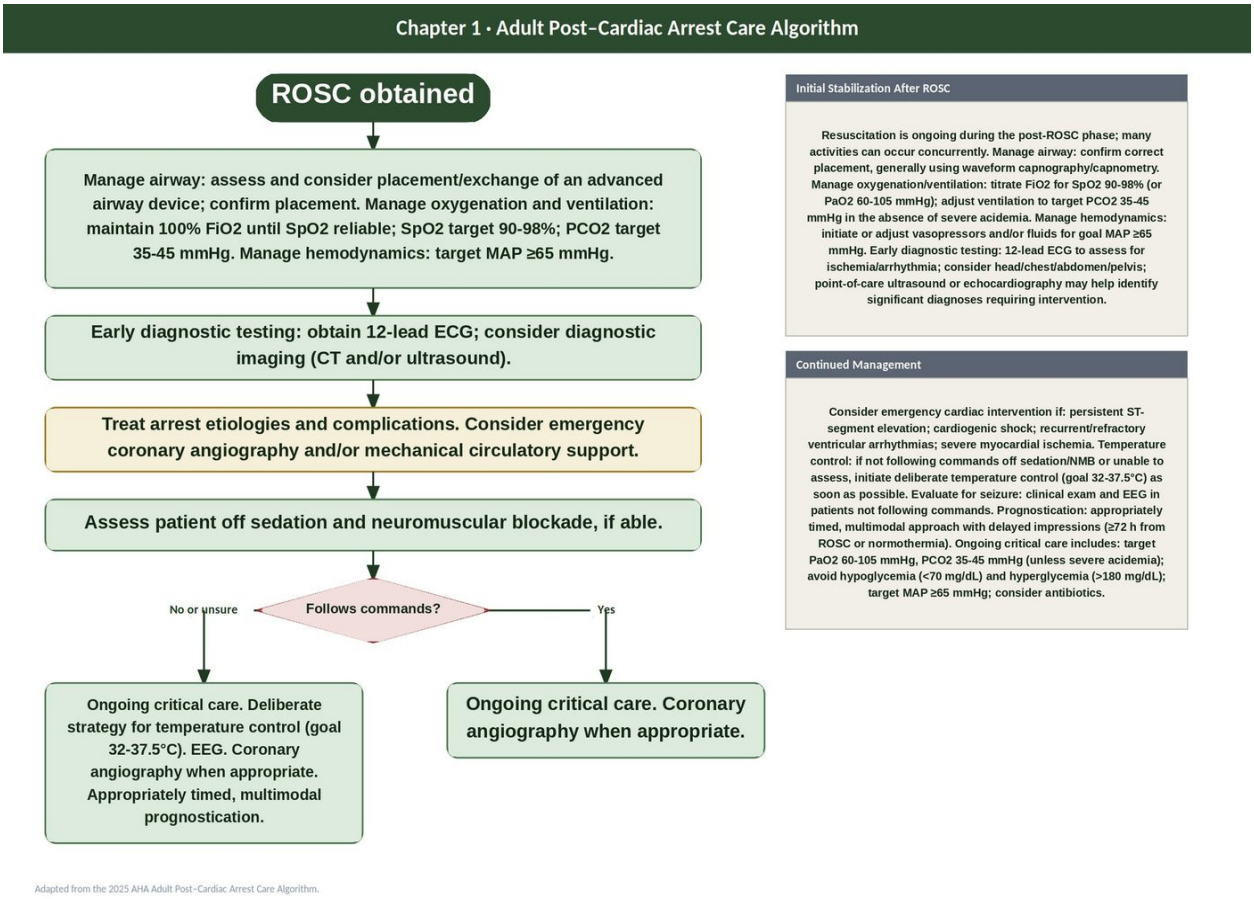
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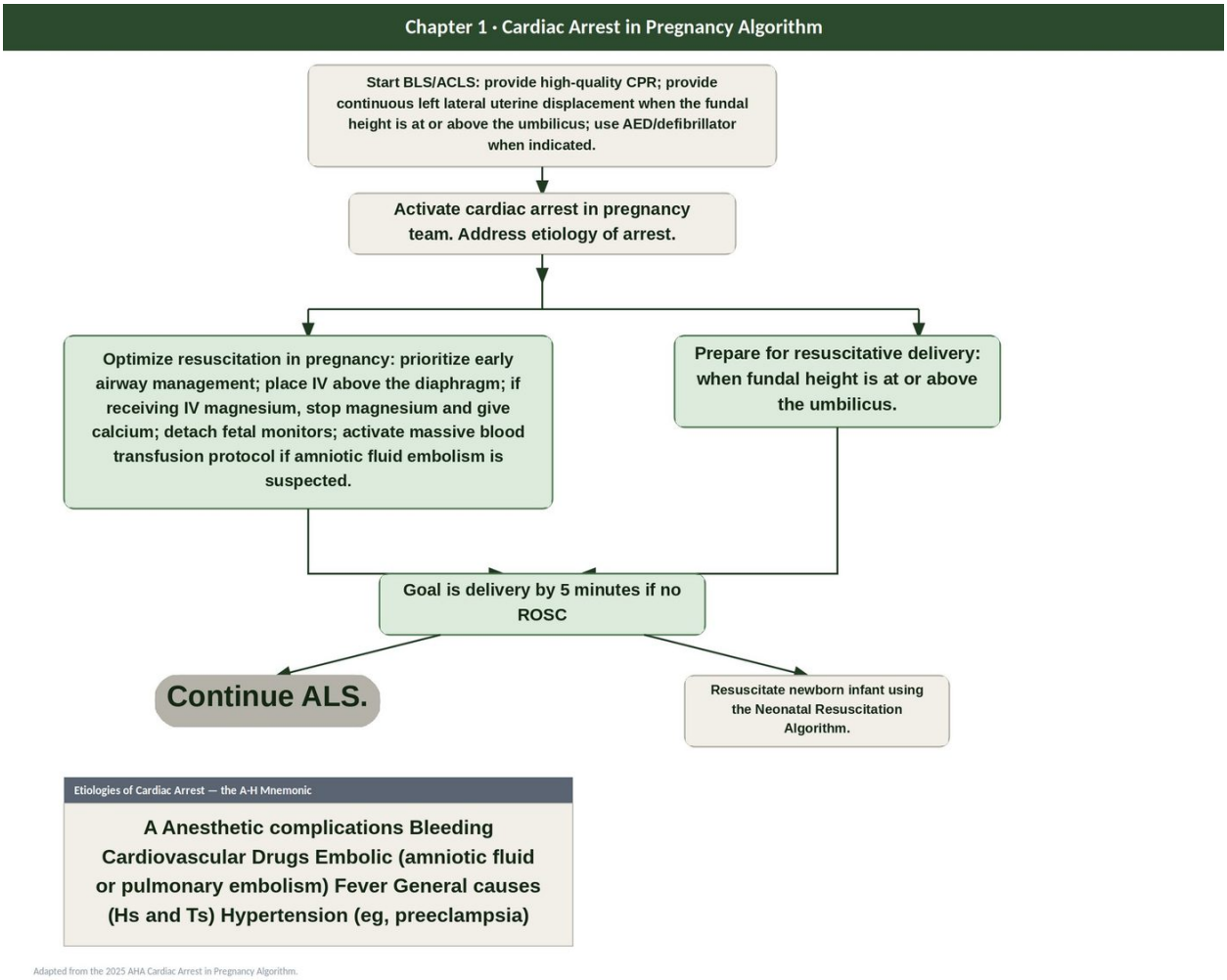
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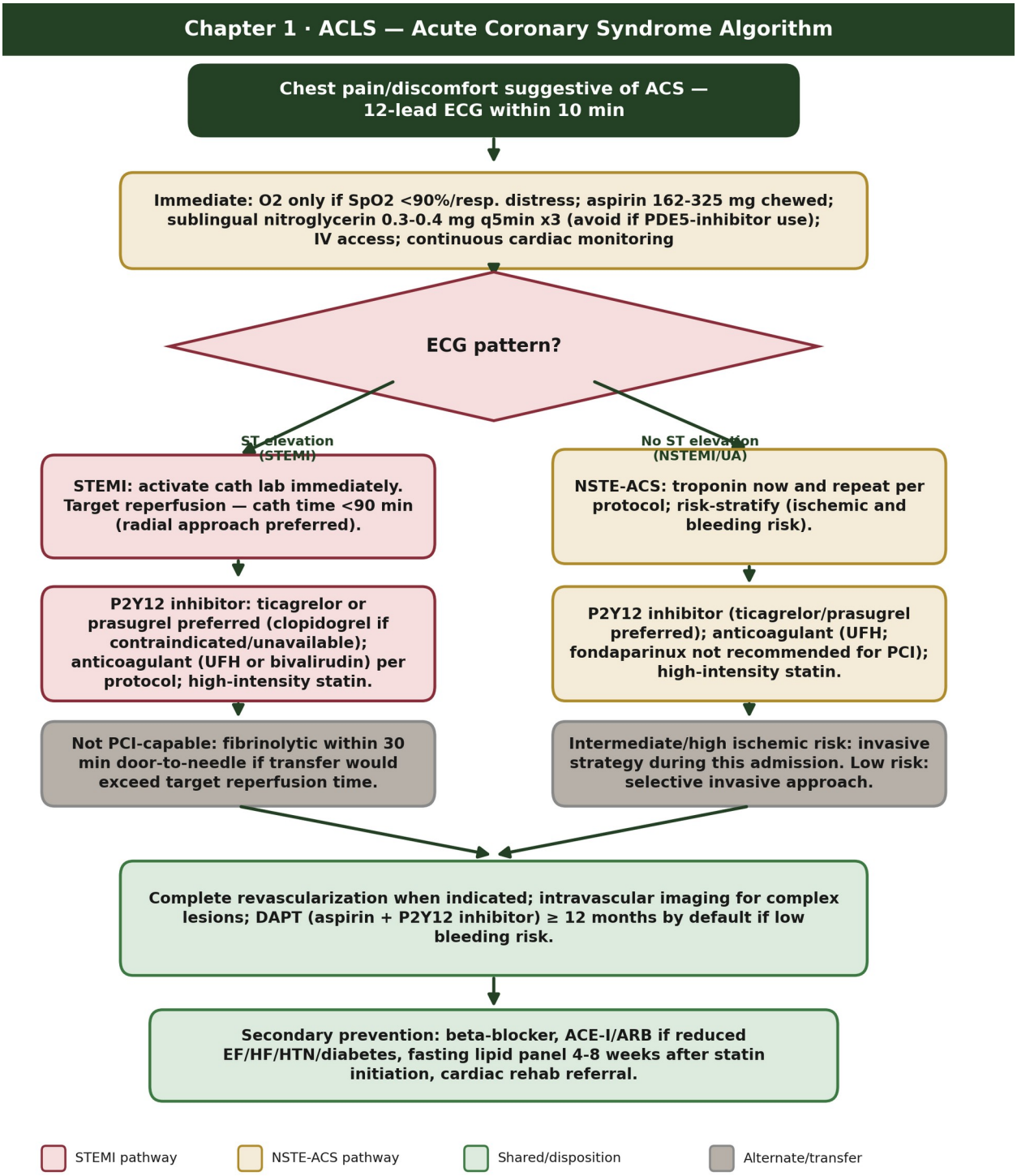
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Chapter 1 · ACLS — Acute Coronary Syndrome Algorithm

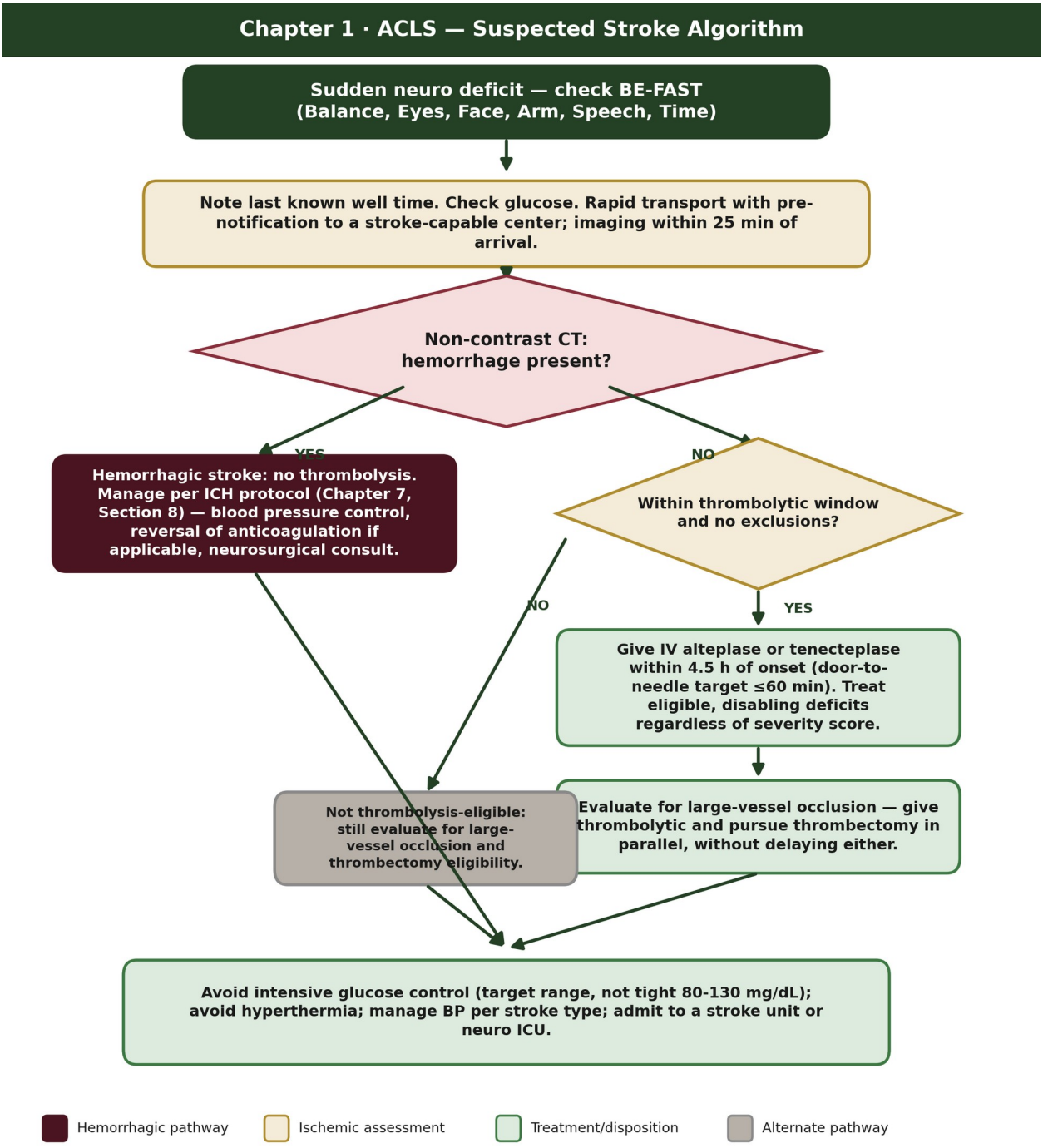
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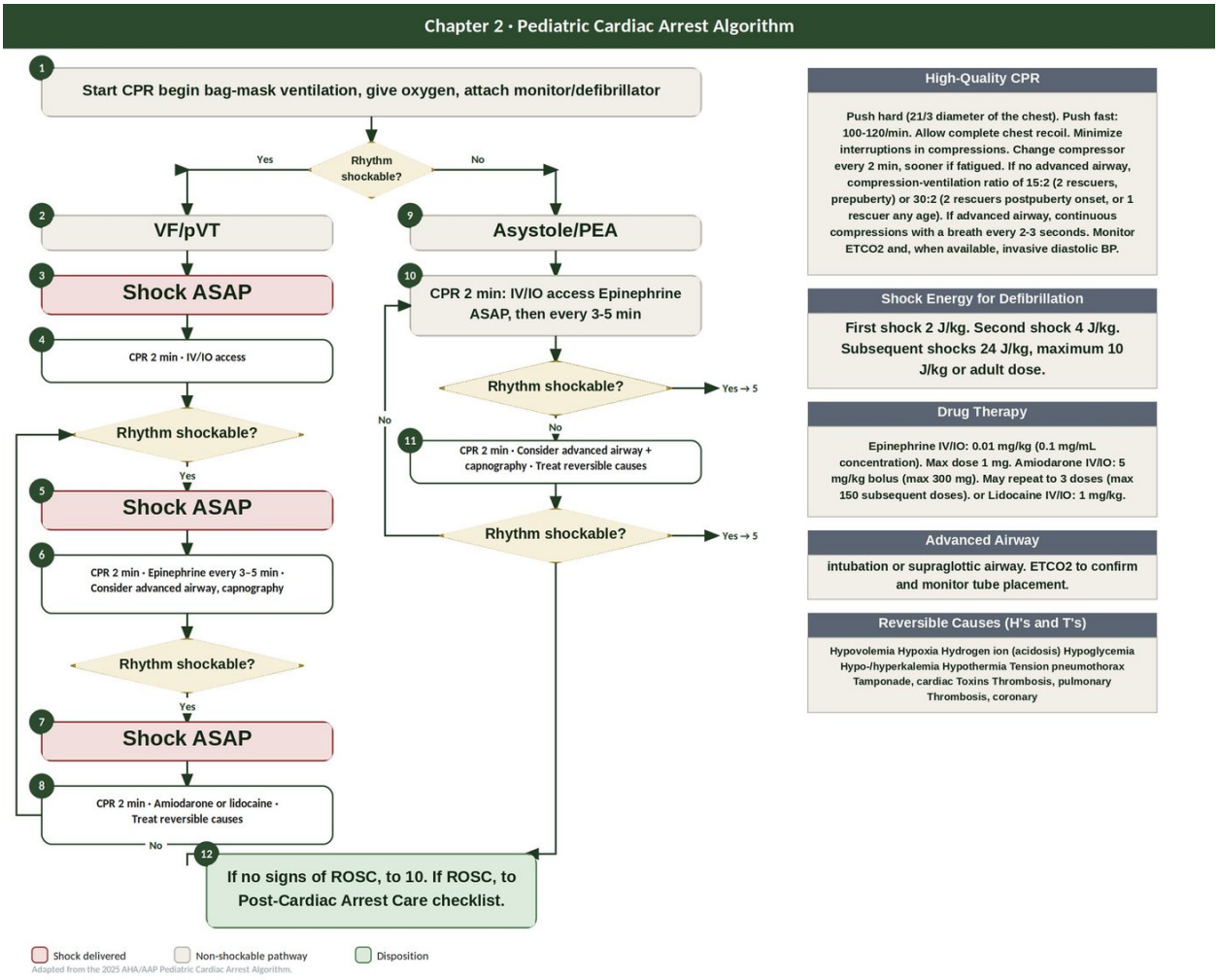
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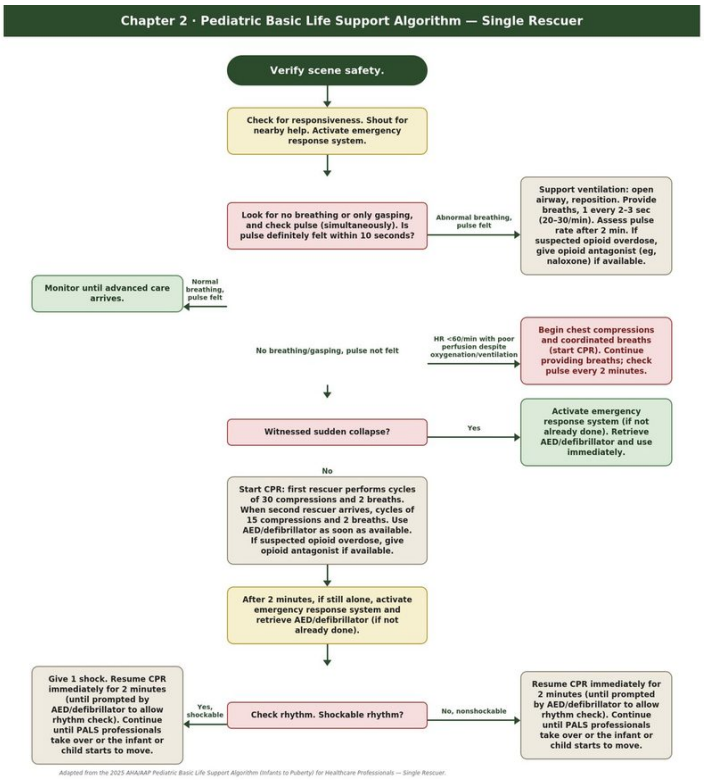
Chapter 2 · PALS — Pediatric Cardiac Arrest Algorithm

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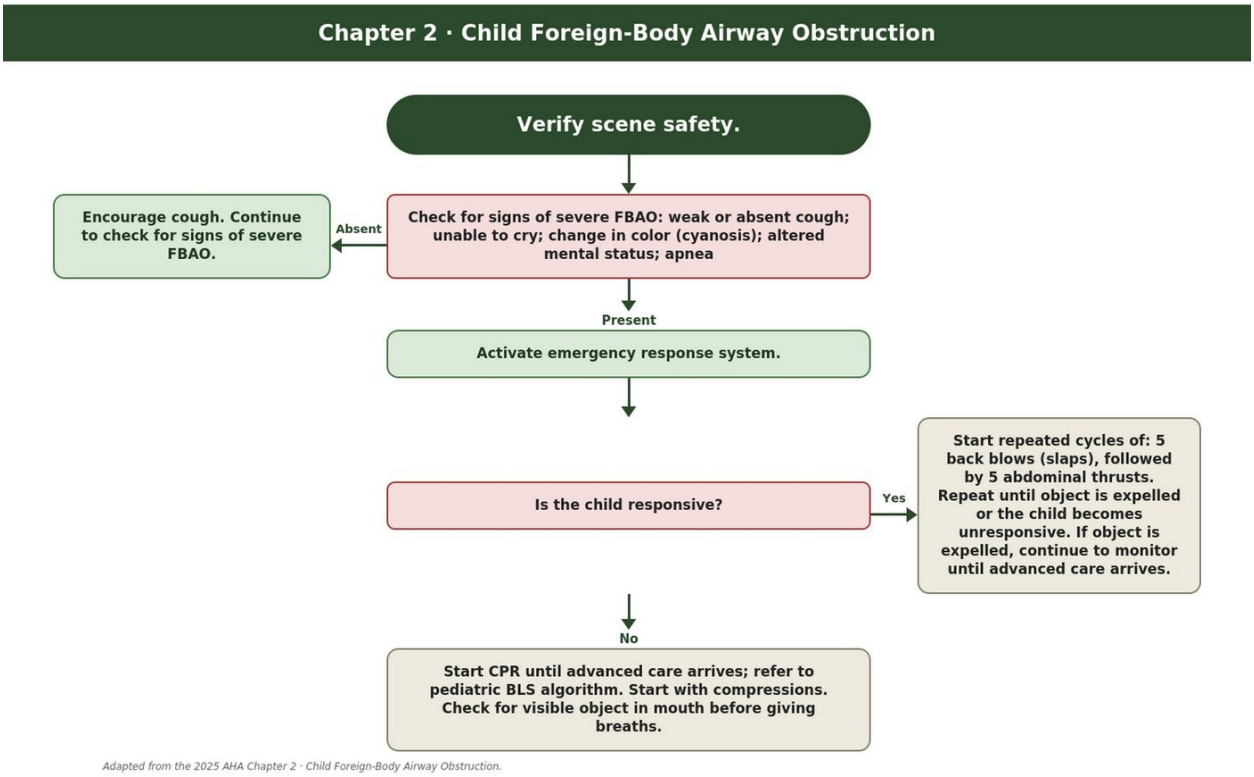


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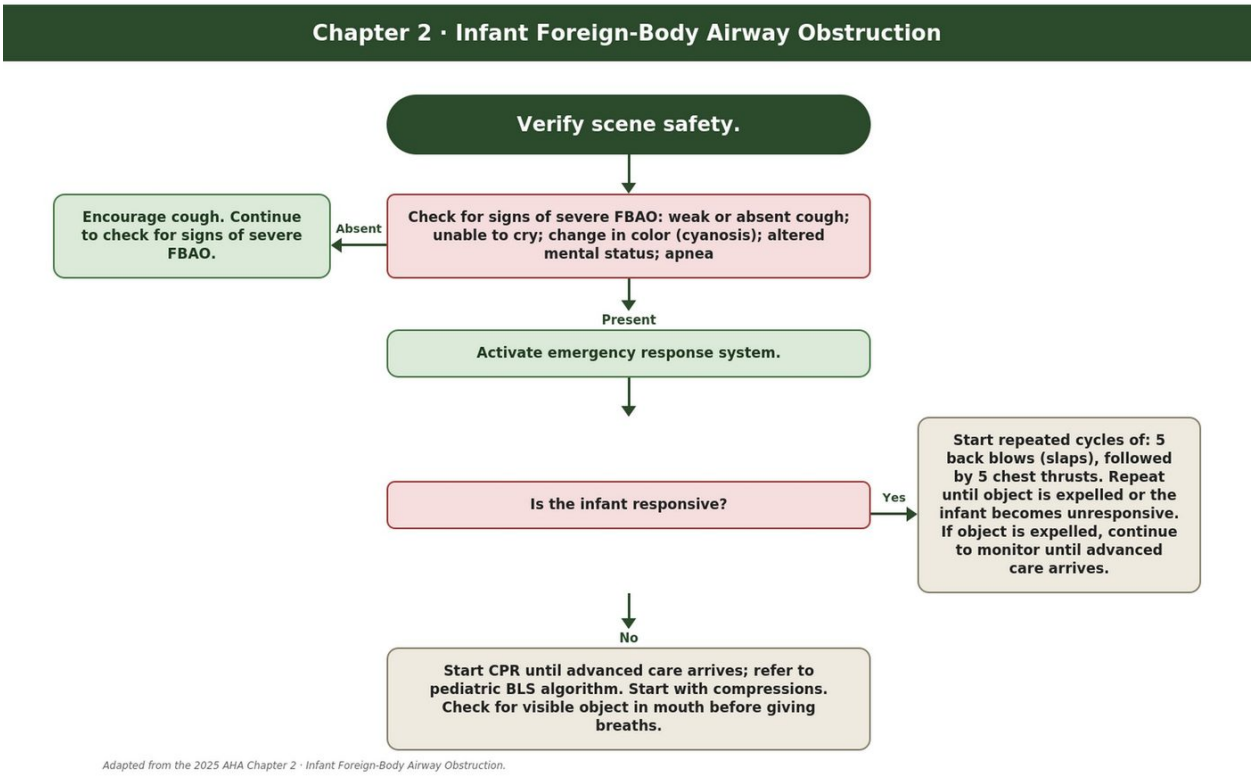


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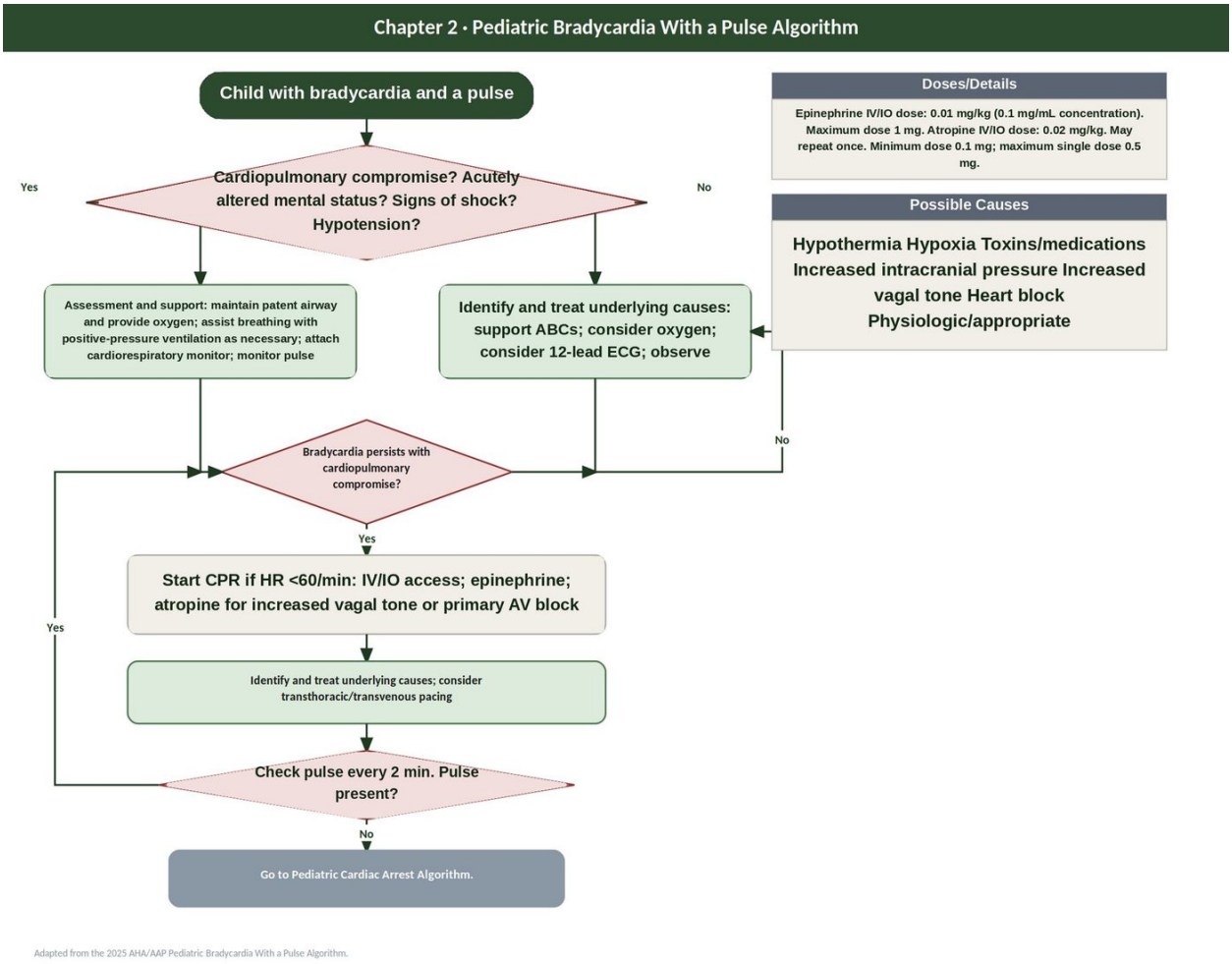
Chapter 2 · PALS — Infant Foreign-Body Airway Obstruction



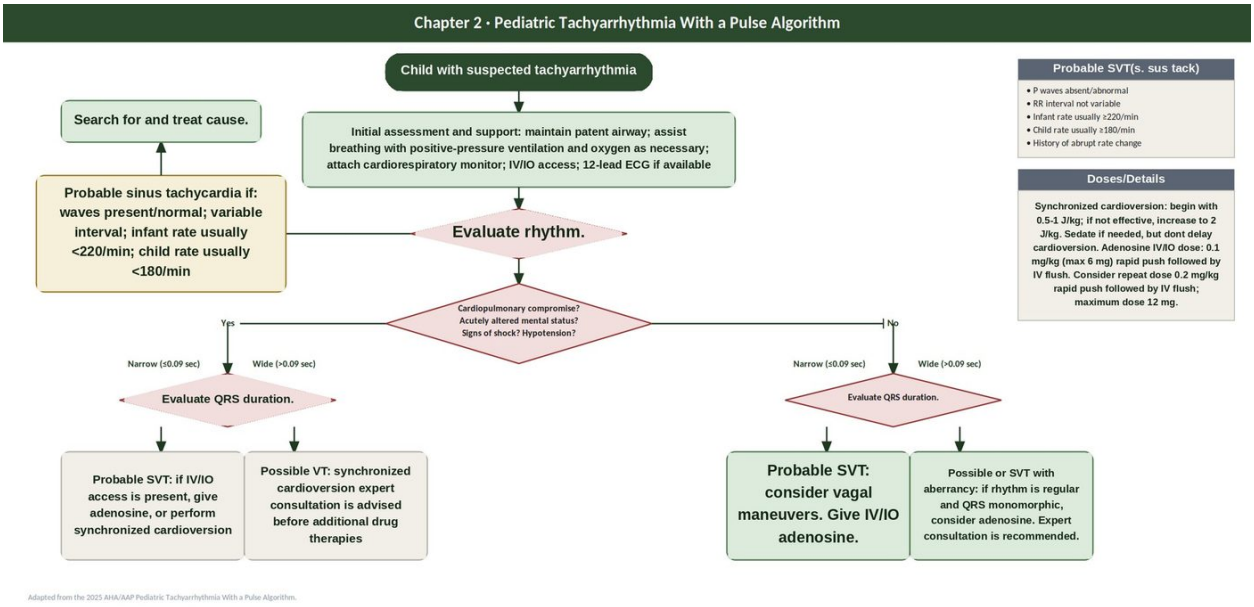
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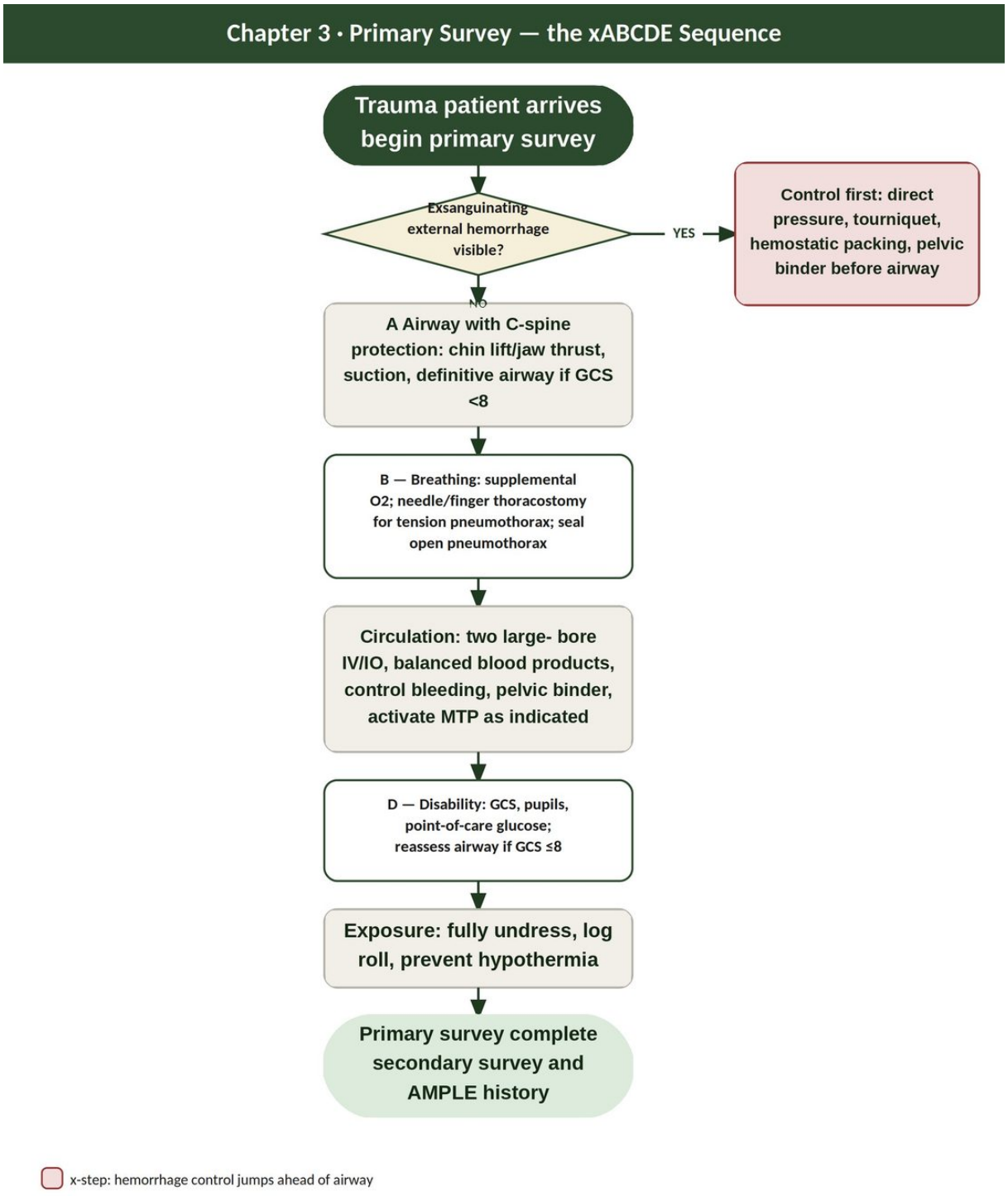


Chapter 2 · PALS — Pediatric Tachyarrhythmia With a Pulse Algorithm



Chapter 3 · ATLS — Primary Survey — the xABCDE Sequence

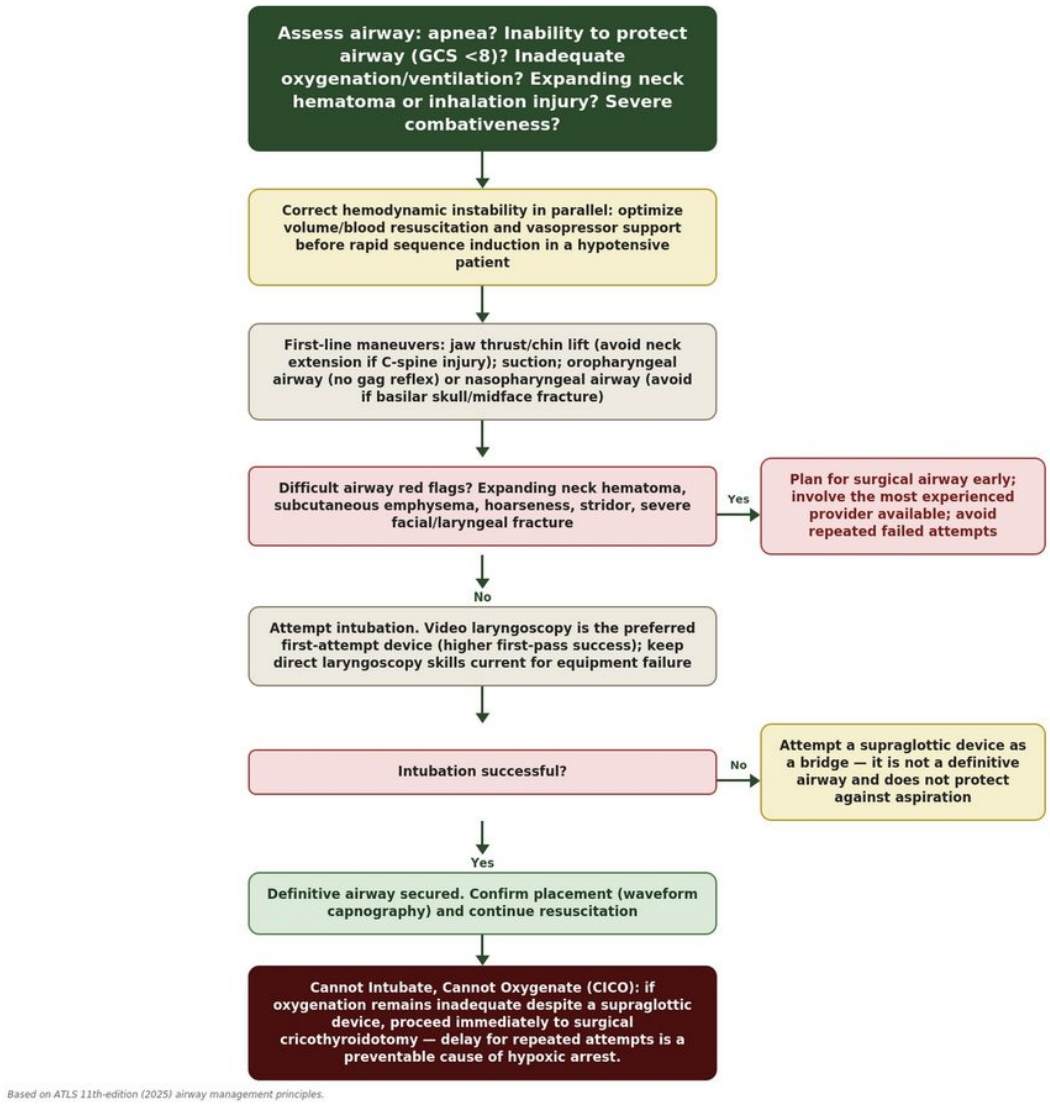
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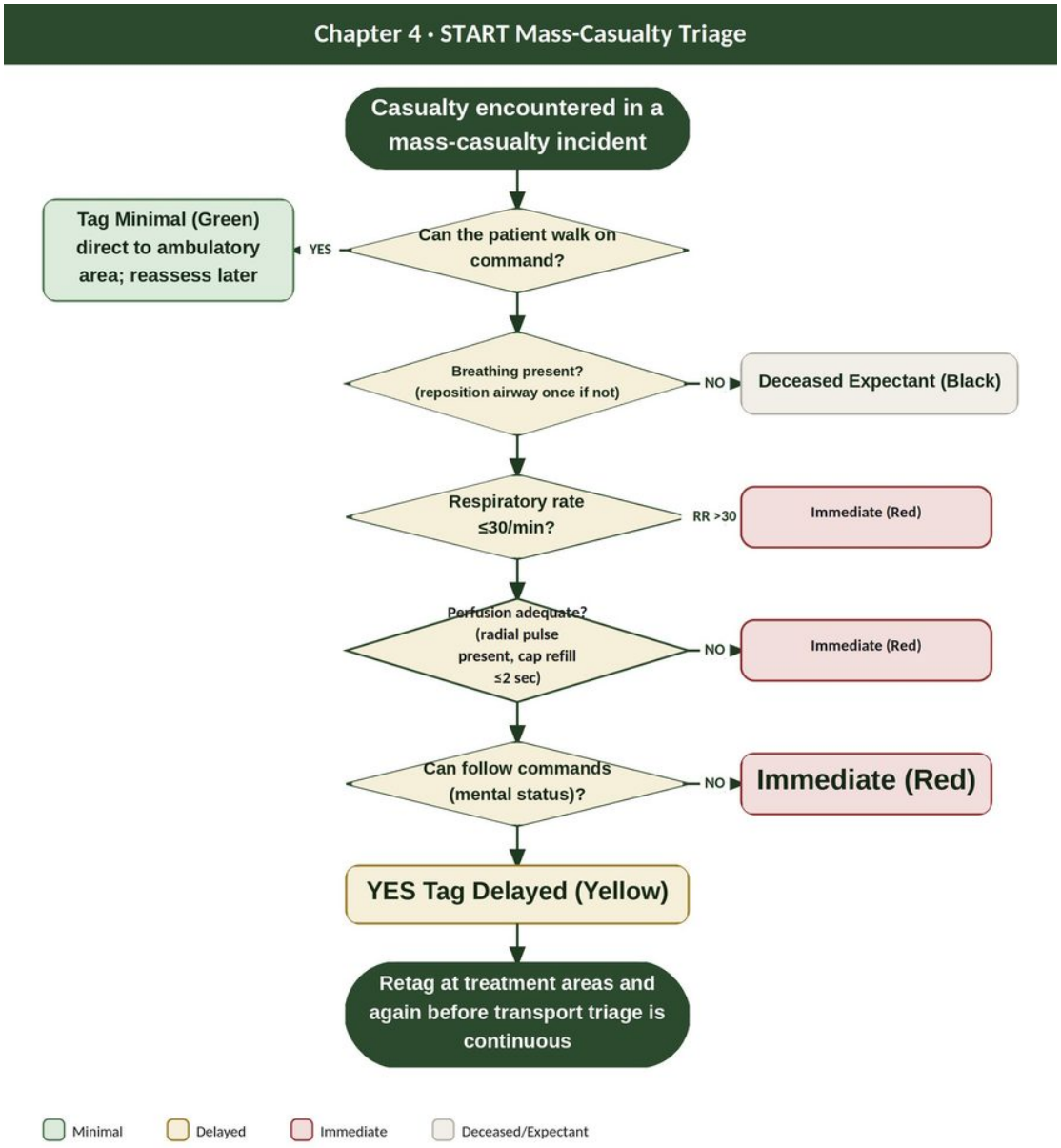
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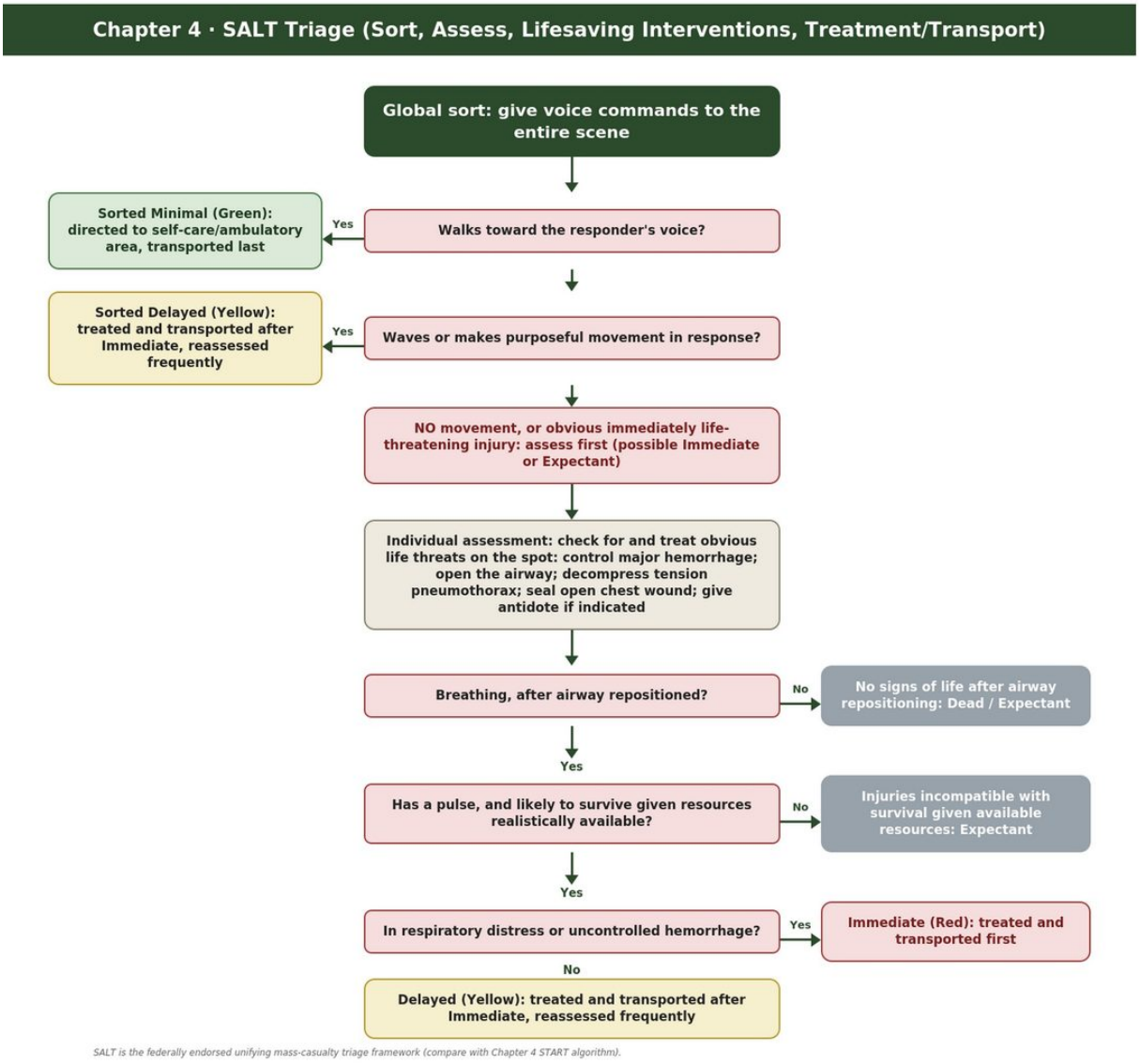
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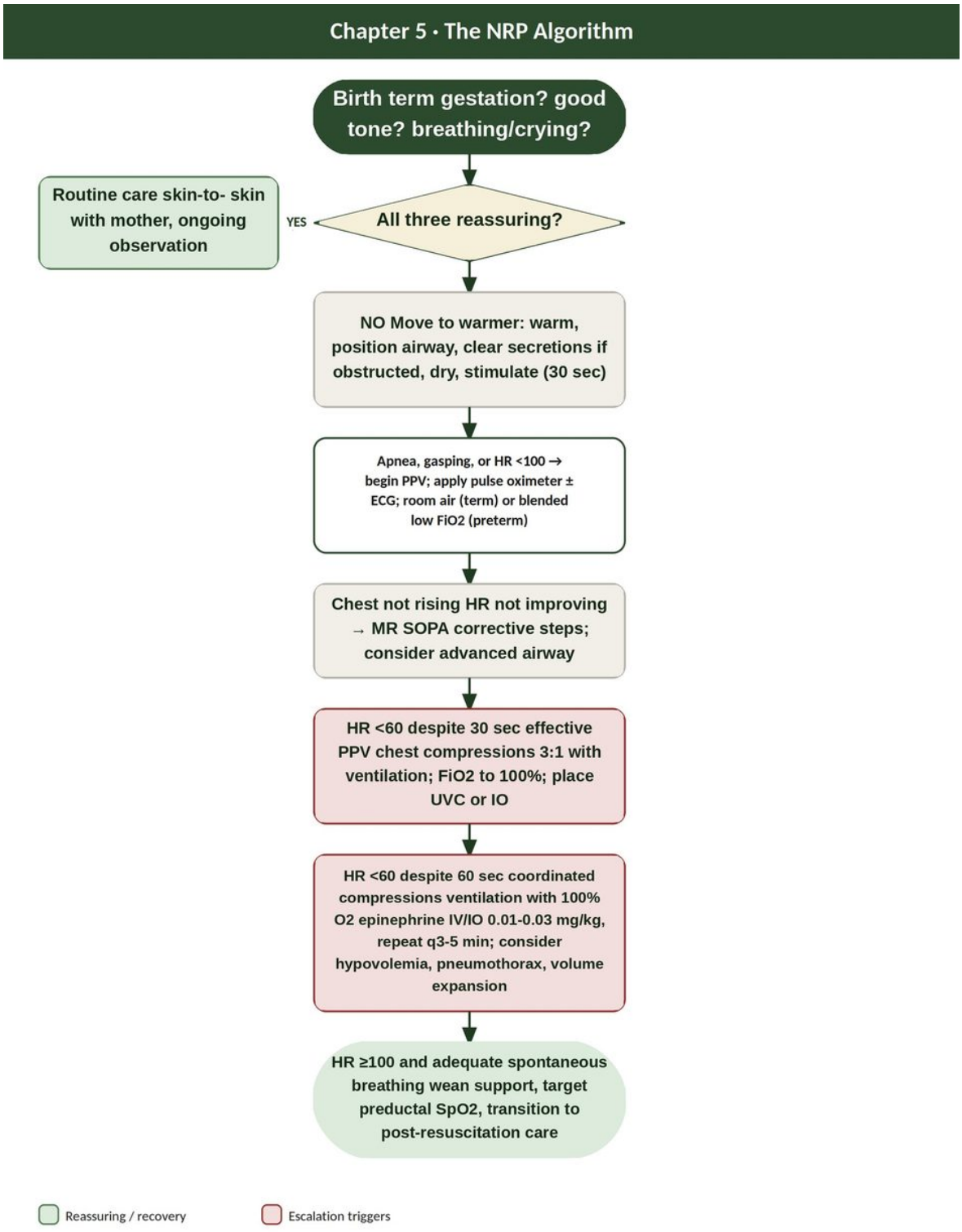
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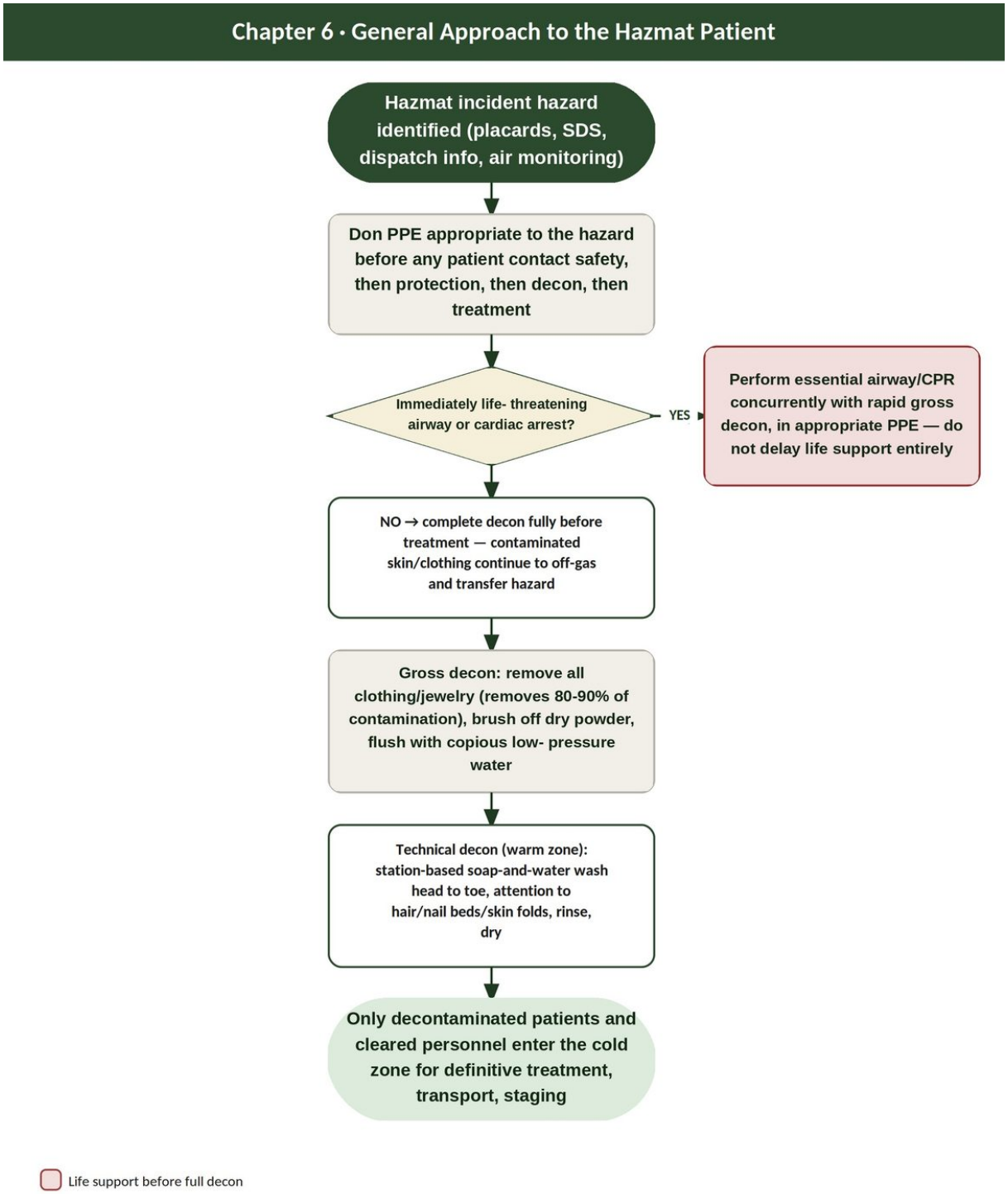
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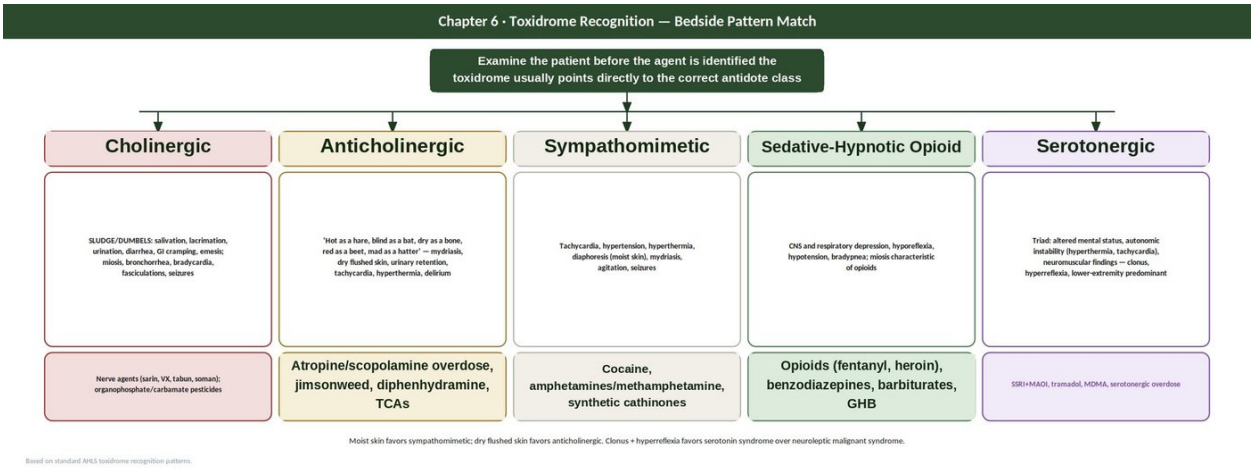
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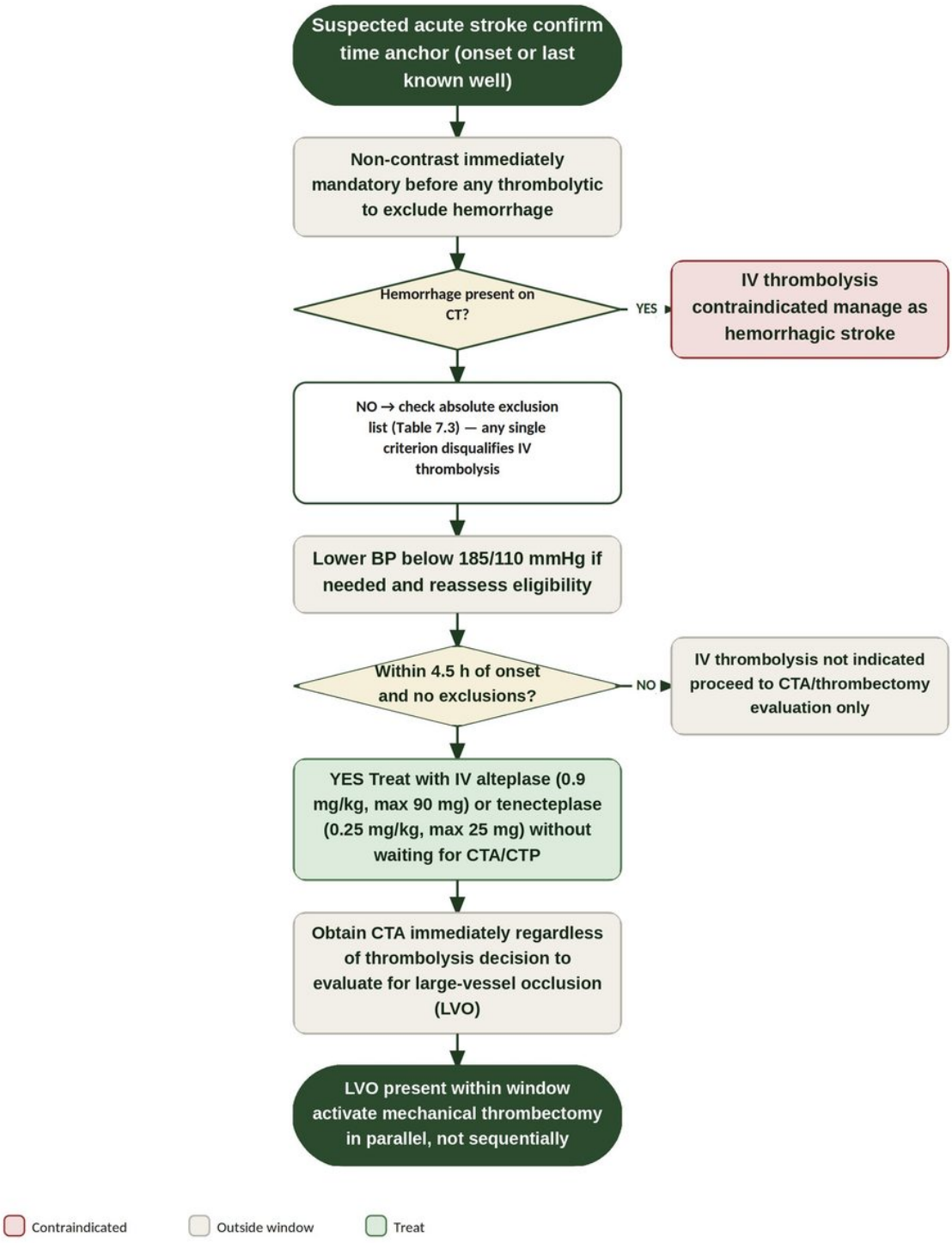
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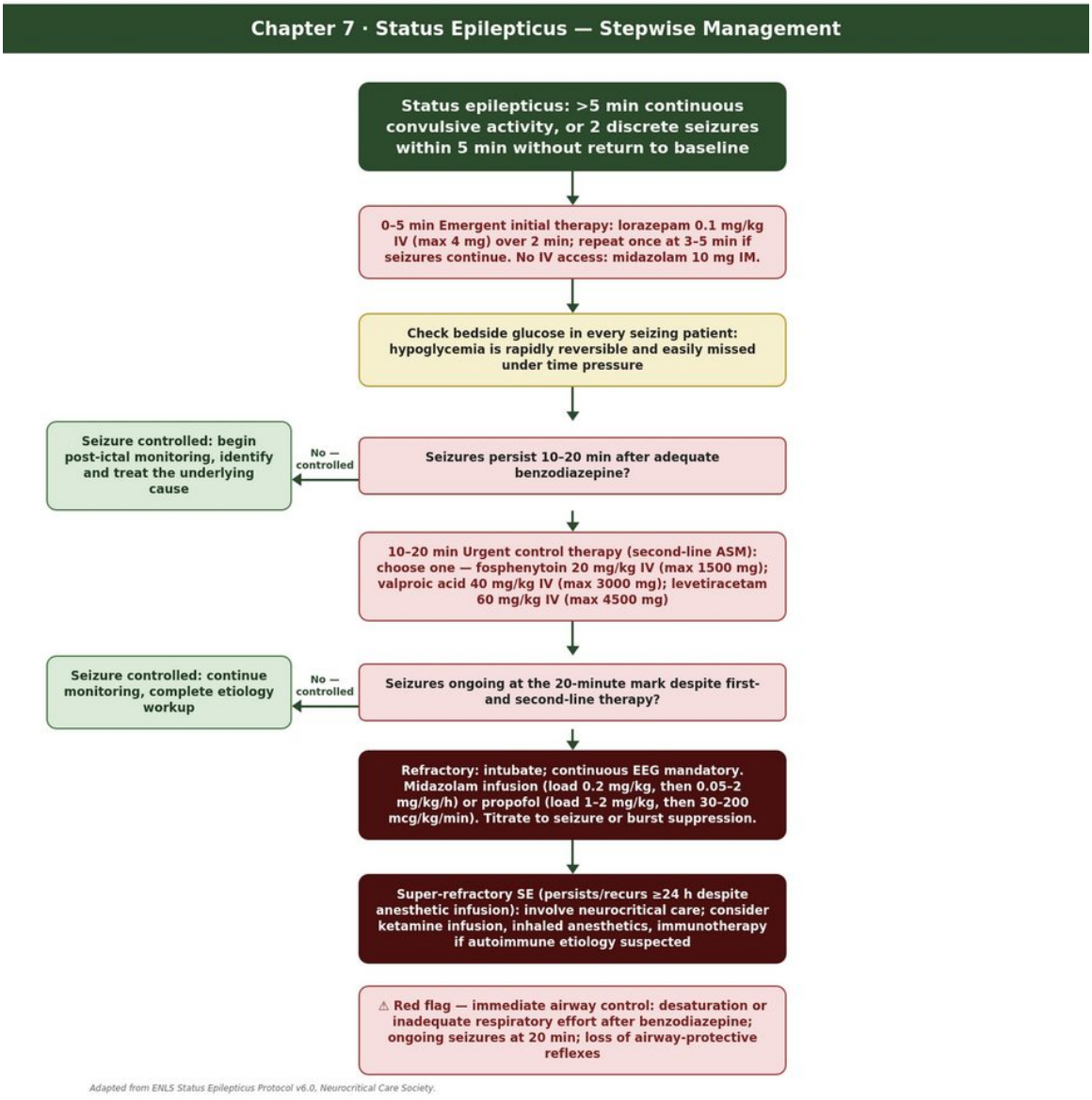
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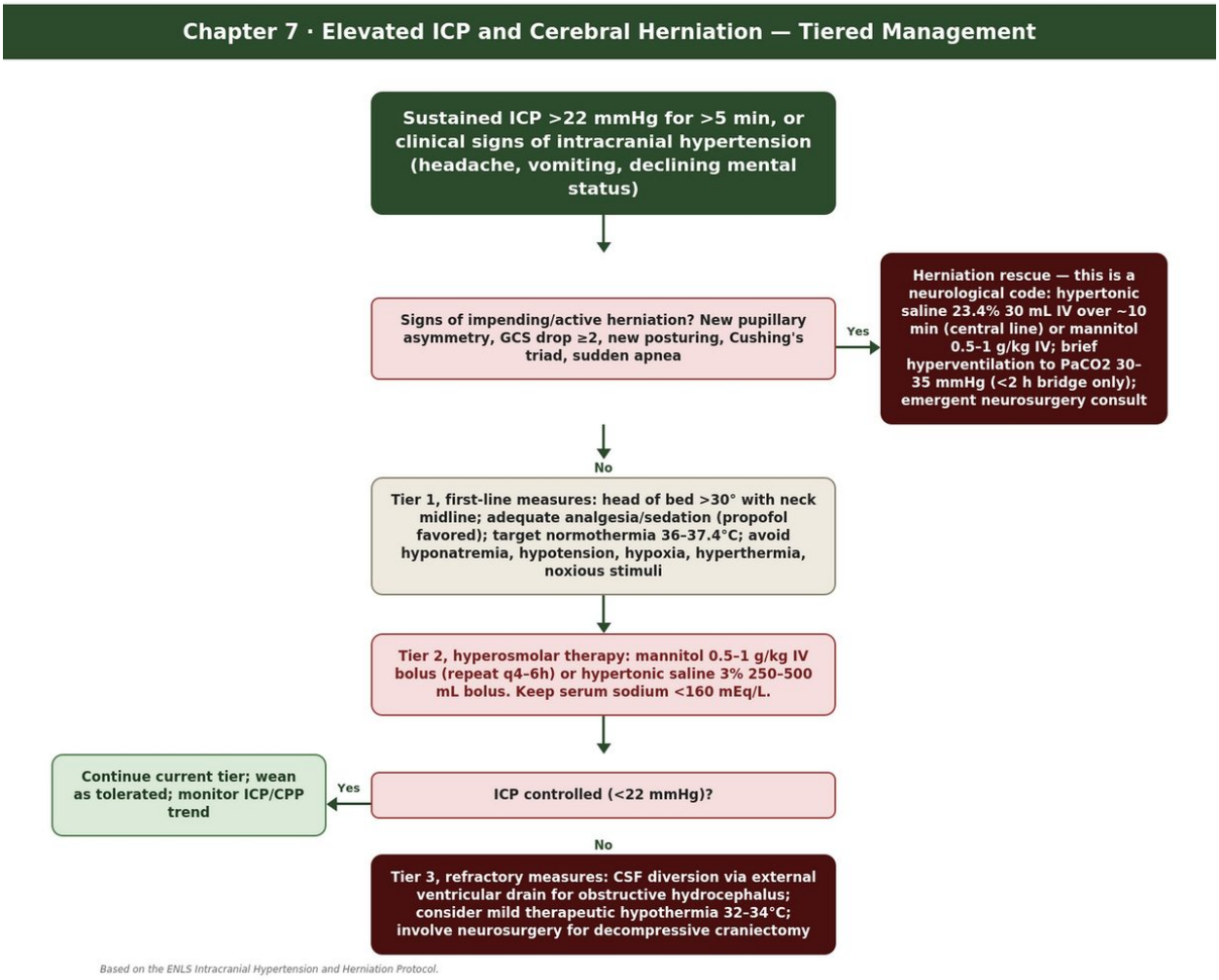
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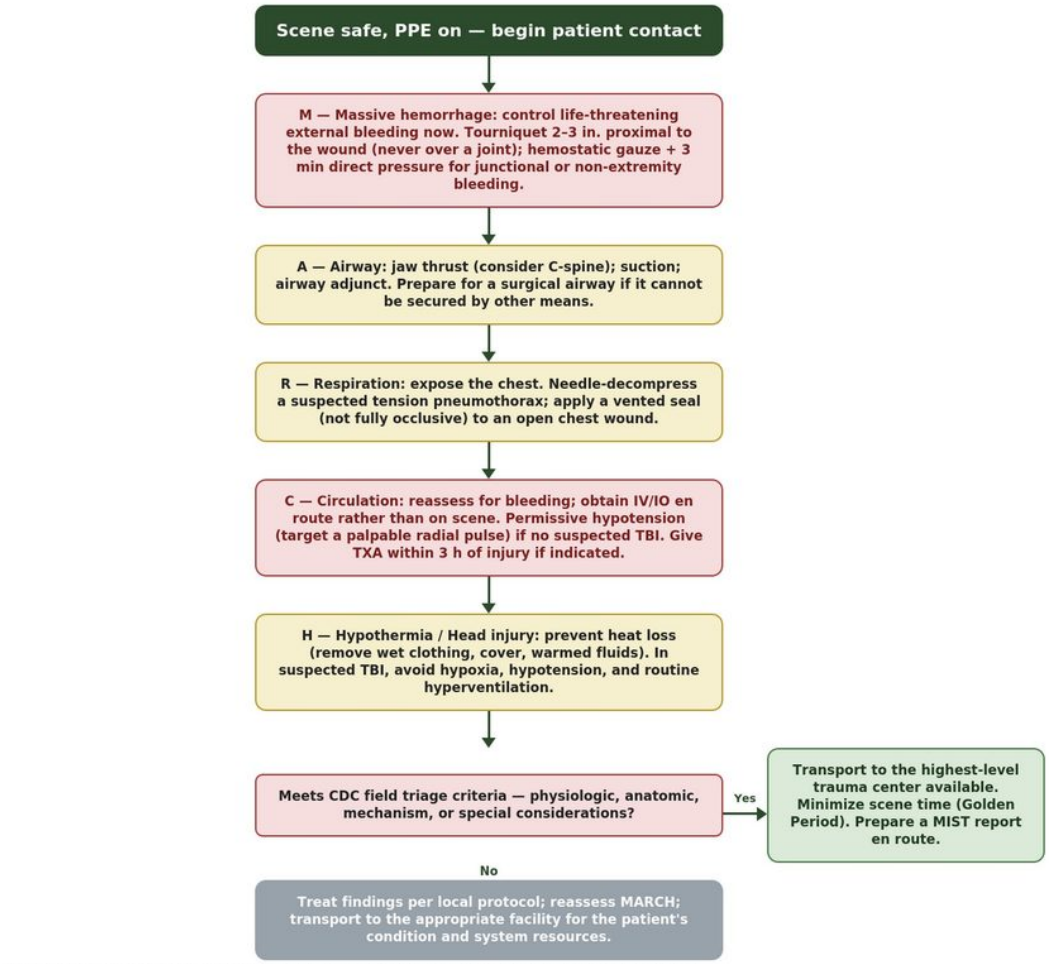
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