

[MC/1018/EN] - BASICS OF FIRST AID 2

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Useful Info

Slides

All the slides can be downloaded as pdf documents here (teacher webpage at unica.it)

Exam

In-progress MCQ tests on Mentimeter that focus on the Advanced Life Support (ALS) algorithm and Special Causes of Cardiac Arrest.



Practical activities

Hands-on training IS MANDATORY for passing THE COURSE.



Practical activities

The Secretariat of the Dean of the Faculty organizes the practical activities.

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Organisation the Emergency System in Italy

Normative Framework

- Decreto del Presidente della Repubblica (DPR) 27/03/1992 – Presidential Decree establishing emergency care organisation.
- Atto di Intesa Stato-Regioni 1996 – State-Regions Agreement on emergency healthcare standards.
  DPR 10/12/1997 identifies **Medicina e Chirurgia d'Accettazione e d'Urgenza (MeCAU)** – Emergency Medicine and Emergency Admissions – as a medical discipline.
- Decreto Ministeriale (DM) 17/02/2006 establishes the **Scuola di Specializzazione in Medicina di Emergenza Urgenza (MEU)** – Postgraduate School of Emergency Medicine.
- DM 70/2015 mandates a **Struttura Complessa (SC) MeCAU** – Complex Emergency Medicine Unit – in all **Dipartimenti di Emergenza e Accettazione (DEA)** – Emergency and Admissions Departments – and defines standards for:
  – **Osservazione Breve Intensiva (OBI)** – Short-Stay Observation Unit
  – **Terapia Sub-Intensiva (TSI)** – High-Dependency Unit (HDU)
- DLgs 81/2008 – Workplace safety legislation.
- Legge 161/2014 – Working-hours compliance.
- AGENAS – National Agency for Regional Health Services – supports planning for **Pronto Soccorso (PS)** – Emergency Department (ED).



Società Italiana di Medicina di Emergenza Urgenza (SIMEU). Standard di riferimento 2021 per le unità di Medicina di Emergenza Urgenza (Medicina e Chirurgia d'Accettazione e d'Urgenza) nella rete ospedaliera italiana. Modelli organizzativi e personale. Approvato dal Consiglio Direttivo Nazionale SIMEU; 29 maggio 2021.



Core Principles and Objectives

- Ensure adequate response to emergencies in **Pronto Soccorso (PS)** – Emergency Department – and **DEA** – Emergency and Admissions Department.
- Manage non-urgent conditions and act as a filter for hospital admissions.
- Guarantee equity and service quality in all **MEU – Emergency Medicine Units**.
- Maintain sustainable workloads within legal working-time limits.
- Prevent work-related stress as required by **DLgs 81/2008**.
- Adapt organisation to epidemiological demands, including COVID-19

Società Italiana di Medicina di Emergenza Urgenza (SIMEU). Standard di riferimento 2021 per le unità di Medicina di Emergenza Urgenza (Medicina e Chirurgia d'Accettazione e d'Urgenza) nella rete ospedaliera italiana. Modelli organizzativi e personale. Approvato dal Consiglio Direttivo Nazionale SIMEU; 29 maggio 2021.



Helicopter Emergency Medical Service (HEMS): Functions and Clinical Role

- Provides rapid advanced medical care for critically ill or injured patients directly at the incident site.
- Stabilises life-threatening conditions before transport, including airway management, ventilation, circulatory support, analgesia, and trauma interventions.
- Ensures fast access to definitive care for time-dependent conditions such as myocardial infarction, stroke, major trauma, sepsis, and obstetric emergencies.
- Reduces transport times where road access is slow or not feasible, supporting residents of remote, rural, inland, and island areas.
- Integrates with the 118 Emergency Dispatch Centres to complement ground ambulances and maintain regional coverage.
- Provides safe transport to the most appropriate hospital, including direct transfer to hub centres for specialised care.

Elisoccorso. AREUS - Azienda Regionale Emergenza Urgenza Sardegna. Accessed November 19, 2025. https://www.areus.sardegna.it/azienda/la-struttura-organizzativa/elisoccorso/

HEMS interventions

- HEMS interventions (on-scene emergency missions)
  Deliver advanced medical treatment at the scene and transport to the best-suited hospital.
- Search and Rescue missions
  Recover individuals in hostile or inaccessible environments with immediate health risks.
- Secondary urgent transfers (Helicopter Air Ambulance)
  Move patients from spoke hospitals to hub centres within Sardinia or outside the region for higher-level care.
- Special missions
  - Neonatal transfers with incubators and specialist staff.
  - Maternal transfers for high-risk pregnancy complications.
  - Transport of transplant teams, organ recipients, blood products, and antidotes.
  - Support for mass-casualty incidents and major emergencies.
- HEMS crew
  Aeronautical team, technical rescue personnel, and highly trained medical and nursing staff.



Elisoccorso. AREUS - Azienda Regionale Emergenza Urgenza Sardegna. Accessed November 19, 2025. https://www.areus.sardegna.it/azienda/la-struttura-organizzativa/elisoccorso/

ED Triage

Triage

Triage assigns limited resources to unlimited medical needs. The process addresses mismatches in timing, volume, and location between patient needs and available capacity.
Emergency medicine has limited control over the rate and number of presentations. Population trends are predictable, but individual arrivals are not.

- Workload stratification by triage category improves prediction.
- Major incidents can create sudden surges in demand.
- Triage operates continuously and is reinforced at key points such as scene extrication, ED arrival, hospital admission, and entry to the operating theatre.

Purpose

Triage aims to protect life and health with fairness and efficient resource use. The method identifies who can safely wait and who needs immediate care. Rising demand and access block intensify the need for reliable triage.

Historical development

Early ED triage was intuitive nursing practice. Nurses reorganised queues to prioritise patients unable to wait. The first systematic record originates from Baltimore in the early 1960s, but early systems lacked structure and consensus on categories. In some departments, clerical staff performed triage or patients self selected medical or surgical queues. Growing demand led departments to adopt structured triage systems with category ranges from two to ten levels.

Performance focus

As systems evolved, emergency departments adopted performance evaluation. These evaluations linked timely, appropriate care with accurate workload classification.

The Triage and the colour Codes: the health traffic light

- When arriving at the Accident and Emergency department, there will be the evaluation of the patient's level of urgency by trained nurses. A colour code will be given to establish the priority of access to treatment on the basis of the seriousness of the case. This does not depend on the arrival time at the hospital.

  This approach is called "Triage" and it aims to avoid delays for urgent cases.

- The Triage does not aim at reducing waiting times, but at ensuring that patients in extremely serious conditions do not have to waste precious time.



Code red: very critical, danger of death, maximum priority, immediate access to treatment;



Code yellow: fairly critical, high level of risk, potential danger of death; treatment cannot be delayed;



Code green: not very critical, no risk of condition worsening, treatment can de delayed;



Code white: not critical, non-urgent patients.

IMPORTANT

At the Accident and Emergency department priority is given to the most serious patient regardless of time of arrival.

Arriving at the Accident and Emergency department by ambulance does not mean you will be visited more quickly.

TRIAGE: modalità e tempi di rivalutazione				
Codice		Denominazione	Tempo massimo di attesa	Modalità di Rivalutazione
Numero	Colore			
1	ROSSO	EMERGENZA	ACCESSO IMMEDIATO ALLE AREE DI TRATTAMENTO: NESSUNA RIVALUTAZIONE	
2	ARANCIONE	URGENZA	ACCESSO ENTRO 15 MIN	OSSERVAZIONE DIRETTA O VIDEO MEDIATA CON MONITORAGGIO COSTANTE DELLE CONDIZIONI
3	AZZURRO	URGENZA DIFFERIBILE	ACCESSO ENTRO 60 IN	RIPETIZIONE DI PARTE O TUTTE LE FASI DI VALUTAZIONE: - A GIUDIZIO DELL'INFERMIERE DI TRIAGE - A RICHIESTA DEL PAZIENTE - UNA VOLTA TRASCORSO IL TEMPO DI ATTESA MASSIMO RACCOMANDATO
4	VERDE	URGENZA MINORE	ACCESSO ENTRO 120 MIN	
5	BIANCO	NON URGENZA	ACCESSO ENTRO 240 MIN	

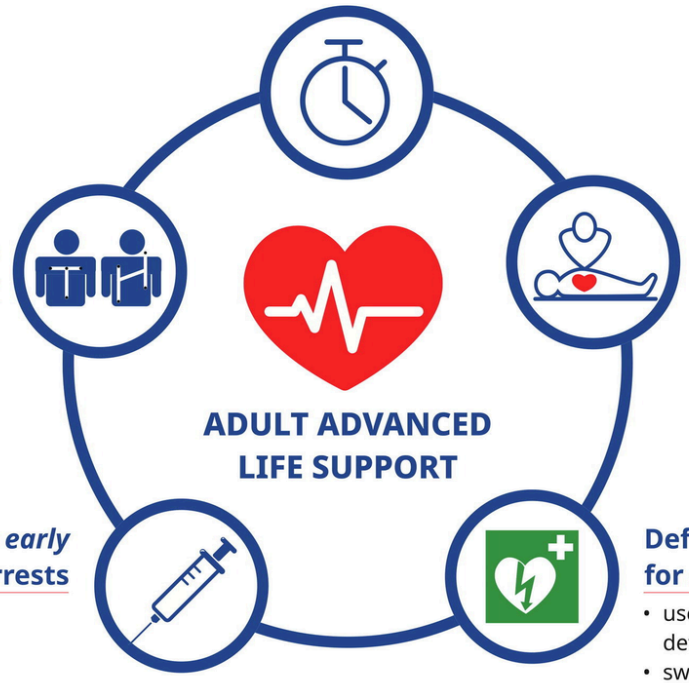
Overview of the Advanced Life Support Algorithm

ADULT ADVANCED LIFE SUPPORT
KEY MESSAGES

GUIDELINES 2025
EUROPEAN RESUSCITATION COUNCIL©

Start advanced life support *early*

- every second counts!



Identify and treat reversible causes *without delay*

Ensure *effective* ventilation and high-quality chest compressions

Give IV adrenaline *early* for non-shockable cardiac arrests

Defibrillate *early* for shockable rhythms

- use *correct* antero-lateral defibrillator pad placement
- switch to antero-posterior pads if 3 shocks are ineffective

Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769

In-hospital cardiac arrest

Prevention of in-hospital cardiac arrest IHCA occurs in about 1.5 patients per 1000 hospital admissions.

Two strategies aim to prevent cardiac arrest and reduce the need for CPR.

Patient-focused decisions

- Use structured discussions to determine if CPR is appropriate for each patient.
- Apply shared decision-making and advanced care planning as recommended by ERC 2025 Ethics in Resuscitation. • Integrate resuscitation decisions with emergency treatment plans to clarify goals and avoid omission of indicated treatments. Physiological deterioration
- Most IHCA events follow identifiable deterioration.
- Early recognition allows timely intervention and prevention of cardiac arrest.

In-hospital chain of survival

- Staff education.
- Monitoring.
- Recognition of deterioration.
- Call for help.
- Clinical response.

This structure aligns with the ILCOR COSTR review of rapid response systems, UK guidance on early warning scores, ILCOR's Ten Steps Toward Improving In-Hospital Cardiac Arrest Care, and SCCM guidance on managing deterioration outside the ICU.



Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769

Recognition

- Early warning score systems help track a patient's condition and standardise recognition of acute illness.
- EWS tools include predefined graded responses and escalation pathways.
- These systems may improve identification of deterioration and reduce time to emergency activation.
- Clinical concern from nurses and other clinicians can signal deterioration even without abnormal scores.

The call for help

- All staff must feel authorised to call for help.
- Training should include the use of structured tools such as SBAR for clear communication.
- Rapid response systems such as MET, RRT, or CCOT provide support for critically ill patients.
- Any team member can activate these systems according to predefined criteria.
- Some hospitals encourage patients and families to activate the team as part of patient safety initiatives.



Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769

Crisis Resource Management

CRISIS RESOURCE MANAGEMENT



CRISIS RESOURCE MANAGEMENT

Call for Help Early

- Call for help early enough to make a difference
- Err on the side of getting more help
- Mobilize early personnel with special skills if they may be needed

Designate Leadership

- Establish clear leadership
- Inform team members who is in charge
- 'Followers' should be active in asking who is leading

Anticipate and Plan

- Plan & prepare for high work-load periods during low work-load periods
- Know where you are likely headed during the crisis and make backup plans early

Know the Environment

- Maintain situational awareness
- Know how things work and where things are
- Be aware of strengths and vulnerabilities of environment

Establish Role Clarity

- Determine who will do what
- Assign areas of responsibility appropriate to knowledge, skills, and training
- Active followers may offer specific roles

Use All Available Information

- Monitor multiple streams of data and information
- Check and cross check information

Distribute the Workload

- Assign specific tasks to team members according to their abilities
- Revise the distribution if there is task overload or failure

Allocate Attention Wisely

- Eliminate or reduce distractions
- Monitor for task saturation & data overload
- Avoid getting fixated
- Recruit others to help w/ monitoring

Communicate Effectively

- Command and request clearly
- Seek confirmation of request (close the loop)
- Avoid "thin air" statements
- Foster input and atmosphere of open information exchange among all personnel

Mobilize Resources

- Activate all helpful resources including equipment and additional personnel

Use Cognitive Aids

- Be familiar with content, format, and location
- Support the effective use of cognitive aids

ANESTHESIA & ANALGESIA

Goldhaber-Fiebert SN, Howard SK. Implementing emergency manuals: can cognitive aids help translate best practices for patient care during acute events? *Anesth Analg*. 2013;117(5):1149. doi:10.1213/ANE.0b013e318298867a

TeamSTEPPS

https://www.ahrq.gov/teamstepps-program/index.html



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Team Strategies & Tools to Enhance Performance & Patient Safety

Communication

Module 1

Agency for Healthcare Research and Quality

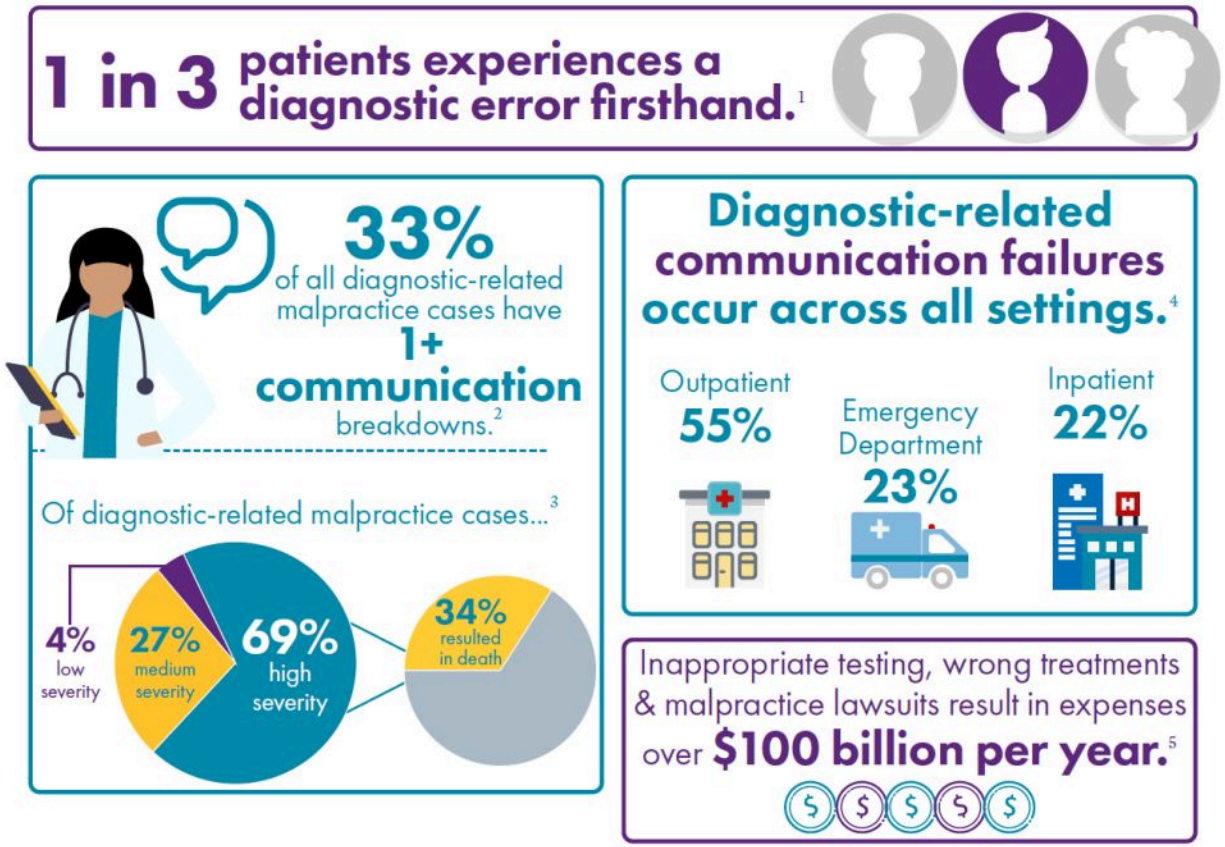
Effective Communication

- Effective communication skills:
  - Are vital for patient safety.
  - Enable team members to effectively relay information, build trust, and provide reassurance and emotional support.
  - Are the mechanism by which most TeamSTEPPS strategies and tools are executed.



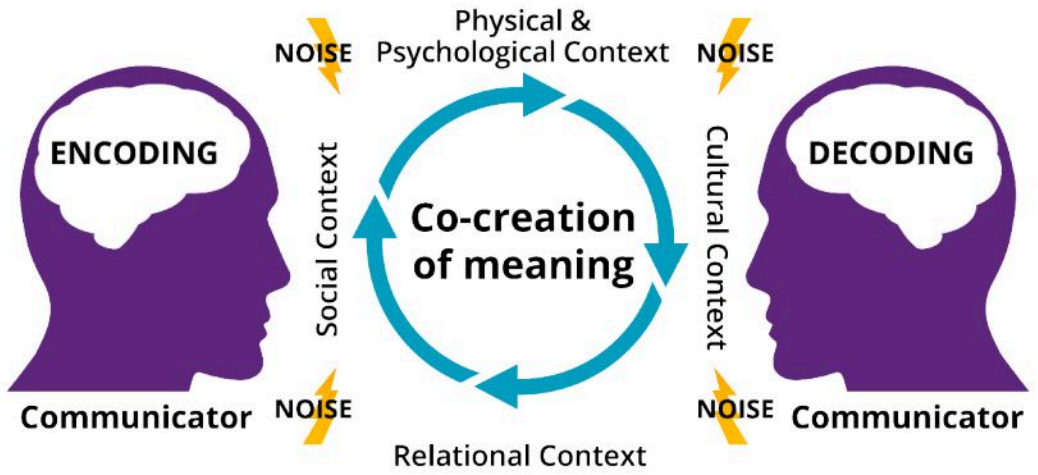
Importance of Communication

- Joint Commission data identify communication failures as a common cause of sentinel events.
- AHRQ-funded research identifies communication and inadequate information transfer as the top two causes of medical errors.
- Diagnostic errors are common, harmful, and often the result of communication breakdowns.



Communication Is…

- Both sending and receiving messages.
- The co-creation of meaning that may differ from the intended meaning.
- Affected by noise, context, and communicator assumptions.
- The mode by which most TeamSTEPPS strategies and tools are executed.



Standards of Effective Communication



Complete

Communicate all relevant information



Clear

Convey information in plain language



Brief

Communicate information in a concise manner



Timely

Offer and request information in an appropriate timeframe



Respectful

Use communication to foster psychological safety and affirm other team members, not just to give instructions or share information

Communication Challenges

- Language barriers
- Distractions, stress, and fatigue
- Physical proximity
- Personalities
- Workload
- Differing backgrounds, vocabularies, and priorities
- Varying communication styles
- Conflict
- Lack of information verification
- Shift changes

Information Exchange Tools

Communication

- Situation – Background – Assessment – Recommendation or Request (SBAR)
- Call-Out
- Check-Back
- Teach-Back
- Handoffs (including I-PASS)

SBAR Provides…

- A framework for team members to effectively communicate information to one another.



Situation

What is going on with the patient?



Background

What is the clinical background and context?



Assessment

What do I think the problem is?



Recommendation or Request

What would I recommend?

What do I need from you?

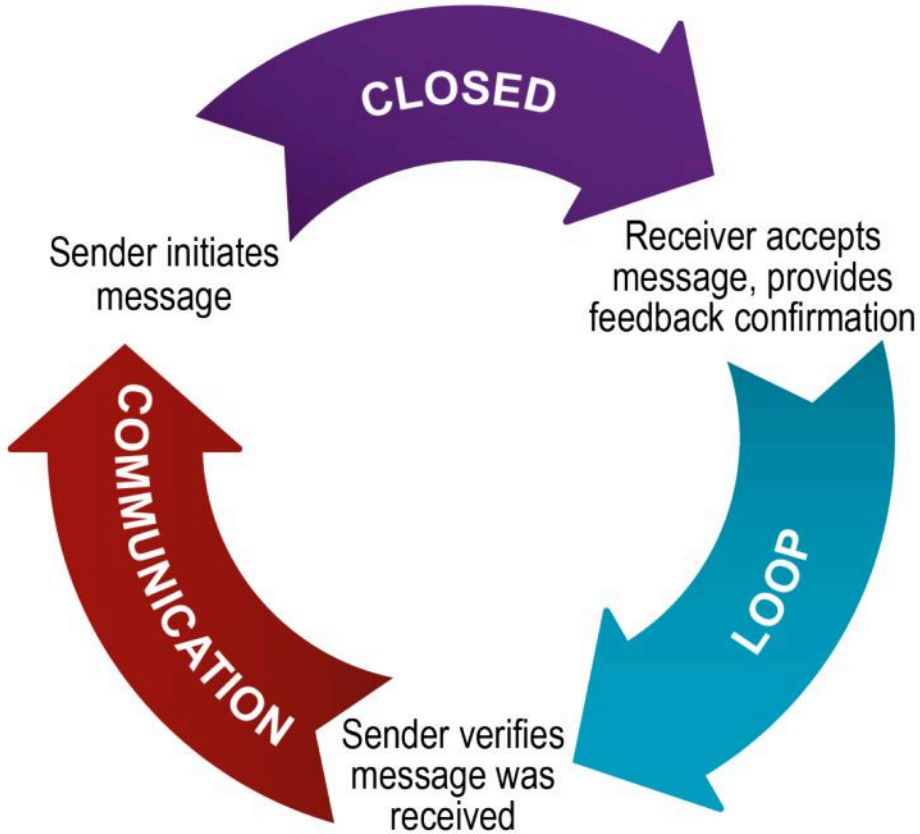
Call-Out Is…

- A strategy used to communicate important or critical information:
  - It informs all team members simultaneously during emerging situations.
  - It helps team members anticipate next steps.

Check-Back Is…

- Sometimes also called a Repeat-Back.

TeamSTEPPS also includes a Teach-Back tool, which can be used with patients and family caregivers.



Handoff Is…

Communication

- The transfer of information during transitions in care across the continuum:
  - Includes an opportunity to ask questions, clarify, and confirm.
  - Is relevant during shift changes, transfers between departments, and care team transitions.
  - Is sometimes done virtually or with e-handoff functions within an electronic health record.
- Handoffs include:
  - Transfer of responsibility and accountability.
  - Clarity of information.
  - Verbal communication of information.
  - Acknowledgment by receiver.
  - Opportunity to review.

I-PASS: A Common Handoff Tool

Communication



Illness Severity
- Stable, watcher, unstable

Patient Summary
- Summary statement
- Events leading up to admission or care transition
- Hospital course or treatment plan
- Ongoing assessment
- Contingency plan

Action List
- To-do list
- Timelines and ownership

Situation Awareness & Contingency Planning
- Know what's going on
- Plan for what might happen

Synthesis by Receiver
- Receiver summarizes what was heard
- Asks questions
- Restates key actions/to-do items

Other Handoff Tools

- ANTICipate
  - **A**dministrative Data; **N**ew clinical information; **T**asks to be performed; **I**llness severity; **C**ontingency plans for changes
- SHARQ
  - **S**ituation; **H**istory; **A**ssessment; **R**ecommendations/Result; **Q**uestions

Tools and Strategies Summary

Communication

BARRIERS	TOOLS and STRATEGIES	OUTCOMES

• Inconsistency in Team Membership
• Lack of Time
• Lack of Information Sharing
• Hierarchy
• Defensiveness
• Conventional Thinking
• Complacency
• Varying Communication Styles
• Conflict
• Lack of Coordination and Follow-Up
• Distractions
• Fatigue and Burnout
• Workload
• Misinterpretation of Cues
• Lack of Role Clarity
	• Communication
	  • SBAR
	  • Call-Out
	  • Check-Back
	  • Handoff
	  • Teach-Back
	  • I-PASS
		• Shared Mental Model
		• Adaptability
		• Team Orientation
		• Mutual Trust
		• Reduced Burnout
		• Psychological Safety
		• Effective Team Performance
		• Safe, Highly Reliable, Patient-Centered Care



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Team Leadership

Module 2

Agency for Healthcare Research and Quality

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Understanding Teams and Team Structure

Team Structure

Team Leadership

- Teamwork cannot occur in the absence of a clearly defined team.
- Understanding a team's structure and how multiple teams interact is critical for implementation planning.



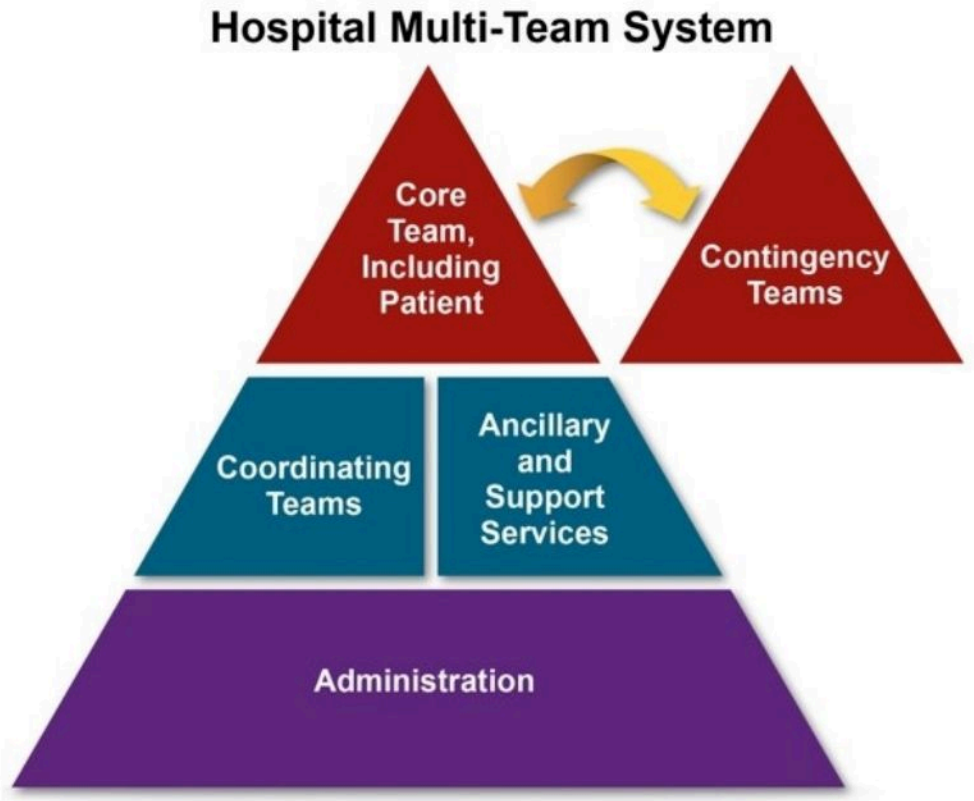
What Defines a Team?

Team Leadership

- Two or more people who interact dynamically, interdependently, and adaptively toward a common and valued goal, have specific roles or functions, and have a time-limited membership
- Teams can be:
  - In-person or virtual.
  - Temporary or permanent.
  - Frequently changing due to staff schedules and assignments.

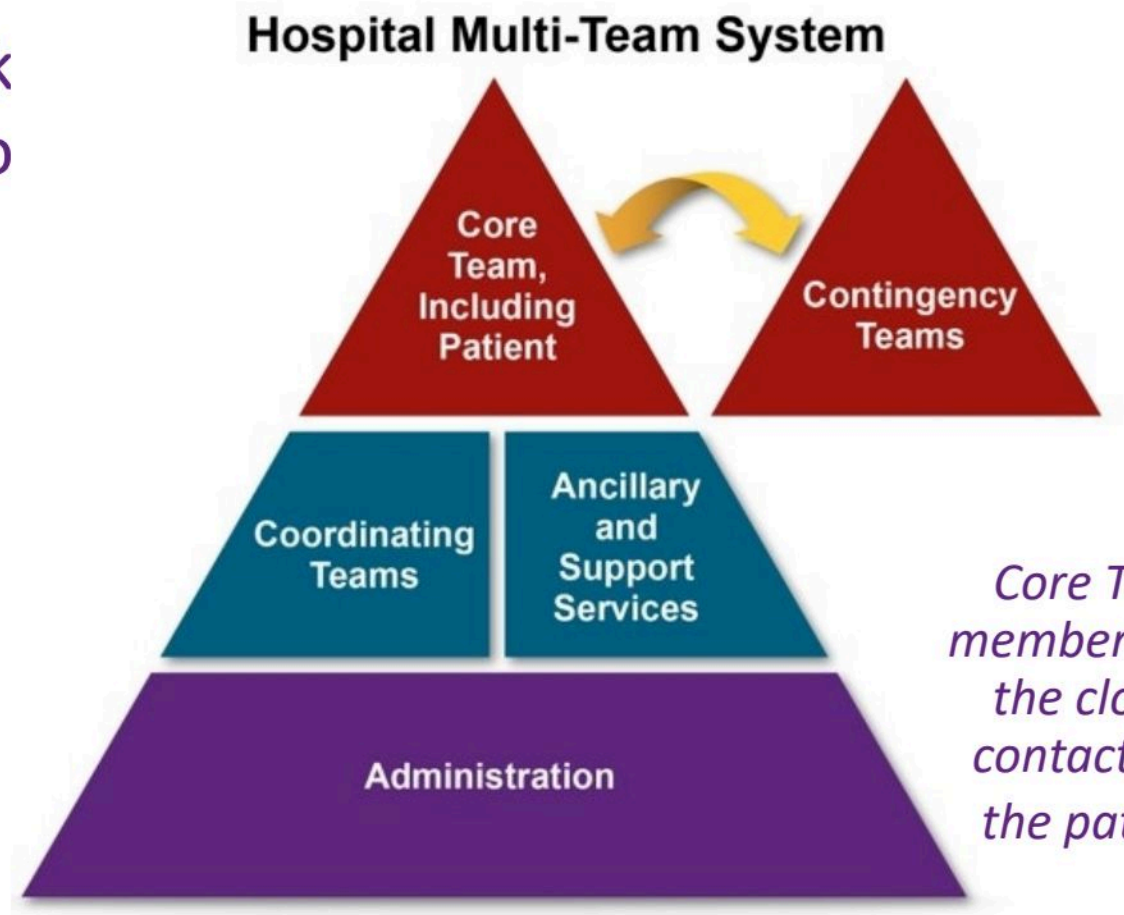
Multi-Team System (MTS) for Patient Care

Team Leadership



A Core Team Is…

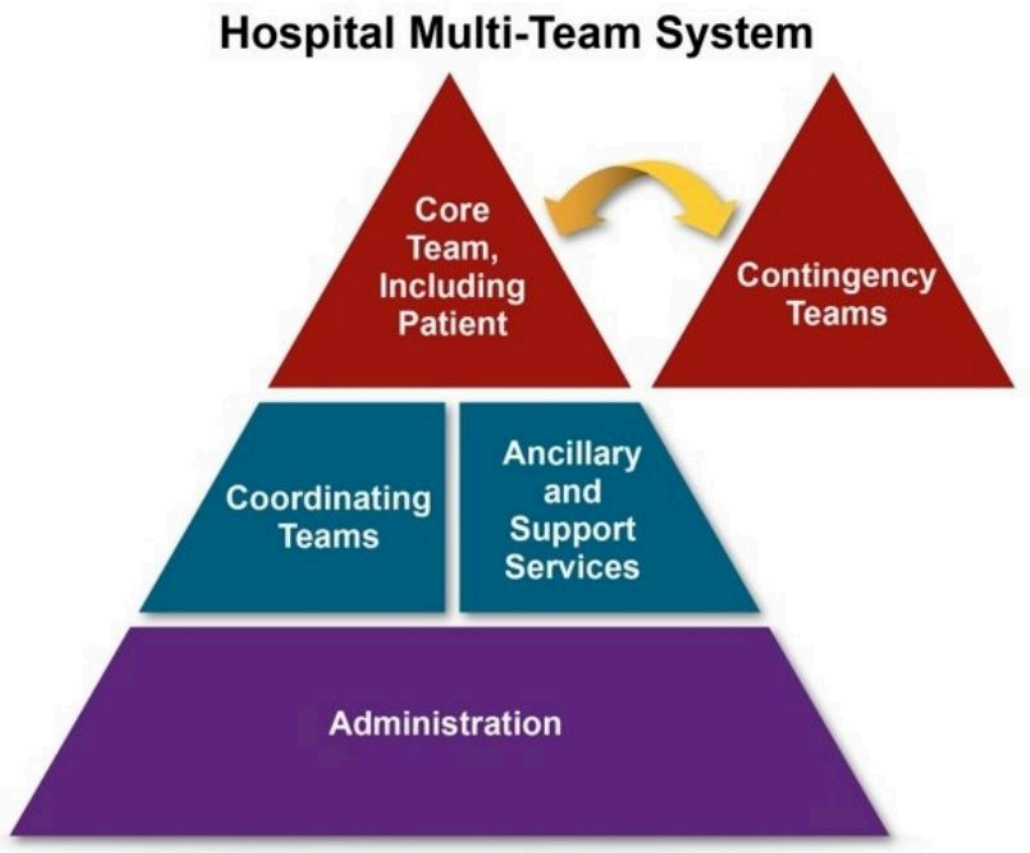
- A group of care providers who work interdependently to manage a set o assigned patients from point of assessment to disposition.



Core Team members have the closest contact with the patient.

A Contingency Team Is…

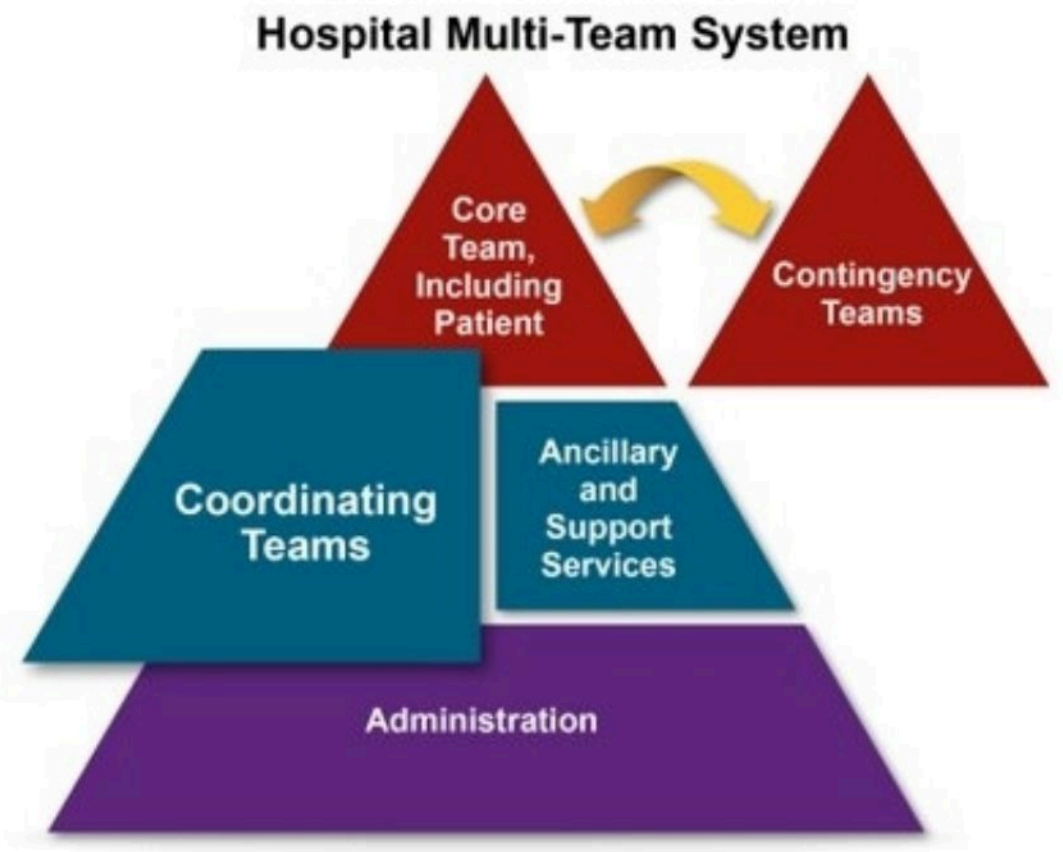
- A time-limited team formed for emergent or specific events and composed of members from various teams.



A Coordinating Team Is…

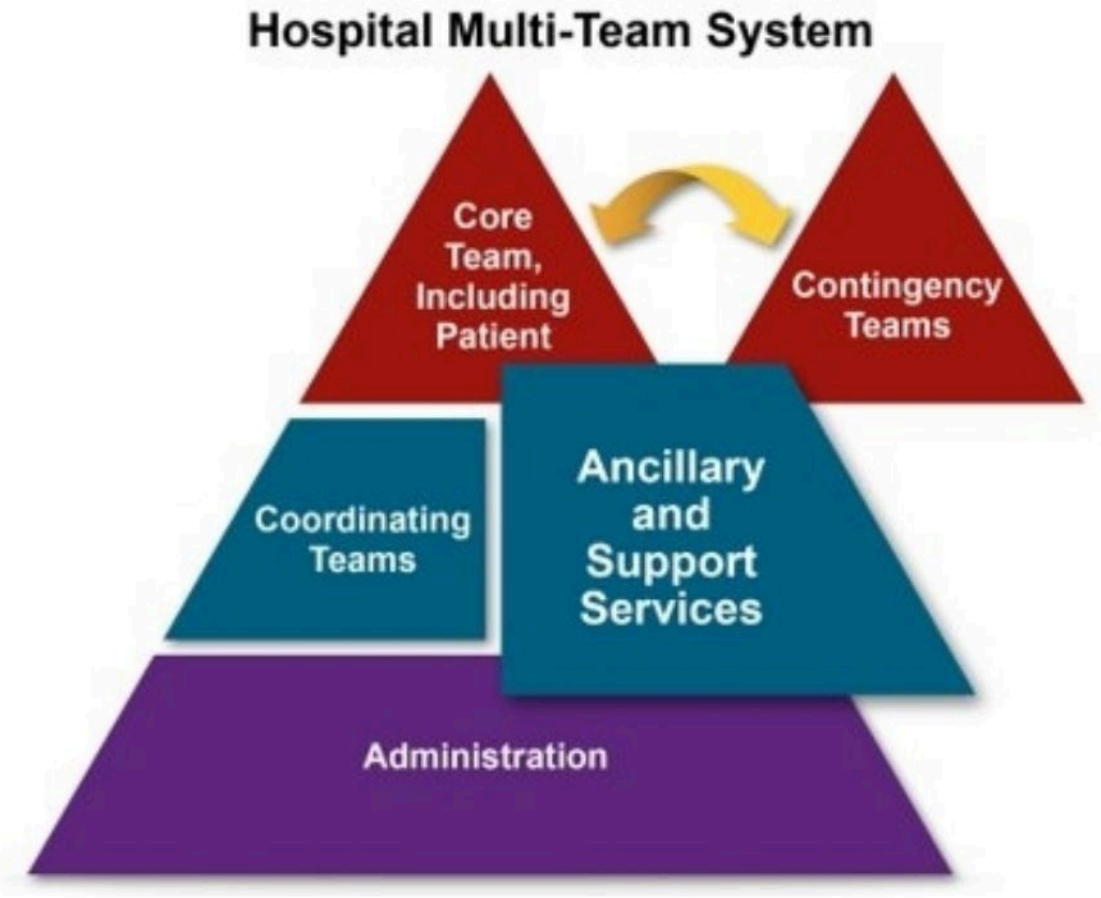
Team Leadership

- A team comprising those work area members who are responsible for managing the operational environment that supports the Core Team.



Ancillary and Support Services

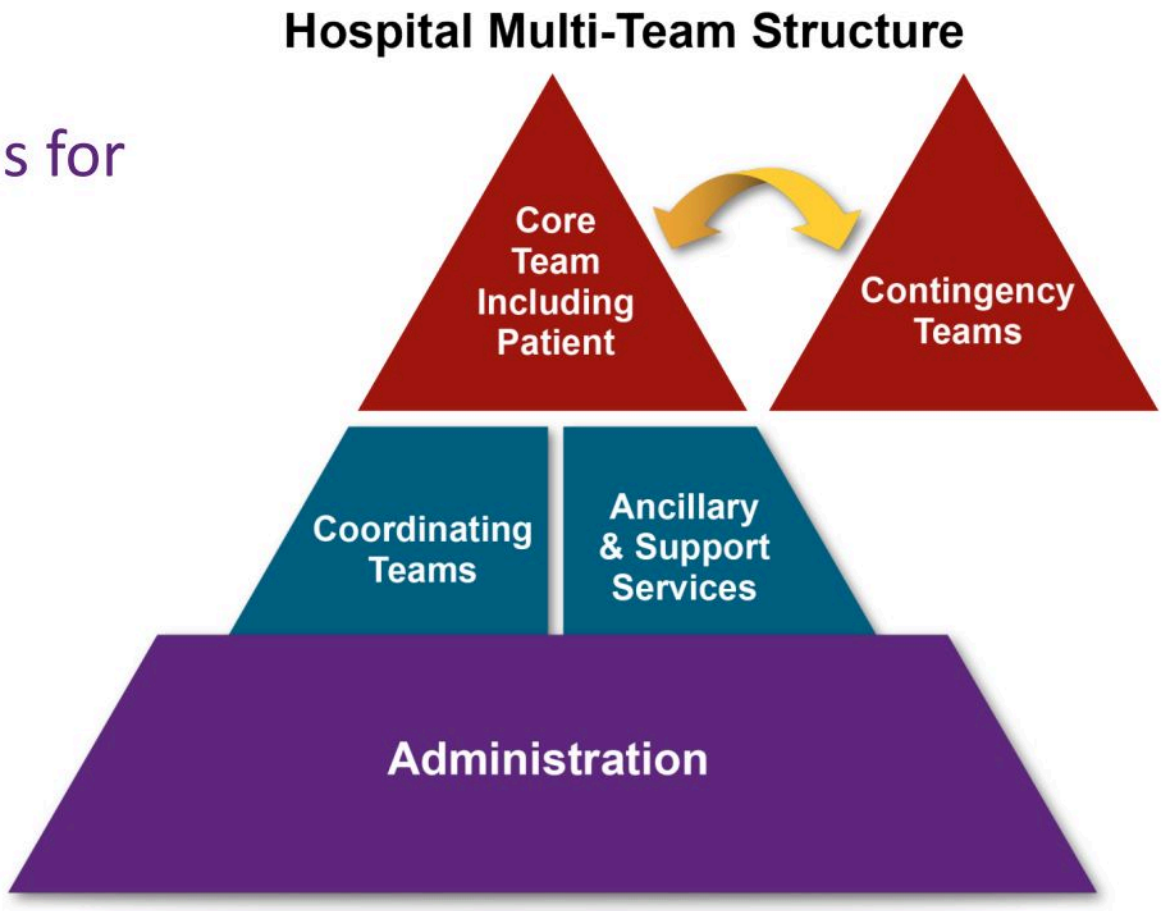
- Ancillary Services provide direct, task-specific, time-limited care to patients.
- Support Services provide indirect service-focused tasks that help facilitate the optimal healthcare experience for patients and their families.



The Role of Administration Is To…

Team Leadership

- Establish and communicate vision.
- Develop policies and set expectations for staff related to teamwork.
- Support and encourage staff during implementation and culture change.
- Hold teams accountable for team performance.
- Define the culture of the organization.



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Leading Teams

Leadership

- Ensures the patient remains the focus for all team decisions
- Holds a teamwork system together
- Ensures a plan is conveyed, reviewed, and updated
- Is facilitated by communicating, continuously monitoring the situation, and fostering an environment of mutual support



Types of Team Leaders

- **Designated:** The person assigned to lead and organize a team, establish clear goals, and facilitate open communication and teamwork among team members
- **Situational:** Any team member with the skills to manage the situation at hand

Effective Team Leaders

Team Leadership

- Ensure the patient's needs and preferences are understood and prioritized.
- Define, assign, share, monitor, and modify a plan.
- Review the team's performance.
- Establish "rules of engagement."
- Manage and allocate resources effectively.
- Provide feedback regarding assigned responsibilities and progress toward the goal.
- Facilitate information sharing.
- Encourage team members to assist one another.
- Facilitate conflict resolution.
- Model effective teamwork.

Defining the Plan

Team Leadership

- When developing a plan, team leaders should consider:
  - **Patient:** What does the patient need and how can the team best provide it?
  - **Time:** How much time is available to complete all the necessary tasks and activities?
  - **People:** Do available staff have the necessary knowledge and skills to perform their roles?
  - **Equipment:** Is the needed equipment available and working?
  - **Information:** Has all the needed information been collected and reviewed?

Assigning Tasks and Responsibilities

Team Leadership

- Determine the tasks and roles to be assigned.
- Determine which roles must be filled and allocate tasks appropriately.
- Communicate clear expectations of what team members need to do.
- Request feedback.

Brief Checklist Tool

Team Leadership



Brief Checklist

____ Who is on the team?

____ Do all members understand and agree upon goals?

____ Are roles and responsibilities understood?

____ What is our plan of care?

____ What is provider availability throughout the shift?

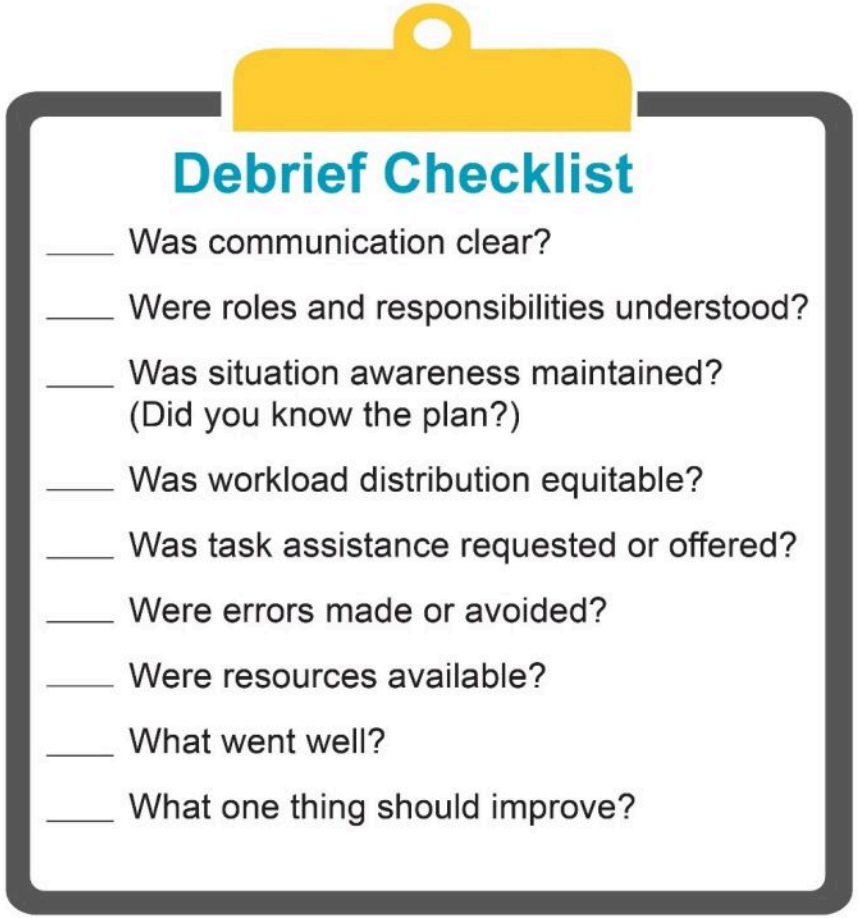
____ How is workload shared among team members?

____ What resources are available?

Reviewing the Team's Performance: Debrief

Team Leadership

- Debriefs foster process improvement:
  - Brief, informal information exchange and feedback sessions
  - Occur after an event or shift
  - Designed to improve teamwork skills
  - Designed to improve outcomes:
    - An accurate recounting of key events
    - Analysis of why the event occurred
    - Discussion of lessons learned and reinforcement of successes
    - Revised plan to incorporate lessons learned



Facilitating Conflict Resolution

Team Leadership

- Effective leaders:
  - Facilitate conflict resolution to avoid compromising patient safety and quality of care.
  - Do not allow interpersonal or irrelevant issues to negatively affect the team.
  - Help team members master conflict resolution techniques.

Promoting and Modeling Teamwork

Team Leadership

- Effective leaders cultivate desired team behaviors and skills through:
  - Open sharing of information.
  - Role modeling and effective cuing of team members to use prescribed teamwork behaviors and skills.
  - Constructive and timely feedback.
  - Facilitation of briefs, huddles, debriefs, and conflict resolution.
  - Mitigation of conflict within the team.

Tools and Strategies Summary

Team Leadership

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• Lack of Role Clarity
	• Communication
	  ○ SBAR
	  ○ Call-Out
	  ○ Check-Back
	  ○ Handoff
	  ○ Teach-Back
	  ○ I-PASS
	• Leading Teams
	  ○ Brief
	  ○ Huddle
	  ○ Debrief
		• Shared Mental Model
		• Adaptability
		• Team Orientation
		• Mutual Trust
		• Reduced Burnout
		• Psychological Safety
		• Effective Team Performance
		• Safe, Highly Reliable, Patient-Centered Care



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Situation Monitoring

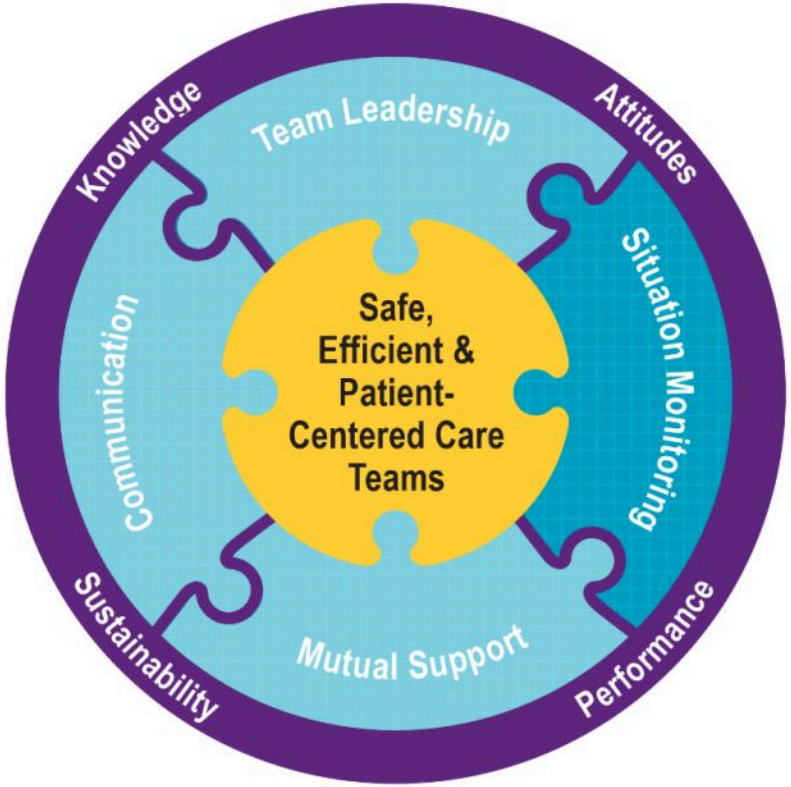
Module 3

Agency for Healthcare Research and Quality

Situation Monitoring…

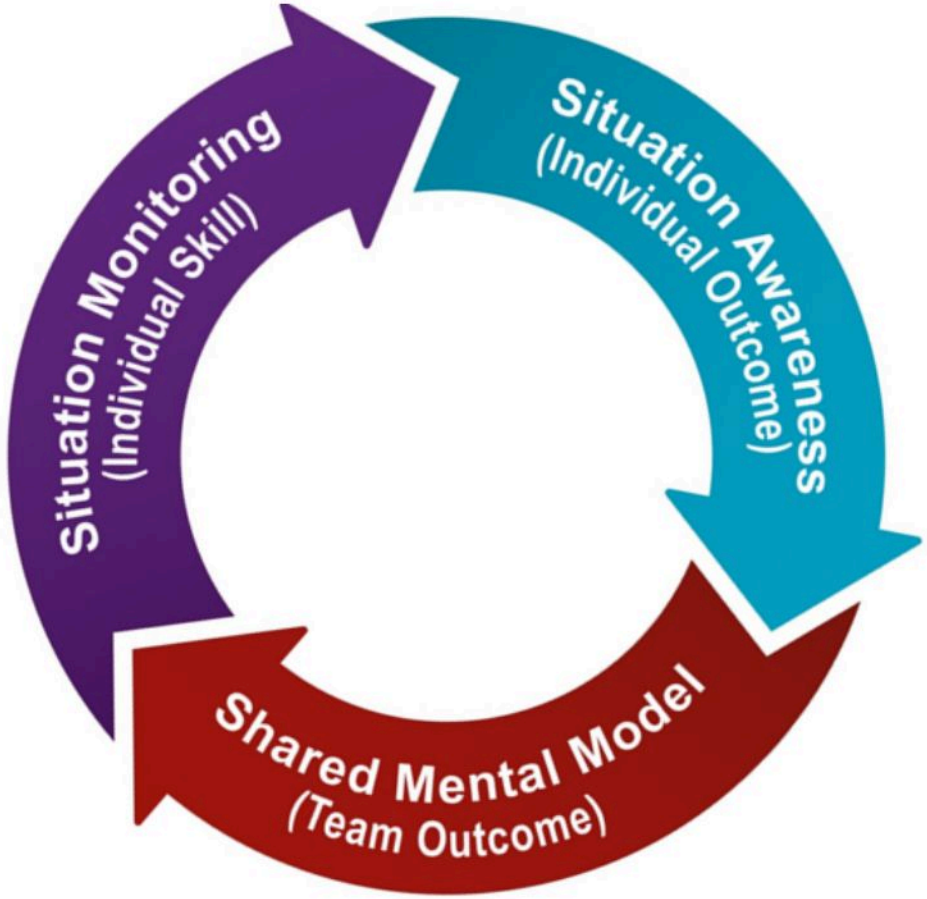
Situation Monitoring

- Ensures new or changing information is identified for communication and decision making.
- Leads to effective support of fellow team members.



Situation Monitoring: A Continuous Process

Situation Monitoring



Situation Awareness Is…

Situation Monitoring

- The state of knowing the current conditions affecting one's work.
- It includes knowing…
  - Status of the patient.
  - Status of other team members.
  - Environmental conditions.
  - Current progress towards goals.

Situation Awareness Small Group Discussion

Situation Monitoring

Goal:

- Identify factors that promote and prevent situation awareness.

Process:

- In your group, compile a list of factors that affect situation awareness in your team.
- Create a list of ways other team members help or hurt your situation awareness.
- Discuss what group members do to help get and share information with patients to foster situation awareness.



Conditions That Maximize Situation Awareness

Situation Monitoring

- When team members:
  - Share information with the team
  - Request information from others
  - Direct information to specific team members
  - Include patient or family in communication
  - Use resources fully (e.g., status board, automation)
  - Maintain documentation
  - Know and understand where to focus attention
  - Know and understand the plan
  - Inform team members when the plan has changed

How Shared Mental Models Help Teams

Situation Monitoring

- Lead to mutual understanding of situation
- Lead to more effective communication
- Enable backup behaviors
- Help ensure understanding of each other's roles and how they interplay
- Enable better prediction and anticipation of team needs
- Create commonality of effort and purpose

A Shared Mental Model Is…

Situation Monitoring

- The perception of, understanding of, or knowledge about a situation or process that is shared among team members through communication.

Who Lacks the Shared Mental Model?



When and How To Share?

Situation Monitoring

When:

- Briefs
- Huddles
- Debriefs
- Transitions in care

How:

- SBAR
- Call-outs
- Check-backs

Tools To Support Situation Monitoring

Situation Monitoring

Tool	Primary Use
STEP	Patient Status
I'M SAFE	Team Member Status
Cross-Monitoring	Status of Environment and Other Team Members
STAR	Status of Specific Activities or Tasks

Status of Patient

Situation Monitoring

Status of Patient

Team Members

Environment

Progress Toward Goal

- Patient History
- Vital Signs
- Medications
- Physical Exam
- Plan of Care
- Psychosocial Condition
- Patient Preferences or Concerns
- If Patient Has Limited English Proficiency

Team Members

Situation Monitoring

Status of Patient

Team Members

Environment

Progress Toward Goal

- Fatigue
- Workload
- Task Performance
- Skill Level
- Stress Level

Environment

Situation Monitoring

Status of Patient

Team Members

Environment

Progress Toward Goal

- Facility information, including occupancy levels
- Administrative information and available staff
- Human resources
- Triage acuity
- Community or facility disease outbreaks
- Equipment status

Progress Toward Goal

Situation Monitoring

Status of Patient

Team Members

Environment

Progress Toward Goal

- Call a Huddle!
- Status of Team's Patients
- Goal of Team
- Tasks/Actions That Are or Need To Be Completed
- Plan Still Appropriate

Tools for Situation Monitoring: I'M SAFE Checklist

Situation Monitoring



Tools for Situation Monitoring: Cross-Monitoring

Situation Monitoring

- What it is:
  - "Watching each others' back" to ensure team members' actions are consistent with the plan and advocating for the patient as needed when risks arise and help is needed.
- Why it matters:
  - The plan may be unclear or misunderstood.
  - The plan may be forgotten or changed.
  - When plans are unclear or not implemented, a shared mental model is lost and patients are at risk.
- How to do it:
  - Make monitoring each other a group norm that's expected and not a sign of mistrust.
  - Encourage asking questions to confirm that actions match the plan.
  - Express concerns when actions aren't consistent with the plan.
  - Involve the patient whenever relevant.

Tools for Situation Monitoring: STAR

Situation Monitoring

- What it is:
  - A four-step mnemonic tool that is used extensively to elicit and share key information about activities and their consequences.
- Key elements include:
  - Stop: Pause to focus on the immediate task.
  - Think: Think methodically and identify the correct action.
  - Act: Perform the act.
  - Review: Confirm anticipated result has occurred or apply contingency if required.
- Practice example:
  - Pick a task you were asked to perform by a family member or friend in the past month that didn't quite go as planned. Use STAR to explain what you did and what you could have done differently to have achieved a better outcome.

Tools and Strategies Summary

Situation Monitoring

BARRIERS	TOOLS and STRATEGIES	OUTCOMES

• Inconsistency in Team Membership
• Lack of Time
• Lack of Information Sharing
• Hierarchy
• Defensiveness
• Conventional Thinking
• Complacency
• Varying Communication Styles
• Conflict
• Lack of Coordination and Follow-Up
• Distractions
• Fatigue and Burnout
• Workload
• Misinterpretation of Cues
• Lack of Role Clarity
	• Communication
	  ○ SBAR
	  ○ Call-Out
	  ○ Check-Back
	  ○ Handoff
	  ○ Teach-Back
	  ○ I-PASS
	• Leading Teams
	  ○ Brief
	  ○ Huddle
	  ○ Debrief
	• Situation Monitoring
	  ○ STEP
	  ○ I'M SAFE
	  ○ Cross-Monitoring
	  ○ STAR
		• Shared Mental Model
		• Adaptability
		• Team Orientation
		• Mutual Trust
		• Reduced Burnout
		• Psychological Safety
		• Effective Team Performance
		• Safe, Highly Reliable, Patient-Centered Care



TeamSTEPPS®
Team Strategies & Tools to Enhance Performance & Patient Safety

Mutual Support

Module 4

Agency for Healthcare Research and Quality

Mutual Support Is…

Mutual Support

- Dependent on information gathered through situation monitoring.
- Facilitated by the communication of information.
- Enhanced by leaders who encourage and model mutually supportive behaviors.
- Vital to supporting both patients and other care providers.



Mutual Support Involves…

Mutual Support

- Team members who:
  1. Assist each other.
  2. Provide and receive feedback.
  3. Exert assertive advocacy behaviors when patient safety is threatened.
  4. Respond to conflict promptly and constructively.

Mutual Support Discussion

Mutual Support

Ask



- How do I increase self-awareness to be aware of the impact of actions on me, others/team/patient, and the environment?

Listen



- What are ways I demonstrate support to each team member?

Act



- How do we create and sustain an environment in which our team members can ask for assistance when overloaded or uncertain?

Task Assistance

Mutual Support

- Team members foster a climate in which it is expected that assistance will be actively **sought** and **offered** as a method for reducing error risks and workplace stress and burnout.

What Is Feedback?

Mutual Support

- Feedback is information provided that can positively or negatively impact team performance.
- It may be:
  - Nonverbal (not paying attention, looking upset, smiling and affirming what's being said).
  - Verbal (making a suggestion, agreeing, or disagreeing).
  - Intentional (a message you were deliberately sending).
  - Unintentional (a reaction you were not trying to communicate).

Types of Formative Feedback

Mutual Support

- Formal
  - Retrospective and typically scheduled in advance
  - Has an evaluative quality
  - Examples: collaborative discussions, case conferences, individual performance reviews
- Informal
  - Typically in real time
  - Provided on an ongoing basis
  - Focuses on knowledge and practical skills development
  - Examples: huddles, debriefs

Characteristics of Effective Formative Feedback

Mutual Support

- Effective formative feedback is:
  - Appreciative.
  - Timely.
  - Respectful.
  - Specific.
  - Directed toward improvement:
    - Helps prevent the same problem from occurring in the future.
  - Considerate.
  - Patient focused.

Advocacy and Assertion

Mutual Support

- Advocate for the patient:
  - Invoked when team members' viewpoints don't coincide with that of a decision maker and patient safety is at risk.
- Assert a corrective action in a **firm** and **respectful** manner.

How would members of your team react to advocacy and assertion from a patient or family caregiver on the team?

The Assertive Statement

Mutual Support

- Respects and supports authority.
- Clearly asserts concerns and suggestions.
- Is nonthreatening and ensures critical information is addressed.
- Five-Step Process:
  1. Open the discussion.
  2. State the concern.
  3. State the problem—real or perceived.
  4. Offer a solution.
  5. Obtain an agreement on next steps.

Two-Challenge Rule

Mutual Support

- Empowers any team member to "**stop the line**" if they sense or discover a breach of safety.
- This action should never be taken lightly, because it requires immediate cessation of the process and resolution of the safety issue.

Two-Challenge Rule (continued)

Mutual Support

- Invoked when an initial assertion is ignored…
  - It is your responsibility to assertively restate the concern.
  - The team member being challenged must acknowledge that they heard and understood your concern.
  - If the response does not clarify and alleviate concern, rephrase the anticipated danger.
  - If the outcome is still not acceptable:
    - Take a stronger course of action.
    - Engage other team members.
    - Use supervisor or chain of command.

Please Use CUS Words
but *only* when appropriate!

Mutual Support



I am **C**ONCERNED!

I am **U**NCOMFORTABLE!

This is a **S**AFETY ISSUE!

"Stop the Line"

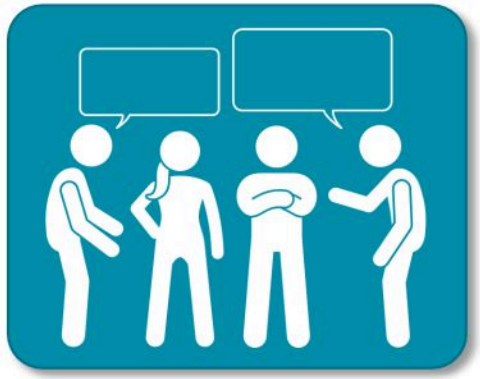
Advocacy and Assertion Scenario

Mutual Support

- A medical floor nurse is assigned to a patient who has had a myocardial infarction. The attending physician provides the final treatment, reviews the clinical situation, and determines that the patient is well enough to be discharged.
- Before the patient is discharged, the nurse checks the patient's vitals one last time. The nurse finds it unusual that the blood pressure and heart rate are substantially elevated.

Conflict in Teams

Mutual Support



Informational Conflict
(We have different information.)



Interpersonal Conflict
(Hostile and harassing behavior)

Two-Challenge Rule

DESC Script

Conflict Resolution: DESC Script

Mutual Support

- A constructive approach for managing and resolving conflict



Describe the specific situation or behavior; provide concrete data.

Express how the situation makes you feel/what your concerns are.

Suggest alternatives and seek agreement.

Consequences should be stated in terms of impact on the patient and established team goals; strive for consensus.

DESC It

- How to “DESC It!”
  - Have timely discussion.
  - Work on win-win.
  - Frame problem in terms of your own experience.
  - Choose a private location.
  - Use “I” statements; avoid blaming statements.
  - Remember: Critique is not criticism.
  - Focus on what is right, not who is right.

Tools and Strategies Summary

Mutual Support

BARRIERS	TOOLS and STRATEGIES	OUTCOMES

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Knowledge
Team Leadership
Attitudes
Communication
Safe,
Efficient &
Patient-
Centered Care
Teams
Situation Monitoring
Sustainability
Mutual Support
Performance

Introduction to SBAR in Healthcare

Shahid S, Thomas S. Situation, background, assessment, recommendation (SBAR) communication tool for handoff in health care – a narrative review. Saf Health. 2018;4(1):7. doi:10.1186/s40886-018-0073-1

What is SBAR?

SBAR stands for Situation, Background, Assessment, Recommendation. It was first developed by the US Navy for handoffs in submarine operations.

SBAR in Healthcare

Kaiser Permanente introduced SBAR to health care in 2003 to standardise urgent communication between nurses and physicians.

SBAR Components

The Joint Commission defines the components as:

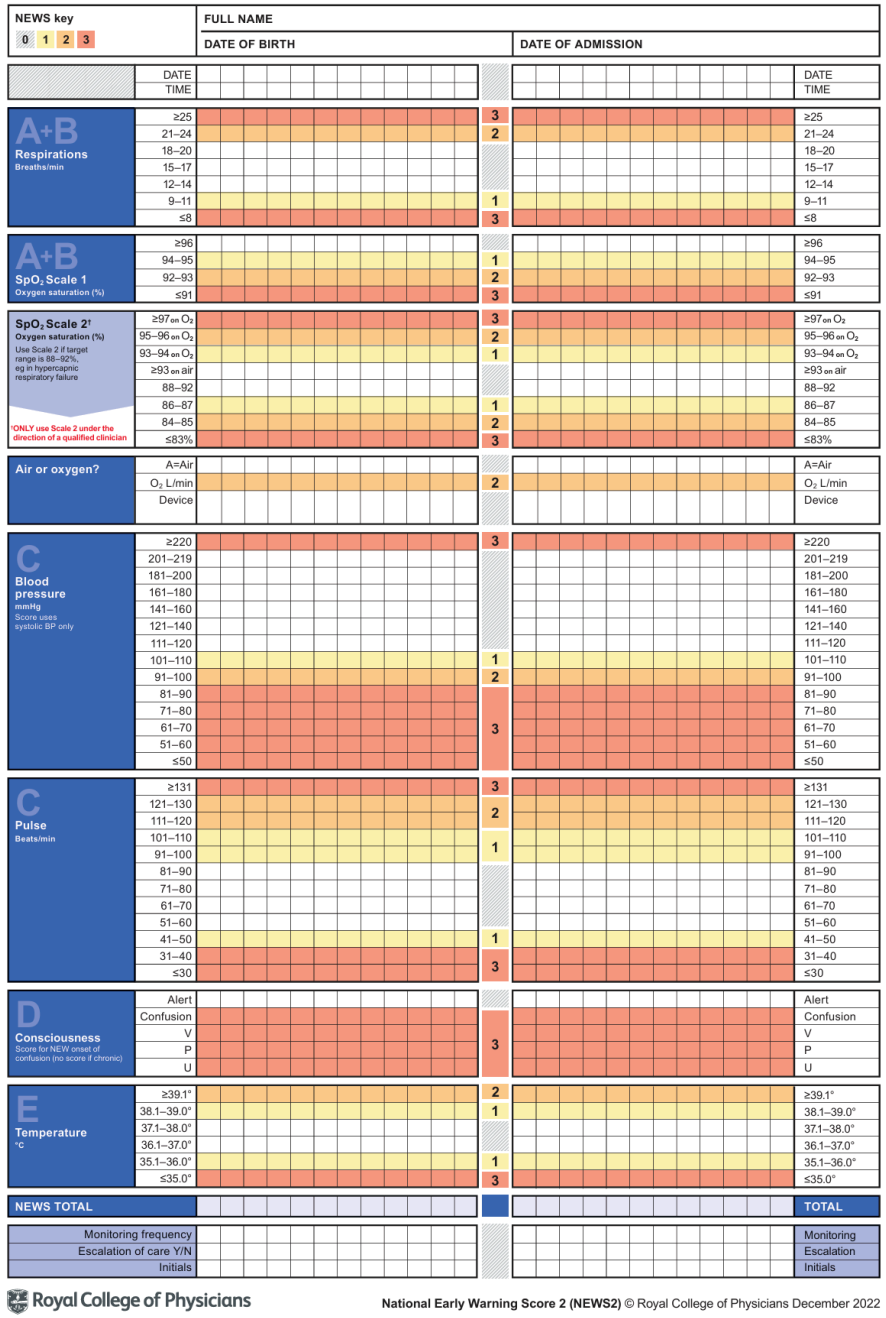
- Situation (the reason for the call),
- Background (relevant clinical information),
- Assessment (the clinician's evaluation of the problem),
- Recommendation (the proposed action).

Benefits of SBAR

SBAR improves clarity in acute care communication and increases provider satisfaction with communication quality. Its use is supported across multiple specialties including anesthesia, perioperative and postoperative medicine, obstetrics, emergency medicine, acute care, pediatrics, and neonatology.

salvatore.sardo@unica.it

Structured clinical monitoring



Royal College of Physicians

National Early Warning Score 2 (NEWS2) © Royal College of Physicians December 2022

National early warning score (NEWS) 2. Accessed November 19, 2025. https://www.rcp.ac.uk/improving-care/resources/national-early-warning-score-news-2/

Chart 1: The NEWS scoring system

Physiological parameter	Score 3	2	1	0	1	2	3
Respiration rate (per minute)	≤8		9–11	12–20		21–24	≥25
SpO_2 Scale 1 (%)	≤91	92–93	94–95	≥96			
SpO_2 Scale 2 (%)	≤83	84–85	86–87	88–92 ≥93 on air	93–94 on oxygen	95–96 on oxygen	≥97 on oxygen
Air or oxygen?		Oxygen		Air			
Systolic blood pressure (mmHg)	≤90	91–100	101–110	111–219			≥220
Pulse (per minute)	≤40		41–50	51–90	91–110	111–130	≥131
Consciousness				Alert			CVPU
Temperature (°C)	≤35.0		35.1–36.0	36.1–38.0	38.1–39.0	≥39.1	

National early warning score (NEWS) 2. Accessed November 19, 2025. https://www.rcp.ac.uk/improving-care/resources/national-early-warning-score-news-2/

Chart 2: NEWS thresholds and triggers

NEW score	Clinical risk	Response
Aggregate score 0–4	Low	Ward-based response
Red score Score of 3 in any individual parameter	Low–medium	Urgent ward-based response*
Aggregate score 5–6	Medium	Key threshold for urgent response*
Aggregate score 7 or more	High	Urgent or emergency response**

* Response by a clinician or team with competence in the assessment and treatment of acutely ill patients and in recognising when the escalation of care to a critical care team is appropriate.

**The response team must also include staff with critical care skills, including airway management.

National early warning score (NEWS) 2. Accessed November 19, 2025. https://www.rcp.ac.uk/improving-care/resources/national-early-warning-score-news-2/

Chart 4: Clinical response to the NEWS trigger thresholds

NEW score	Frequency of monitoring	Clinical response
0	Minimum 12 hourly	• Continue routine NEWS monitoring
Total 1–4	Minimum 4–6 hourly	• Inform registered nurse, who must assess the patient • Registered nurse decides whether increased frequency of monitoring and/or escalation of care is required
3 in single parameter	Minimum 1 hourly	• Registered nurse to inform medical team caring for the patient, who will review and decide whether escalation of care is necessary
Total 5 or more Urgent response threshold	Minimum 1 hourly	• Registered nurse to immediately inform the medical team caring for the patient • Registered nurse to request urgent assessment by a clinician or team with core competencies in the care of acutely ill patients • Provide clinical care in an environment with monitoring facilities
Total 7 or more Emergency response threshold	Continuous monitoring of vital signs	• Registered nurse to immediately inform the medical team caring for the patient – this should be at least at specialist registrar level • Emergency assessment by a team with critical care competencies, including practitioner(s) with advanced airway management skills • Consider transfer of care to a level 2 or 3 clinical care facility, ie higher-dependency unit or ICU • Clinical care in an environment with monitoring facilities

National early warning score (NEWS) 2. Accessed November 19, 2025. https://www.rcp.ac.uk/improving-care/resources/national-early-warning-score-news-2/

Table 2. AUROC for Identifying Intensive Care Unit Transfer or Death Within 24 Hours by Hospital Campus

Hospital campus	Encounters, No.	AUROC (95% CI)					
		MEWS	EDI	RI	NEWS	NEWS2	eCART
All	362 926	0.757 (0.750-0.764)	0.808 (0.802-0.812)	0.828 (0.823-0.834)	0.829 (0.824-0.835)	0.831 (0.826-0.836)	0.895 (0.891-0.900)
A	63 783	0.788 (0.773-0.800)	0.836 (0.824-0.847)	0.852 (0.842-0.863)	0.848 (0.837-0.861)	0.846 (0.837-0.861)	0.903 (0.895-0.912)
B	32 352	0.806 (0.786-0.827)	0.856 (0.838-0.872)	0.881 (0.869-0.897)	0.870 (0.853-0.888)	0.874 (0.862-0.892)	0.931 (0.919-0.945)
C	40 562	0.740 (0.722-0.754)	0.784 (0.771-0.797)	0.796 (0.783-0.807)	0.796 (0.783-0.807)	0.801 (0.789-0.816)	0.881 (0.871-0.891)
D	10 130	0.720 (0.696-0.752)	0.773 (0.741-0.795)	0.792 (0.770-0.813)	0.798 (0.775-0.822)	0.795 (0.769-0.817)	0.871 (0.850-0.888)
E	62 457	0.747 (0.723-0.765)	0.800 (0.783-0.818)	0.813 (0.795-0.829)	0.831 (0.815-0.851)	0.829 (0.813-0.846)	0.885 (0.872-0.898)
F	10 944	0.736 (0.716-0.752)	0.727 (0.710-0.742)	0.746 (0.729-0.763)	0.784 (0.768-0.801)	0.780 (0.763-0.797)	0.866 (0.853-0.882)
G	142 698	0.744 (0.730-0.755)	0.814 (0.806-0.823)	0.834 (0.828-0.843)	0.828 (0.816-0.838)	0.831 (0.822-0.841)	0.894 (0.887-0.902)

Abbreviations: AUROC, area under the receiver operating characteristics curve; eCART, eCARTv5; EDI, Epic Deterioration Index; MEWS, Modified Early Warning Score; NEWS, National Early Warning Score; RI, Rothman Index.

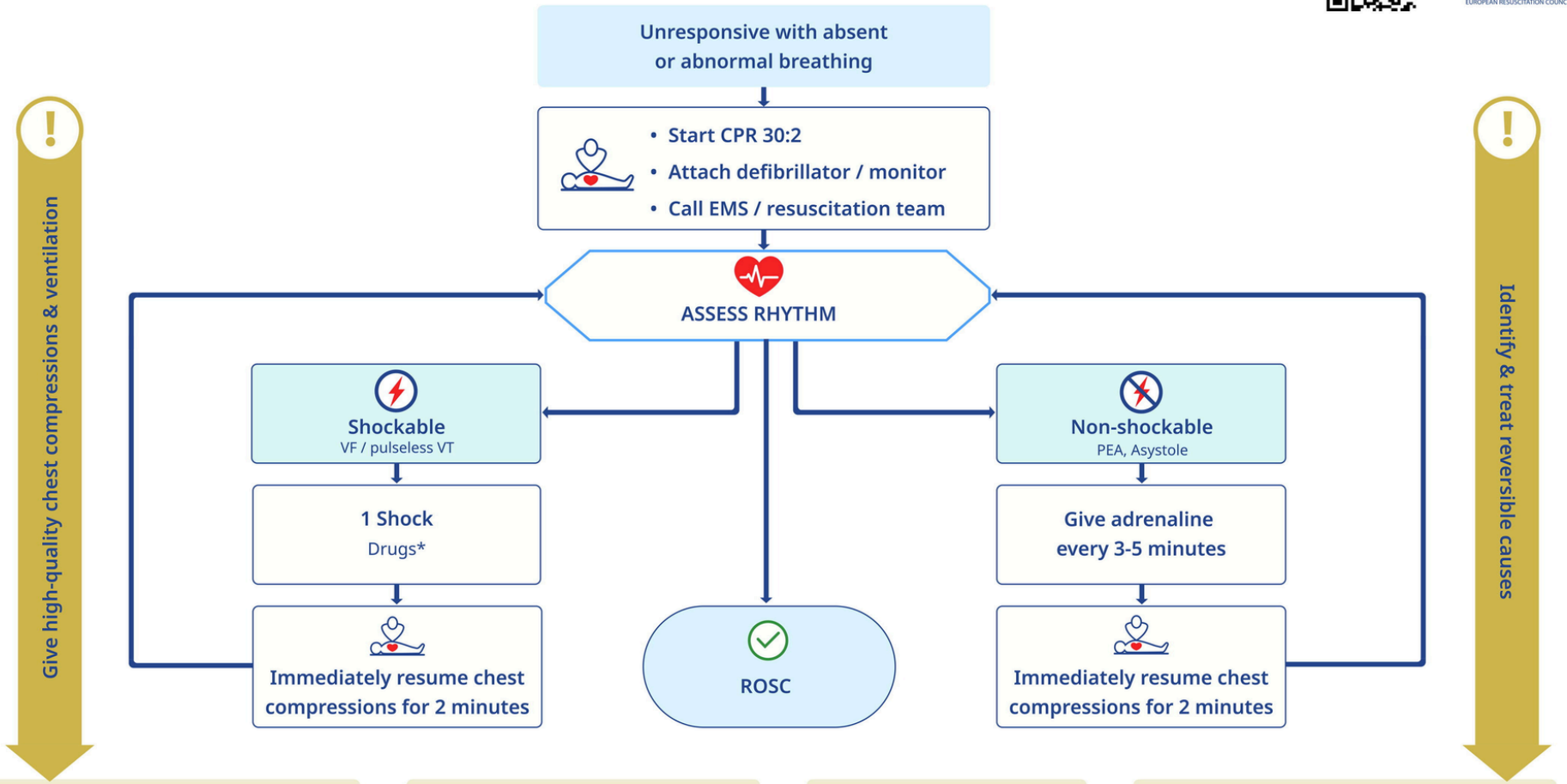
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Overview of the Advanced Life Support Algorithm

GUIDELINES 2025
EUROPEAN RESUSCITATION COUNCIL



- Ensure optimal defibrillator pad placement
- Give oxygen
- Continuous compressions if tracheal tube or supraglottic airway
- Use waveform capnography
- Minimise interruptions to chest compressions
- Early IV access (IO if IV not possible)

* Drugs

- Give 1st adrenaline after 3 shocks, then every 3-5 minutes
- Give amiodarone after 3 shocks

Consider

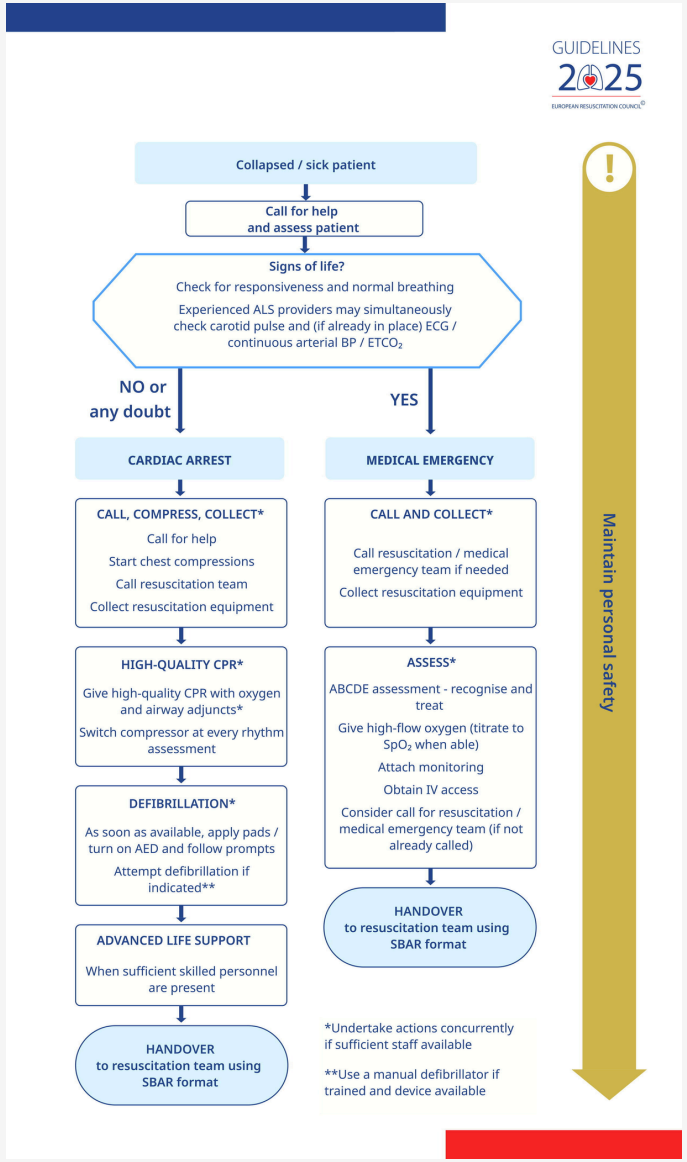
- Changing pads to antero-posterior after 3 shocks
- Mechanical chest compressions to facilitate transfer / treatment
- Ultrasound to identify reversible causes
- Extracorporeal CPR and, or Coronary angiography/percutaneous coronary intervention

Immediately after ROSC

- Use ABCDE approach
- Aim for SpO_2 of 94-98% and normal $PaCO_2$
- Aim SBP > 100 mmHg
- 12 Lead ECG
- Identify and treat cause
- Temperature control

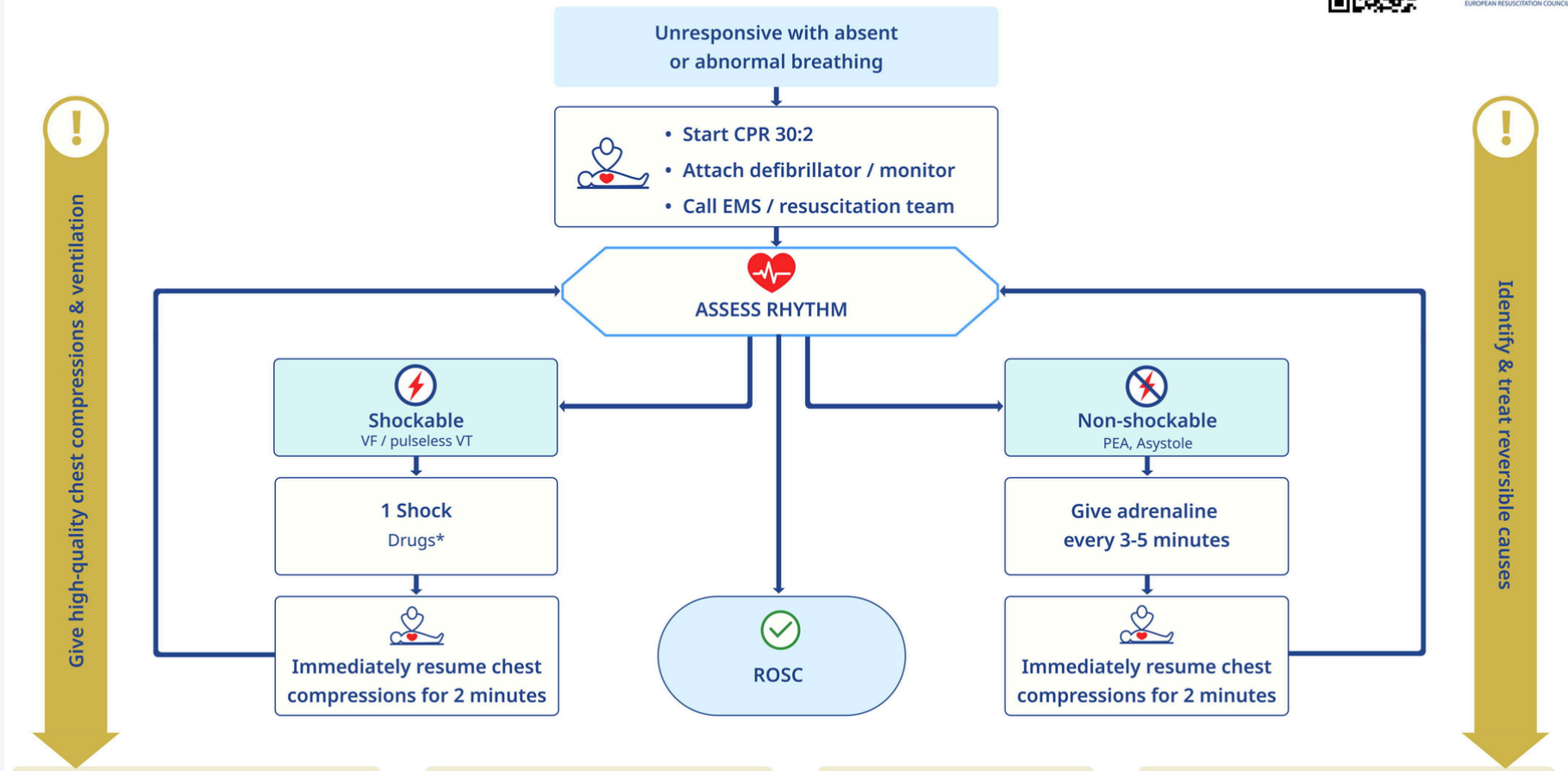
- Hypoxia
- Hypovolaemia
- Hyper-hypokalaemia / metabolic
- Hypothermia, hyperthermia
- Toxins
- Tamponade (cardiac)
- Tension pneumothorax
- Thrombosis (coronary / pulmonary)

Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769



Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769

GUIDELINES 2025
EUROPEAN RESUSCITATION COUNCIL



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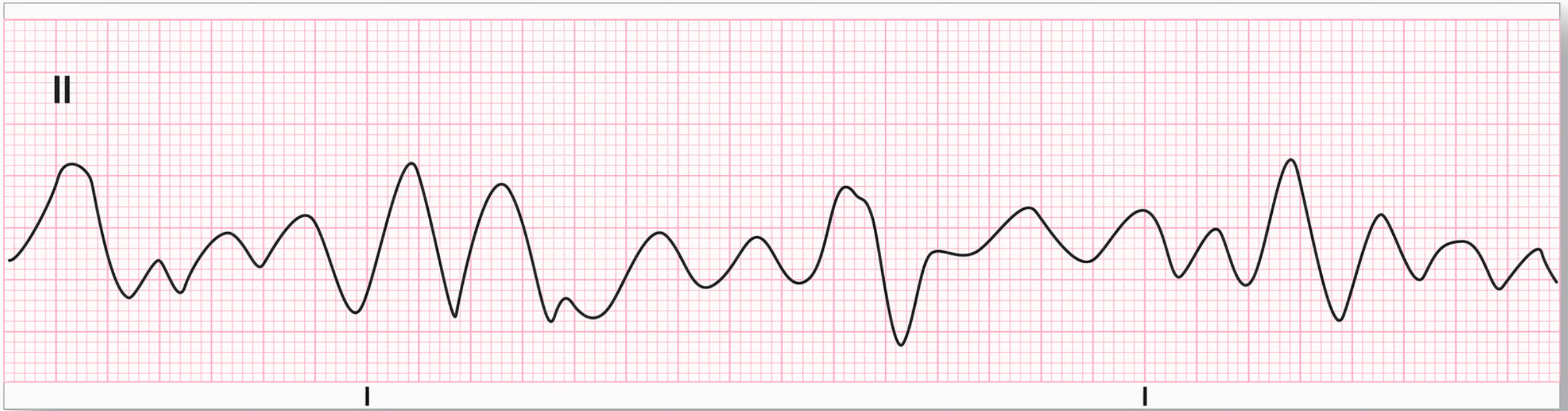
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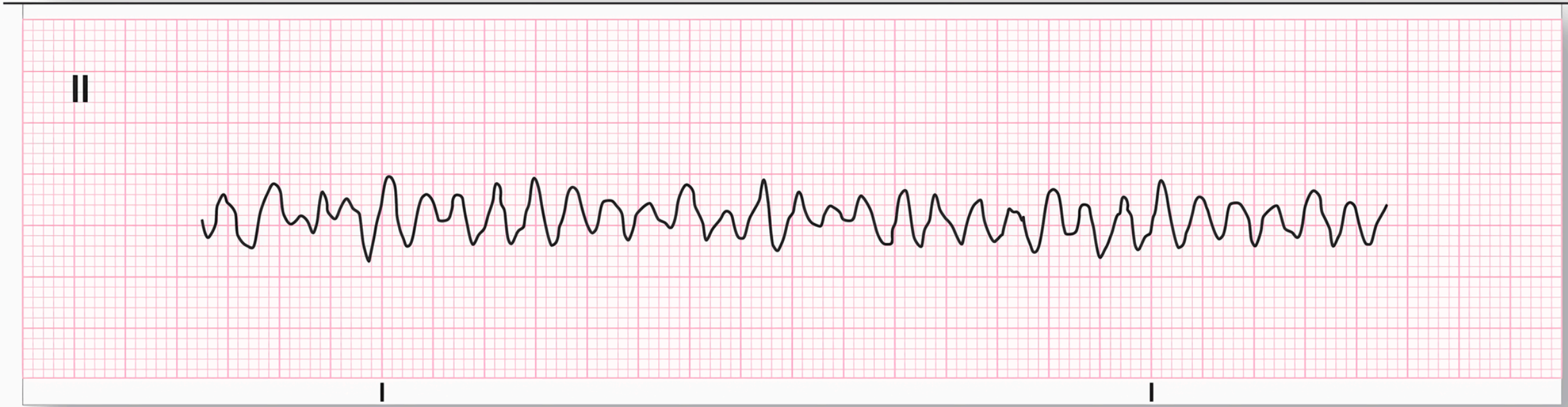
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- Identify and treat cause
- Temperature control

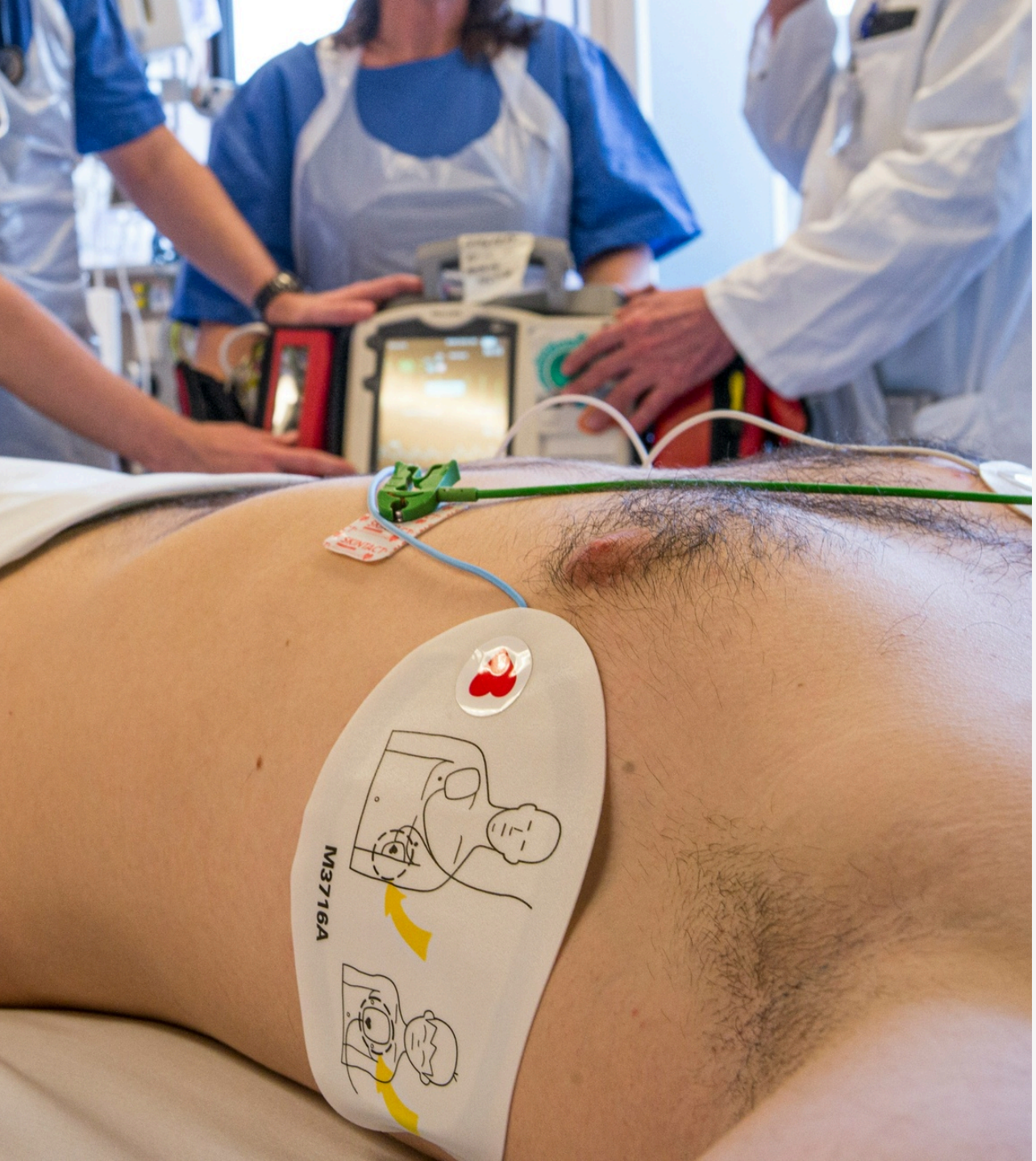
- Hypoxia
- Hypovolaemia
- Hyper-hypokalaemia / metabolic
- Hypothermia, hyperthermia
- Toxins
- Tamponade (cardiac)
- Tension pneumothorax
- Thrombosis (coronary / pulmonary)

Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769





Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.



High quality CPR and defibrillation.

Continue CPR while the defibrillator is brought to the bedside and the pads are applied. High quality chest compressions increase the probability of successful defibrillation.

Early shock delivery.

Give a shock as soon as it is indicated.

Deliver shocks with the shortest possible pause in compressions.

Continue compressions during charging, aim for an interruption of less than five seconds, then resume compressions immediately.

Defibrillation.

Attempt immediate defibrillation for ventricular fibrillation of any amplitude, including fine VF. Post shock actions.

Resume chest compressions immediately after the shock. If there are clear signs of return of spontaneous circulation such as regained consciousness, purposeful movement, an arterial waveform or a sudden rise in end tidal carbon dioxide, pause compressions for rhythm assessment and, if appropriate, a pulse check.

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Safe and effective defibrillation.

Reduce fire risk by removing oxygen masks, nasal cannulae or bag mask devices and placing them at least one metre from the chest.

When a mechanical ventilator is in use, direct any oxygen exhaust away from the chest.

Keep a self inflating bag or the ventilator circuit attached to a supraglottic airway or tracheal tube. Defibrillator charging.

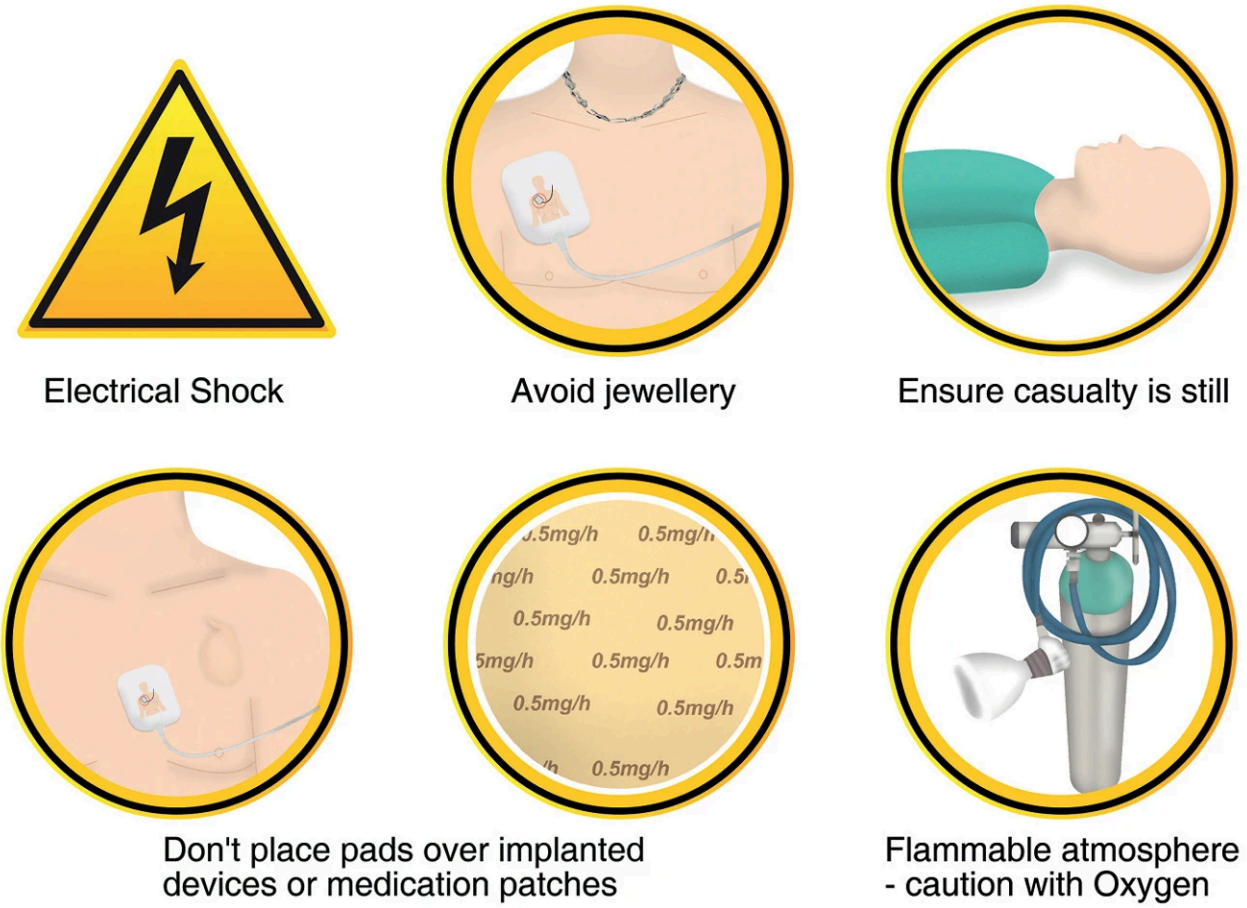
Charging the defibrillator in anticipation of each rhythm check can reduce hands off time. This approach is acceptable if it does not prolong the peri shock pause. Mechanical compressions.

A shock from a manual defibrillator can be delivered safely without interrupting mechanical chest compressions.

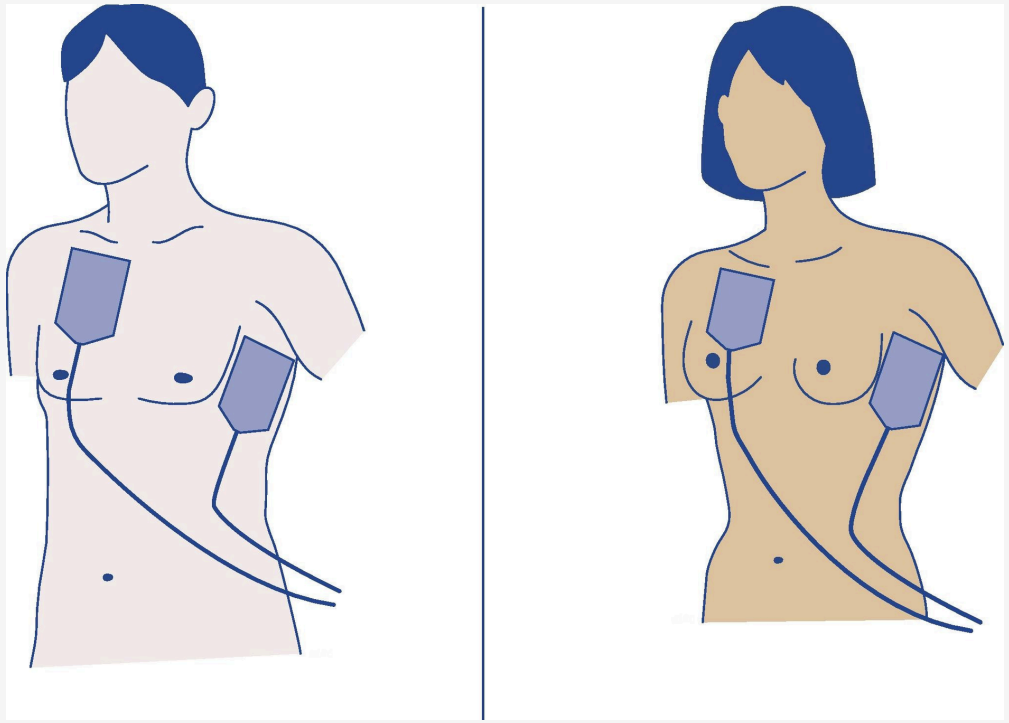
Manual compressions.

Do not defibrillate during manual chest compressions. The practice is unsafe for rescuers even when wearing clinical gloves.

Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769



Hammett E. How to save a life with CPR and an AED. BDJ Team. 2019;6(2):19037. doi:10.1038/bdjteam.2019.37



Defibrillation pads and paddles.

Current evidence does not identify a specific pad or paddle size that offers clear benefit for adult external defibrillation.

Pads versus paddles.

Pads are preferred when available. They support continuous monitoring, allow the operator to stand clear, and reduce pauses before and after shocks because they enable hands free use. Good pad contact reduces the risk of arcing and fire.

Paddles.

When using paddles, apply firm pressure to both paddles to improve skin contact, reduce transthoracic impedance and reduce the risk of arcing.

Pad position.

The antero lateral position is the first choice.

The apical pad must be placed below the armpit in the mid axillary line.

Consider an antero posterior position for vector change after three failed shocks in refractory shockable rhythms. Place the anterior pad to the left of the sternum, avoiding breast tissue. Place the posterior pad at the same level, centred just medial to the left scapula.

Special situations.
For patients with an implantable pacemaker or defibrillator, place the pads more than eight centimetres from the device or use an alternative position.
Use an alternative pad position when the patient is prone, such as the bi axillary position, or in refractory shockable rhythms.

Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769

Energy levels and number of shocks.

Use single shocks followed by a two minute cycle of chest compressions.

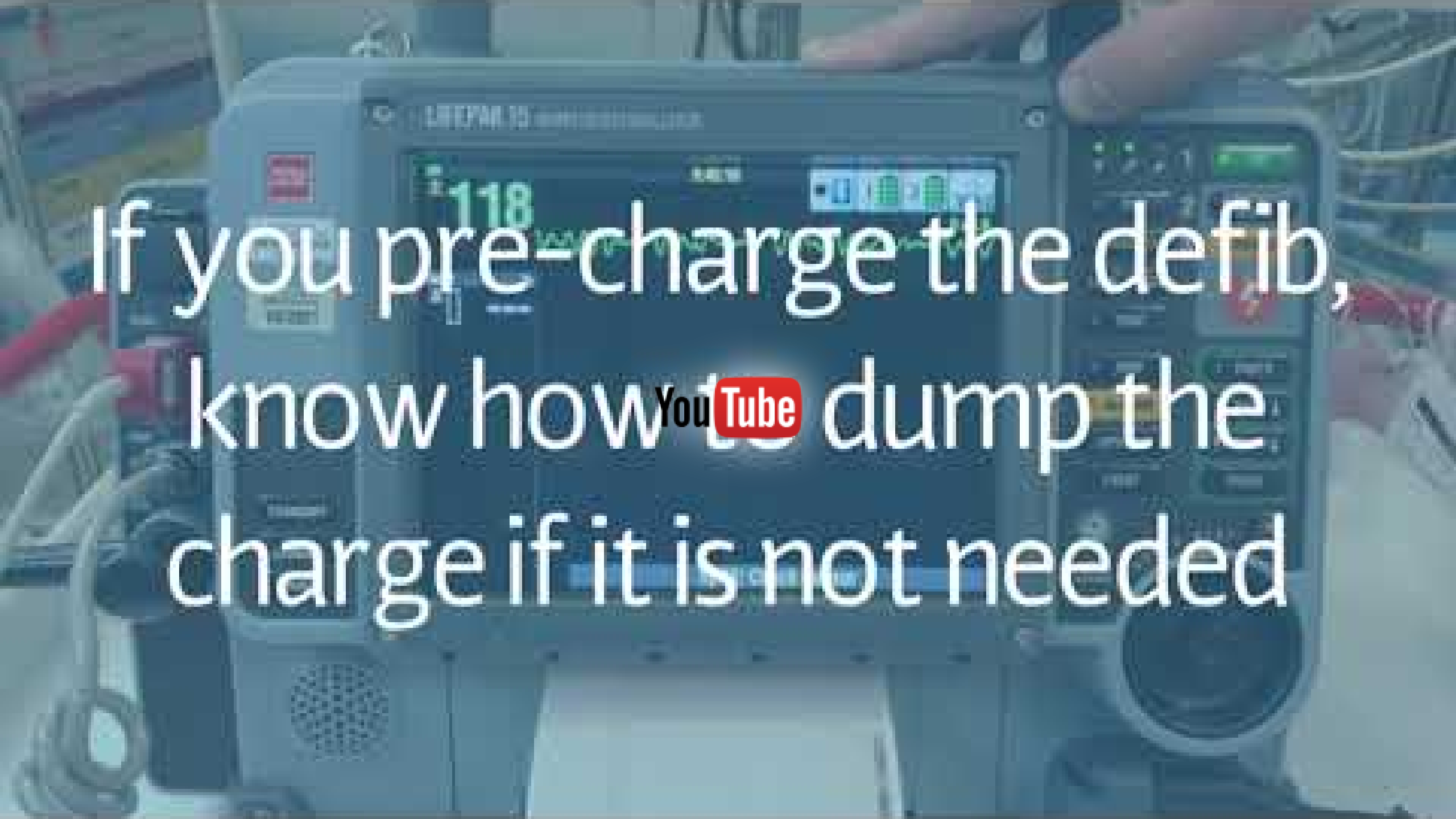
Use up to three stacked shocks only when VF or pulseless VT occurs during a witnessed, monitored arrest with a defibrillator already present, such as during cardiac catheterisation or in a high dependency setting. These count as the initial shock cycle for drug timing.

Energy settings.

For biphasic waveforms, excluding pulsed biphasic, use at least 150 joules for the first shock.
For pulsed biphasic, use 130 to 150 joules for the first shock.
If the first shock fails and the device can deliver higher energy, increase the energy for subsequent shocks.
If the rescuer does not know the recommended settings, use the highest adult setting for all shocks.
Use standard energy levels in obese patients.

Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769




If you pre-charge the defib,
know how to dump the
charge if it is not needed

Refractory ventricular fibrillation.

Consider increasing the shock energy after an unsuccessful shock.

Refractory VF is defined as continuous ventricular fibrillation after three consecutive shocks. After confirming correct antero lateral pad placement, consider changing the defibrillation vector by switching to an alternative pad position such as antero posterior.

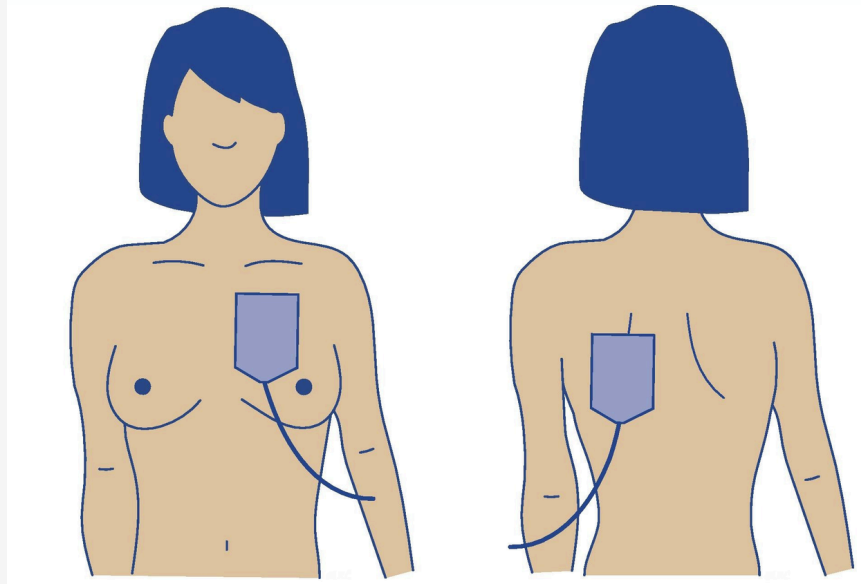
After the third failed shock, prepare a new set of pads for placement at the next rhythm check.

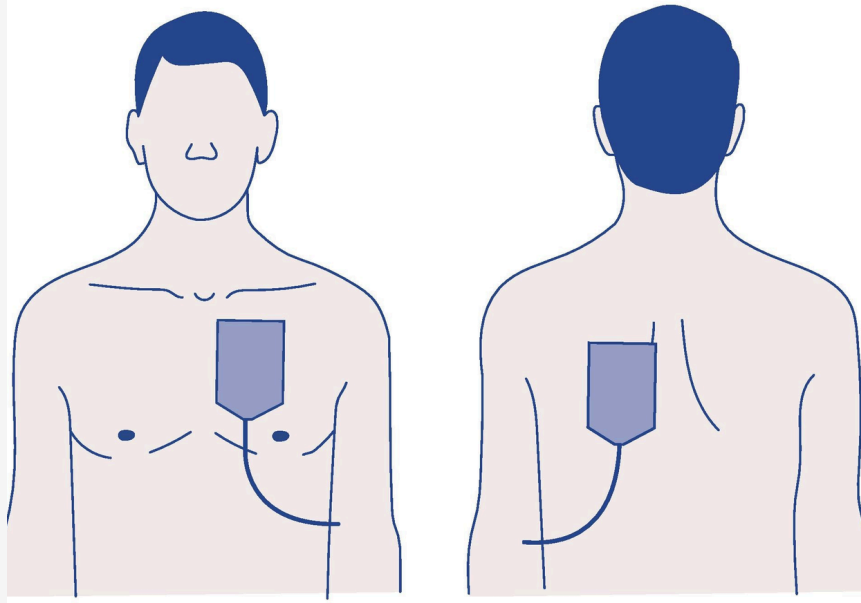
Shave the areas needed for pad placement if this reduces transthoracic impedance.

Dual sequential defibrillation uses both antero lateral and antero posterior pad positions with two defibrillators discharged in rapid sequence.

The method has been proposed for refractory shockable rhythms, but the evidence is limited and the technique is operationally complex.

Routine use is not recommended by the ERC.





Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769

Patients with actively discharging implantable cardioverter defibrillators.

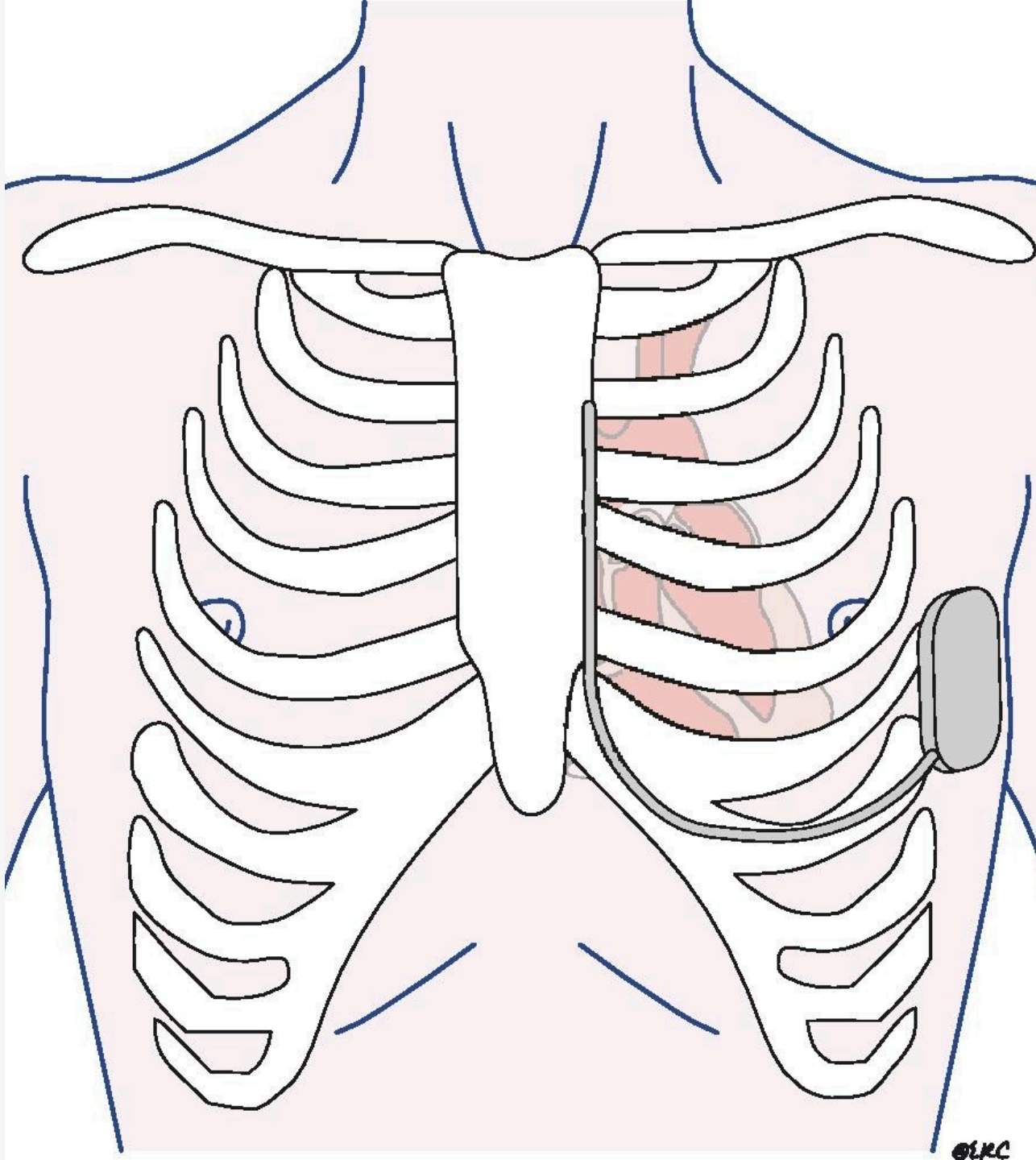
Rescuers may feel a noticeable shock in the arms if an ICD delivers a discharge during external chest compressions, even when clinical gloves are used.

f an ICD does not terminate a shockable rhythm, give standard external shocks.

Place defibrillation pads or paddles more than eight centimetres from the device.

If the ICD is detecting arrhythmias incorrectly and delivering inappropriate shocks, place a magnet over the device to stop further shocks. This does not disable pacing if pacing is active.

Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769





Airway and ventilation.

- Begin CPR with basic airway techniques and progress stepwise according to rescuer skill until ventilation is effective. Provide the highest feasible inspired oxygen concentration during CPR.
- Start ventilation as early as possible and use an appropriate rate and tidal volume to avoid hypoventilation and hyperventilation.
- Optimise bag mask ventilation by ensuring a good mask seal and maintaining airway patency.
- Use a two person technique if needed.
- Give each inspiratory breath over one second and confirm visible chest rise. When a supraglottic airway is used, prefer an i gel over a laryngeal tube.

Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769

Advanced airway management.

- Attempt tracheal intubation only if the rescuer has a high success rate and can use continuous waveform capnography.
- **Expert consensus defines a high success rate as more than 95 percent within two attempts.**
- Aim to limit the interruption in chest compressions for intubation to less than five seconds.
- Use direct or video laryngoscopy according to local protocols and rescuer experience.
- Prefer video laryngoscopy when available.
- Use a sustained end tidal carbon dioxide trace on waveform capnography to exclude oesophageal intubation.
- After placing a tracheal tube or supraglottic airway, **ventilate at ten breaths per minute** and **continue chest compressions without pausing.**
- When using a supraglottic airway, if gas leakage prevents effective ventilation, pause compressions and ventilate using a compression ventilation ratio of thirty to two.
- If mechanical ventilation is used, select a volume controlled mode during compressions. Set a tidal volume of six to eight millilitres per kilogram of predicted body weight or use a volume that produces visible chest rise. Use the highest inspired oxygen concentration, a respiratory rate of ten breaths per minute, an inspiratory time of one to two seconds, positive end expiratory pressure of zero to five centimetres of water, **a peak pressure alarm set to sixty to seventy centimetres of water and turn off the flow trigger.** Switch to manual ventilation if mechanical ventilation is ineffective. If standard airway techniques fail, appropriately trained rescuers should perform a surgical cricothyroidotomy to provide oxygenation and ventilation.

Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769



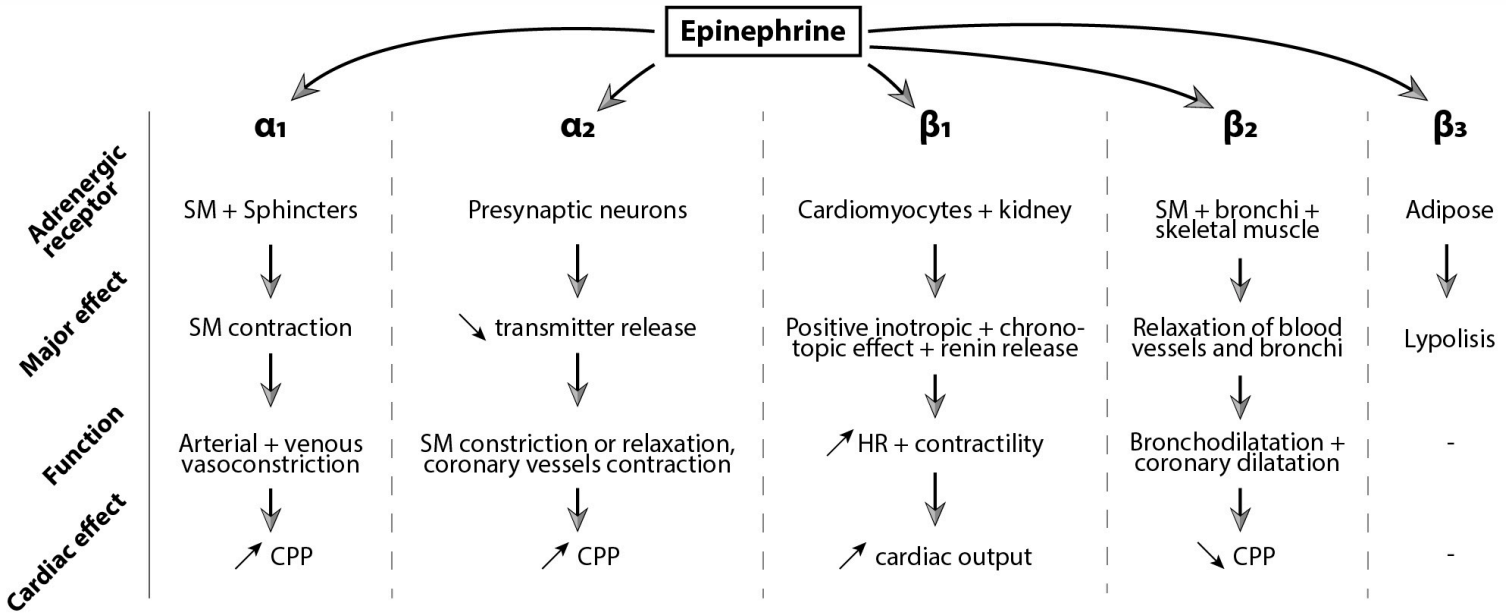


Pharmacology of the Advanced Life Support Algorithm

Adrenaline

- Adrenaline has been part of international resuscitation guidelines since the 1960s.
- Its α1-mediated vasoconstriction increases aortic diastolic pressure and improves coronary and cerebral perfusion pressure.
- Higher coronary perfusion pressure is associated with increased likelihood of return of spontaneous circulation (ROSC).
- Despite physiological benefits during cardiopulmonary resuscitation, no human data show improved long-term neurological outcome with adrenaline.
- Observational studies report worse neurological outcomes in patients who receive adrenaline, but these findings are confounded by indication bias.
- Global coronary and cerebral flow rises with adrenaline, but microcirculatory flow may fall.
- After ROSC, high circulating adrenaline can produce tachycardia, increased oxygen demand, and ventricular arrhythmias.

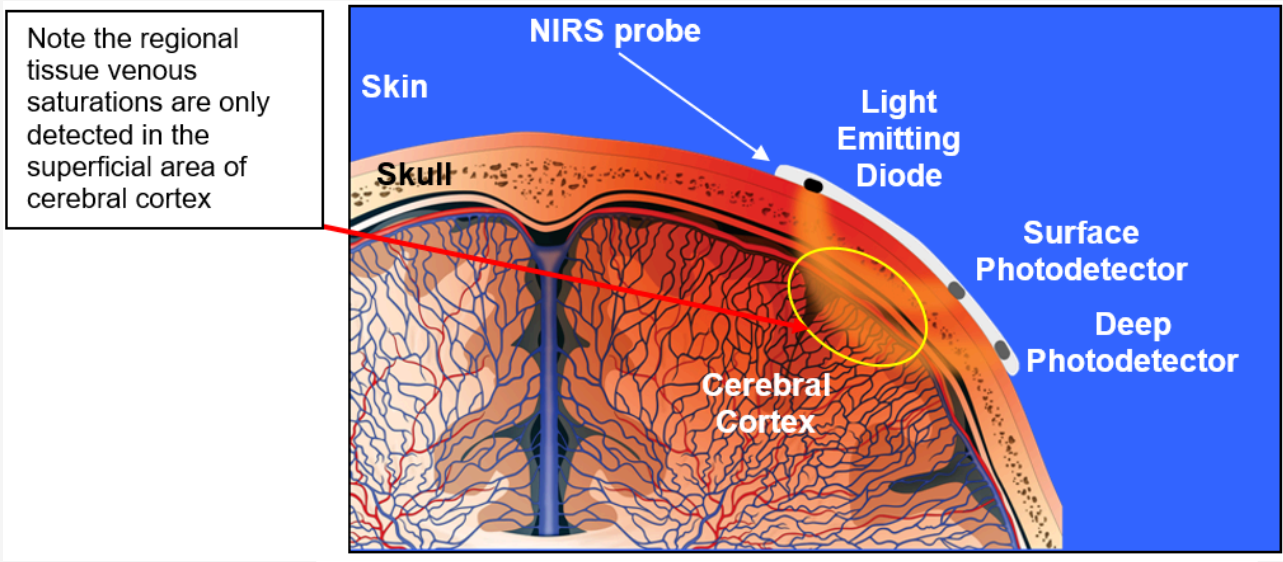
Gough CJR, Nolan JP. The role of adrenaline in cardiopulmonary resuscitation. Crit Care. 2018;22(1):139. doi:10.1186/s13054-018-2058-1



Jaeger D, Marquez AM, Kosmopoulos M, et al. A narrative review of drug therapy in adult and pediatric cardiac arrest. *Rev Cardiovasc Med*. 2023;24(6):163. doi:10.31083/j.rcm2406163

Human physiologic effects of adrenaline during cardiac arrest

- In a study of 100 patients monitored with invasive aortic and right-atrial lines during CPR, higher coronary perfusion pressure (CPP) strongly predicted return of spontaneous circulation (ROSC). No patient with CPP below 15 mmHg achieved ROSC.
- A near-infrared spectroscopy (NIRS) study of 36 in-hospital arrests evaluated 89 adrenaline doses. Mean regional cerebral oxygenation (rSO_2) rose slightly after adrenaline, but rSO_2 was already increasing before dosing, and adrenaline did not significantly change the trend.
- The sensitivity of NIRS for detecting adrenaline-related changes in cerebral oxygenation remains uncertain.



https://www.clinicalguidelines.scot.nhs.uk/ggc-paediatric-guidelines/ggc-paediatric-guidelines/anaesthetics/near-infrared-spectroscopy-nirs-guideline-1072/

- Adrenaline also increased transitions from ROSC to ventricular tachycardia or ventricular fibrillation, and from ROSC back to PEA, indicating greater electrical instability.
- Overall, adrenaline raises CPP and improves the chance of achieving ROSC, but it also increases post-ROSC arrhythmias and rhythm instability.

Gough CJR, Nolan JP. The role of adrenaline in cardiopulmonary resuscitation. Crit Care. 2018;22(1):139. doi:10.1186/s13054-018-2058-1

Adrenaline

- Randomised pig studies show that adrenaline increases arterial pressure and cerebral perfusion pressure, but often reduces peripheral and cerebral oxygenation.
- A 36-pig study comparing 20 or 30 µg/kg adrenaline with placebo showed higher aortic and cerebral perfusion pressures, but lower SpO_2 and cerebral tissue oximetry in the adrenaline groups.
- A 6-pig study found that 40 µg/kg adrenaline increased aortic, coronary, and cerebral perfusion pressures, yet decreased coronary blood flow.
- Microcirculatory imaging studies consistently show impaired microvascular blood flow after adrenaline compared with vasopressin or placebo.
- Cerebral oxygen tension falls and carbon dioxide tension rises after adrenaline in multiple models, despite higher aortic pressure.
- In piglets, adrenaline combined with vasopressin improved blood pressure during CPR, but post-resuscitation cerebral blood flow fell and neuronal injury increased.
- A dog study showed higher myocardial blood flow with adrenaline but marked reductions in renal flow and increased myocardial lactate.
- Bolus adrenaline causes transient surges in cerebral blood flow, whereas infusions produce more stable flow profiles.
- Overall, adrenaline reliably increases mean aortic pressure, but its effects on coronary, cerebral, and microcirculatory blood flow are variable, and microvascular perfusion is often impaired.





Gough CJR, Nolan JP. The role of adrenaline in cardiopulmonary resuscitation. Crit Care. 2018;22(1):139. doi:10.1186/s13054-018-2058-1

Problem
Is the problem a priority?

JUDGEMENT	RESEARCH EVIDENCE	ADDITIONAL CONSIDERATIONS
○ No ○ Probably no ○ Probably yes ● Yes ○ Varies ○ Don't know	Cardiac arrest, both in the out-of-hospital and in-hospital setting, is relatively common and carries a very high morbidity and mortality.	

Desirable Effects
How substantial are the desirable anticipated effects?

JUDGEMENT	RESEARCH EVIDENCE	ADDITIONAL CONSIDERATIONS
○ Trivial ○ Small ● Moderate (survival) ○ Large ○ Varies ○ Don't know	For epinephrine compared with placebo, improvements in return of spontaneous circulation and survival at hospital admission are substantial. The improvement in survival (hospital discharge, 30-days, 3 months, 6 months, and 12 months) is moderate. Whether there is improvement in survival with favorable neurological outcome remains uncertain. The desirable effects appear more pronounced in non-shockable compared with shockable rhythms.	Additional considerations that were raised included the impact of increased return of spontaneous circulation on organ donation.

Epinephrine compared to placebo – Any rhythm (Jacobs 2011, Perkins 2018)

Outcome	RR (95% CI)	RD (95% CI)
Return of spontaneous circulation	3.09 (2.82 to 3.39)	243 more per 1000 (from 211 more to 277 more)
Survival at hospital discharge	1.44 (1.11 to 1.86)	10 more per 1000 (from 2 more to 19 more)
Favorable neurological outcome at hospital discharge	1.21 (0.90 to 1.62)	4 more per 1,000 (from 2 fewer to 12 more)

Epinephrine compared to placebo – Shockable rhythms (Jacobs 2011, Perkins 2018)

Outcome	RR (95% CI)	RD (95% CI)
Return of spontaneous circulation	1.68 (1.48 to 1.92)	185 more per 1,000 (from 130 more to 250 more)
Survival at hospital discharge	1.23 (0.94 to 1.62)	22 more per 1,000 (from 6 fewer to 60 more)
Favorable neurological outcome at hospital discharge	1.05 (0.76 to 1.45)	4 more per 1,000 (from 21 fewer to 39 more)

Epinephrine compared to placebo – Non-shockable rhythms (Jacobs 2011, Perkins 2018)

Outcome	RR (95% CI)	RD (95% CI)
Return of spontaneous circulation	4.45 (3.91 to 5.08)	254 more per 1,000 (from 214 more to 301 more)
Survival at hospital discharge	2.56 (1.37 to 4.80)	7 more per 1,000 (from 2 more to 16 more)
Favorable neurological outcome at hospital discharge	1.80 (0.80 to 4.07)	2 more per 1,000 (from 1 fewer to 9 more)

Undesirable Effects
How substantial are the undesirable anticipated effects?

JUDGEMENT	RESEARCH EVIDENCE	ADDITIONAL CONSIDERATIONS
○ Trivial ● Small ○ Moderate ○ Large ○ Varies ○ Don't know	There is no evidence in clinical trials that epinephrine specifically contributes to cerebral injury beyond its effect of increasing overall survival, including in patients who may have sustained neurological damage.	Epinephrine likely increases the number of survivors with both favorable and unfavorable neurological outcomes, as observed in the PARAMEDIC2 trial.[1,2] This apparent increase in survivors with unfavorable neurological outcome should not be interpreted as epinephrine directly causing unfavorable neurological outcomes, but rather reflects its efficacy in restoring circulation in patients who may already have sustained significant cerebral injury due to prolonged cardiac arrest.

Epinephrine compared to placebo – Any rhythm (Jacobs 2011, Perkins 2018)

Outcome	RR (95% CI)	RD (95% CI)
Favorable neurological outcome at hospital discharge	1.21 (0.90 to 1.62)	4 more per 1,000 (from 2 fewer to 12 more)
Favorable neurologic outcome at 3 months *	1.30 (0.94-1.80)	5 more per 1,000 (from 1 fewer to 13 more)
Favorable neurologic outcome at 6 months *	1.34 (0.96 to 1.88)	5 more per 1,000 (from 1 fewer to 13 more)

* Perkins 2018 only

Certainty of evidence
What is the overall certainty of the evidence of effects?

JUDGEMENT	RESEARCH EVIDENCE	ADDITIONAL CONSIDERATIONS
○ Very low ○ Low ● Moderate (survival) ○ High ○ No included studies	The certainty of evidence varies by outcome. There is high certainty of evidence for return of spontaneous circulation and survival at hospital admission; moderate certainty of evidence for survival at hospital discharge, 3 months, and 6 months; and low to moderate certainty of evidence for favorable neurological outcome at hospital discharge, 3 months, and 6 months.	The variation in the certainty of evidence by outcome was largely due to the event rate for each outcome. There was higher statistical power to evaluate differences in return of spontaneous circulation (a more common event) than survival with favorable neurological outcome (a much less common event). The certainty of evidence for favorable neurological outcome at 3 months and 6 months was also lessened by a loss to follow-up.

Comparison (OHCA)	Outcome		
	Return of spontaneous circulation	**Survival at hospital discharge**	**Favorable neurological outcome at hospital discharge**
Epinephrine compared to placebo – Any rhythm	⊕⊕⊕⊕ High	⊕⊕⊕◯ Moderate	⊕⊕⊕◯ Moderate
Epinephrine compared to placebo – Shockable rhythms	⊕⊕⊕◯ Moderate	⊕⊕⊕◯ Moderate	⊕⊕◯◯ Low
Epinephrine compared to placebo – Non-shockable rhythms	⊕⊕⊕⊕ High	⊕⊕⊕◯ Moderate	⊕⊕◯◯ Low

Drug class.
Class III antiarrhythmic agent.

Primary electrophysiological effect.

- **Blocks potassium rectifier currents** responsible for phase 3 repolarisation.
- **Prolongs action potential duration and effective refractory period.**
- **Reduces myocardial excitability and interrupts re entry and ectopic tachyarrhythmias.**

ECG effects include QTc prolongation and widening of the QRS complex.

Additional mechanisms.

- Beta adrenergic receptor blockade, mainly beta 1.
- Sodium channel blockade.
- Calcium channel blockade.

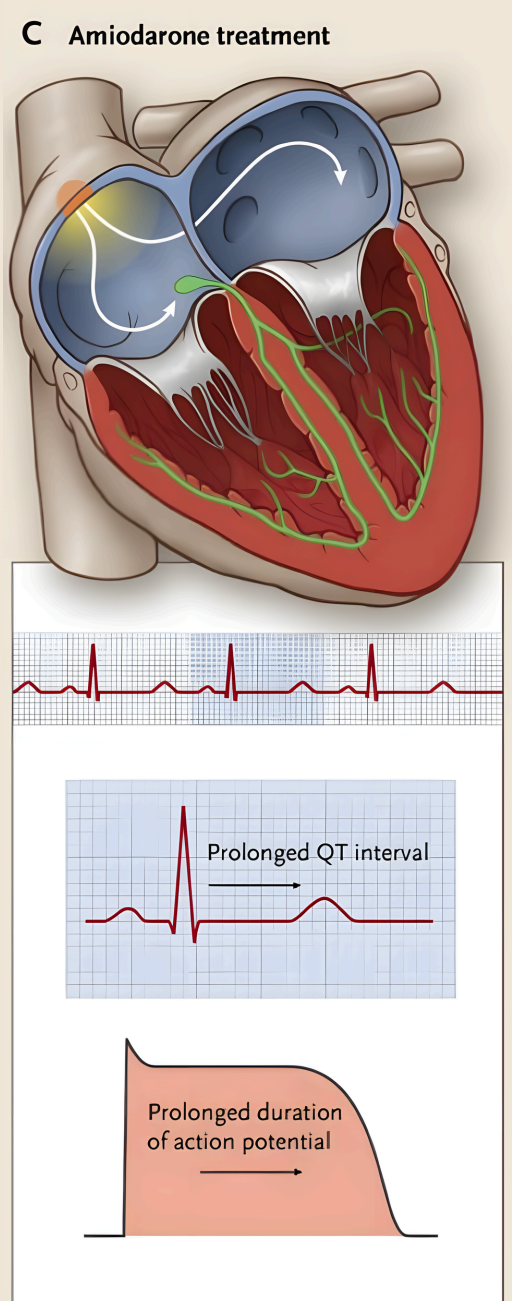
Functional cardiac effects.

- Reduced sinoatrial node automaticity.
- Slowed atrioventricular node conduction.
- Suppression of ectopic pacemaker activity.

Relevant adverse electrophysiological effects.

- Bradycardia.
- Hypotension.
- Risk of QT prolongation and torsades de pointes.

Florek JB, Lucas A, Girzadas D. Amiodarone. In: *StatPearls*. StatPearls Publishing; 2025. Accessed December 16, 2025.
http://www.ncbi.nlm.nih.gov/books/NBK482154/



Zimetbaum P. Amiodarone for atrial fibrillation. *N Engl J Med*. 2007;356(9):935-941.
doi:10.1056/NEJMct065916

ABCDE assessment of the critically patient

Approach to the critically ill patient

- Apply a structured ABCDE assessment to identify and treat immediate threats.
- Perform a full initial assessment and repeat assessments at regular intervals.
- Address life threatening problems before progressing to the next step.
- Evaluate the response to each intervention.
- Identify when additional support is required and call for help early.
- Use the full team so that assessment, monitoring, and access can occur in parallel.
- Communicate clearly using structured formats such as SBAR or RSVP.
- Aim to stabilise the patient and achieve early clinical improvement to allow time for definitive diagnosis and treatment.
- Allow time for interventions to take effect before reassessing.

The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

Resuscitation Council UK

- First steps in initial assessment • Ensure your own safety and use protective equipment such as apron and gloves when needed.
  • Observe the patient briefly to determine whether they appear unwell.
  • If awake, ask how they feel. If collapsed or unconscious, shake gently and ask if they are alright. Normal verbal response indicates an open airway, adequate breathing, and cerebral perfusion. Short sentences suggest respiratory difficulty. Lack of response signals critical illness.
  • Perform a rapid look, listen, and feel assessment, taking about thirty seconds. This helps identify patients who require urgent assistance. Ask a colleague to call for help.
  • If unresponsive and not breathing normally, start CPR. Occasional gasps do not indicate normal breathing. If trained, check for a pulse, but if unsure, begin CPR.
  • Attach monitoring early. Use pulse oximetry, ECG, and non invasive blood pressure measurement.
  • Insert an intravenous cannula promptly and collect blood samples for investigations.

The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

Resuscitation Council UK

A - Airway

Airway obstruction is an emergency.

Get expert help immediately.

Untreated, airway obstruction causes hypoxia and risks damage to the brain, kidneys and heart, cardiac arrest, and death.

The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

Resuscitation Council UK

Look for the signs of airway obstruction

Airway obstruction causes paradoxical chest and abdominal movements (‘see-saw’ respirations) and the use of the accessory muscles of respiration.

Central cyanosis is a late sign of airway obstruction.

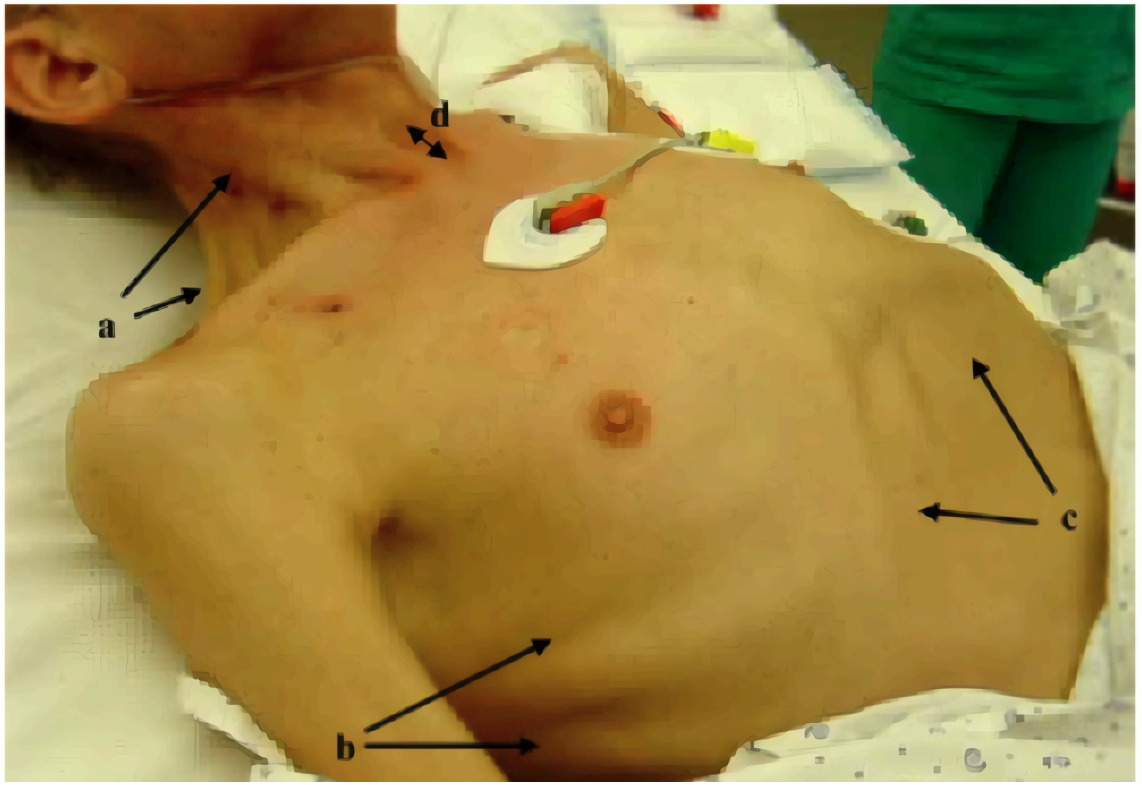
In complete airway obstruction, there are no breath sounds at the mouth or nose.

In partial obstruction, air entry is diminished and often noisy.

In the critically ill patient, depressed consciousness often leads to airway obstruction.

The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

Resuscitation Council UK



Jossen-Racine E, Wittebole X, Roeseler J, Portuesi V, Michotte JB. Rôle du kinésithérapeute respiratoire dans un service d’accueil des urgences pour adultes. *Réanimation*. 2011;20(6):508-515. doi:10.1007/s13546-011-0312-5

a. Contraction of the neck muscles, which serve as accessory inspiratory muscles.

b. Inspiratory indrawing of the intercostal spaces.

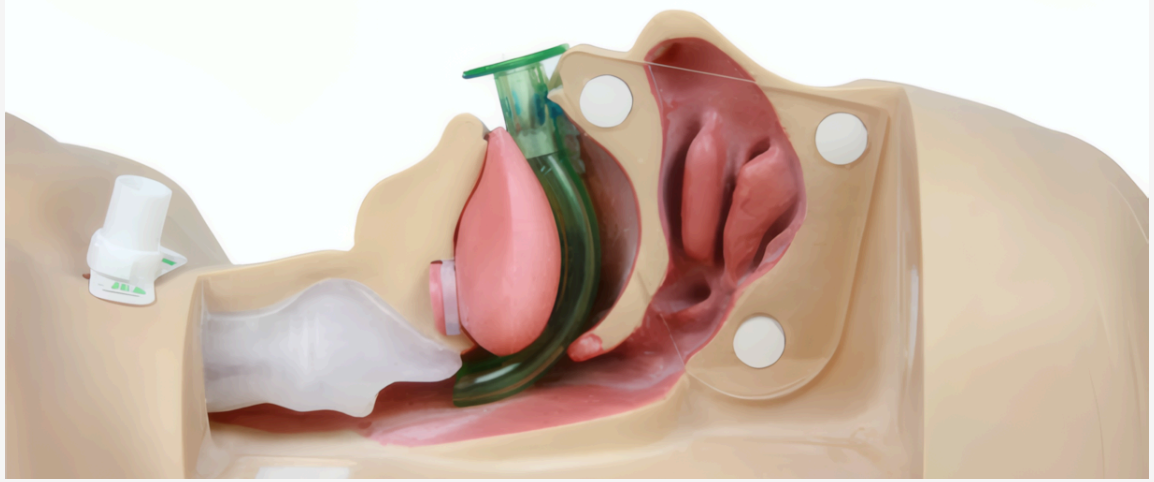
c. Inward movement of the abdominal wall during inspiration, known as paradoxical breathing.

d. Campbell’s sign, defined as a reduction in the distance between the cricoid cartilage and the sternal manubrium during inspiration.


YouTube



Smyth MA, Goor S van, Hansen CM, et al. European resuscitation council guidelines 2025 adult basic life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110771





Treat airway obstruction as a medical emergency

- Obtain expert help immediately.
- Untreated, airway obstruction causes hypoxaemia (low PaO2) with the risk of hypoxic injury to the brain, kidneys and heart, cardiac arrest, and even death.
- In most cases, only simple methods of airway clearance are required (e.g. airway opening manoeuvres, airways suction, insertion of an oropharyngeal or nasopharyngeal airway).
- Tracheal intubation may be required when these fail.

The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

Resuscitation Council UK

Give oxygen at high concentration

- Provide high-concentration oxygen using a mask with oxygen reservoir.
- Ensure that the oxygen flow is sufficient (usually 15 L min-1) to prevent collapse of the reservoir during inspiration.
- If the patient’s trachea is intubated, give high concentration oxygen with a self-inflating bag.
- In acute respiratory failure, aim to maintain an oxygen saturation of 94–98%. In patients at risk of hypercapnic respiratory failure (see below) aim for an oxygen saturation of 88–92%.

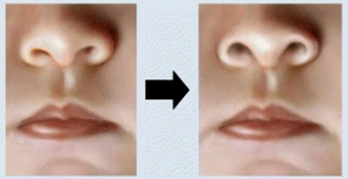
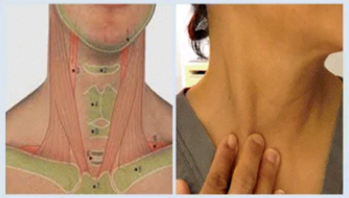


The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

Resuscitation Council UK

B - Breathing

- Identify and treat immediately life threatening causes such as acute severe asthma, pulmonary oedema, tension pneumothorax, and massive haemothorax.
- Look, listen, and feel for signs of respiratory distress, including sweating, central cyanosis, accessory muscle use, and abdominal breathing.
- Measure respiratory rate. Normal rate is 12 to 20 per minute. A rate above 25 per minute or a rising rate signals deterioration.
- Assess depth, rhythm, and symmetry of chest expansion.
- Note chest deformities, jugular venous distension, presence and patency of chest drains, and abdominal distension.
- Record inspired oxygen concentration and the SpO2. Normal SpO2 does not exclude severe hypercapnia.
- Listen near the patient's mouth. Rattling suggests secretions. Stridor or wheeze indicates partial obstruction.

ELEMENT	METHOD	POINTS
 Respiratory Rate	By Counting (bpm)	≤ 20 = 1 21-25 = 2 26-30 = 3 > 30 = 4
 Nasal Flaring (inspiration)	By Observation	1
 Sternocleido-mastoid Use (inspiration)	By Palpation	1
 Abdominal Muscles Use (expiration)	By Palpation	1

Resuscitation Council UK

The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

Apigo M, Schechtman J, Dhliwayo N, Al Tameemi M, Gazmuri RJ. Development of a work of breathing scale and monitoring need of intubation in COVID-19 pneumonia. *Crit Care (Lond Engl)*. 2020;24(1):477. doi:10.1186/s13054-020-03176-y


YouTube

• Percuss the chest. Hyper resonance suggests pneumothorax. Dullness suggests consolidation or pleural fluid.
• Auscultate the chest. Bronchial breathing indicates consolidation with open airways. Reduced or absent sounds suggest pneumothorax, pleural effusion, or complete obstruction.
• Check tracheal position. Deviation suggests mediastinal shift.
• Feel for surgical emphysema or crepitus, which suggests pneumothorax.
• Give oxygen to all critically ill patients. For COPD, start with controlled oxygen via a Venturi mask and aim for SpO2 between 88 and 92 percent, adjusting based on previous arterial blood gas values.
• If breathing is inadequate or absent, provide bag mask ventilation and call for expert help.
• Consider non invasive ventilation in cooperative patients with no airway obstruction. This is especially useful in acute COPD exacerbations to avoid intubation.

Resuscitation Council UK
The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

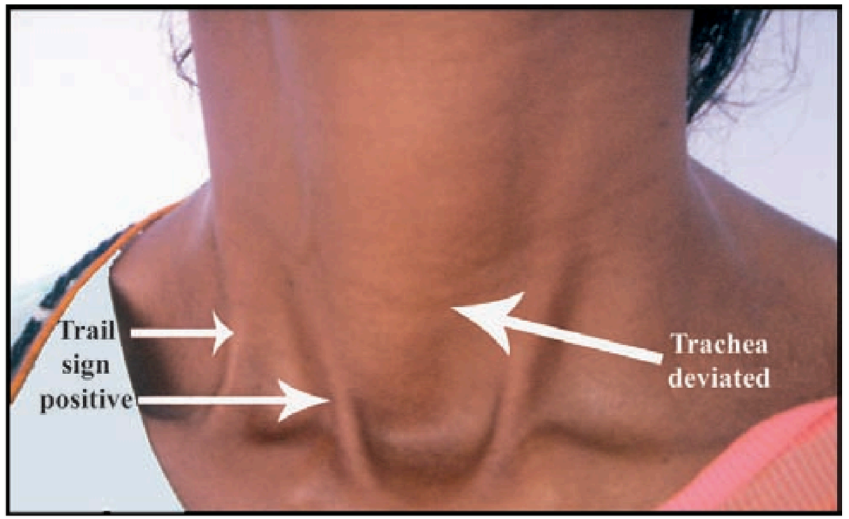
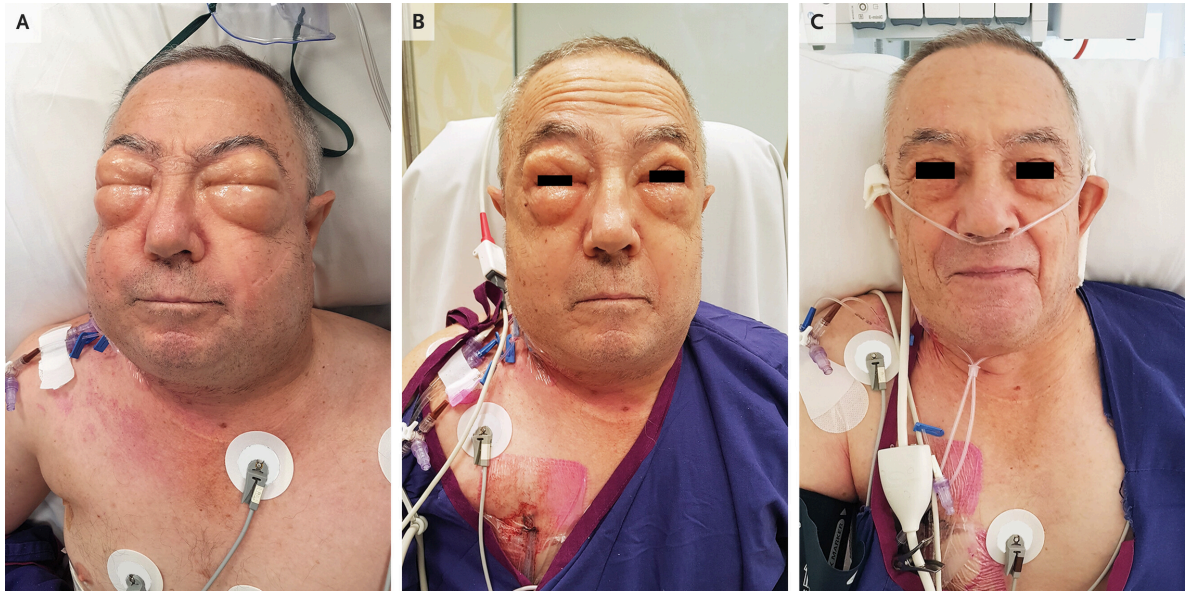


Fig. 4.5: Trail's sign—clavicular head of sternocleidomastoid muscle is prominent on the left side

Vijayasundaram L DT. *Clinical Medicine Made Easy®*. 1/e.; 2008.



Rasiah S, Whitchurch M. Severe subcutaneous emphysema. *N Engl J Med*. 2022;387(9):e18. doi:10.1056/NEJMicm2200611

Respiratory Sound	Clinical Characteristics	Clinical Correlation
Normal tracheal sound	Hollow, nonmusical, heard in both phases.	Indicates upper airway patency; changes with obstruction.
Normal lung sound	Soft, nonmusical, inspiratory and early expiratory.	Reduced in hypoventilation, airway narrowing, effusion, or pneumothorax.
Bronchial breathing	Tracheal-like, heard in both phases.	Suggests consolidation or fibrosis.
Stridor	High pitched, musical.	Indicates upper airway obstruction; phase identifies level.
Wheeze	High pitched, musical.	Suggests airway narrowing; widespread in asthma or COPD.
Rhonchus	Low pitched, snoring quality.	Often due to secretions; may clear with coughing.
Fine crackle	Short, nonmusical, late inspiratory.	Seen in fibrosis, heart failure, or pneumonia.
Coarse crackle	Longer, nonmusical, inspiratory and expiratory.	Often related to secretions, e.g., chronic bronchitis.
Pleural friction rub	Harsh, biphasic.	Indicates pleural inflammation.
Squawk	Short musical sound with crackles.	Seen in distal airway disease and sometimes pneumonia.

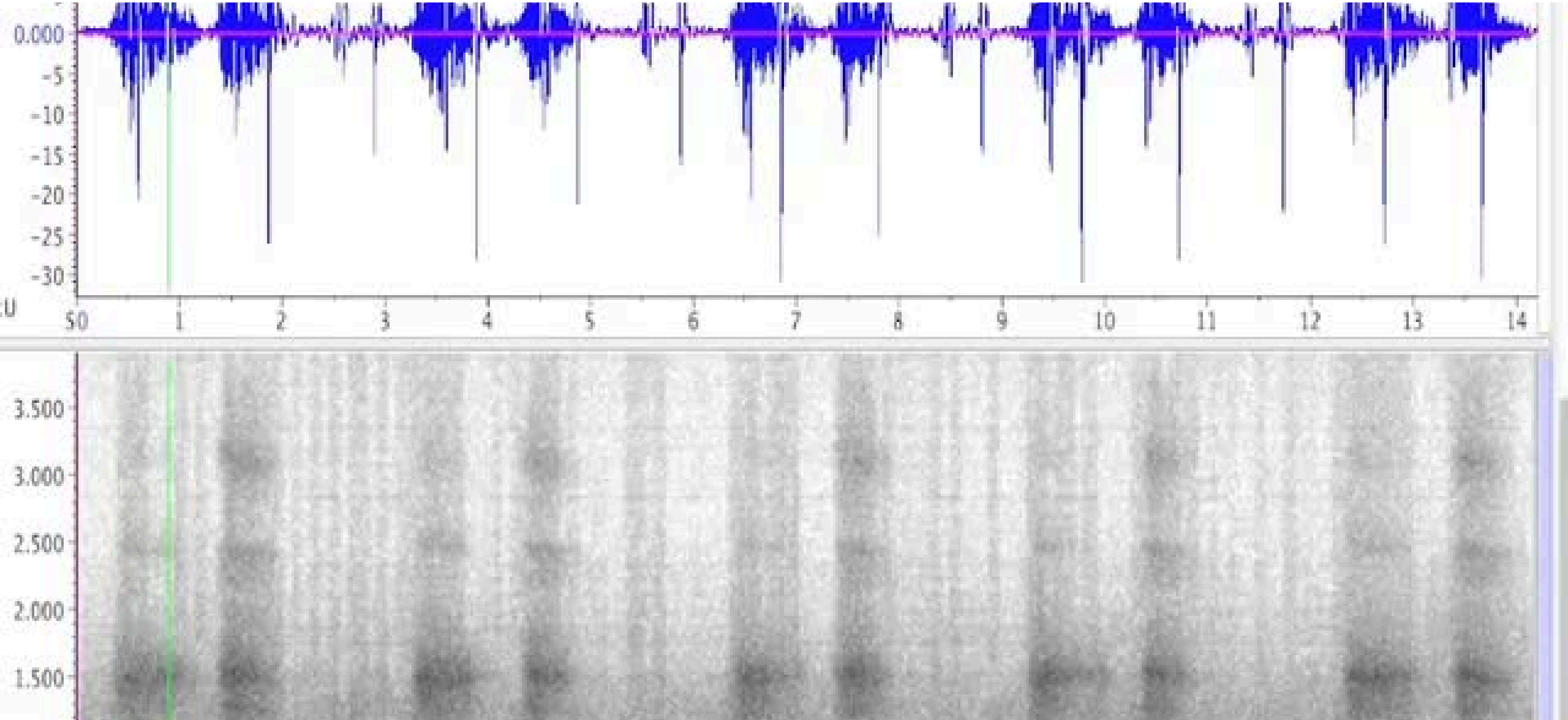
Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370(8):744-751. doi:10.1056/NEJMra1302901



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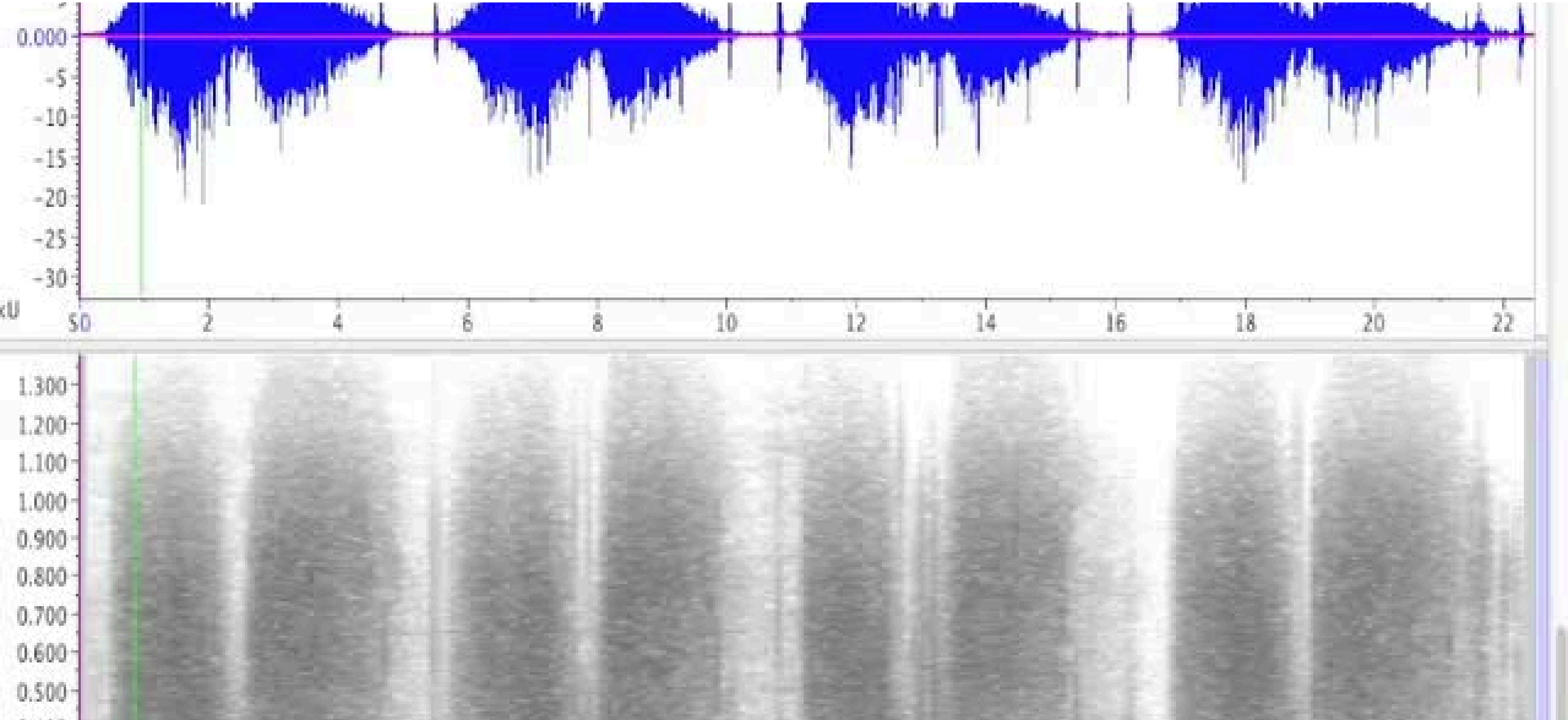
Normal tracheal sound

Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370(8):744-751. doi:10.1056/NEJMra1302901



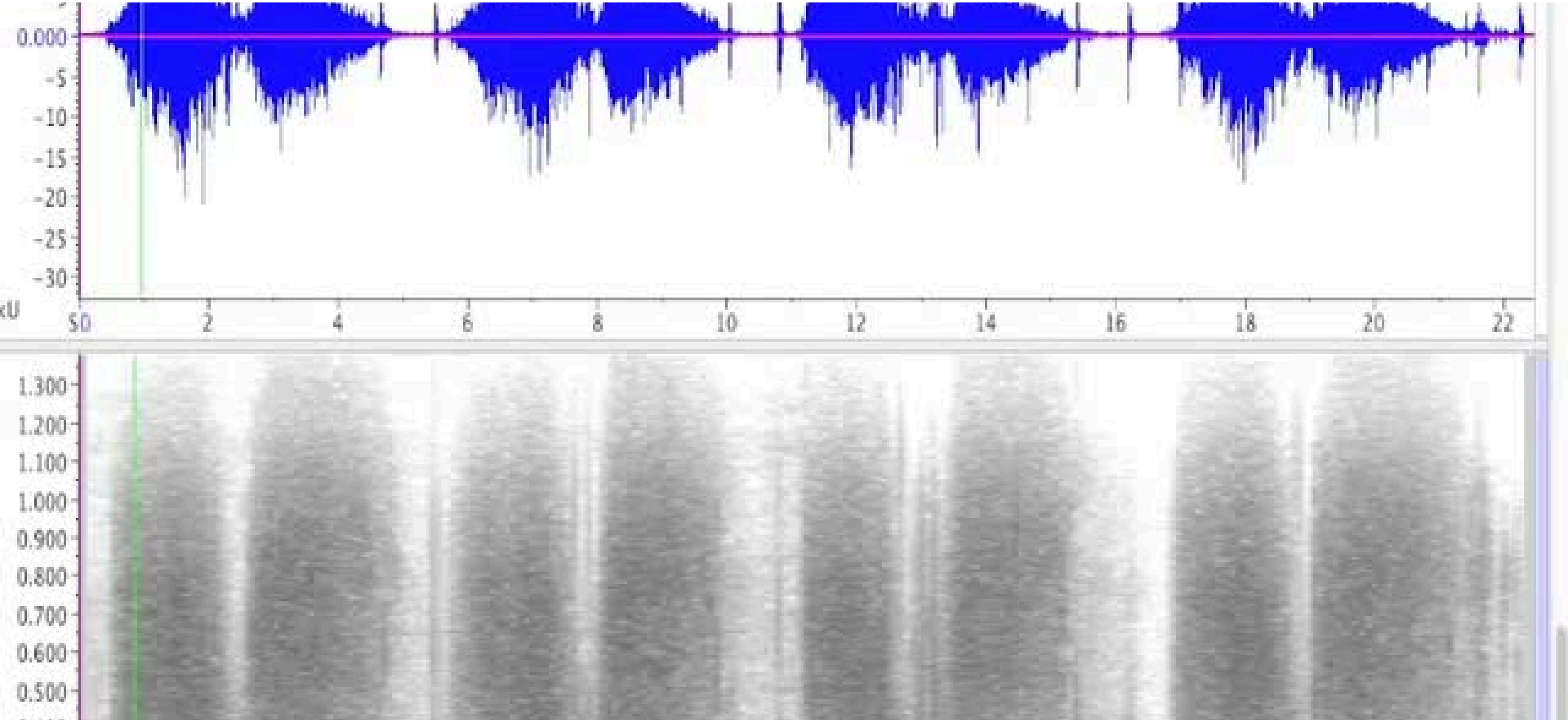
Normal lung sound

Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370(8):744-751. doi:10.1056/NEJMra1302901



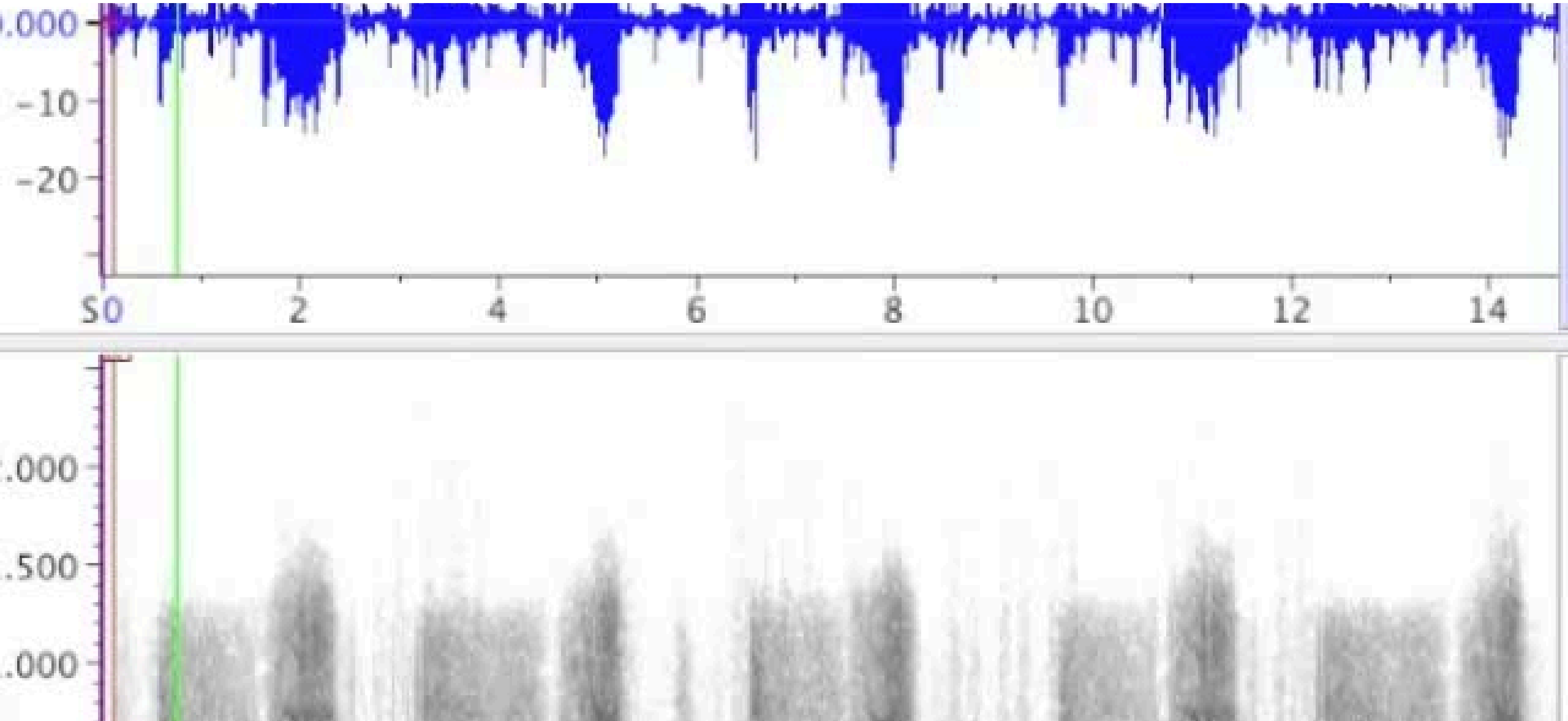
Bronchial breathing

Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370(8):744-751. doi:10.1056/NEJMra1302901



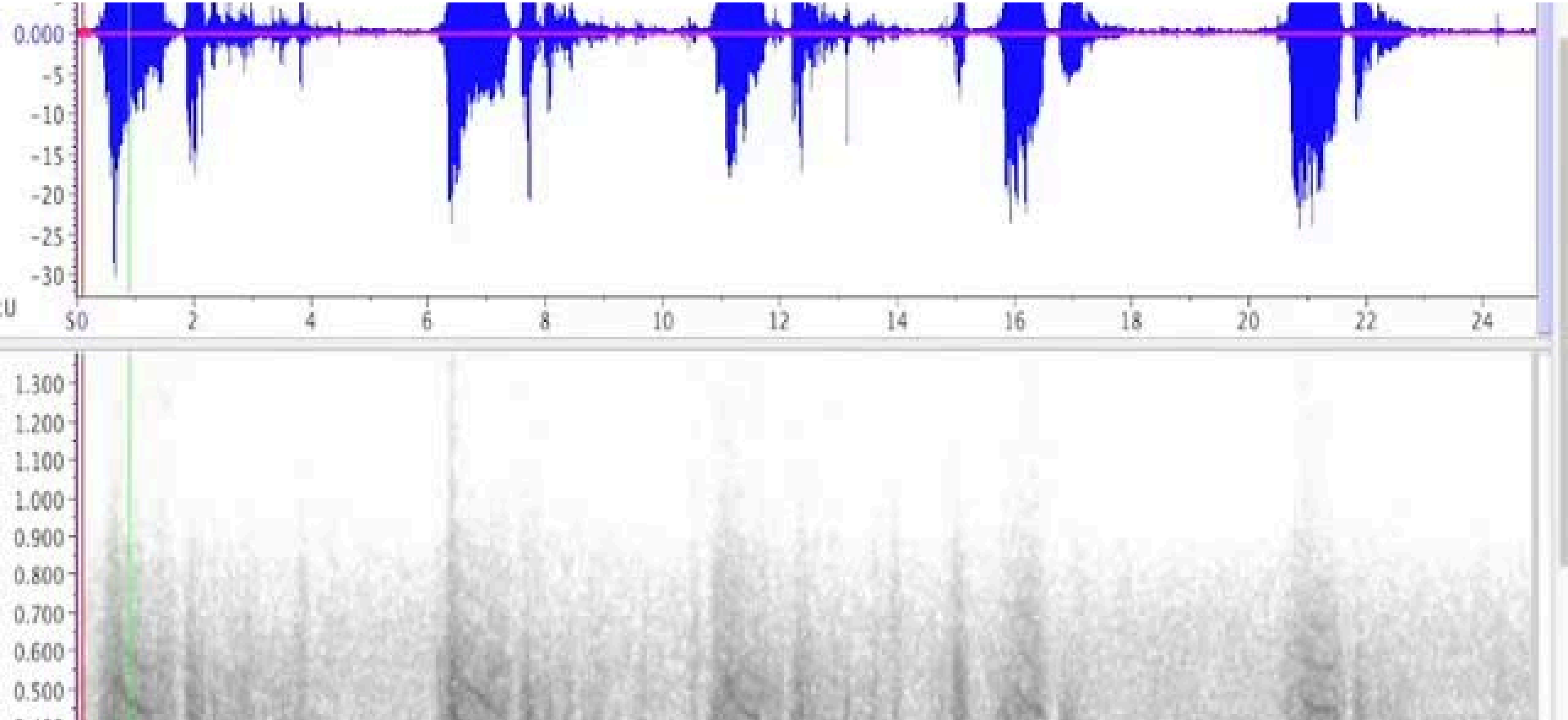
Stridor

Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370(8):744-751. doi:10.1056/NEJMra1302901



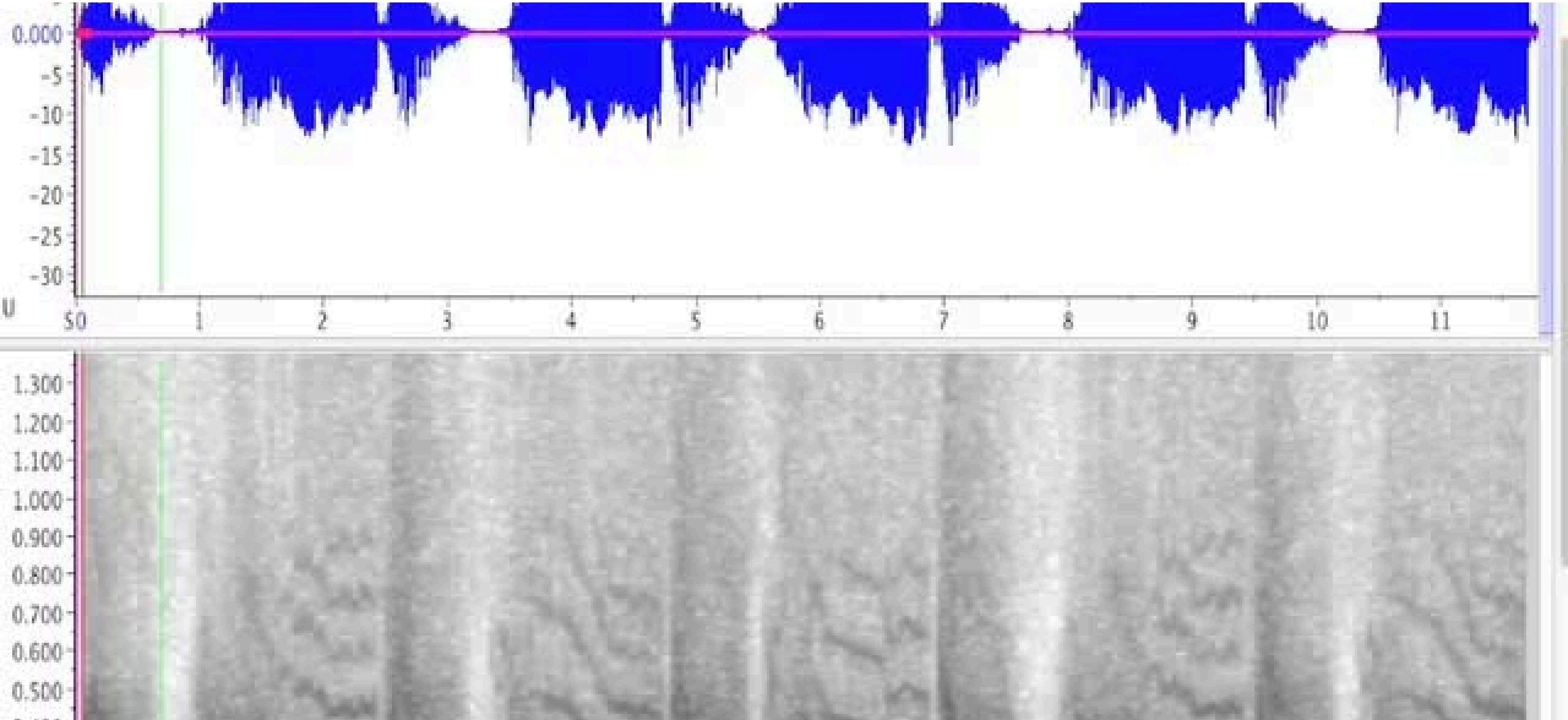
Ronchi

Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370(8):744-751. doi:10.1056/NEJMra1302901



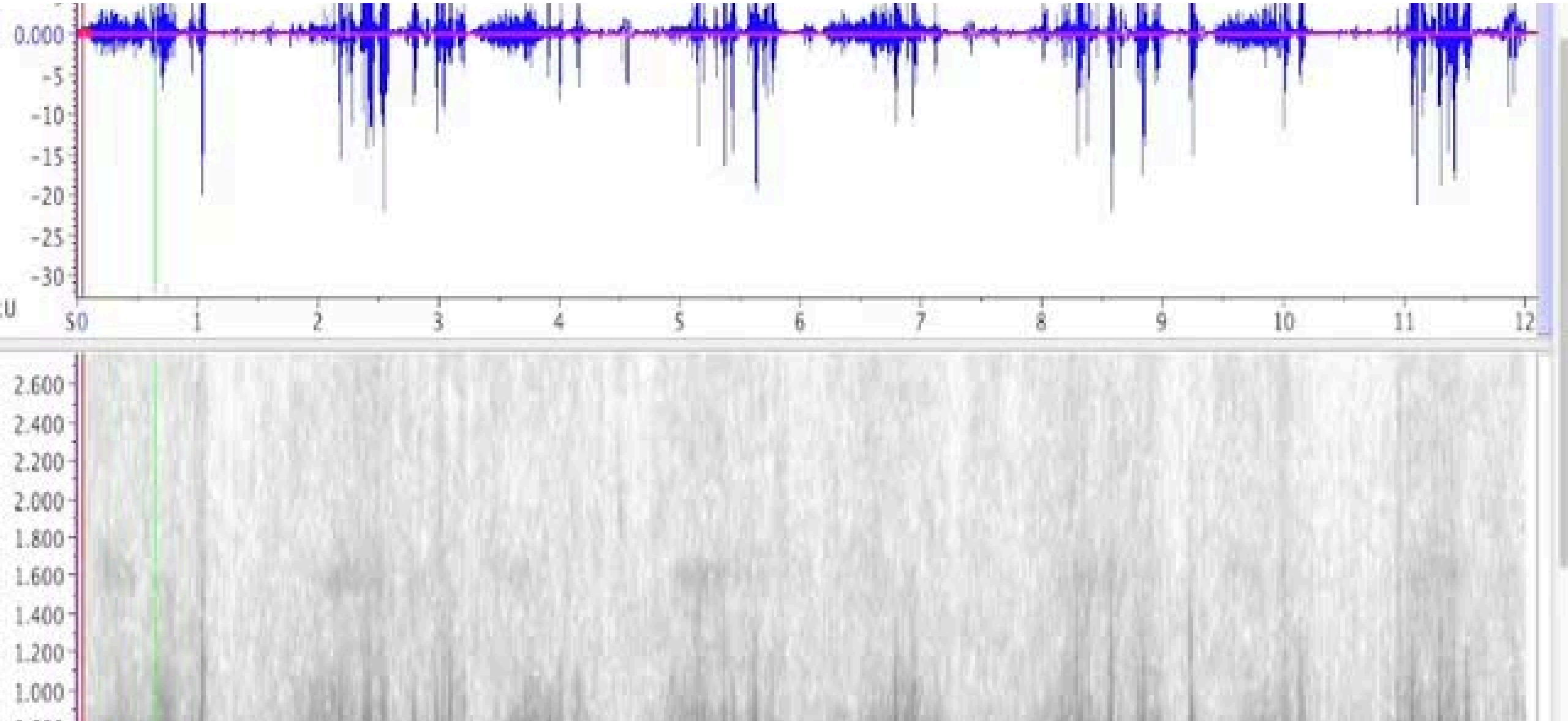
Wheezing

Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370(8):744-751. doi:10.1056/NEJMra1302901



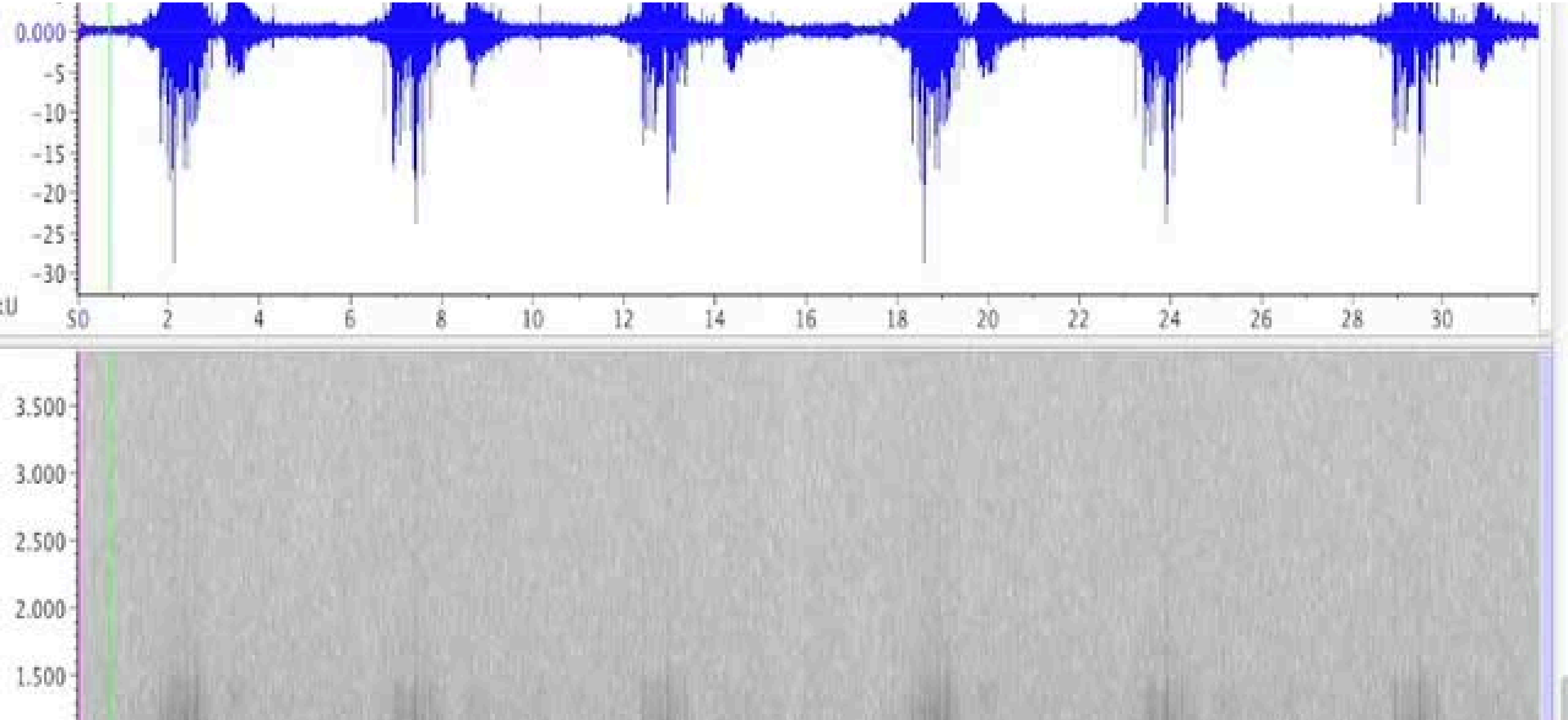
Coarse crackles

Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370(8):744-751. doi:10.1056/NEJMra1302901



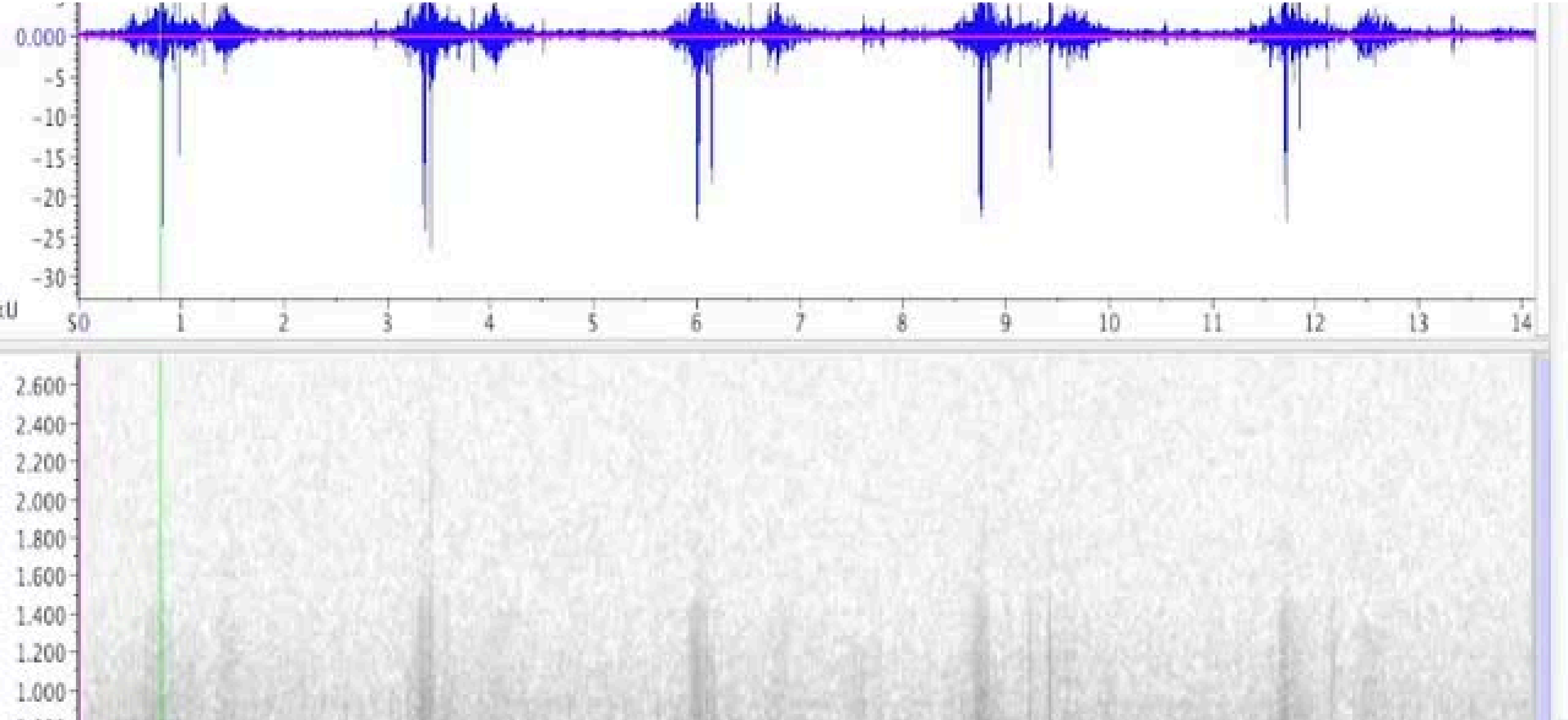
Fine crackles

Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370(8):744-751. doi:10.1056/NEJMra1302901



Pleural friction rub

Bohadana A, Izbicki G, Kraman SS. Fundamentals of lung auscultation. *N Engl J Med*. 2014;370(8):744-751. doi:10.1056/NEJMra1302901



Respiratory Failure and Oxygen Delivery

- **Respiratory Failure**

  Respiratory failure may occur due to dysfunction in ventilation, intrapulmonary gas exchange, gas transport, or tissue gas exchange.

- **Ventilation**

  Ventilation refers to the movement of gases between the environment and the lungs.

- **Gas Transport**

  Gas transport depends on the delivery of adequate quantities of oxygenated blood to the metabolizing tissues.

- **Tissue Gas Exchange**

  Tissue gas exchange involves the extraction and use of oxygen, as well as the release of carbon dioxide, by the peripheral tissues.

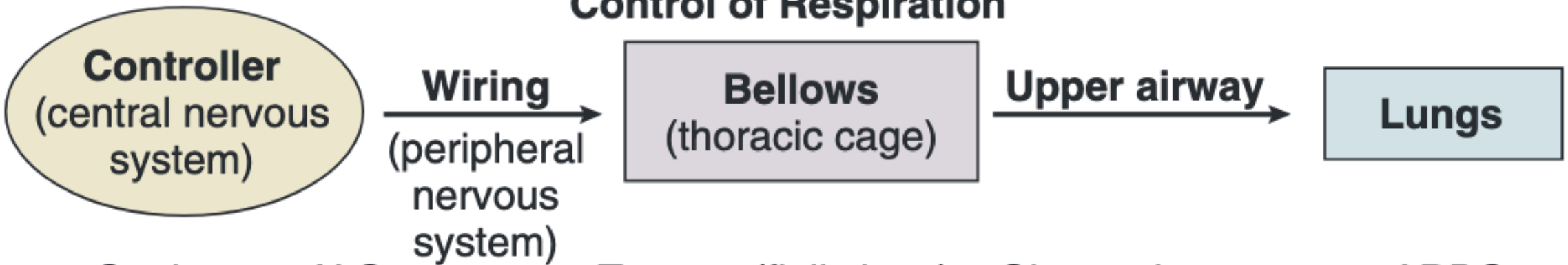
- **Failure of Oxygen Uptake**

  Failure of oxygen uptake refers to the inability of the tissues to extract and use oxygen for aerobic metabolism, as seen in septic shock and cyanide poisoning.

Marini JJ, Dries DJ. Critical Care Medicine: The Essentials and More. Fifth edition. Wolters Kluwer; 2019.

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Control of Respiration



Controller	Wiring	Bellows	Upper airway	Lungs
Stroke	ALS	Trauma (flail chest)	Obstruction	ARDS
Drugs	GBS	Obesity	Laryngeal Edema	CHF
Infection	Polio	Kyphoscoliosis	Infection (epiglottitis, laryngitis)	COPD
	Trauma	Pleural Effusion		Emboli
	Myasthenia	Pneumothorax		Infection
	Botulism	Eschar	Vocal cord paresis	Fibrosis
				Resection
	Critical illness	Critical illness	Obstructive sleep Apnea	
	Neuropathy	Myopathy		

Parrillo JE, Dellinger RP. Critical Care Medicine: Principles of Diagnosis and Management in the Adult. Fifth edition. Elsevier; 2019.

Type I or Hypoxemic Respiratory Failure

Definition

PaO2 < 60 mmHg (8 kPa) with normal or low PaCO2 and normal or high pH. This type involves impairment of oxygen transfer in the lungs, leading to hypoxemia.

Causes

1. Ventilation/Perfusion (V/Q) mismatch,
2. Shunt,
3. Hypoventilation (respiratory pump failure),
4. Impaired diffusion,
5. Low inspired oxygen fraction.

Type I or Hypoxemic Respiratory Failure is the most common form of respiratory failure, where lung disease is severe enough to interfere with pulmonary oxygen exchange, but overall ventilation is maintained.

Duška F, Al-Haddad M, Cecconi M, eds. *Intensive Care Fundamentals: Practically Oriented Essential Knowledge for Newcomers to ICUs*. Springer International Publishing; 2023. doi:10.1007/978-3-031-21991-7

Type II or Hypercapnic Respiratory Failure



Definition

Inadequate ventilation leading to the retention of CO2, with hypercapnia (PaCO2 > 45 mmHg, 6 kPa) and hypoxemia (PaO2 < 60 mmHg, 8 kPa). The value of pH depends on the concentration of HCO3−, which varies based on the duration of hypercapnia.



Causes

1. Increased CO2 production (e.g., fever, sepsis, burns, overfeeding),
2. Decreased CO2 elimination by alveolar ventilation,
3. Decreased CNS drive (CNS lesion, overdose, anesthesia),
4. Neuromuscular disease (e.g., Myasthenia Gravis,
5. Amyotrophic Lateral Sclerosis, Guillain-Barré syndrome, myopathies),
6. Increased work of breathing (WOB) leading to respiratory muscle fatigue and inadequate ventilation, Asthma/COPD,
7. Pulmonary fibrosis,
8. Kyphoscoliosis,
9. Increased physiologic dead space (hypovolemia, poor cardiac output, alveolar overdistension)

Duška F, Al-Haddad M, Cecconi M, eds. Intensive Care Fundamentals: Practically Oriented Essential Knowledge for Newcomers to ICUs. Springer International Publishing; 2023. doi:10.1007/978-3-031-21991-7

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Causes of Type III (Peri-operative) Respiratory Failure



Causes of Type III (Peri-operative) Respiratory Failure

Atelectasis due to low functional residual capacity (FRC) resulting from abnormal abdominal wall mechanics, often leading to type I or type II respiratory failure.



Abnormal Abdominal Wall Mechanics

Peri-operative respiratory failure can be caused by abnormal abdominal wall mechanics that lead to reduced functional residual capacity (FRC) and atelectasis.



Atelectasis and Respiratory Failure

Atelectasis, or the collapse of alveoli, can occur due to the low FRC, resulting in type I (hypoxemic) or type II (hypercapnic) respiratory failure.

Duška F, Al-Haddad M, Cecconi M, eds. Intensive Care Fundamentals: Practically Oriented Essential Knowledge for Newcomers to ICUs. Springer International Publishing; 2023. doi:10.1007/978-3-031-21991-7

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Type IV (Shock) Respiratory Failure



Causes of Type IV (Shock) Respiratory Failure

Respiratory failure secondary to cardiovascular instability, where patients are intubated and ventilated during the resuscitation process to stabilize gas exchange.



Intubation and Ventilation

Patients are placed on mechanical ventilation to support respiratory function and stabilize gas exchange while undergoing resuscitation for shock.



Goal of Treatment

The primary goal is to stabilize the patient's cardiovascular status and improve tissue perfusion, with respiratory support as a crucial component of the resuscitation process.

Duška F, Al-Haddad M, Cecconi M, eds. Intensive Care Fundamentals: Practically Oriented Essential Knowledge for Newcomers to ICUs. Springer International Publishing; 2023. doi:10.1007/978-3-031-21991-7

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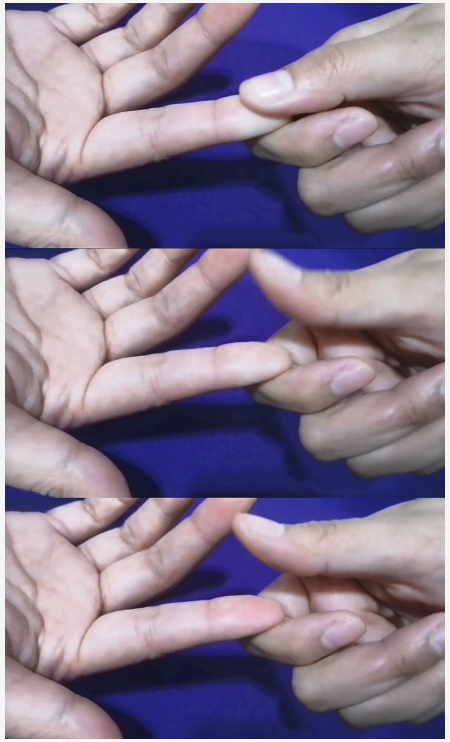
TABLE 9.2 Classification of Acute Respiratory Failure (Modified From[10])

	Type I	Type II	Type III	Type IV
Mechanism of hypoxemia	Low FiO_2 ventilation/perfusion (V/Q) mismatch Shunting Reduced diffusing capacity	Hypoventilation	Shunting Hypoventilation V/Q mismatch	Hypoperfusion or inadequate oxygenation of peripheral tissues
Location of pathologic process	Inhaled air composition Alveolar–capillary unit Oxygen-carrying capacity of blood	Airway Central nervous system (CNS) Neuromuscular system Chest wall	Alveolar–capillary unit collapse with regional hypoventilation	Cardiovascular system Peripheral tissues
Clinical syndromes	Cardiogenic pulmonary edema Acute respiratory distress syndrome (ARDS) Pneumonia Interstitial lung disease Pulmonary embolism Pulmonary hypertension Atelectasis Alveolar hemorrhage Carbon monoxide poisoning Anatomic shunts	Chronic obstructive pulmonary disease (COPD) Asthma CNS depression (intoxication) CNS trauma or injury Neuromuscular disorders Skeletal disorders Obesity–hypoventilation syndrome	ARDS Thoracic or upper abdominal surgery or trauma Inadequate postoperative analgesia Pleural tumor or inflammation Trapped lung Subdiaphragmatic tumor or inflammation Obesity	Septic (distributive) shock Hypovolemic shock Cardiogenic shock Compromised cellular oxidation Hypermetabolic states

Vincent JL, Moore FA, Bellomo R, Marini JJ, eds. Textbook of Critical Care. 8th edition. Elsevier; 2024.

C - Circulation

- Assume hypovolaemia as the primary cause of shock until another cause is identified.
- Give intravenous fluid to patients with cool peripheries and a fast heart rate, unless clear cardiac causes are present.
- Exclude haemorrhage rapidly in surgical patients.
- Assess hand colour for pallor, mottling, or cyanosis.
- Check limb temperature for warmth or coolness.
- Measure capillary refill time. A value above two seconds suggests poor perfusion.
- Assess veins for collapse, which suggests hypovolaemia.
- Measure pulse or heart rate and evaluate presence, rate, quality, rhythm, and symmetry.
- Measure blood pressure. Note diastolic pressure, pulse pressure, and patterns suggesting vasodilation or vasoconstriction.
- Auscultate the heart and check for murmurs, rubs, muffled sounds, or pulse dissociation.



By Thailand2021 - Own work, CC BY-SA 4.0, https://commons.wikimedia.org/w/index.php?curid=127781605



Contou D, Prost N de. Skin mottling. *N Engl J Med*. 2016;375(22):2187-2187. doi:10.1056/NEJMicm1602055

The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

Resuscitation Council UK

- Look for signs of poor cardiac output such as reduced consciousness or low urine output below 0.5 mL per kg per hour.
- Search for external or concealed haemorrhage. Thoracic, abdominal, pelvic, or retroperitoneal bleeding can be significant even with empty drains.
- Treat cardiovascular collapse with fluid resuscitation, haemorrhage control, and restoration of perfusion.
- Insert one or more large bore intravenous cannulae and draw blood for routine investigations and cross match.
- Give a bolus of 500 mL crystalloid over less than fifteen minutes for hypotension. Use smaller boluses in cardiac failure or trauma and reassess frequently.
- Recheck heart rate and blood pressure every five minutes and aim for systolic pressure above 100 mmHg.
- Repeat fluid challenges if needed and seek expert help if response is inadequate.
- Stop or slow fluids if signs of cardiac failure appear and consider inotropes or vasopressors.
- For suspected acute coronary syndrome, obtain a 12 lead ECG early.
- Initial treatment includes aspirin 300 mg, nitroglycerine, oxygen only if SpO2 is below 94 percent, and intravenous morphine titrated slowly.





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D - Disability, Neurologic & Glycaemia

- Common causes include severe hypoxia, hypercapnia, cerebral hypoperfusion, and recent sedative or analgesic drug administration.
- Review airway, breathing, and circulation immediately. Treat hypoxia and hypotension without delay.
- Check the drug chart for reversible drug related causes of reduced consciousness. Use antidotes when appropriate such as naloxone for opioid toxicity.
- Examine the pupils for size, symmetry, and light reactivity.
- Assess conscious level using the ACVPU scale: Alert, new Confusion, response to Voice, response to Pain, or Unresponsive.
- Alternatively assign a Glasgow Coma Scale score. Use supraorbital pressure as a painful stimulus if needed.

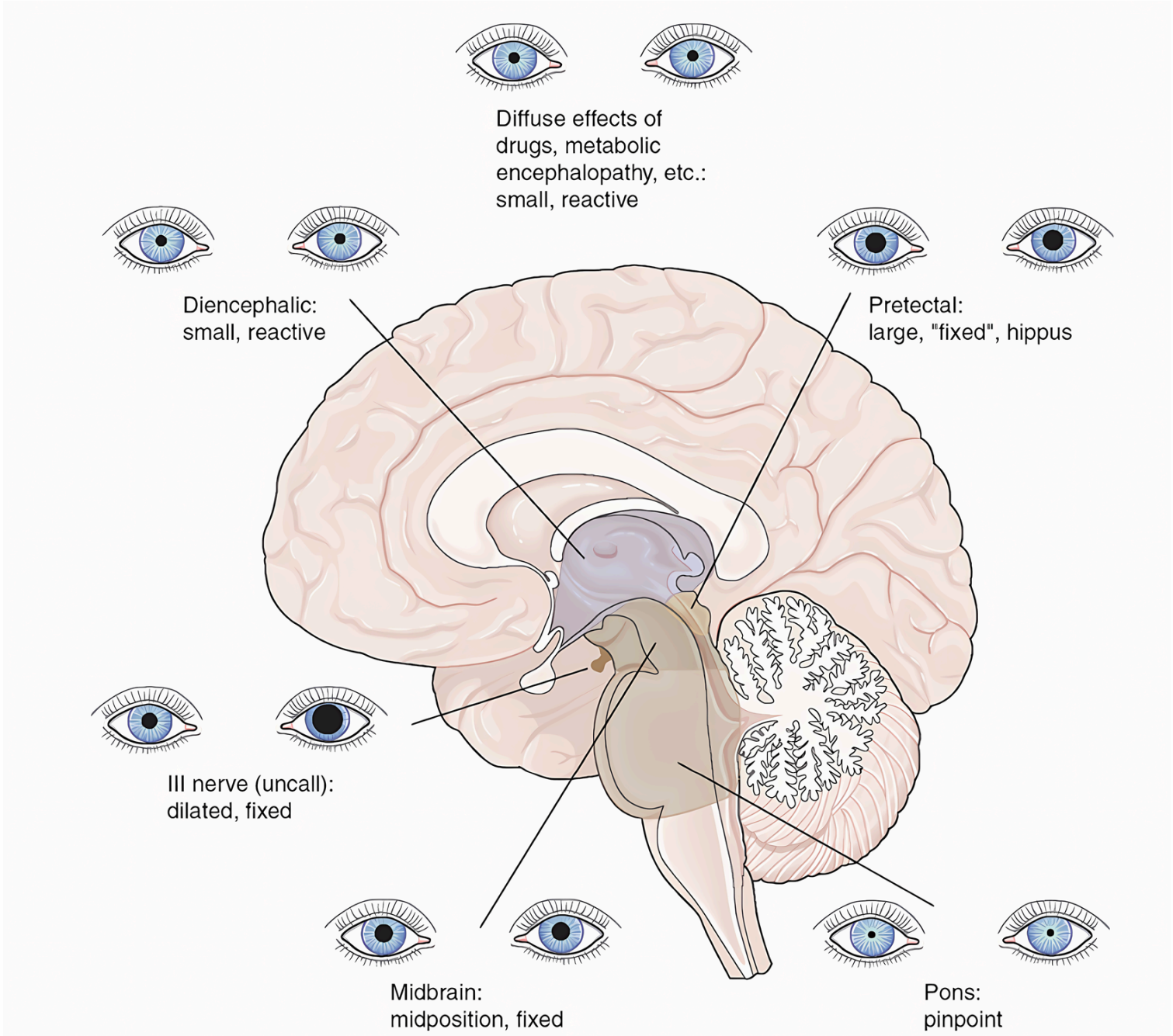
The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

- Measure blood glucose with rapid bedside testing. In peri arrest situations, use venous or arterial sampling because capillary measurements may be unreliable.
- Treat hypoglycaemia according to local protocols. If glucose is below 4.0 mmol per litre in an unconscious patient, give 50 mL of 10 percent glucose intravenously.
- Repeat small boluses of 10 percent glucose as needed up to 250 mL total, monitoring glucose levels and clinical response.
- Consider further glucose administration if no improvement occurs. Follow national guidance for hypoglycaemia in patients with diabetes.
- Place unconscious patients in the lateral position if the airway is unprotected.

The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

Resuscitation Council UK

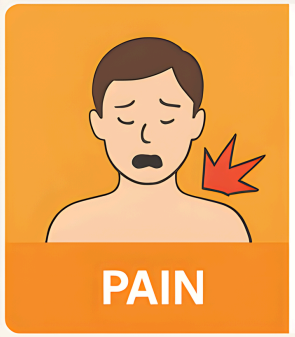




Saper CB, Posner JB, Claassen J, Schiff ND, eds. *Plum and Posner's Diagnosis and Treatment of Stupor and Coma*. Fifth edition. Oxford University Press; 2019.









1. Alert. The patient is awake, responds appropriately, follows commands, opens their eyes spontaneously, and interacts with their surroundings.

2. Verbal. The patient does not open their eyes spontaneously. They open their eyes in response to verbal prompts and respond in a meaningful way.

3. Pain. The patient does not open their eyes spontaneously. They respond only to painful stimulation with movement, vocalisation, or other reactions.

4. Unresponsive. The patient shows no spontaneous response and does not react to verbal or painful stimuli.

Romanelli D, Farrell MW. AVPU scale. In: *StatPearls*. StatPearls Publishing; 2025. Accessed November 20, 2025. http://www.ncbi.nlm.nih.gov/books/NBK538431/

GLASGOW COMA SCALE : Do it this way

GCS
EYES
VERBAL
MOTOR

Institute of Neurological Sciences NHS Greater Glasgow and Clyde



CHECK

For factors Interfering with communication,
ability to respond and other injuries



OBSERVE

Eye opening , content of speech and movements of right and left sides



STIMULATE

Sound: spoken or shouted request
Physical: Pressure on finger tip, trapezius or supraorbital notch



RATE

Assign according to highest response observed

The Glasgow structured approach to assessment of the Glasgow coma scale. Accessed November 17, 2025. https://www.glasgowcomascale.org/

Eye opening

Criterion	Observed	Rating	Score
Open before stimulus	✔	Spontaneous	4
After spoken or shouted request	✔	To sound	3
After finger tip stimulus	✔	To pressure	2
No opening at any time, no interfering factor	✔	None	1
Closed by local factor	✔	Non testable	NT

The Glasgow structured approach to assessment of the Glasgow coma scale. Accessed November 17, 2025. https://www.glasgowcomascale.org/

Verbal response

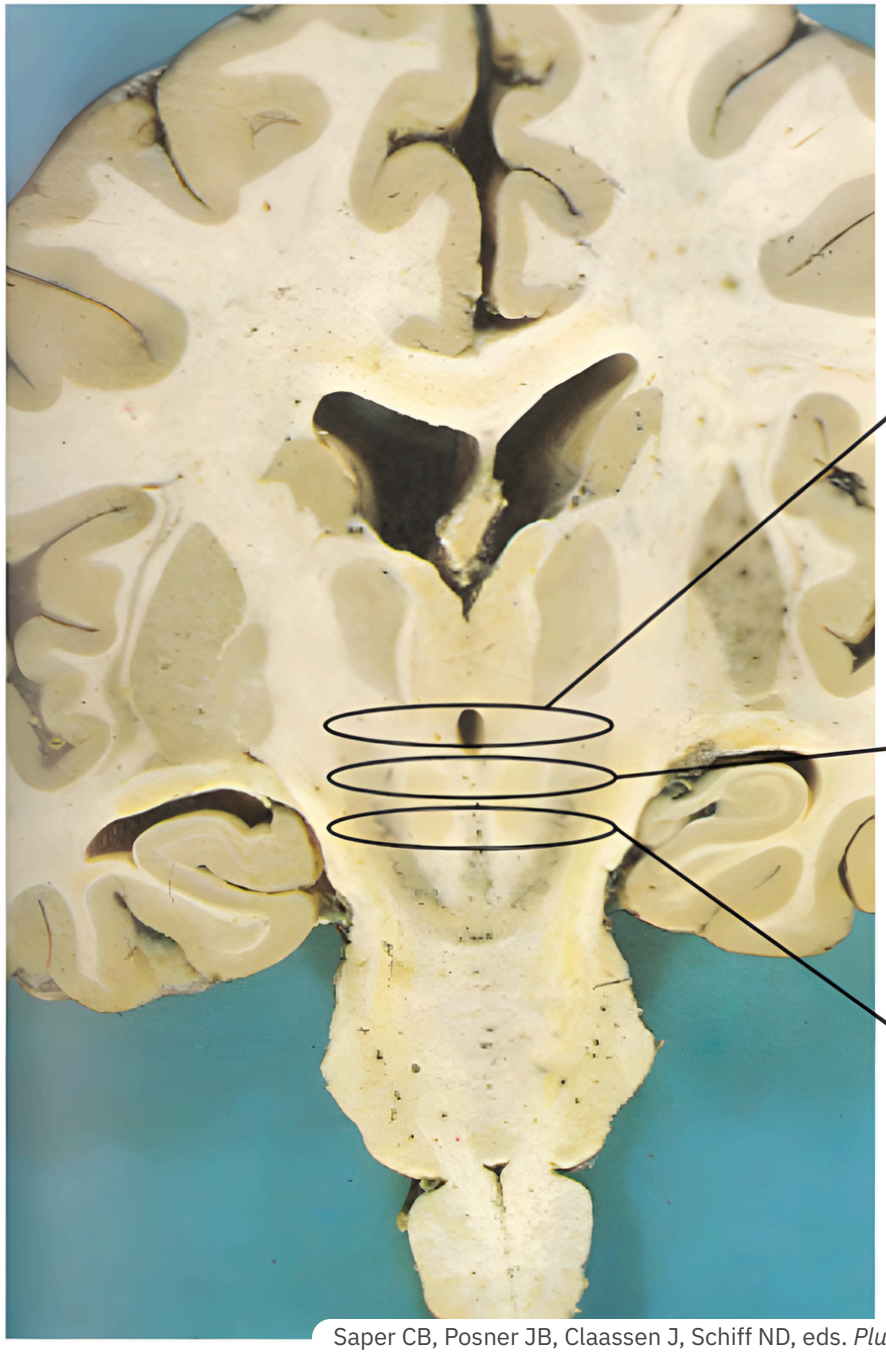
Criterion	Observed	Rating	Score
Correctly gives name, place and date	✔	Orientated	5
Not orientated but communication coherently	✔	Confused	4
Intelligible single words	✔	Words	3
Only moans / groans	✔	Sounds	2
No audible response, no interfering factor	✔	None	1
Factor interferring with communication	✔	Non testable	NT

The Glasgow structured approach to assessment of the Glasgow coma scale. Accessed November 17, 2025. https://www.glasgowcomascale.org/

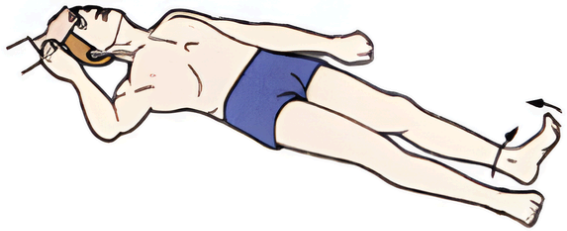
Best motor response

Criterion	Observed	Rating	Score
Obey 2-part request	✔	Obeys commands	6
Brings hand above clavicle to stimulus on head neck	✔	Localising	5
Bends arm at elbow rapidly but features not predominantly abnormal	✔	Normal flexion	4
Bends arm at elbow, features clearly predominantly abnormal	✔	Abnormal flexion	3
Extends arm at elbow	✔	Extension	2
No movement in arms / legs, no interfering factor	✔	None	1
Paralysed or other limiting factor	✔	Non testable	NT

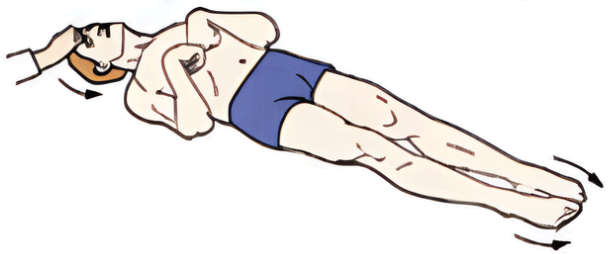
The Glasgow structured approach to assessment of the Glasgow coma scale. Accessed November 17, 2025. https://www.glasgowcomascale.org/



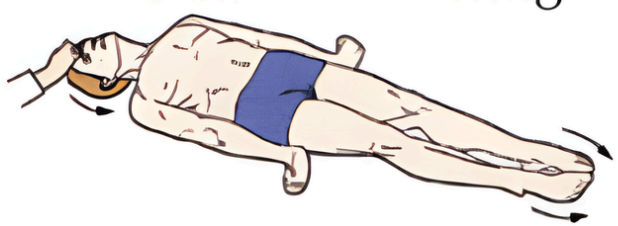
Diencephalic
Paratonia,
Asymmetric Motor



Meso-diencephalic
Decorticate Posturing

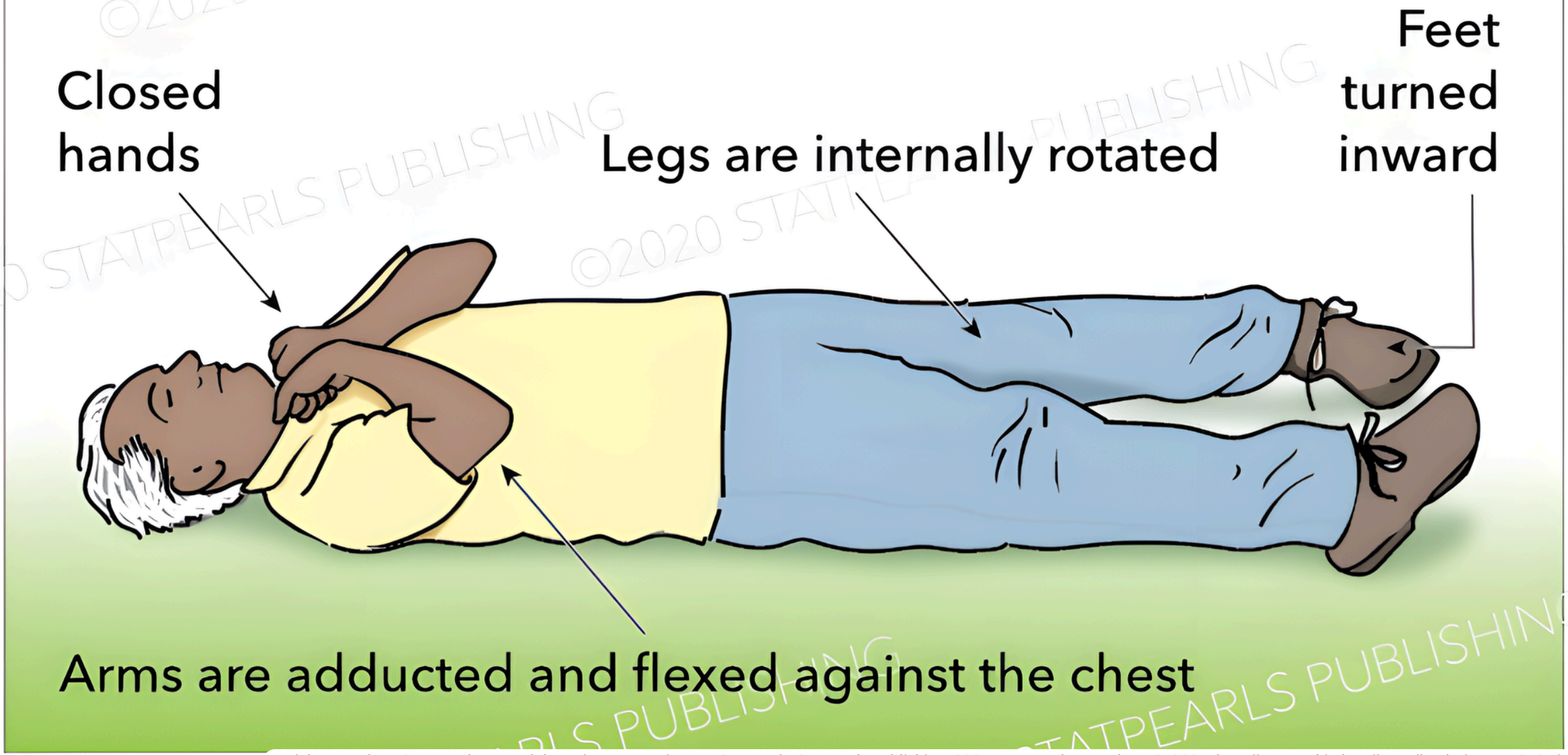


Upper Midbrain
Decerebrate Posturing



Saper CB, Posner JB, Claassen J, Schiff ND, eds. *Plum and Posner's Diagnosis and Treatment of Stupor and Coma*. Fifth edition. Oxford University Press; 2019.

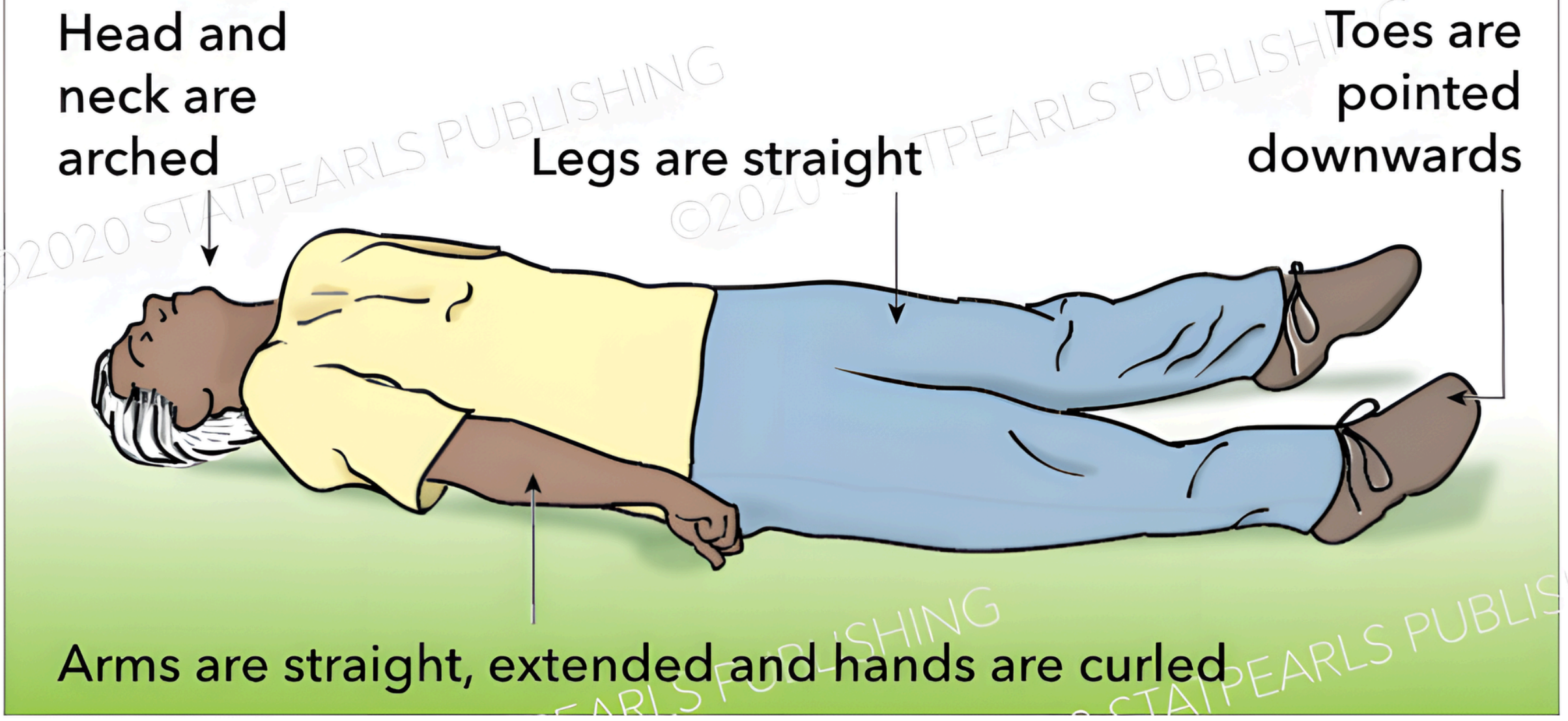
Signs of Decorticate Posturing



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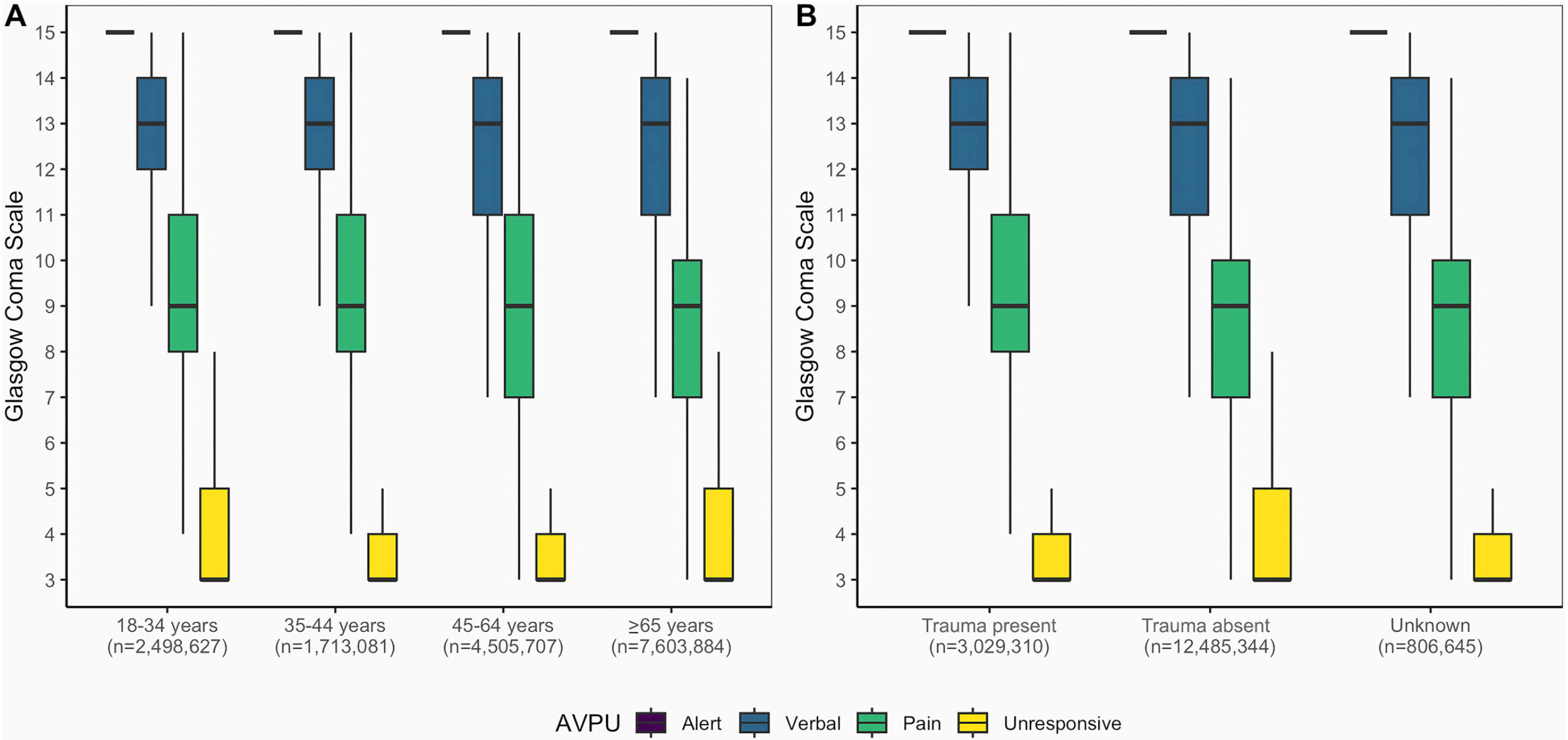
Knight J, Decker LC. Decerebrate and decorticate posturing. In: *StatPearls*. StatPearls Publishing; 2025. Accessed November 18, 2025. http://www.ncbi.nlm.nih.gov/books/NBK559135/

Signs of Decerebrate Posturing



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Knight J, Decker LC. Decerebrate and decorticate posturing. In: *StatPearls*. StatPearls Publishing; 2025. Accessed November 18, 2025. http://www.ncbi.nlm.nih.gov/books/NBK559135/



Ramgopal S, Cash RE, Okubo M, Martin-Gill C. Mapping glasgow coma scale to AVPU scores among adults in the prehospital setting. *Am J Emerg Med*. 2024;86:1-4. doi:10.1016/j.ajem.2024.09.043

Instructions for assessing the FOUR score.

Eye response.

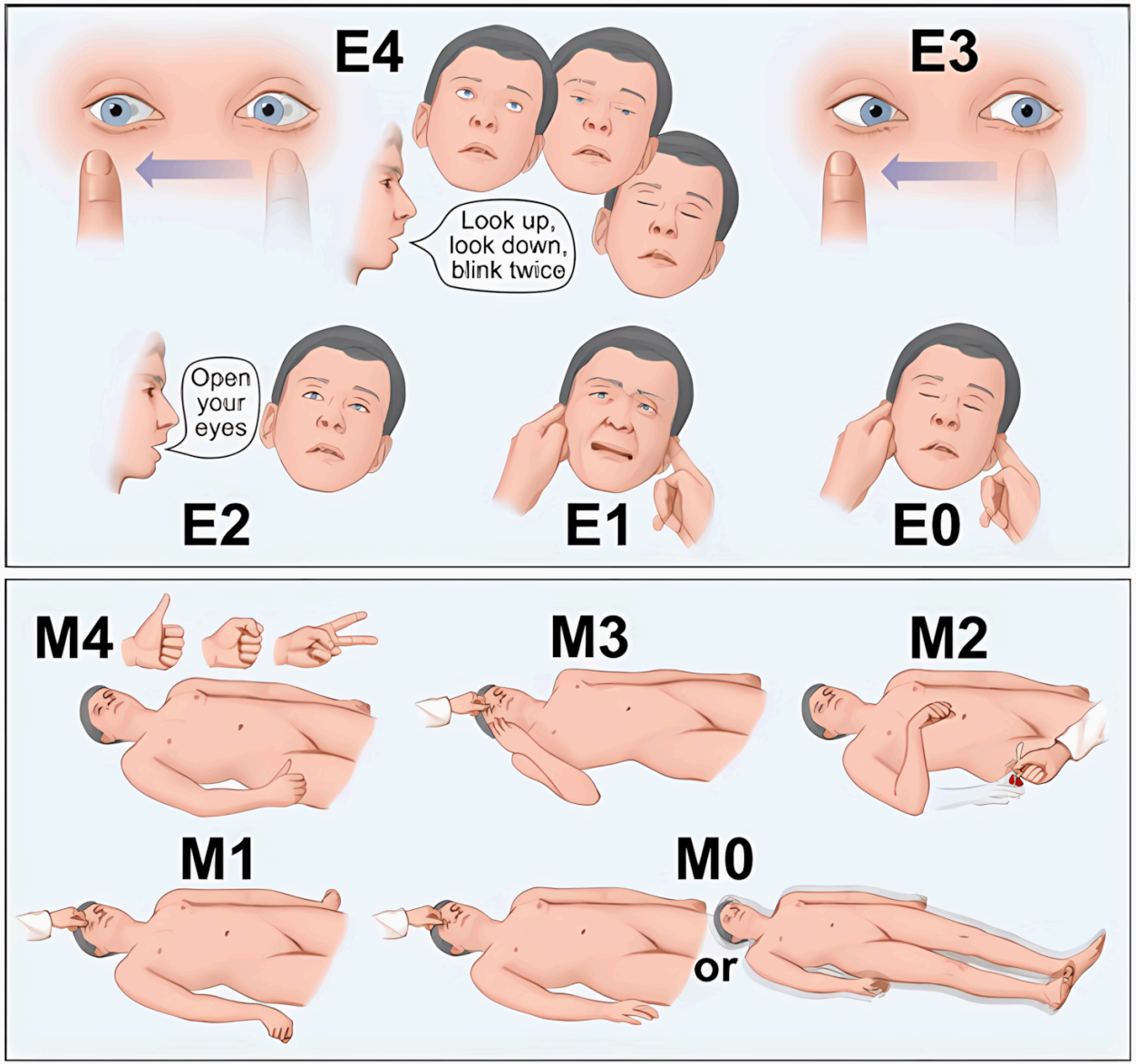
Assess the best response after at least three attempts.

- E4 indicates at least three voluntary eye movements. If the eyelids are closed, open them and assess horizontal tracking, then vertical tracking if needed. Tracking with only one eye is acceptable in cases of swelling or trauma. Two blinks on command also qualify as E4 and help identify locked in syndrome.
- E3 indicates open eyes without voluntary tracking.
- E2 indicates eyelid opening to a loud voice.
- E1 indicates eyelid opening to pain.
- E0 indicates no eyelid opening to pain.

Motor response.

Grade the best response of the arms.

- M4 indicates a clear hand signal with either hand, such as thumbs up, a fist, or a peace sign.
- M3 indicates localisation, such as touching the examiner's hand after painful stimulation of the temporomandibular joint or supraorbital nerve.
- M2 indicates any flexion of the upper limbs.
- M1 indicates extensor posturing.
- M0 indicates no motor response to pain or myoclonus status epilepticus.



Wijdicks EFM, Bamlet WR, Maramattom BV, Manno EM, McClelland RL. Validation of a new coma scale: the FOUR score. Ann Neurol. 2005;58(4):585-593. doi:10.1002/ana.20611

Instructions for assessing the FOUR score.

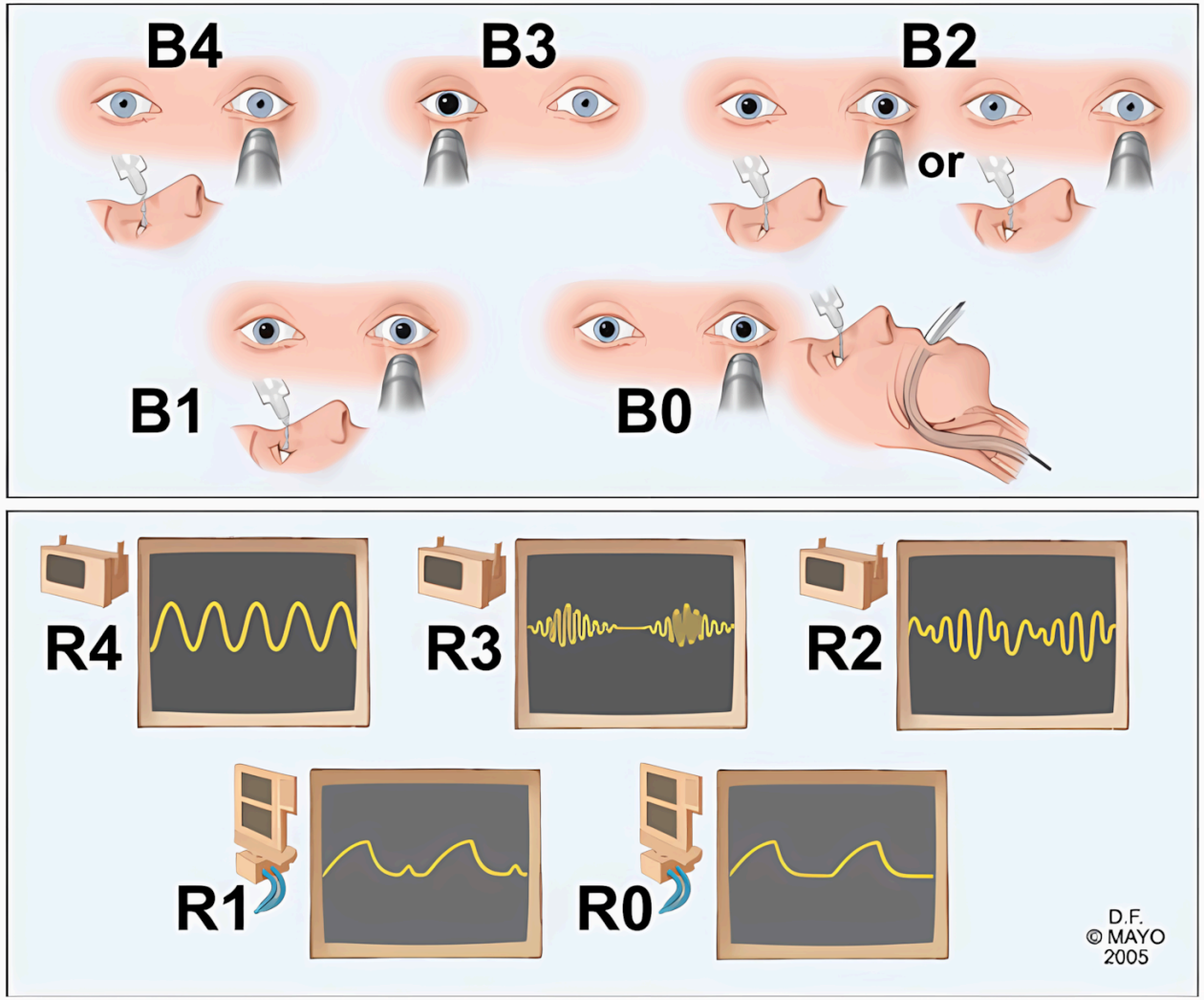
Brainstem reflexes.

Assess the best response.

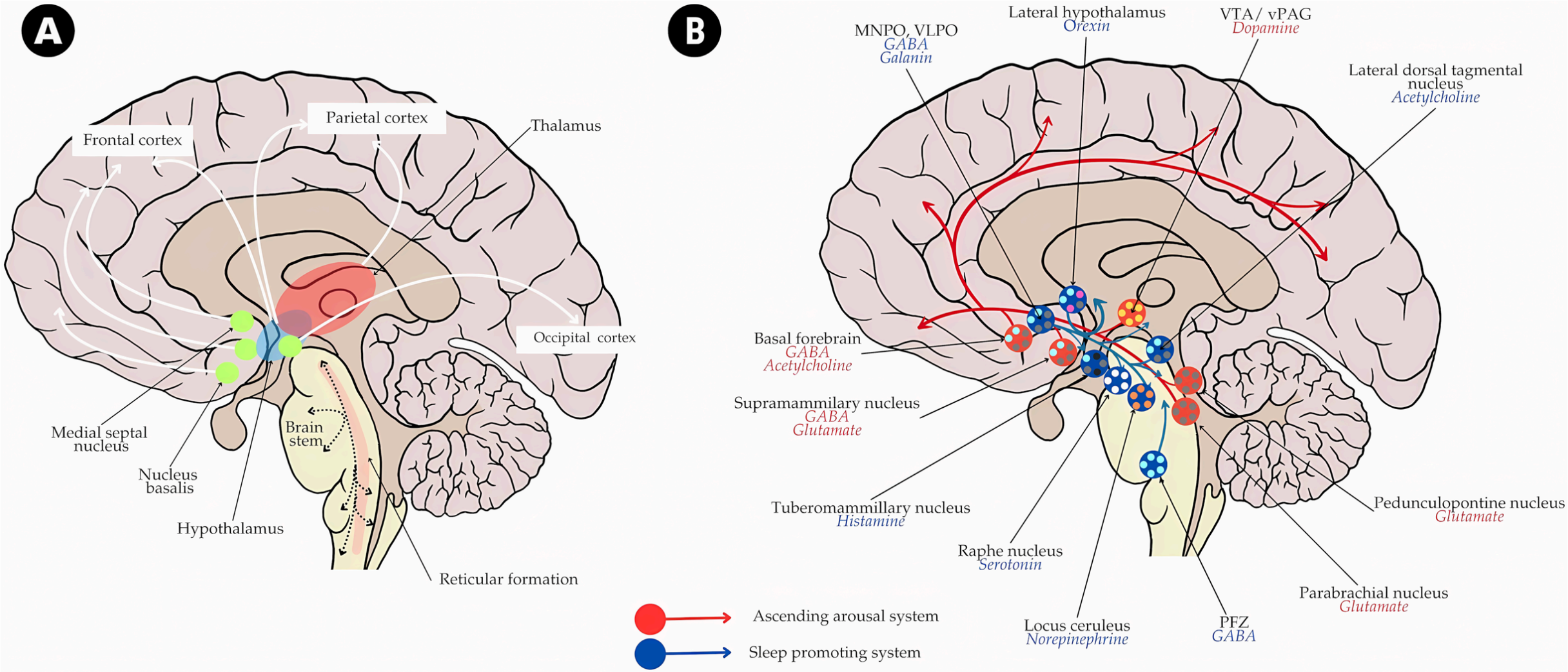
- Examine pupillary and corneal reflexes.
- Test the corneal reflex with two to three drops of sterile saline from a short distance or with a sterile cotton swab.
- Test the cough reflex to tracheal suction only if both pupillary and corneal reflexes are absent.
- B4 indicates both reflexes present.
- B3 indicates one pupil wide and fixed.
- B2 indicates absence of either reflex.
- B1 indicates absence of both pupil and cornea reflexes.
- B0 indicates absence of pupil, cornea, and cough reflexes.

Respiration.

- Assess spontaneous breathing in a non intubated patient.
- Grade as regular breathing for R4,
- Cheyne Stokes for R3, or
- irregular breathing for R2.
- In ventilated patients, assess spontaneous breaths on the ventilator pressure waveform.
- R1 indicates patient triggered breaths.
- When the ventilator entirely drives the rate, R0 may require an apnoea test.

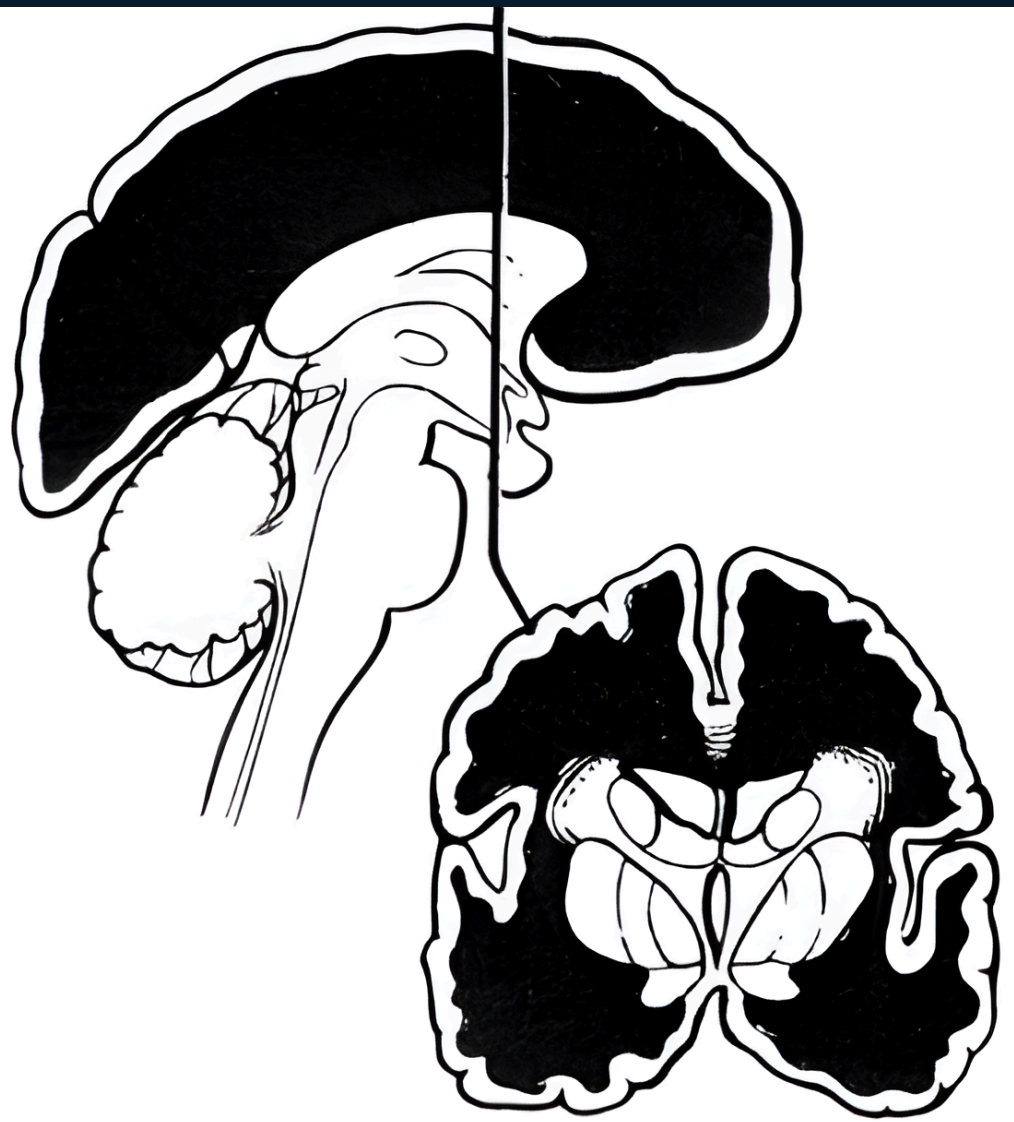


Wijdicks EFM, Bamlet WR, Maramattom BV, Manno EM, McClelland RL. Validation of a new coma scale: the FOUR score. Ann Neurol. 2005;58(4):585-593. doi:10.1002/ana.20611



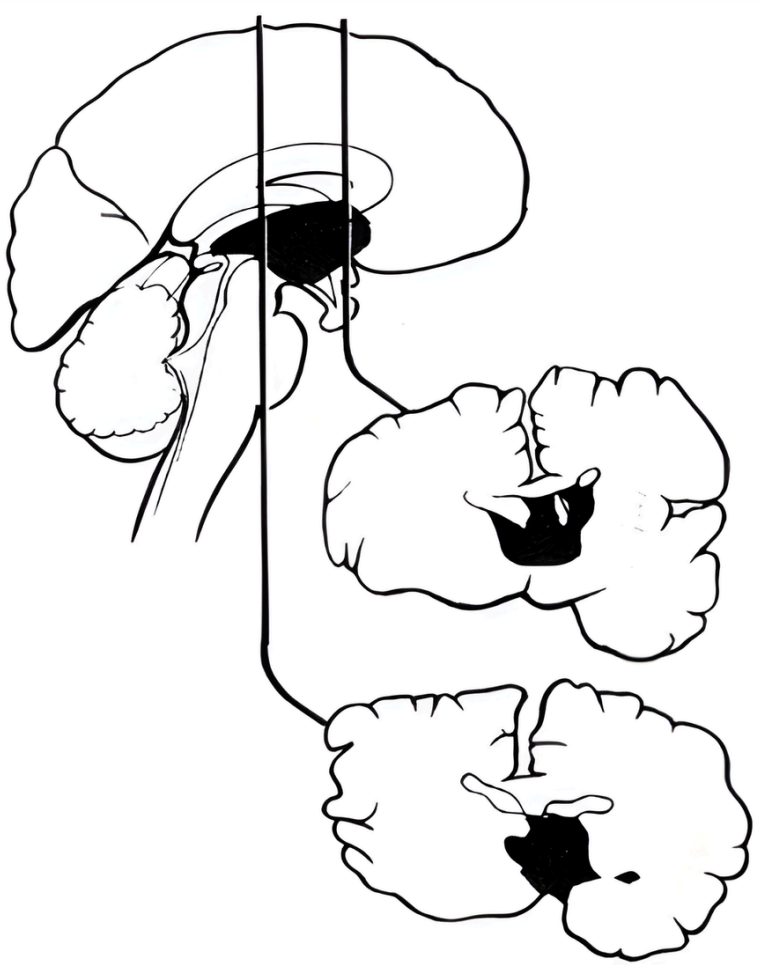
Hein ZM, Al-Zaghal ZAS, Muhammad Ghazali M, et al. Mechanistic insights into the sleep-glymphopathy-cerebral small vessel disease loop: implications for epilepsy pathophysiology and therapy. *Front Neurosci*. 2025;19. doi:10.3389/fnins.2025.1546482

Diffuse hemispheric damage



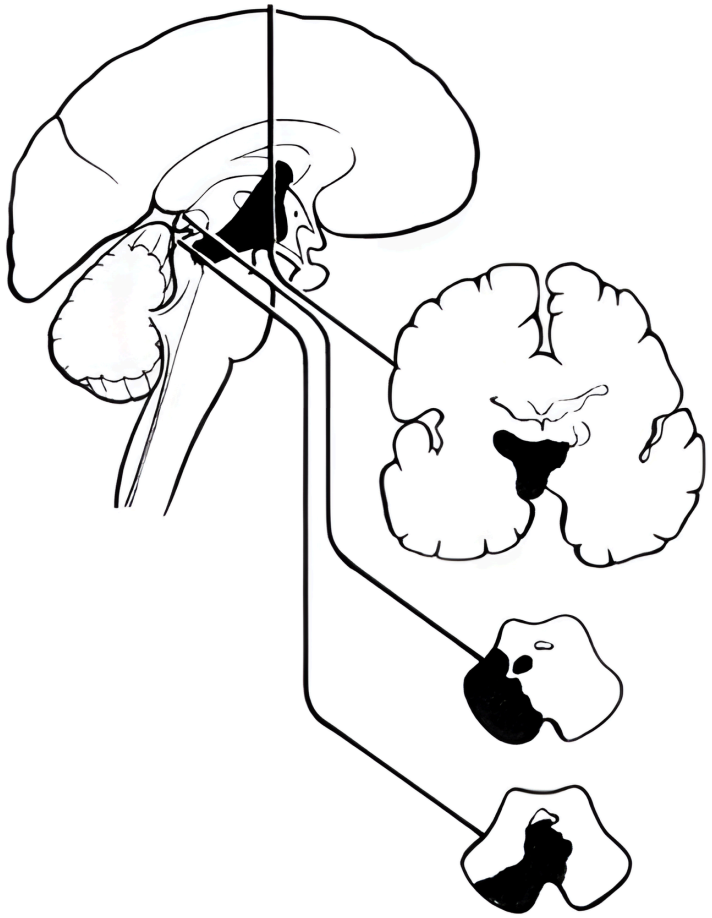
Saper CB, Posner JB, Claassen J, Schiff ND, eds. *Plum and Posner's Diagnosis and Treatment of Stupor and Coma*. Fifth edition. Oxford University Press; 2019.

Diencephalic injury



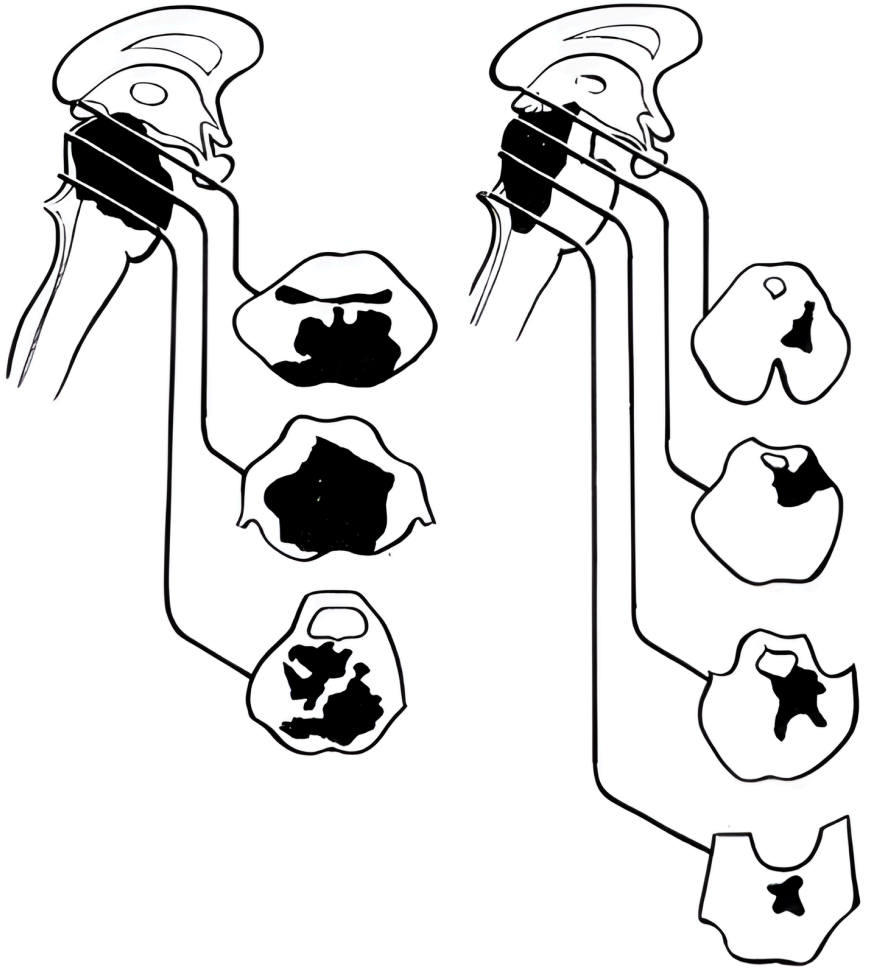
Saper CB, Posner JB, Claassen J, Schiff ND, eds. *Plum and Posner's Diagnosis and Treatment of Stupor and Coma*. Fifth edition. Oxford University Press; 2019.

Damage to the paramedian portion of the upper midbrain and caudal diencephalon



Saper CB, Posner JB, Claassen J, Schiff ND, eds. *Plum and Posner's Diagnosis and Treatment of Stupor and Coma*. Fifth edition. Oxford University Press; 2019.

High pontine and lower midbrain paramedian tegmental injury / Pontine haemorrhage



Saper CB, Posner JB, Claassen J, Schiff ND, eds. *Plum and Posner's Diagnosis and Treatment of Stupor and Coma*. Fifth edition. Oxford University Press; 2019.

E - Exposure & Temperature

To examine the patient properly full exposure of the body may be necessary.

Respect the patient’s dignity and minimise heat loss.

The ABCDE approach | resuscitation council UK. Accessed November 17, 2025. https://www.resus.org.uk/library/abcde-approach

Resuscitation Council UK

F - Fluids, Kidney, Electrolytes



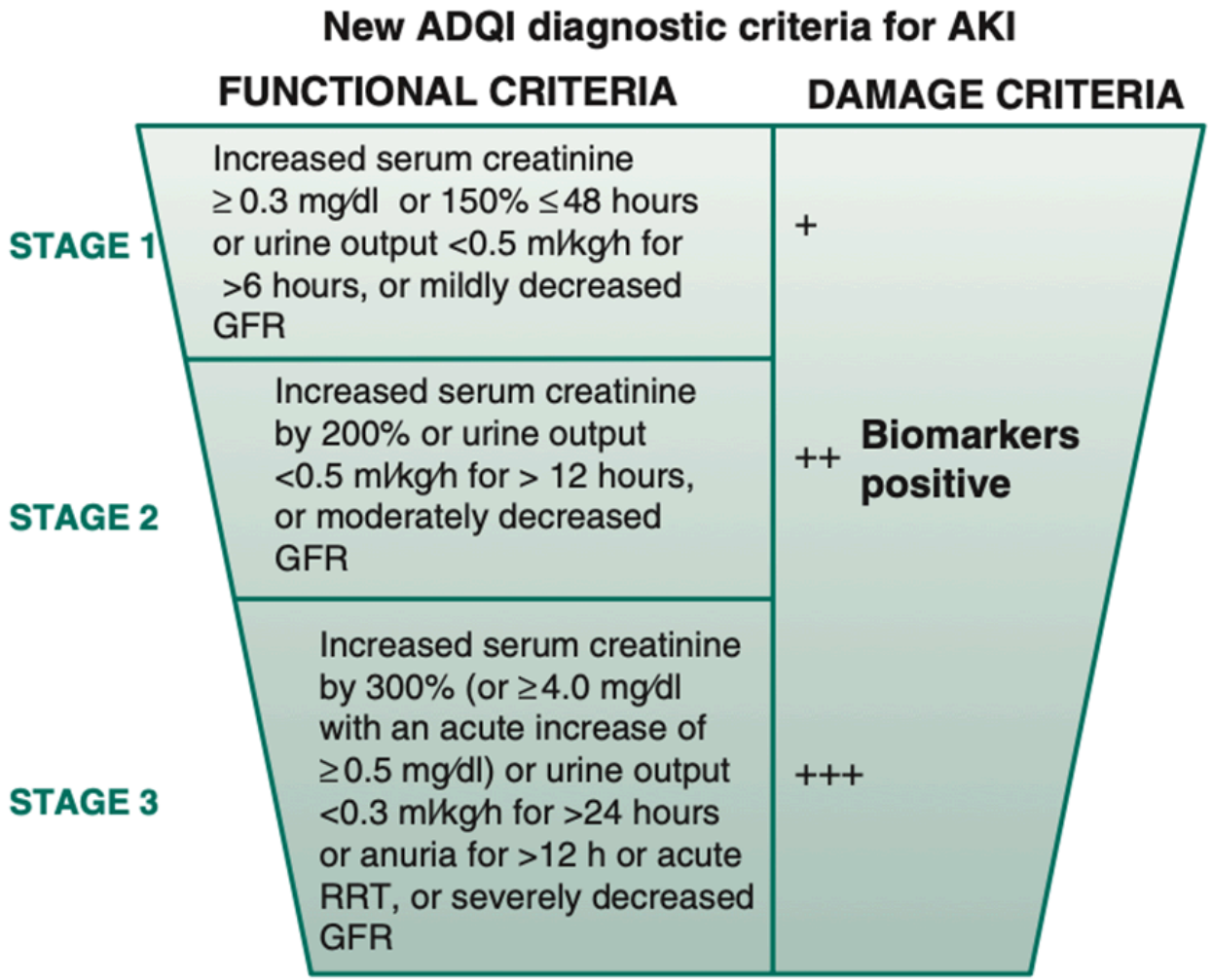
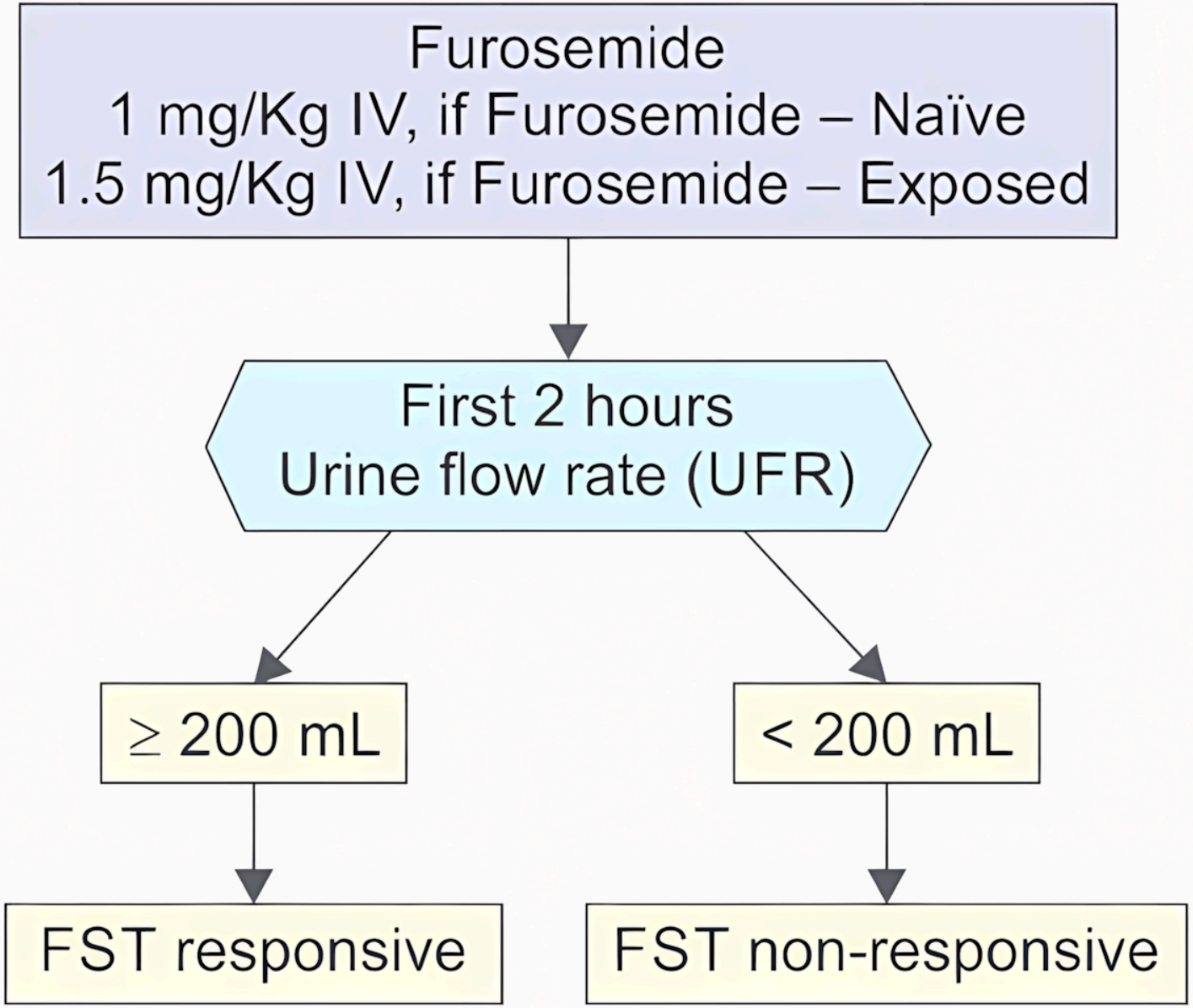


FIGURE 11.4 The new ADQI diagnostic criteria for acute kidney injury. The proposed diagram requires discrete values for biomarkers.[26,27,28] *AKI,* Acute kidney injury; *GFR,* glomerular filtration rate; *RRT,* renal replacement therapy.

Ronco C, Bellomo R, Kellum J, Ricci Z. Critical Care Nephrology E-Book. Elsevier Health Sciences; 2017.

Flowchart 1: Furosemide stress test

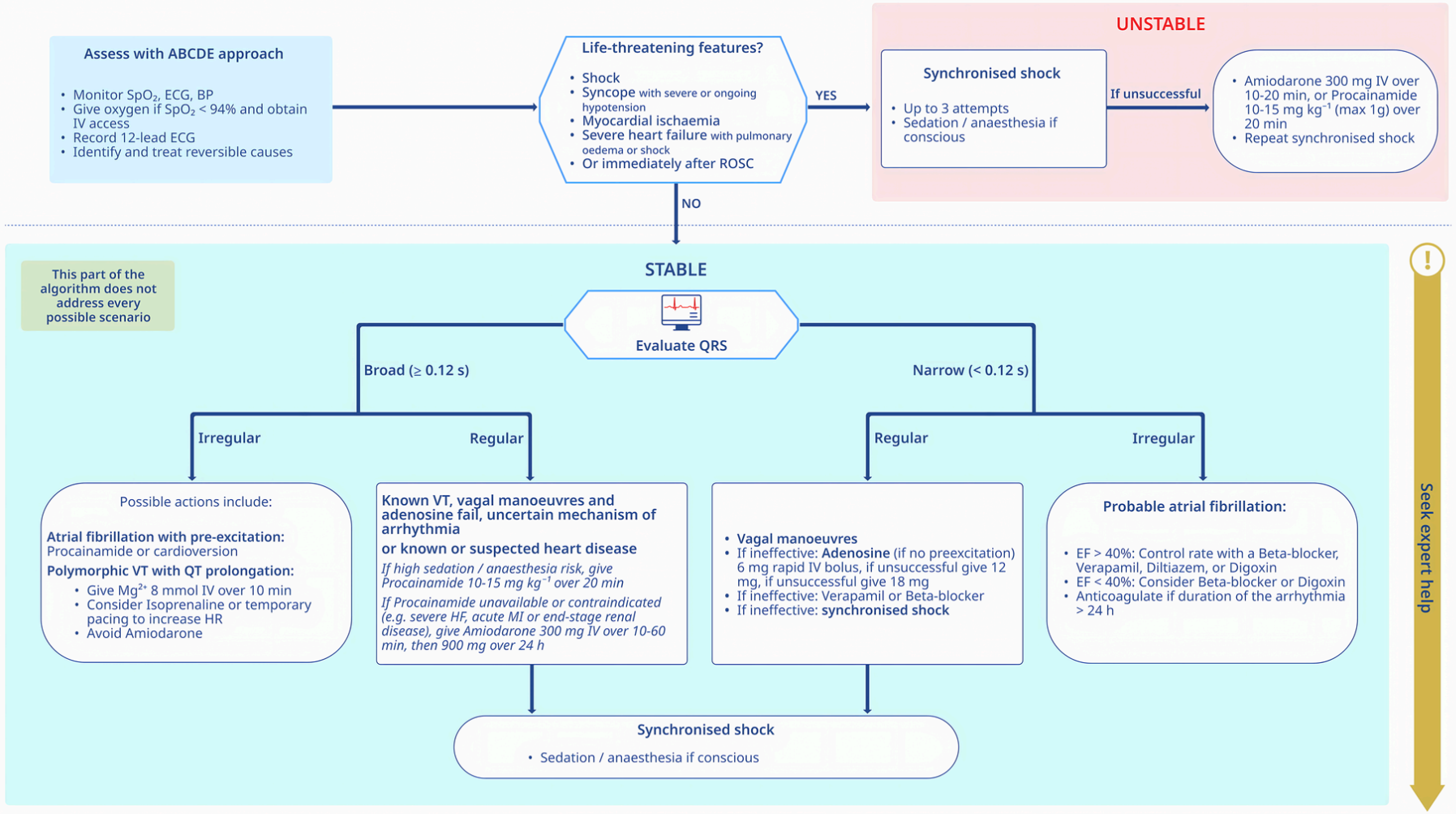


Rajasekaran KK. Furosemide Stress Test in Predicting Acute Kidney Injury Outcomes. Indian Journal of Critical Care Medicine. 2020;24(S3):100-101. doi:10.5005/jp-journals-10071-23381



Tachyarrhythmias

GUIDELINES 2025
EUROPEAN RESUSCITATION COUNCIL

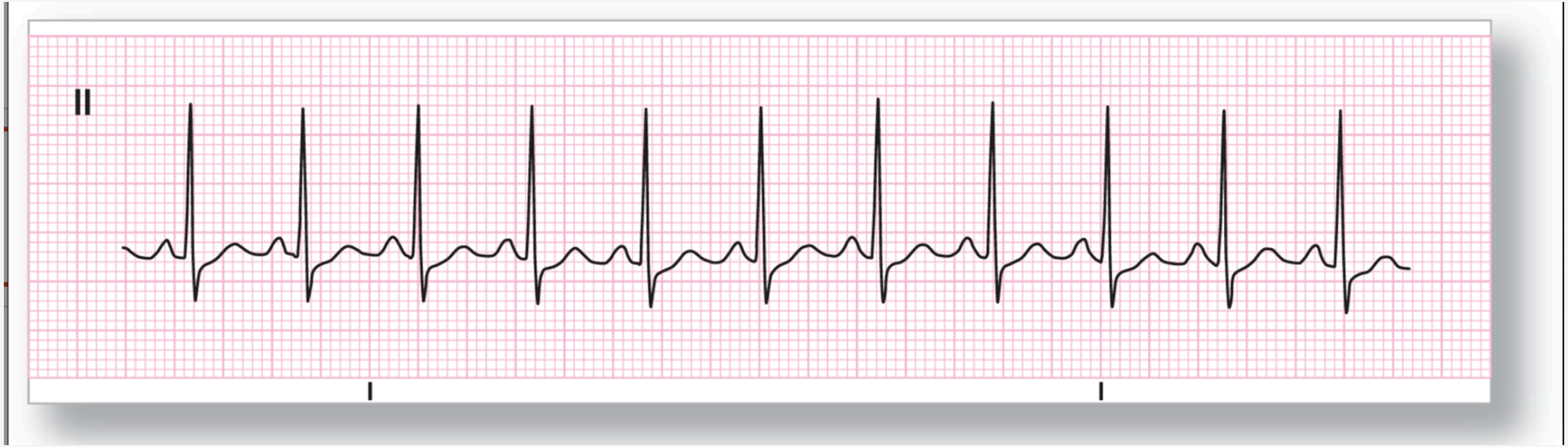


*This algorithm is for peri-arrest tachyarrhythmia of abnormal origin. It is not for sinus tachycardia where the underlying cause should be treated - do not try to normalise the heart rate in sinus tachycardia with antiarrhythmic medication or synchronised cardioversion.

Seek expert help if the tachyarrhythmia and, or life-threatening features persist.

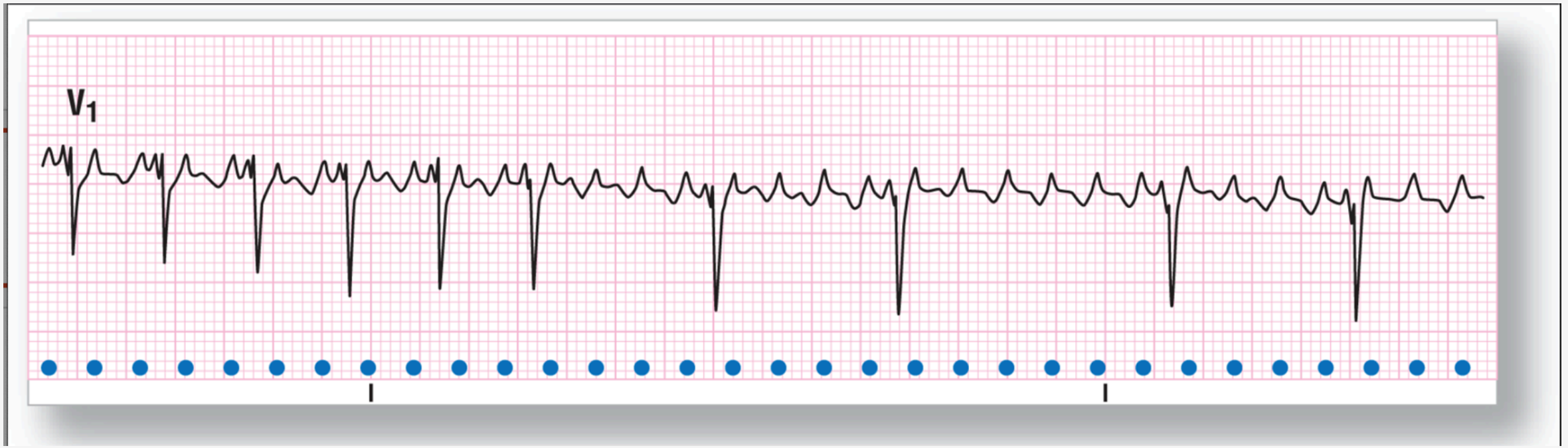
Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110769

Sinus tachycardia



Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Atrial Flutter



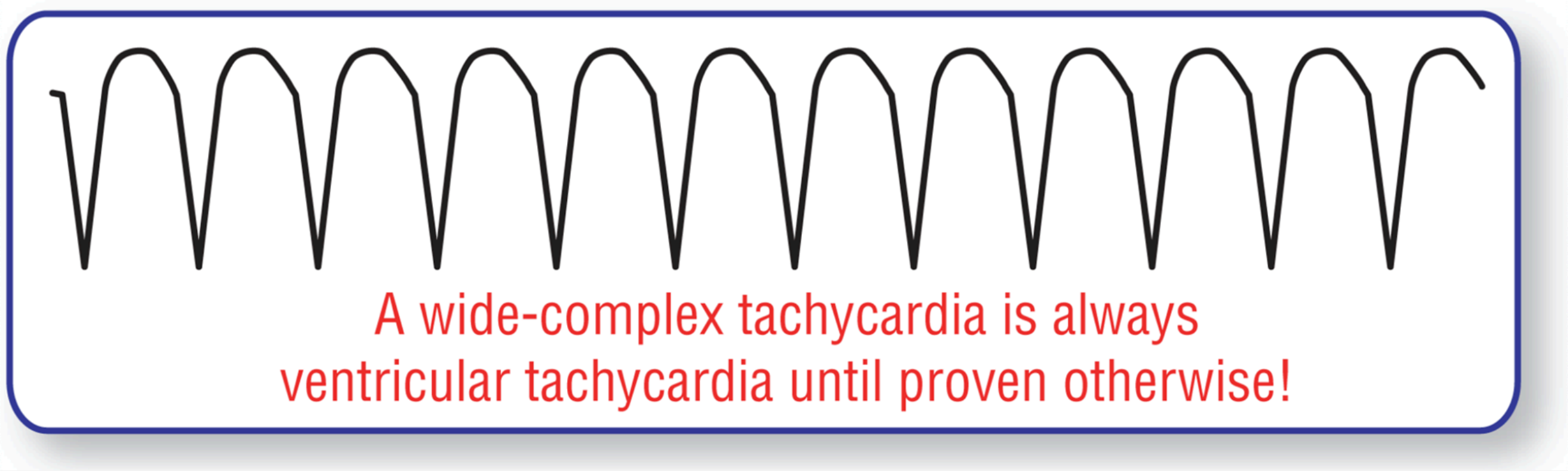
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Atrial Fibrillation



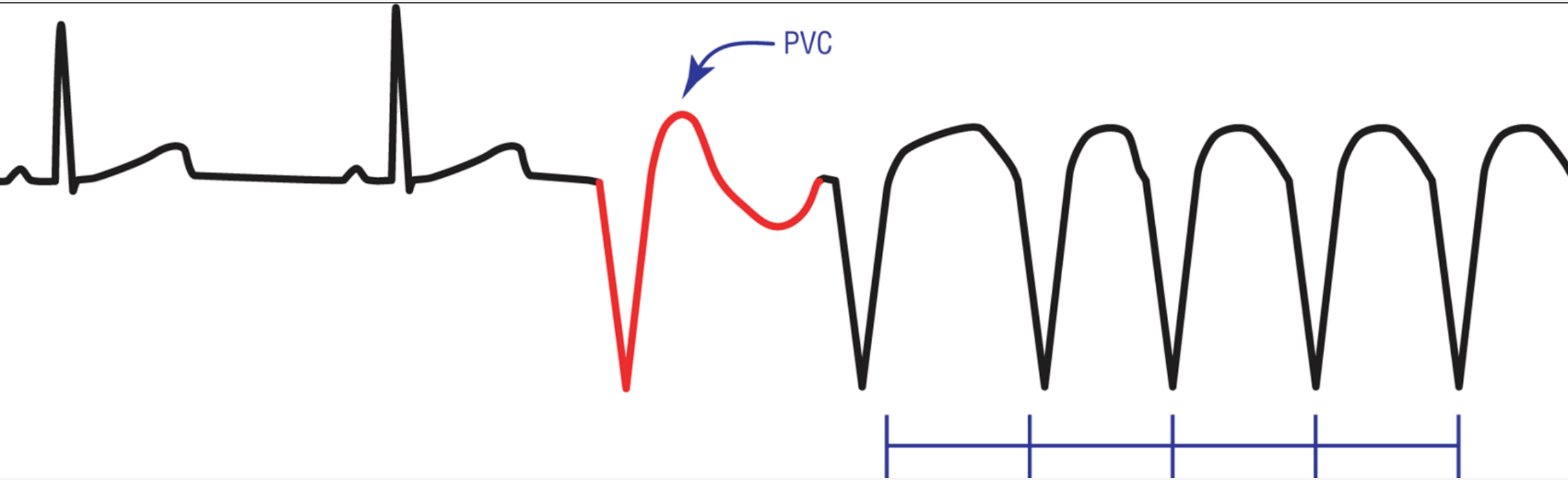
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Wide-complex Tachycardia



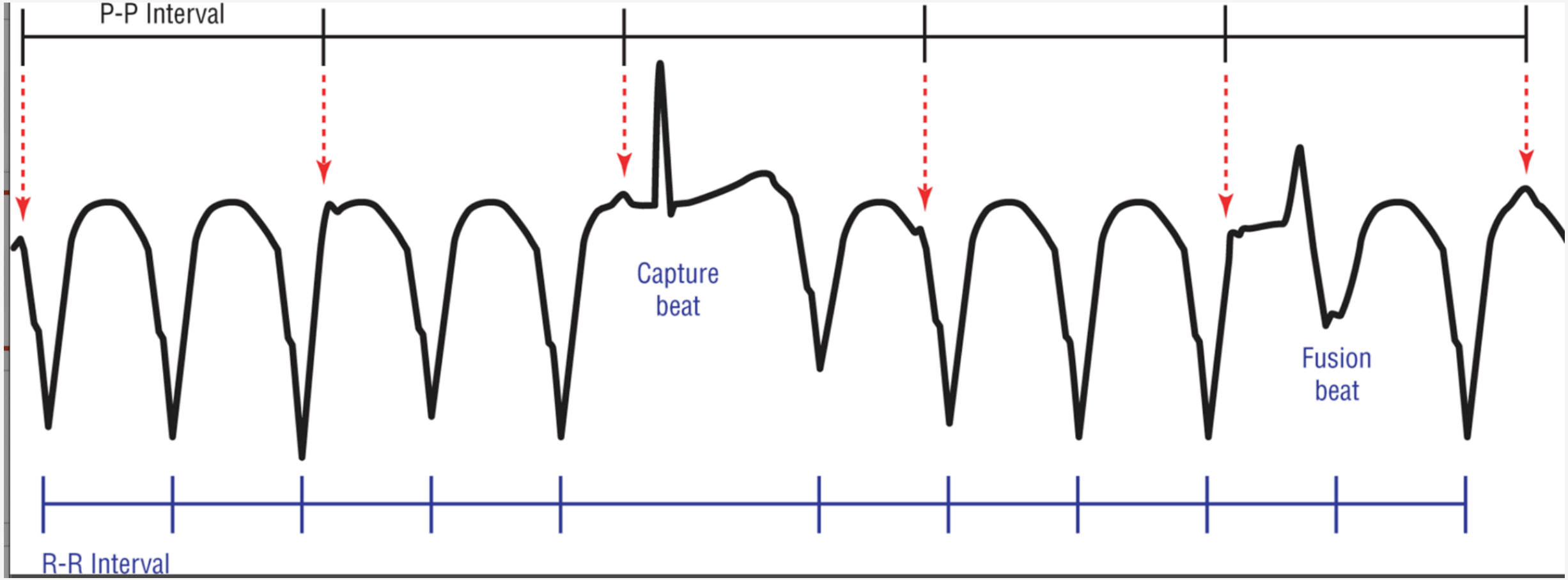
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Ventricular Tachycardia



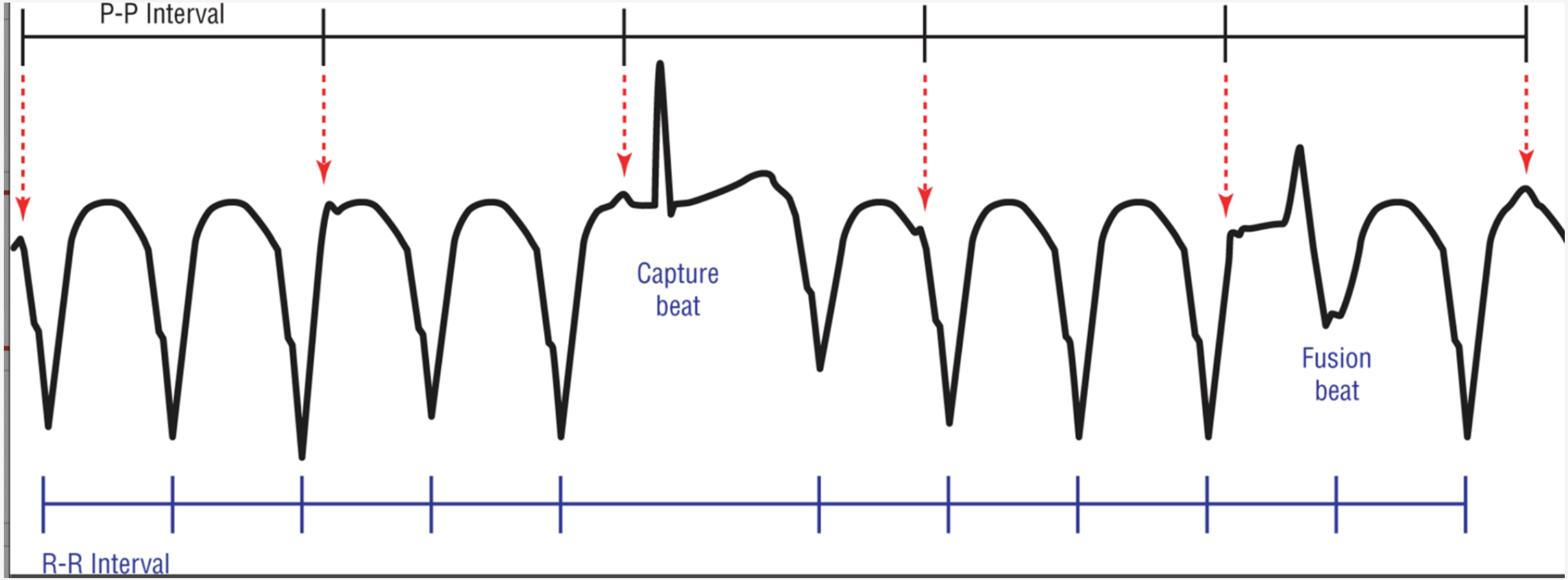
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Ventricular Tachycardia



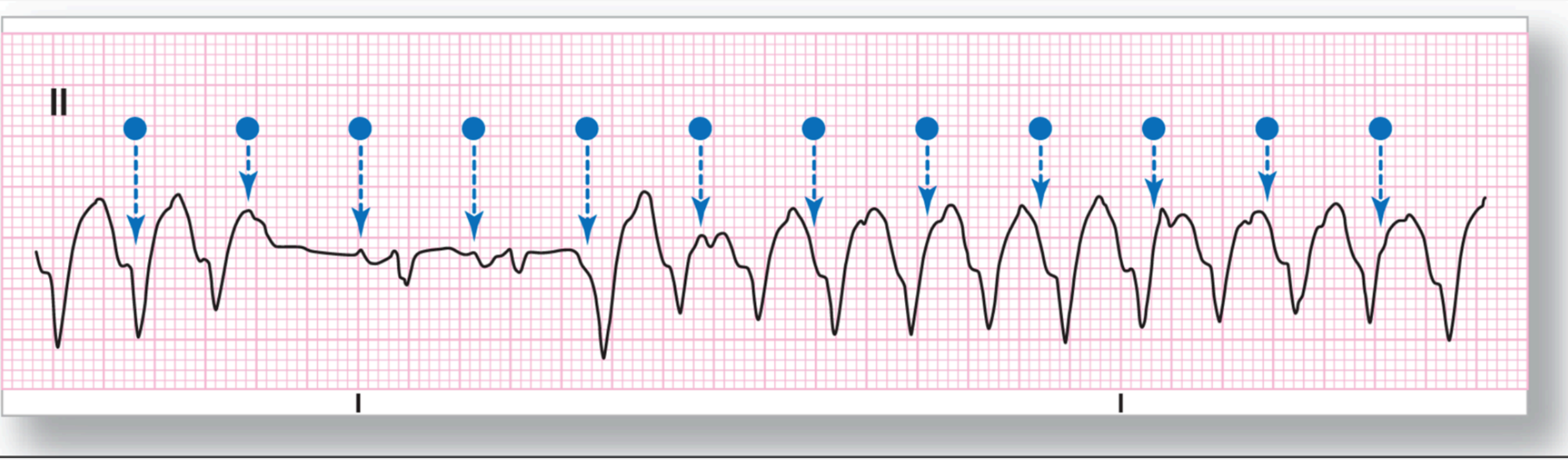
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Ventricular Tachycardia



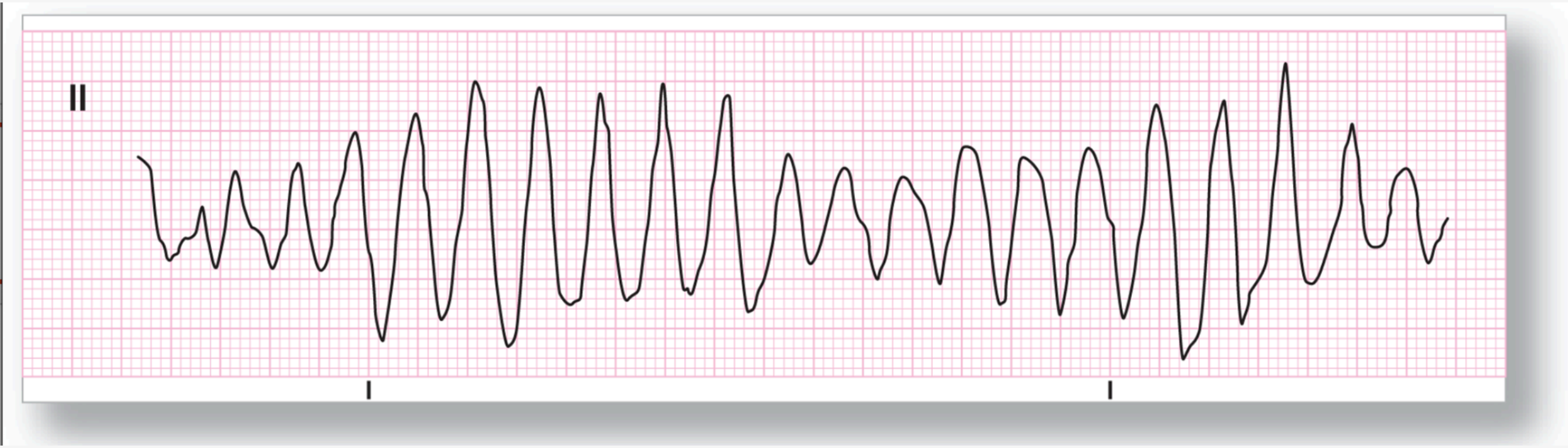
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Ventricular Tachycardia



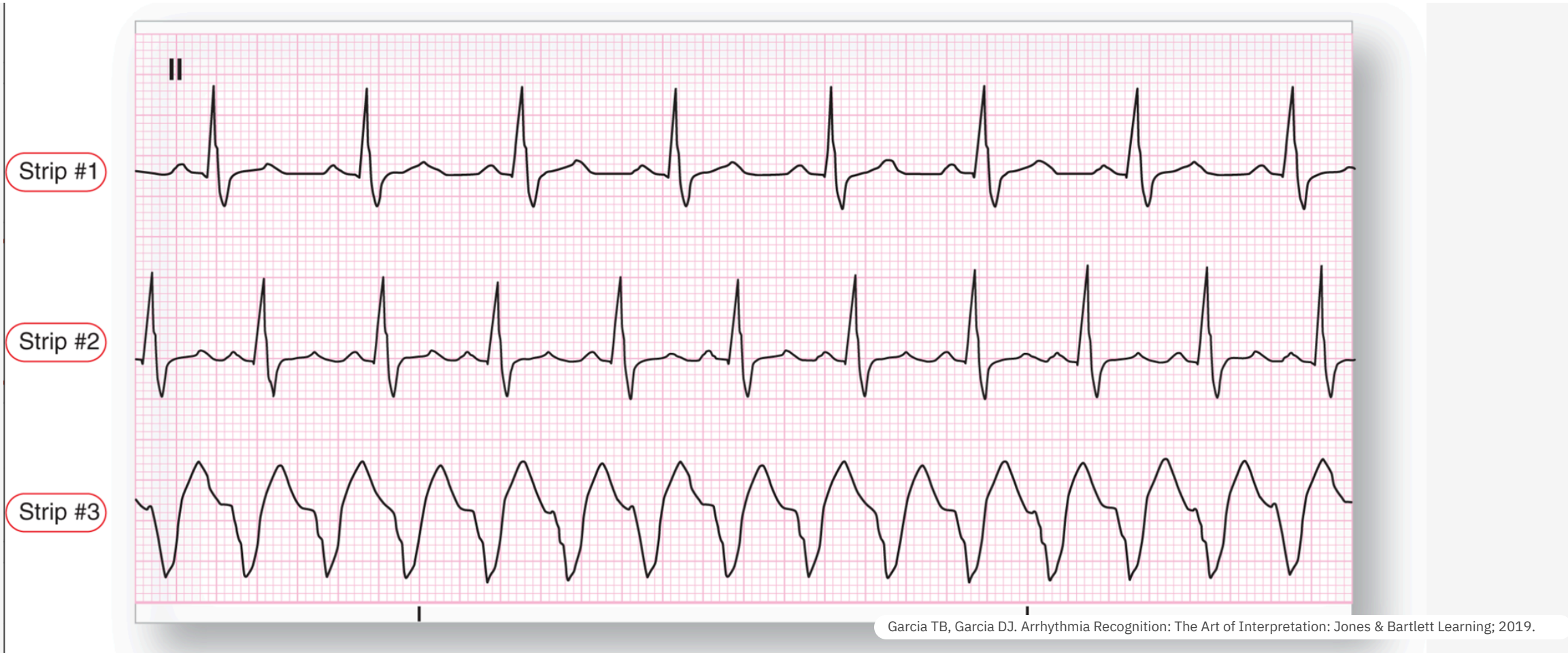
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Ventricular Tachycardia - Torsade de pointes

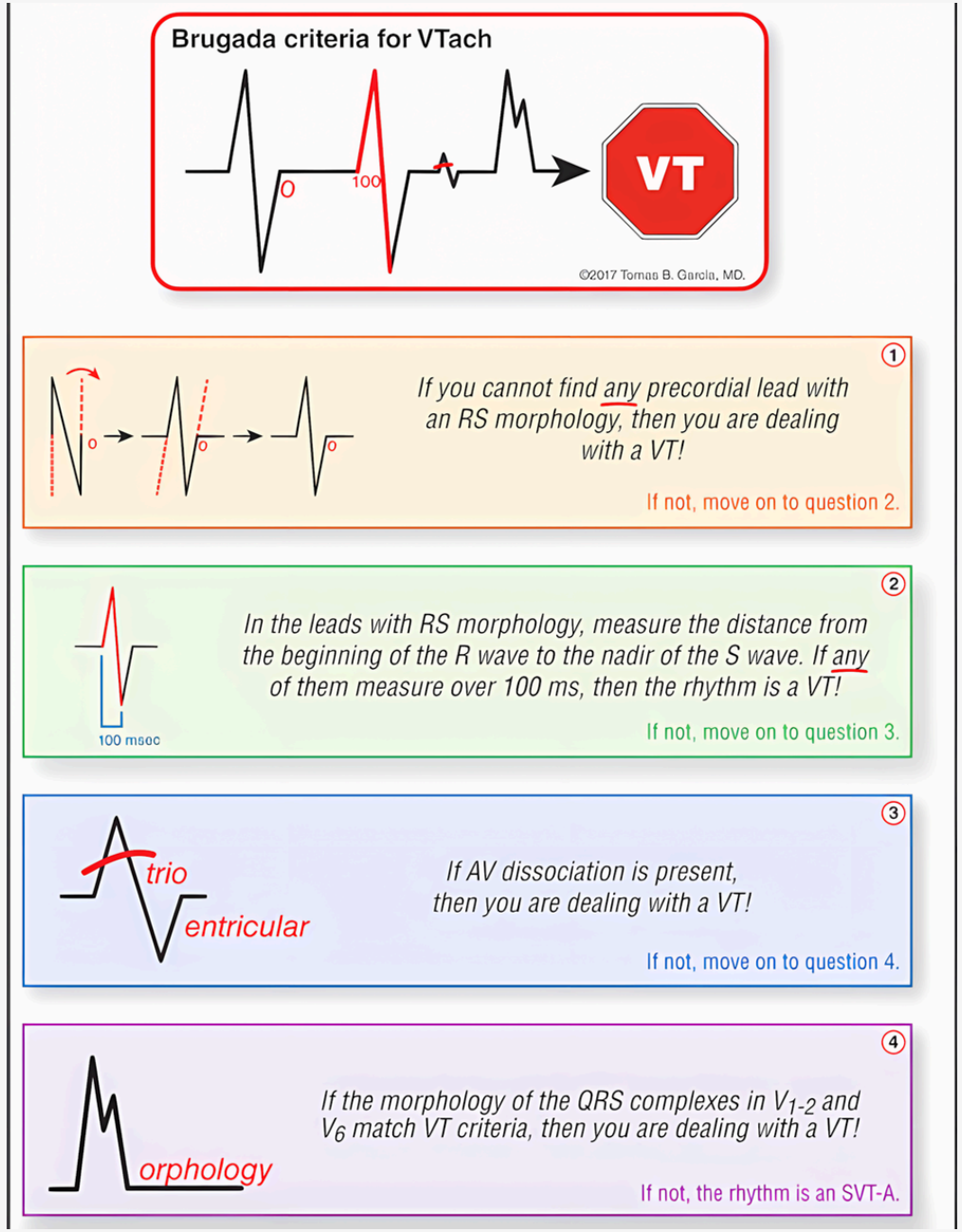


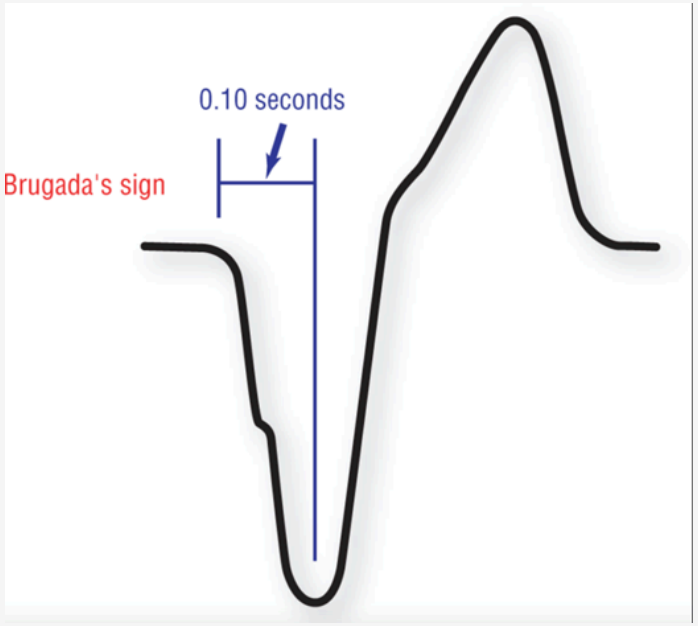
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

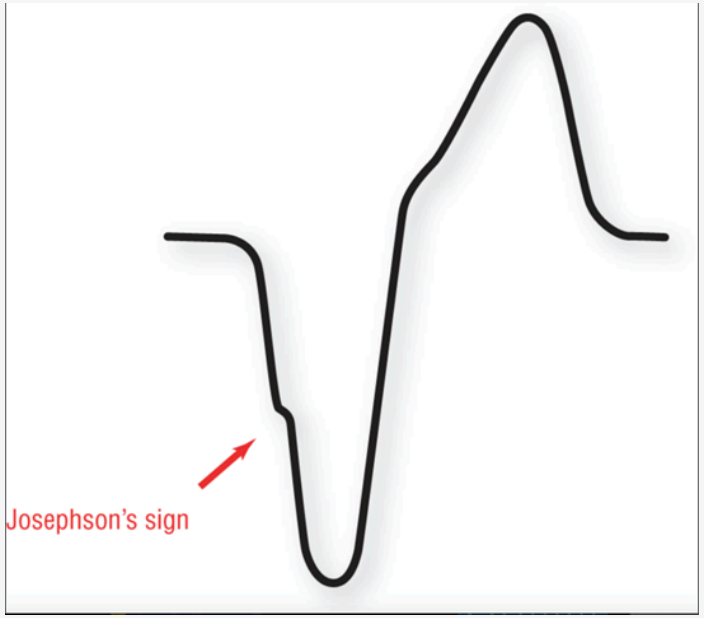
Supraventricular Tachycardia with aberrancy - Right Bundle Branch Block



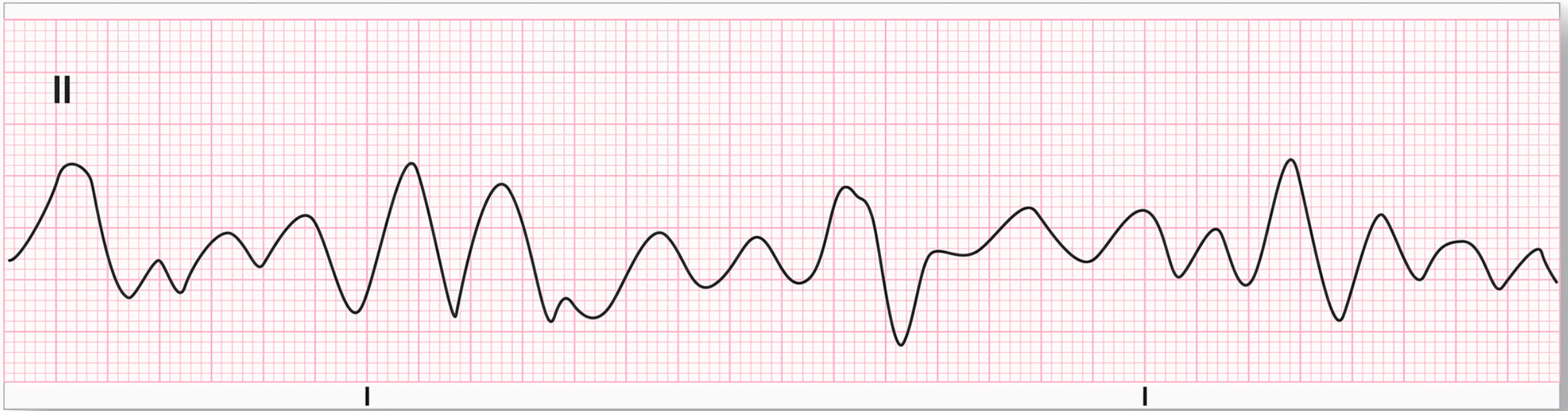
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

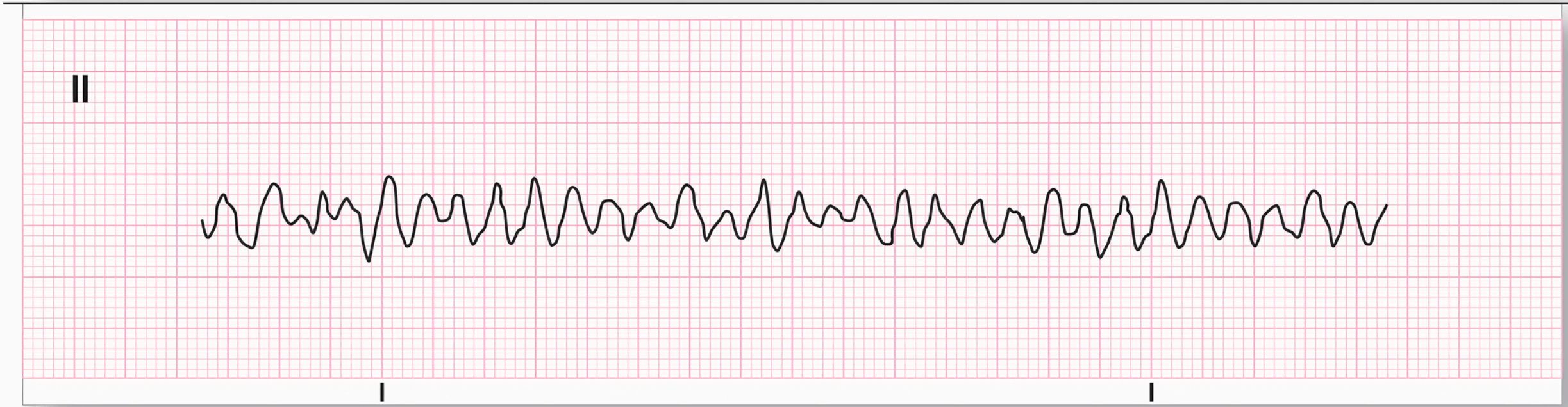






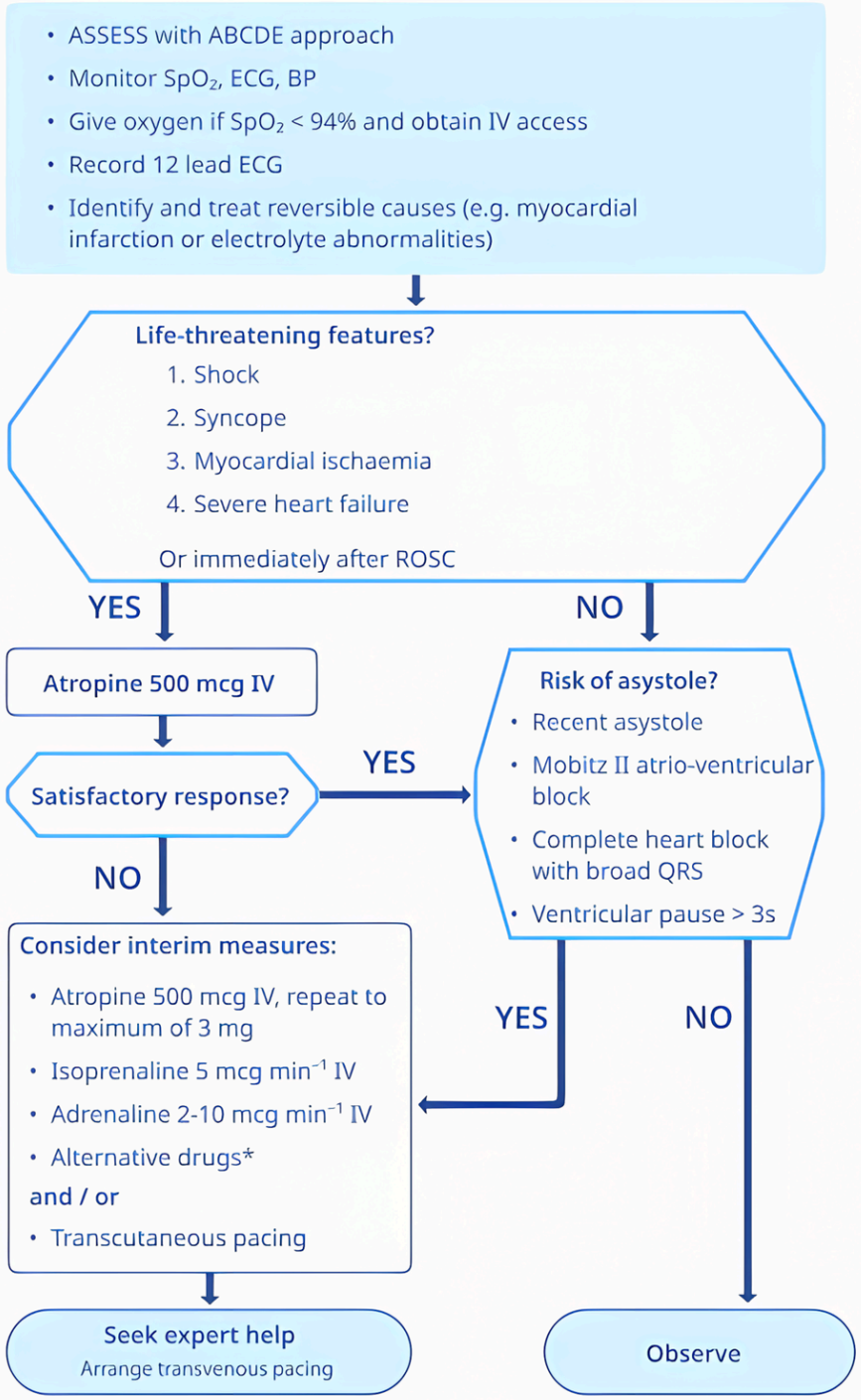
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.





Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: The Art of Interpretation. Jones & Bartlett Learning; 2019.

Bradycardia

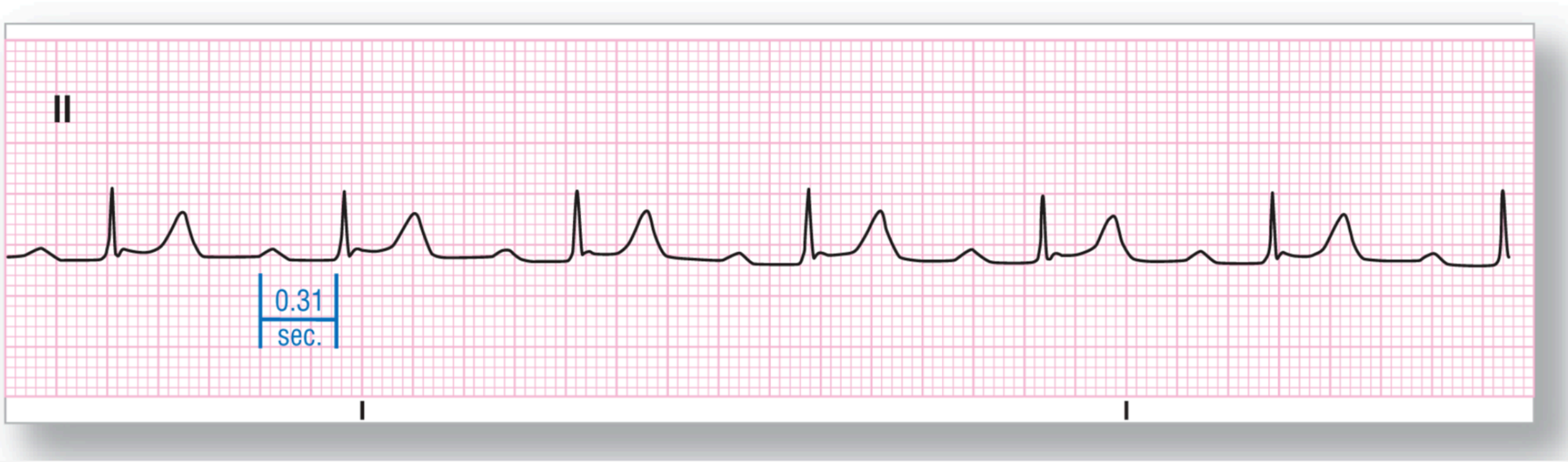


Bradycardia

- If bradycardia is accompanied by adverse signs, give atropine 500 µg IV (IO) and, if necessary, repeat every 3–5 min to a total of 3 mg.
- If treatment with atropine is ineffective, consider second-line drugs. These include isoprenaline (5 µg min^{-1} starting dose), and adrenaline (2–10 µg min^{-1}).
- For bradycardia in patients with cardiac transplant or spinal cord injury, consider giving aminophylline (100–200 mg slow intravenous injection). Do not give atropine to patients with cardiac transplants – it can cause a high-degree atrioventricular block or even sinus arrest – use aminophylline.•Consider giving glucagon if beta-blockers or calcium channel blockers are a potential cause of the bradycardia.
- Do not give atropine to patients with high-degree atrioventricular block and wide QRS. It is ineffective and may worsen the block.
- Consider pacing in patients who are unstable, with symptomatic bradycardia refractory to drug therapies.○Establish early transvenous pacing in unstable patients with symptomatic bradycardia.○Consider transthoracic (transcutaneous) pacing as a bridge to transvenous pacing or when transvenous pacing is not readily available.
- Whenever a diagnosis of asystole is made, check the ECG carefully for the presence of P waves because, unlike true asystole, this is more likely to respond to cardiac pacing.
- If atropine is ineffective and transvenous/transcutaneous pacing is not immediately available, fist pacing can be attempted while waiting for pacing equipment.

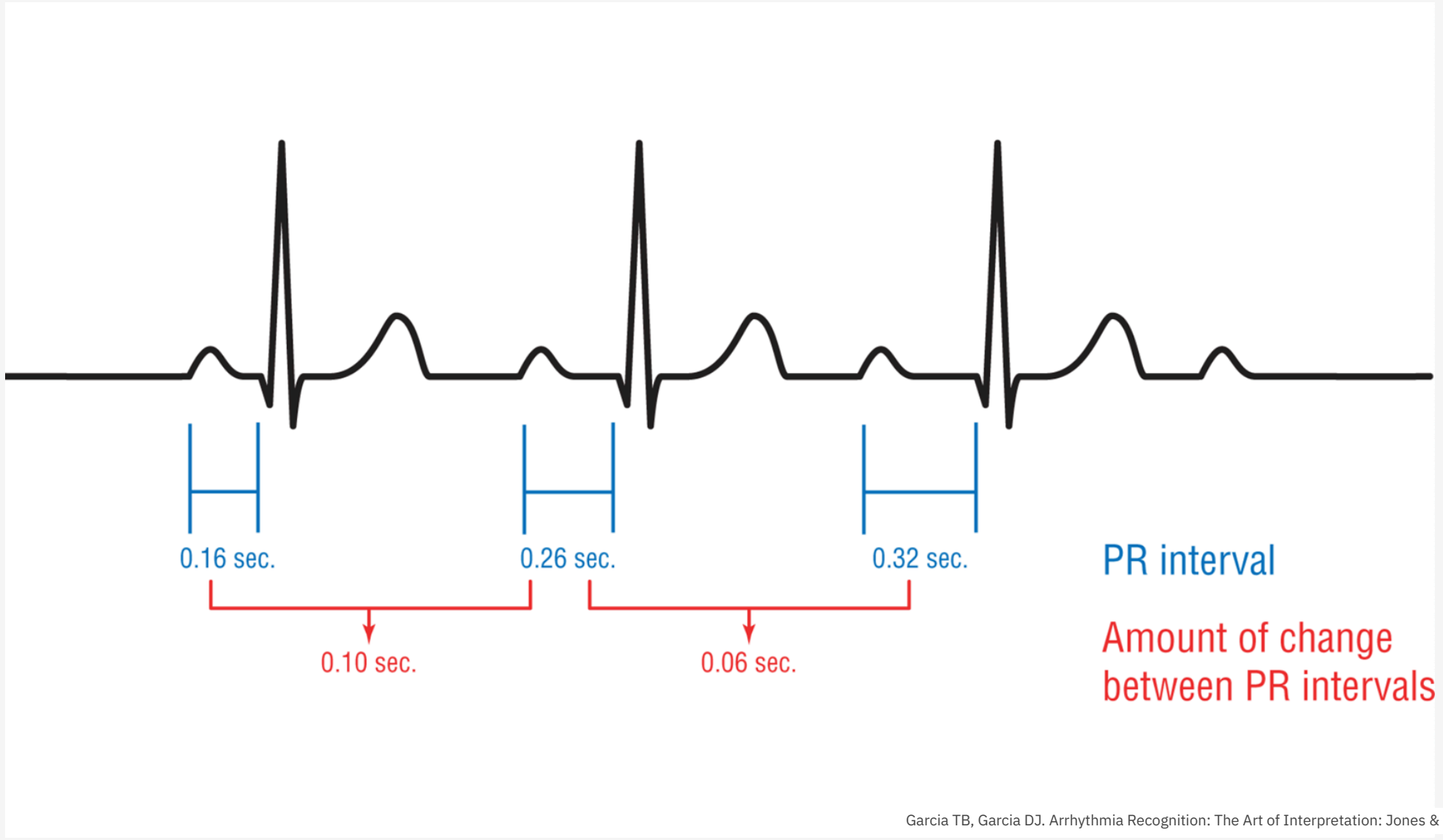
Soar J, Böttiger BW, Carli P, et al. European resuscitation council guidelines 2025 adult advanced life support. Resuscitation. 2025;215. doi:10.1016/j.resuscitation.2025.110769

Atrioventricular Block I



Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Atrioventricular Block II - Mobitz I



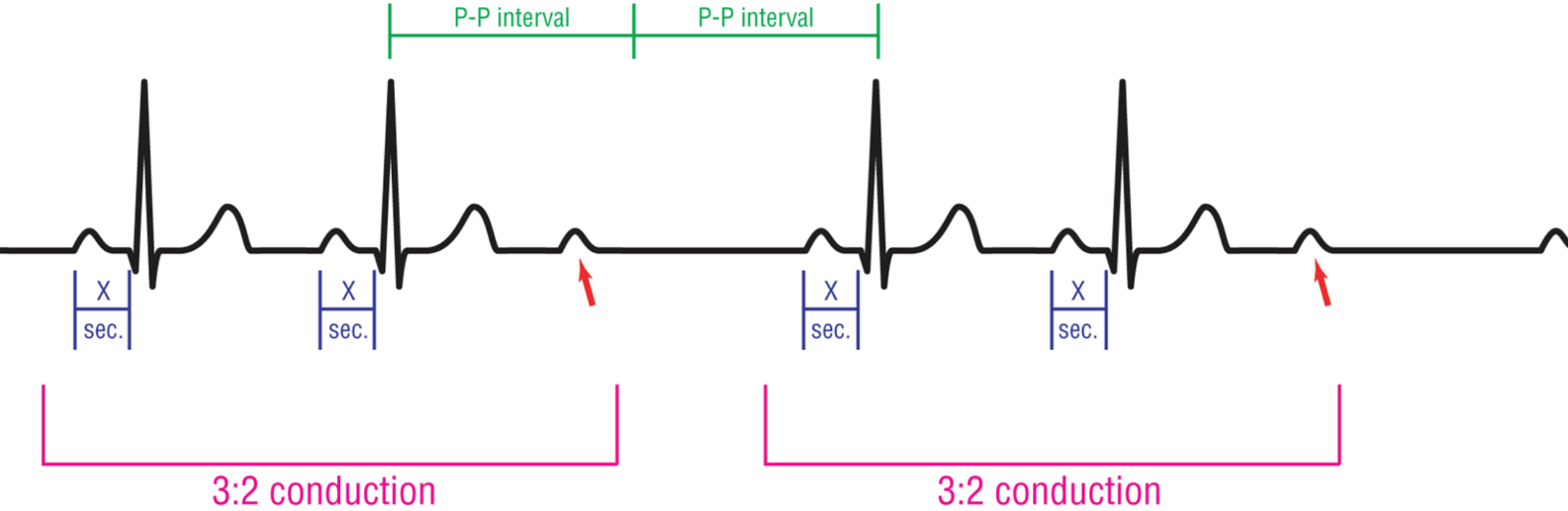
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Atrioventricular Block II - Mobitz I



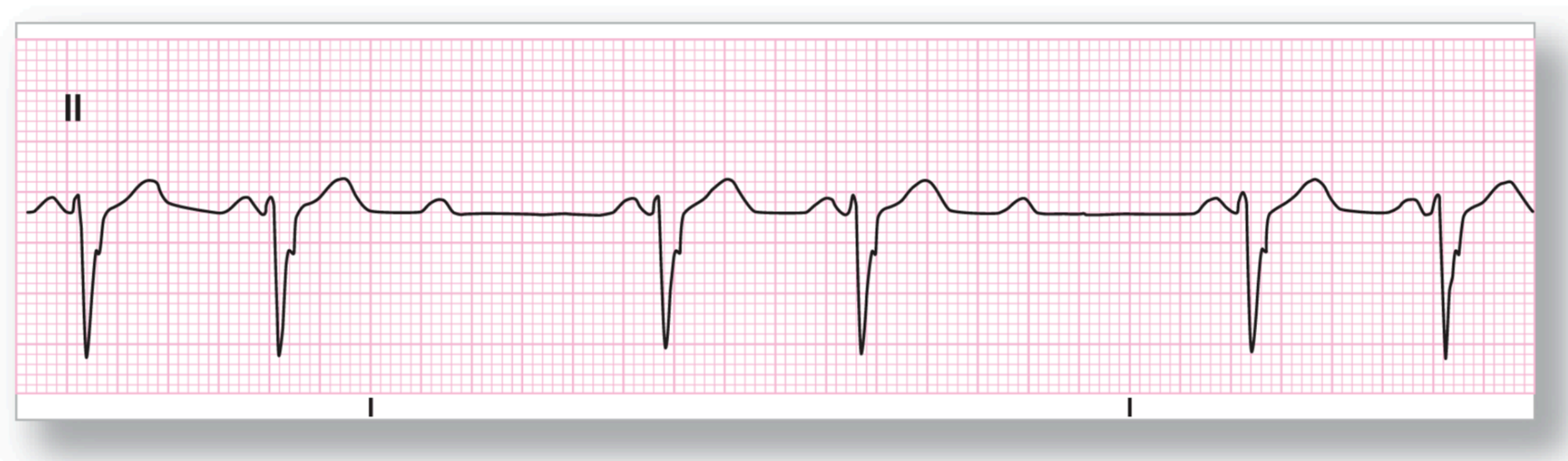
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Atrioventricular Block II - Mobitz II



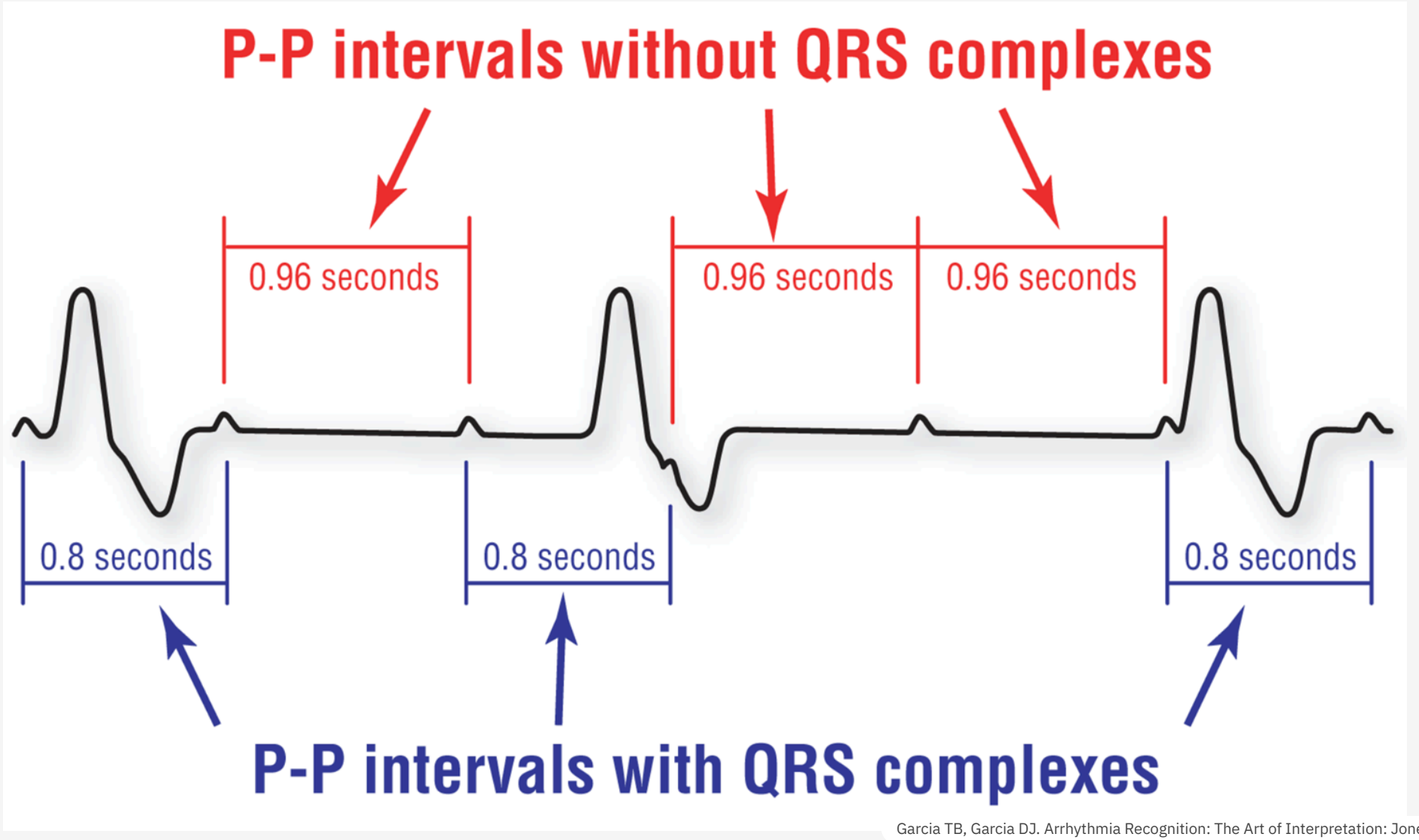
Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Atrioventricular Block II - Mobitz II

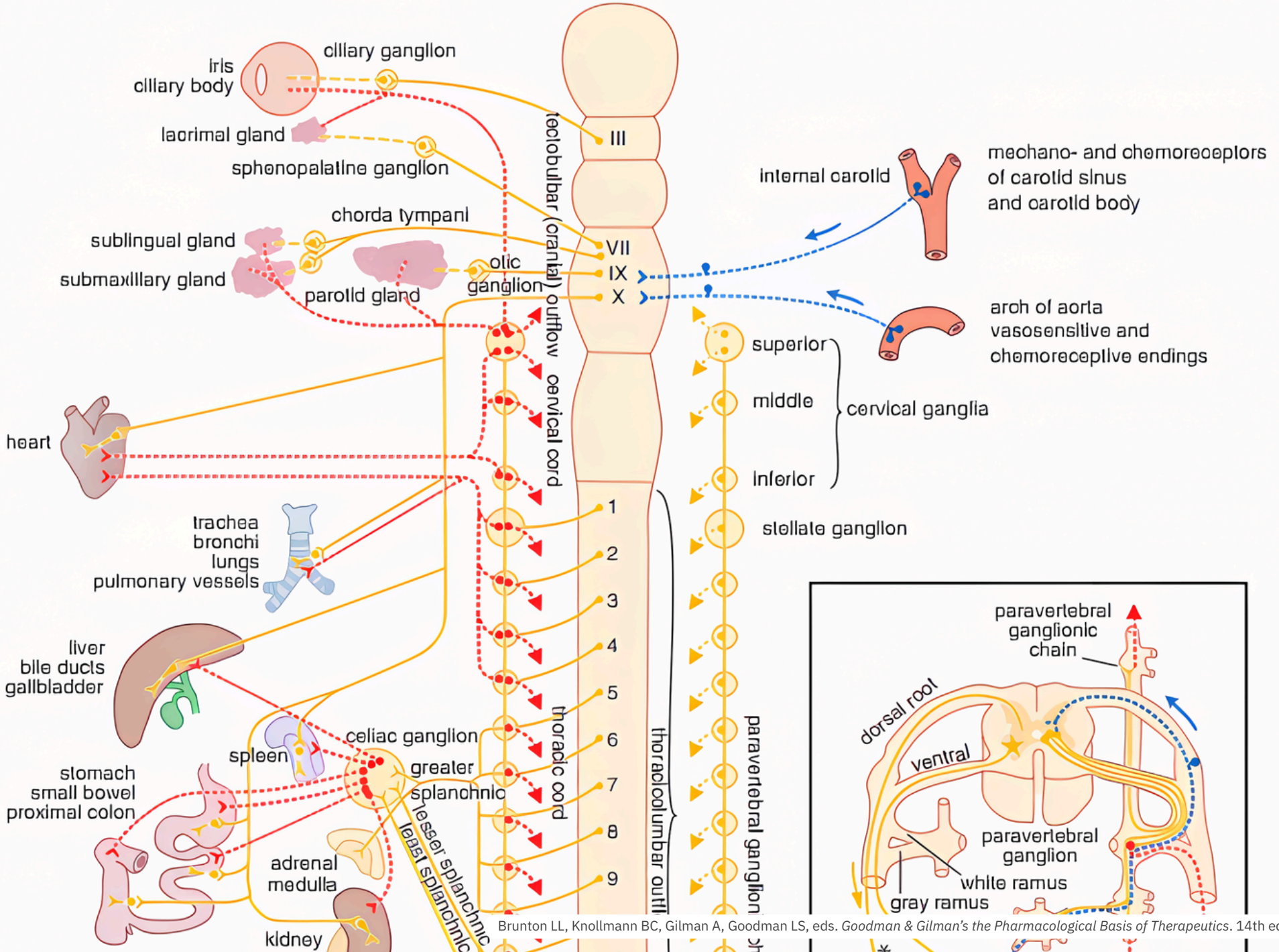


Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.

Atrioventricular Block III



Garcia TB, Garcia DJ. Arrhythmia Recognition: The Art of Interpretation: Jones & Bartlett Learning; 2019.



Brunton LL, Knollmann BC, Gilman A, Goodman LS, eds. *Goodman & Gilman's the Pharmacological Basis of Therapeutics*. 14th ed. McGraw Hill; 2023.

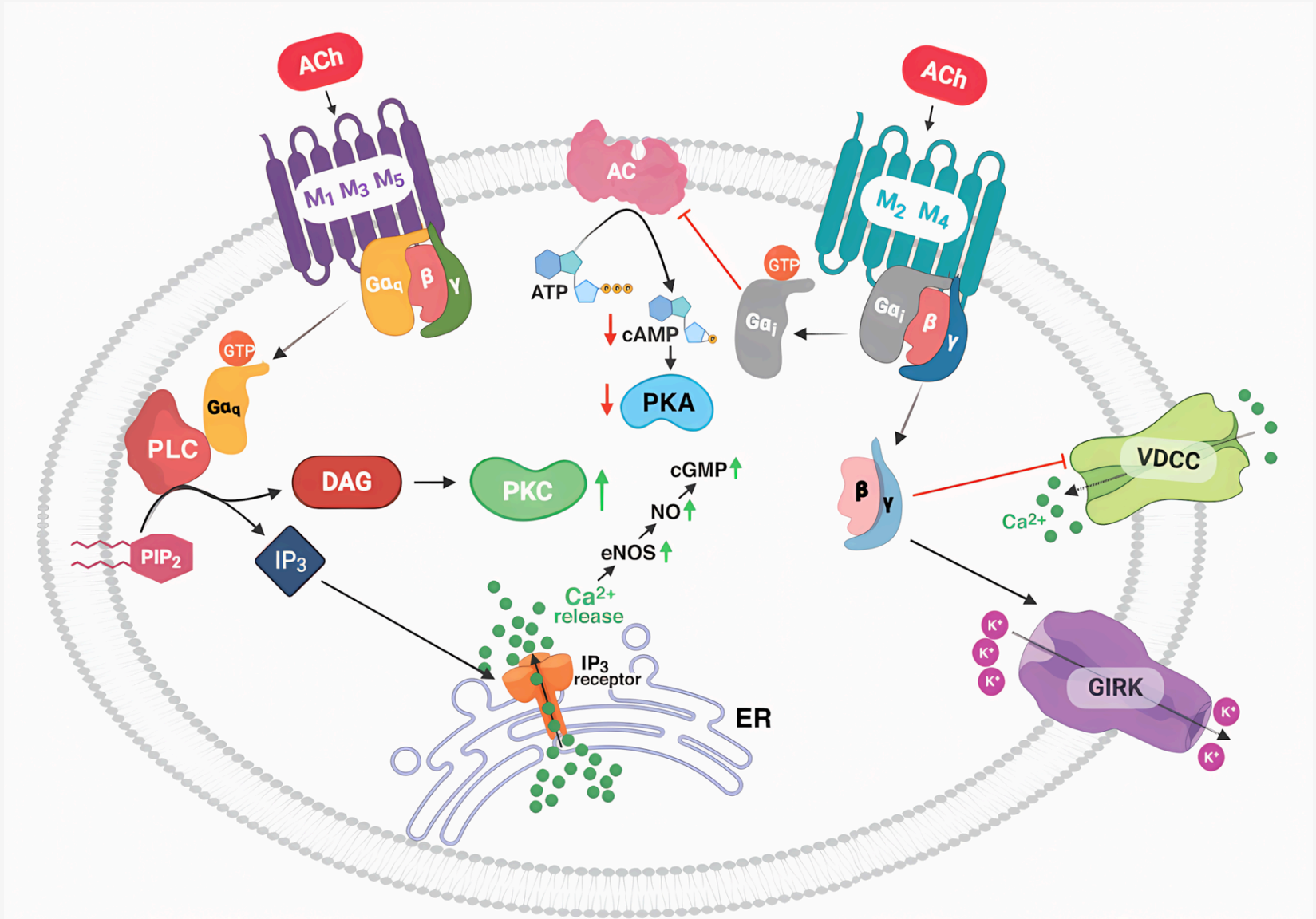
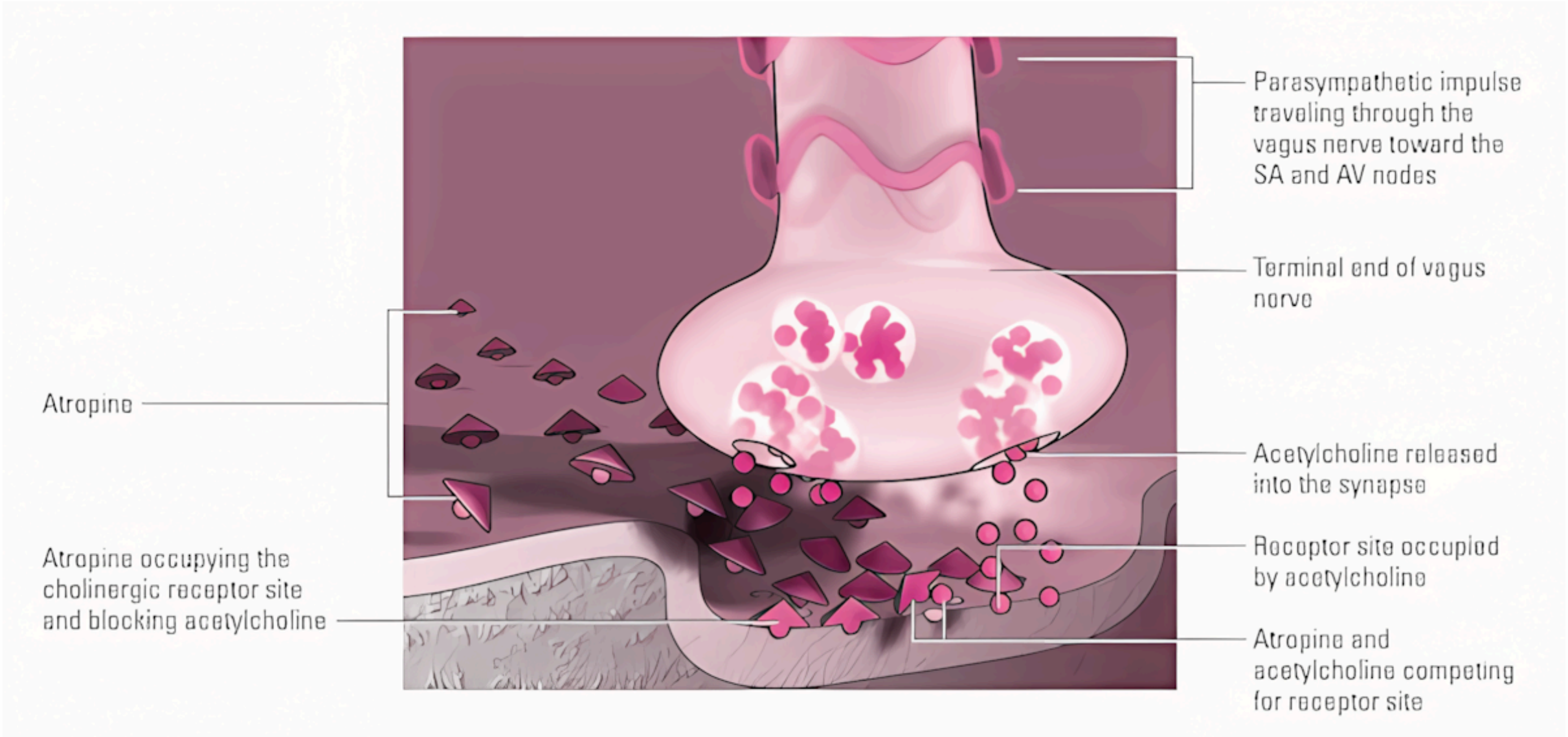


Figure 11–1 *G protein-coupling properties of the M_1–M_5 muscarinic receptors.* Note that the odd-numbered receptors (M_1, M_3, and M_5) selectively couple to G proteins of the G_q family, while the even-numbered receptors (M_2 and M_4) preferentially activate G_i-type G proteins. Some of the major downstream signaling pathways are shown. AC, adenylyl cyclase; DAG, diacylglycerol; IP_3, inositol 1,4,5-trisphosphate; PKA, protein kinase A (cyclic AMP-dependent protein kinase); GIRK channel; G protein-coupled inwardly rectifying K^+ channel; PLC, phospholipase C; PIP_2, phosphatidylinositol 4,5-bisphosphate; VDCC, voltage-dependent Ca^{2+} channel.

Brunton LL, Knollmann BC, Gilman A, Goodman LS, eds. *Goodman & Gilman's the Pharmacological Basis of Therapeutics*. 14th ed. McGraw Hill; 2023.



https://www.nursingcenter.com/blogs-plus/blogs/blogs-post?identifier=Atropine#/post/Atropine

Paradoxical Bradycardia with Low-Dose Atropine



Adult doses < 0.5 mg

Low doses of the medication can paradoxically cause bradycardia, or a slow heart rate, in some patients.



Slow IV pushes

Administering the medication slowly through an IV can also lead to a temporary decrease in heart rate.



Transient effects

The bradycardia caused by low doses or slow administration is likely to be temporary and short-lived.

Patel P, McLendon K, Preuss CV. Atropine. In: StatPearls. StatPearls Publishing; 2025. Accessed December 12, 2025. http://www.ncbi.nlm.nih.gov/books/NBK470551/

Hyper/hypokalaemia and other electrolyte disorders

Hyperkalaemia, non cardiac arrest

- Hyperkalaemia affects 1 to 10 percent of hospitalised patients.
- Patients with chronic kidney disease show higher rates of in hospital major cardiovascular events and arrhythmias when hyperkalaemia occurs.
- Dialysis patients experience severe hyperkalaemia more often than non dialysis patients, 45.8 percent versus 10.3 percent.
- In hospital mortality is higher in hyperkalaemia, 18.1 percent, compared with hypokalaemia, 5 percent, and normokalaemia, 3.9 percent.
- Mortality risk increases 3.9 fold when serum potassium exceeds 6.5 mmol per litre.

Renal and cardiac disease commonly coexist and increase hyperkalaemia risk, often worsened by ACE inhibitors, angiotensin II antagonists, and mineralocorticoid receptor antagonists.

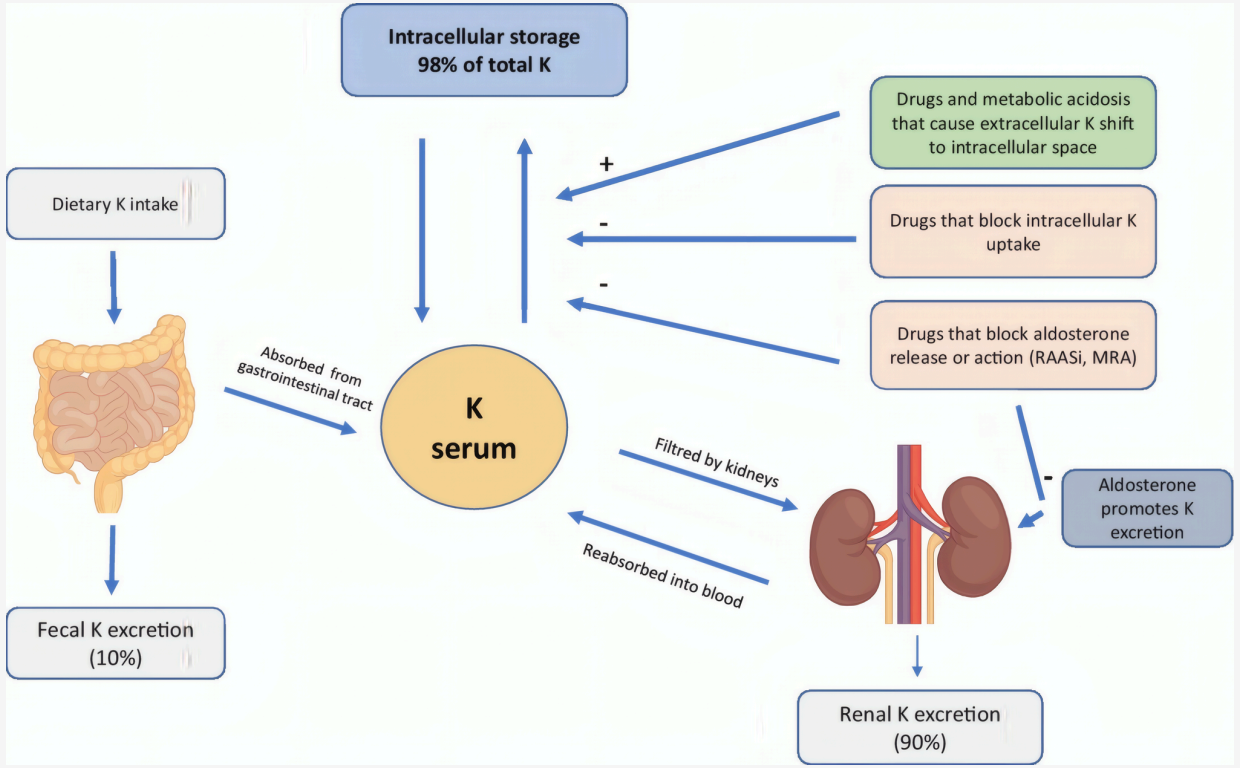
Reducing or stopping these drugs because of hyperkalaemia correlates with poorer outcomes.
New potassium binders such as sodium zirconium cyclosilicate and patiromer support cardio renal protection.

Hyperkalaemia is defined by serum potassium above 5.5 mmol per litre, with severity guiding management.

Classification:

- mild 5.5 to 5.9 mmol per litre,
- moderate 6.0 to 6.4 mmol per litre,
- severe at or above 6.5 mmol per litre, extreme at or above 9.0 mmol per litre.

Risk increases when multiple factors coexist, for example the combination of ACE inhibitors or mineralocorticoid receptor antagonists with chronic kidney disease.



Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Costa D, Patella G, Provenzano M, et al. Hyperkalemia in CKD: an overview of available therapeutic strategies. *Front Med*. 2023;10. doi:10.3389/fmed.2023.1178140

Summary of cardiac action potential with the ionic currents involved



Cellular Electrophysiology

Excitability refers to the conversion of a small triggering stimulus into a large, stereotypical response (the action potential)



Action Potential

The cardiac electrical cycle has been schematically divided in five phases, four of them describing the AP contour and one the diastolic interval



Ionic Currents

Two autoregenerative processes cooperate to determine the threshold: activation of an inward current (INa) and removal of the outward current responsible for stabilizing diastolic potential (IK)

The summary covers the key concepts of cellular electrophysiology, action potential characteristics, and the ionic currents involved in cardiac excitability and action potential generation.

Electrophysiological effects of hyperkalaemia

Systemic hyperkalaemia depolarizes the resting membrane potential by about 18 mV when potassium increases from 4 to 8 mmol per litre.

Action potential duration shortens because extracellular potassium increases potassium channel conductance.

Early hyperkalaemia decreases the effective refractory period. Worsening hyperkalaemia produces postrepolarization refractoriness and prolongs the effective refractory period.

Conduction velocity shows a biphasic pattern.

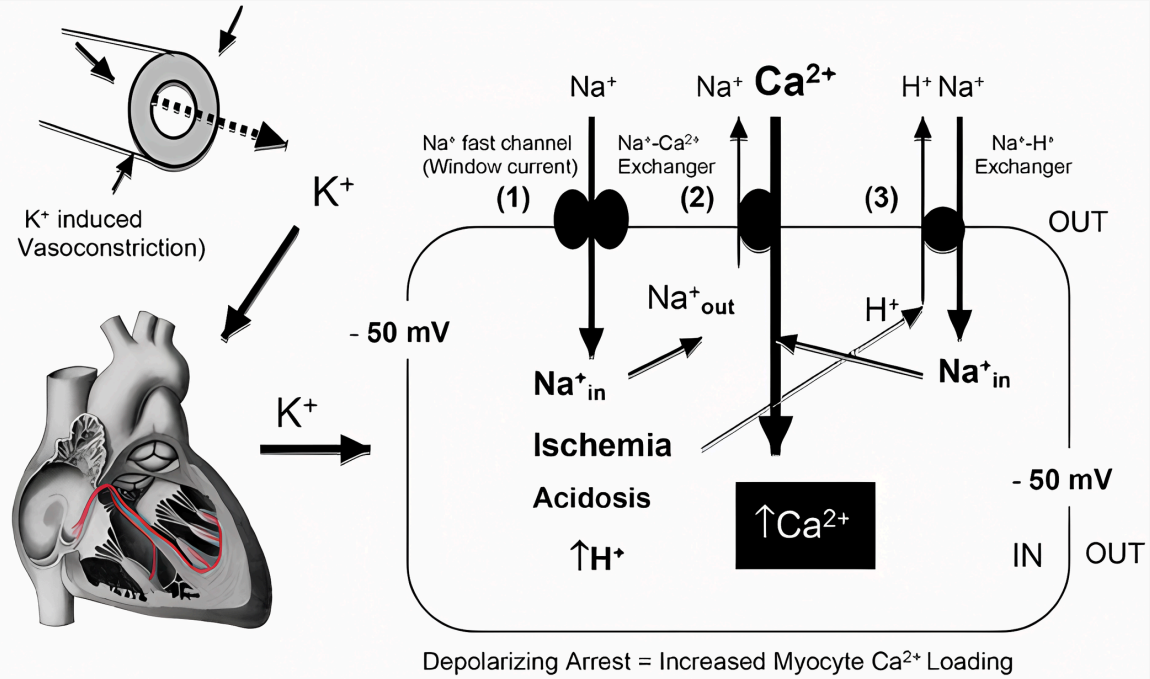
- Mild hyperkalaemia accelerates conduction because the membrane potential moves closer to the sodium channel activation threshold.
- Higher potassium levels reduce sodium channel availability and slow conduction, leading to propagation failure above about 14 mmol per litre.

Hyperkalaemia accentuates conduction velocity restitution because sodium channel recovery is slower at depolarized resting potentials.

Premature beats conduct more slowly even when conduction at normal rates appears preserved.

Weiss JN, Qu Z, Shivkumar K. Electrophysiology of hypokalemia and hyperkalemia. *Circ: Arrhythmia Electrophysiol.* 2017;10(3):e004667. doi:10.1161/CIRCEP.116.004667

"A schematic of the effect of hyperkalemia and prolonged myocardial membrane depolarization on Na^+ entry through the "window current" and the net influx of Ca^{2+} into the myocardial cell via the reversal of the Na^+/Ca^{2+} exchanger $3Na^+$ ions are extruded in exchange for 1 Ca^{2+} entry at a membrane potential of −50 mV]."



Dobson GP, Faggian G, Onorati F, Vinten-Johansen J. Hyperkalemic cardioplegia for adult and pediatric surgery: end of an era? *Front Physiol.* 2013;4. doi:10.3389/fphys.2013.00228

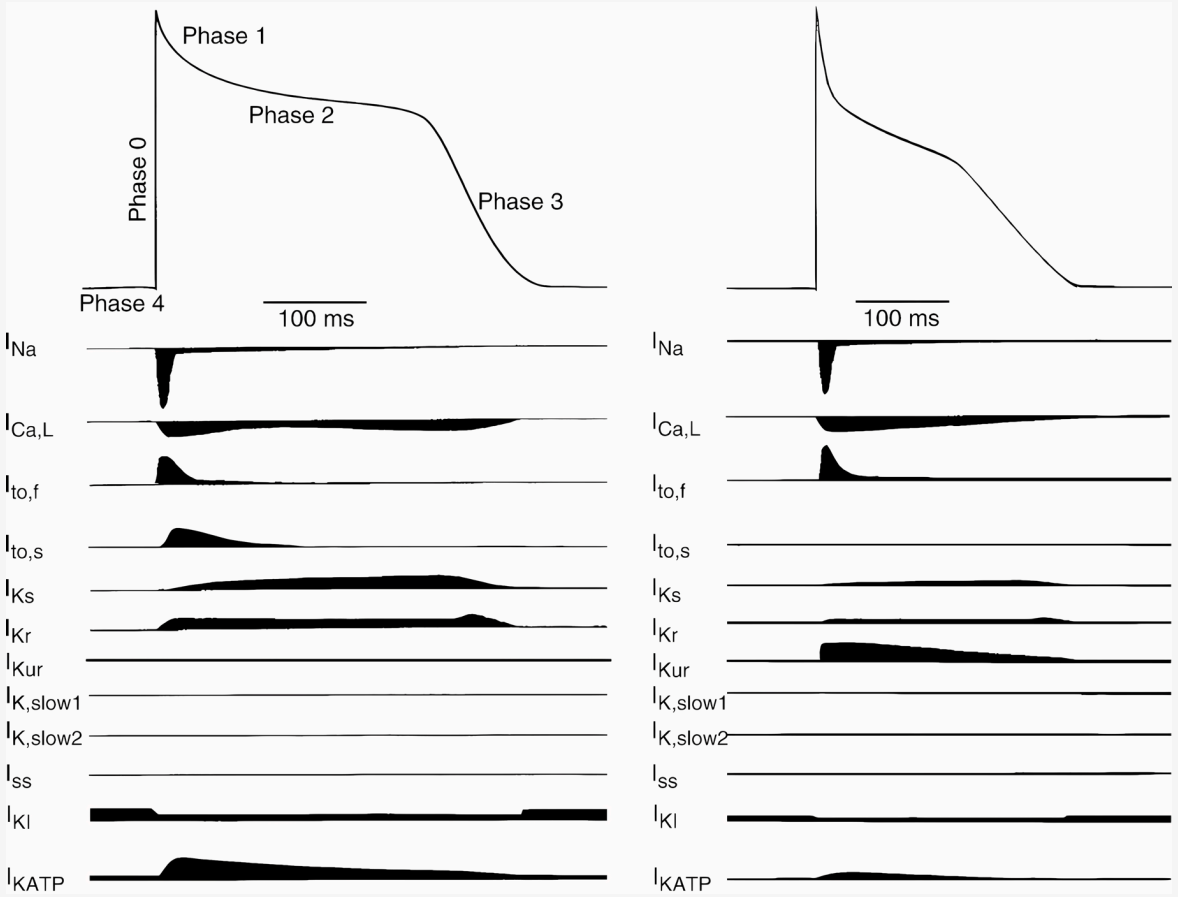
Resting membrane potential, phase 4.
The resting membrane potential is stable and negative, typically around −85 to −90 mV. High potassium permeability through inward rectifier potassium channels (IK1) dominates membrane conductance. The membrane potential remains close to the potassium equilibrium potential, which ensures electrical stability and readiness to respond to an external stimulus.

Rapid depolarisation, phase 0.
When a depolarising stimulus reaches threshold, fast voltage gated sodium channels open. A large inward sodium current (INa) causes a rapid upstroke of the membrane potential toward positive values. This phase determines conduction velocity in working myocardium.

Initial repolarisation, phase 1.
Fast inactivation of sodium channels occurs. A transient outward potassium current (Ito) produces a brief, partial repolarisation. The prominence of this phase varies across myocardial regions.

Plateau phase, phase 2.
Membrane potential remains near 0 mV. A balance exists between inward calcium current through L type calcium channels (ICaL) and outward potassium currents, mainly delayed rectifier potassium currents (IKr and IKs). Calcium entry during this phase triggers calcium induced calcium release from the sarcoplasmic reticulum and initiates contraction.

Final repolarisation, phase 3.
Calcium channels inactivate progressively. Outward potassium currents increase, primarily IKr followed by reactivation of IK1. These currents restore the membrane potential toward the potassium equilibrium potential. Return to resting state.
The membrane potential stabilises again at the resting level. Ionic gradients are maintained by membrane transporters, including the sodium potassium pump and the sodium calcium exchanger, which prepare the cell for the next action potential.



Macfarlane PW, Oosterom A van, Pahlm O, Kligfield P, Janse M, Camm J, eds. *Comprehensive Electrocardiology*. Springer; 2010.

Nerbonne JM, Kass RS. Molecular physiology of cardiac repolarization. *Physiological Reviews*. 2005;85(4):1205-1253. doi:10.1152/physrev.00002.2005

$$V_m = -61.5 \log\left(\frac{[K^+]_i}{[K^+]_o}\right)$$

Clinical effects of altered potassium levels • Hyperkalaemia affects excitability and conduction velocity in all excitable tissues.
• Potassium concentration is higher inside the cell than outside, and this gradient determines the resting membrane potential.
• The Nernst equation describes the relationship: Vm = −61.5 log([K+]i/[K+]o).
• Low extracellular potassium decreases inward rectifier potassium conductance, produces hyperpolarization, and shortens action potential duration.
• High extracellular potassium increases inward rectifier conductance and depolarizes the resting membrane potential. • Mild to moderate hyperkalaemia lowers the resting membrane potential more than the threshold potential. This reduces the distance between the two and increases excitability and conduction velocity.
• Severe hyperkalaemia increases the difference between resting membrane potential and threshold potential, reducing excitability and slowing conduction.
• Effects depend on both magnitude and rate of potassium change.

Campese VM, Adenuga G. Electrophysiological and clinical consequences of hyperkalemia. *Kidney Int Suppl*. 2016;6(1):16-19. doi:10.1016/j.kisu.2016.01.003

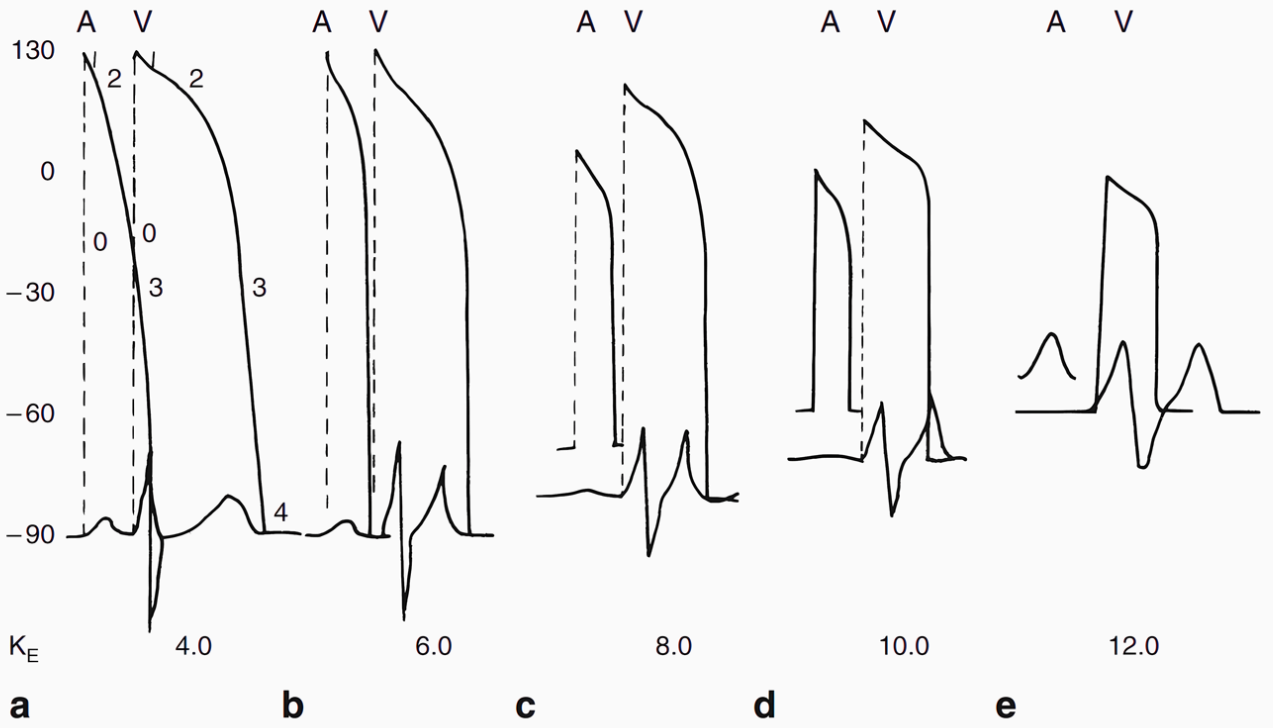
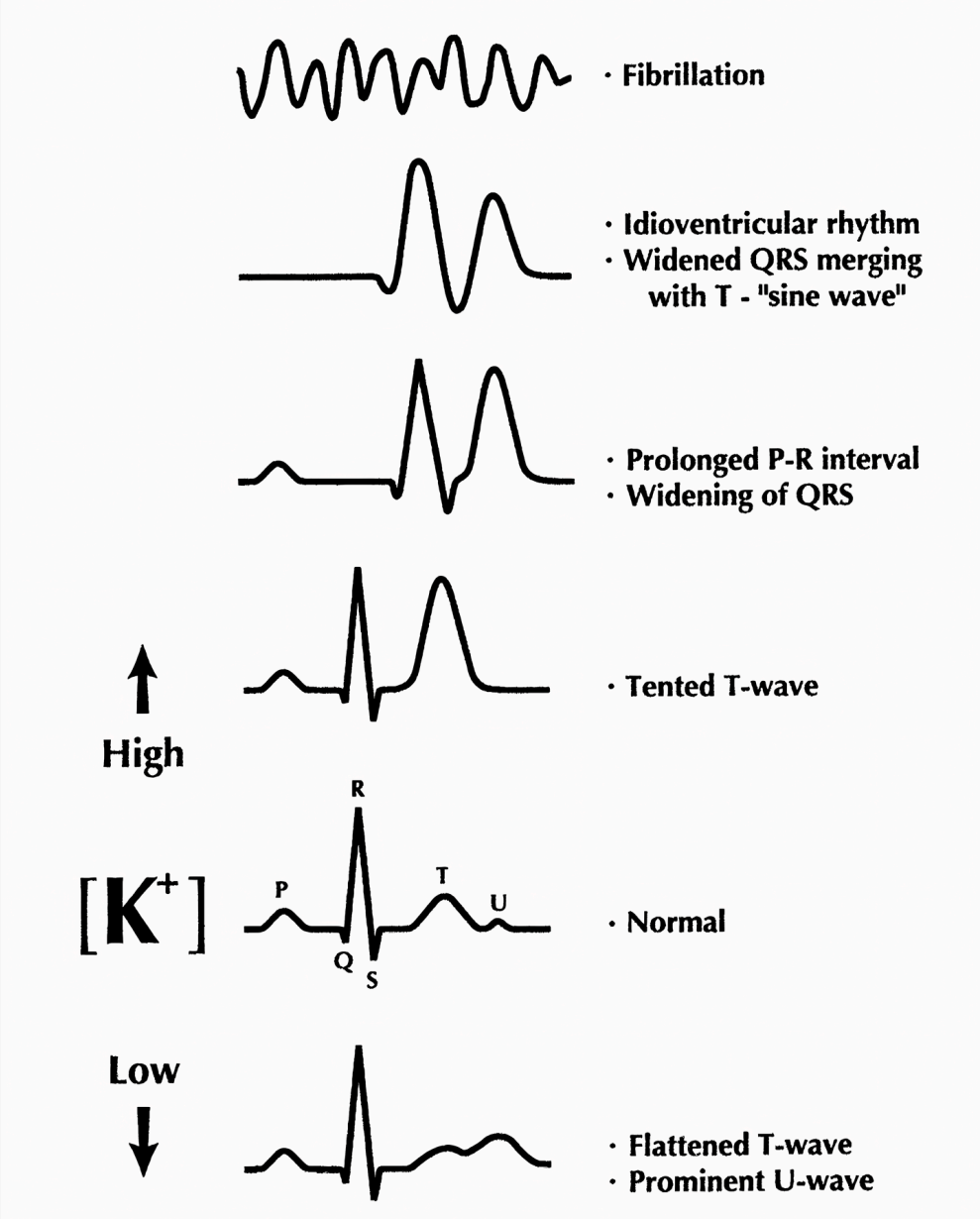


Figure 17.19

Diagram of an atrial (A) and a ventricular (V) action potential (AP) superimposed on the electrocardiogram. The numbers on the left designate the transmembrane potential in millivolts (mV) and the numbers at the bottom, the extracellular concentration of potassium (K_E) in milliequivalents per liter (mEq l^{-1}). In tracings (a) and (b), the resting membrane potential (RMP) is −90 mV, whereas the total amplitude of the AP is 130 mV because the upstroke overshoots the 0 potential by +40 mV. Note the shortening of the AP, the increased velocity of phase 3, the progressive decrease in RMP, and in the amplitude of the AP with increasing K_E. The most rapid upstroke of the AP is shown as a line interrupted by wide spaces ((a) and (b)); the slowest upstroke is shown as a continuous line (atrial AP in (d) and ventricular AP in (e)). Note the decreasing upstroke velocity and the increasing duration of the QRS complex with increasing K_E. In (a) and (b), the atrial and the ventricular AP have the same RMP and the same amplitude, whereas in (c) and (d), the RMP of the atrial AP is less negative than the RMP of the ventricular AP, and the amplitude of the atrial AP is lower than that of the corresponding ventricular AP. See text for further discussion. (After Surawicz [9]. © Mosby, St Louis, Missouri. Reproduced with permission.)

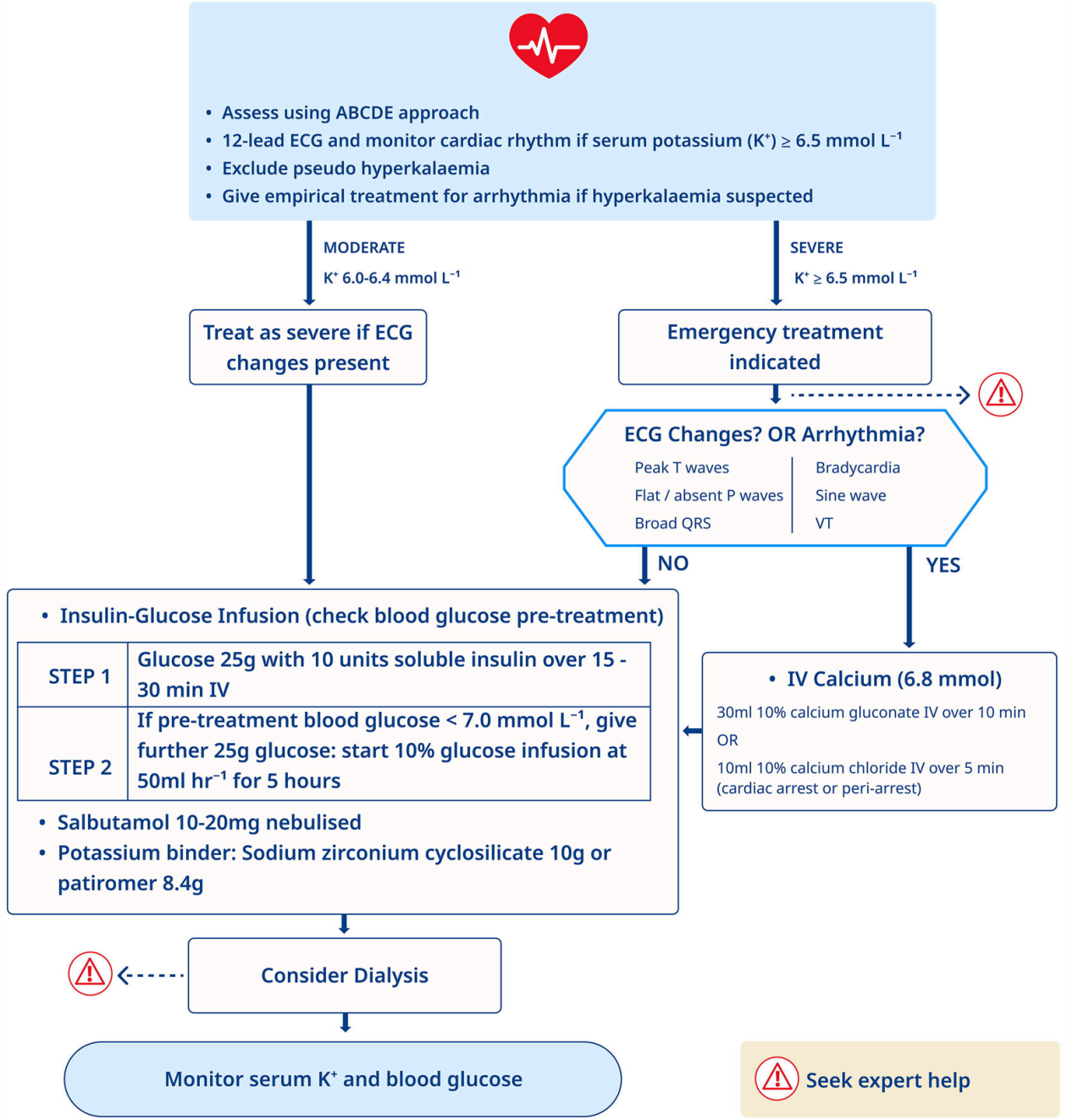


Gennari FJ. Disorders of potassium homeostasis. Hypokalemia and hyperkalemia. *Crit Care Clin*. 2002;18(2):273-288, vi. doi:10.1016/s0749-0704(01)00009-4

Arrhythmia mechanisms

- Early ECG changes include peaked T waves due to global action potential shortening.
- Worsening hyperkalaemia produces P wave broadening and loss, QRS widening, and risks of heart block, asystole, and ventricular arrhythmias.
- Sinus node function is relatively preserved because of low resting potassium conductance. Atrioventricular nodal conduction often deteriorates. His Purkinje pacemaking becomes unreliable in severe hyperkalaemia.
- Reentrant tachyarrhythmias arise from decreased sodium channel availability after premature beats and from conduction velocity restitution.
- Hyperkalaemia increases repolarization reserve, which may favor early repolarization and phase 2 reentry.
- Calcium may exert stabilizing effects by enhancing L type calcium current and reducing all or none repolarization.

Weiss JN, Qu Z, Shivkumar K. Electrophysiology of hypokalemia and hyperkalemia. *Circ: Arrhythmia Electrophysiol*. 2017;10(3):e004667. doi:10.1161/CIRCEP.116.004667



Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Insulin and glucose

- Insulin and glucose Insulin with glucose is the most effective therapy for shifting potassium into cells.
- Ten units of soluble insulin lower serum potassium by about 0.7 to 1.4 mmol per litre.
- Evidence suggests a dose dependent effect and a correlation between baseline potassium and the potassium lowering response.
- Hypoglycaemia is common, with reported incidence up to 28 percent.
- Reducing insulin to five units does not meaningfully reduce hypoglycaemia risk.
- Low pre treatment blood glucose is the most consistent predictor of hypoglycaemia, especially values below 7 mmol per litre.
- Slow infusion of 50 g glucose over four hours still produced hypoglycaemia in about 6 percent of patients. Current ERC algorithms recommend a modified protocol to lower this risk while maintaining efficacy.

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753





Salbutamol

- Salbutamol shifts potassium intracellularly through beta 2 agonist activity.
- Inhaled doses of 10 to 20 mg lower potassium by roughly 0.9 mmol per litre within two hours.
- Reduced effect is reported in patients taking non selective beta blockers and in up to 40 percent of patients with end stage kidney disease.
- Monotherapy is not advised. Combined insulin glucose plus salbutamol produces a greater potassium reduction, about 1.2 mmol per litre.
- Effects last four to six hours and rebound hyperkalaemia may occur.

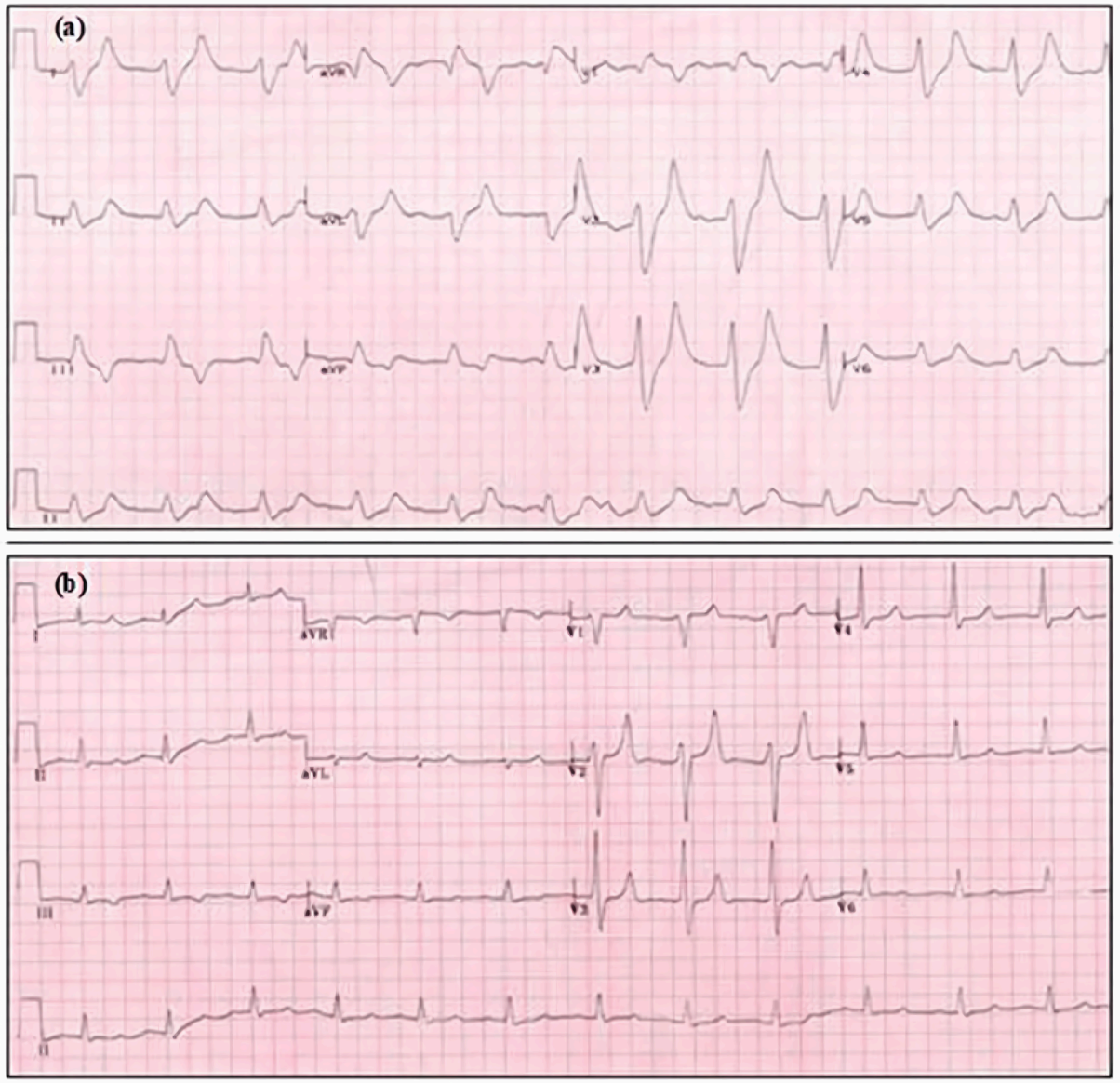
Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Intravenous calcium salts

- Intravenous calcium stabilises cardiac membranes in hyperkalaemia, especially when ECG changes are present.
- Evidence is limited and conflicting, but withholding calcium in severe hyperkalaemia with ECG abnormalities is not advised.
- Calcium may improve ECG abnormalities before potassium lowering therapies take effect.
- Mechanisms include membrane stabilisation and restoration of conduction.
- Current recommendations support use in patients at highest arrhythmic risk.

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753





Effect of IV calcium on ECG in hyperkalaemia.

ECG on admission (a) and following pre-emptive administration of 20 ml 10 % calcium gluconate IV (b) in a patient who presented with a flaccid paralysis. Response to IV calcium shown with narrowing of QRS complex. Blood gas analyser was unavailable and formal lab result later showed a serum K^+ of 9.3 mmol/L. Reproduced with permission from UKKA Hyperkalaemia Guideline.

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Sodium bicarbonate

- Available studies show minimal potassium lowering effect, about 0.1 mmol per litre at one hour.
- Evidence does not support routine use in hyperkalaemia outside cardiac arrest.
- ERC recommends against its routine administration in non arrest patients.

Potassium binders

- Sodium zirconium cyclosilicate and patiromer are effective and well tolerated.
- Sodium zirconium cyclosilicate acts within one hour and lowers potassium by about 1.1 mmol per litre at 48 hours, with greater effect at higher potassium levels.

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753





Indications for dialysis in hyperkalaemia

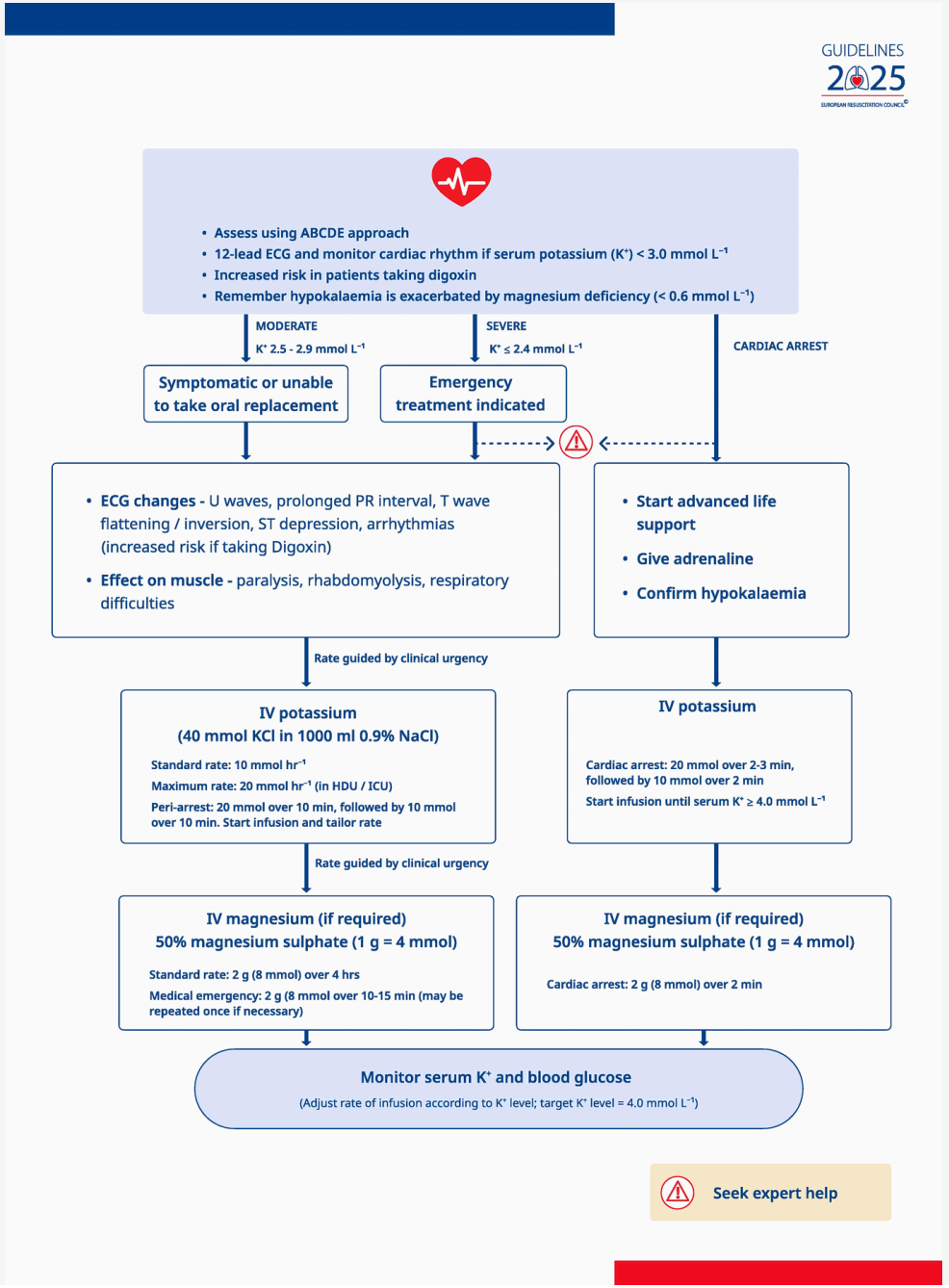
- Severe hyperkalaemia that poses immediate risk, with or without ECG abnormalities or arrhythmias.
- Hyperkalaemia that does not respond to standard medical therapy.
- End stage kidney disease requiring extracorporeal clearance.
- Oliguric acute kidney injury with urine output below 400 millilitres per day.
- Significant tissue breakdown such as rhabdomyolysis.

Rebound hyperkalaemia may occur after dialysis.

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753



Hypokalaemia



Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Hypokalemia: *Physiology*

Authors: Samin Dolatabadi, Ran (Marissa) Zhang, Mannat Dhillon
Reviewers: Meena Assad, Yan Yu*, Juliya Hemmett*
* *MD at time of publication*

Abbreviations:
- **EABV** – Effective Arterial Blood Volume
- **ENaC** – Epithelial Sodium Channel
- **HCL** – Hydrochloric acid
- **HCO_3^-** –Bicarbonate ion

Beta-2 receptor stimulation (e.g. Salbutamol) → ↑ Na^+/K^+ ATPase activity in skeletal muscle cells (moves K^+ into the cell & Na^+ out of cell) → ↑ K^+ entry into skeletal muscle cells

↑ Red blood cell production → ↑ K^+ uptake by new red blood cells

Refeeding Syndrome → ↑ Insulin in response to carbohydrate load

Exogenous insulin → ↑ Na^+/K^+ ATPase activity in skeletal muscle & hepatic cells → ↑ K^+ entry into skeletal muscle & hepatic cells

↑ Intracellular shift of K^+ into cells → **Hypokalemia (Serum [K^+] < 3.5 mmol/L)**

↓ K^+ dietary intake (rare cause in isolation) → ↓ K^+ availability for gastrointestinal absorption → **Hypokalemia (Serum [K^+] < 3.5 mmol/L)**

Vomiting → Gastric acid depletion → ↓HCl → Loss of H+ → Shift in bicarbonate buffer system to ↑ plasma HCO_3^- → Plasma HCO_3^- above reabsorptive capacity of the proximal tubule → ↑ HCO_3^- in the distal tubular lumen of kidneys → ↑ Electronegativity of tubular lumen

Diarrhea Laxatives → Extracellular fluid volume depletion → ↓ EABV

Renin secreting tumour → ↑ Renin secretion

Hyperaldosteronism

Renal artery stenosis → ↓ Afferent arteriole pressure perfusing kidneys

Loop and Thiazide diuretics → ↑ Na^+ & water excretion in kidneys → ↓ EABV

Bartter's and Gittelman's syndrome → ↑ Na^+ & water excretion in kidneys → ↓ EABV

↓ EABV / ↑ Renin secretion / ↓ Afferent arteriole pressure perfusing kidneys → Renin-Angiotensin-Aldosterone System (RAAS) activation → ↑ Aldosterone release from the adrenal cortex → ↑ Expression of ENaC (Na^+ reabsorption) in principal cells of the cortical collecting duct → ↑ Na^+ reabsorption in principal cells → Cl^- left behind in tubular lumen of kidneys → ↑ Electronegativity of tubular lumen → K^+ follows the electrical gradient into tubular lumen → ↑ Renal K^+ secretion → **Hypokalemia (Serum [K^+] < 3.5 mmol/L)**

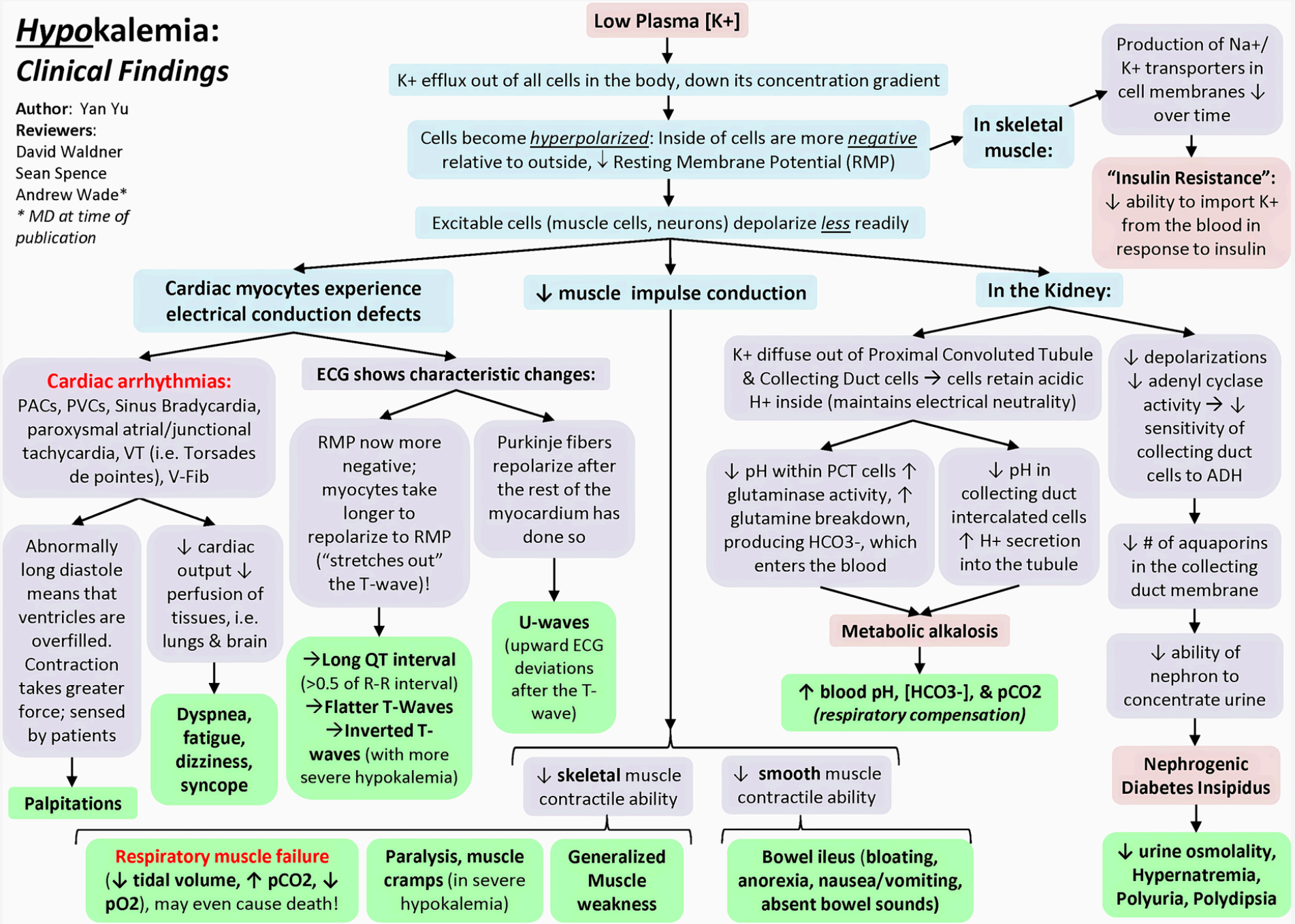
Liddle syndrome → Genetic condition leading to inability to degrade ENaC channels in principal cells of the cortical collecting duct → ↑ Na^+ reabsorption in principal cells → Cl^- left behind in tubular lumen of kidneys

Legend: Pathophysiology | Mechanism | Sign/Symptom/Lab Finding | Complications

Published **March 6, 2019**, updated **Jan 23, 2022** on www https://calgaryguide.ucalgary.ca/Hypokalemia-Physiology/

*Hypo*kalemia: *Clinical Findings*

Author: Yan Yu
Reviewers:
David Waldner
Sean Spence
Andrew Wade*
** MD at time of publication*



Legend: Pathophysiology | Mechanism | Sign/Symptom/Lab Finding | Complications

Published **May 21, 2013** on www.the https://calgaryguide.ucalgary.ca/Hypokalemia:-Clinical-Findings/

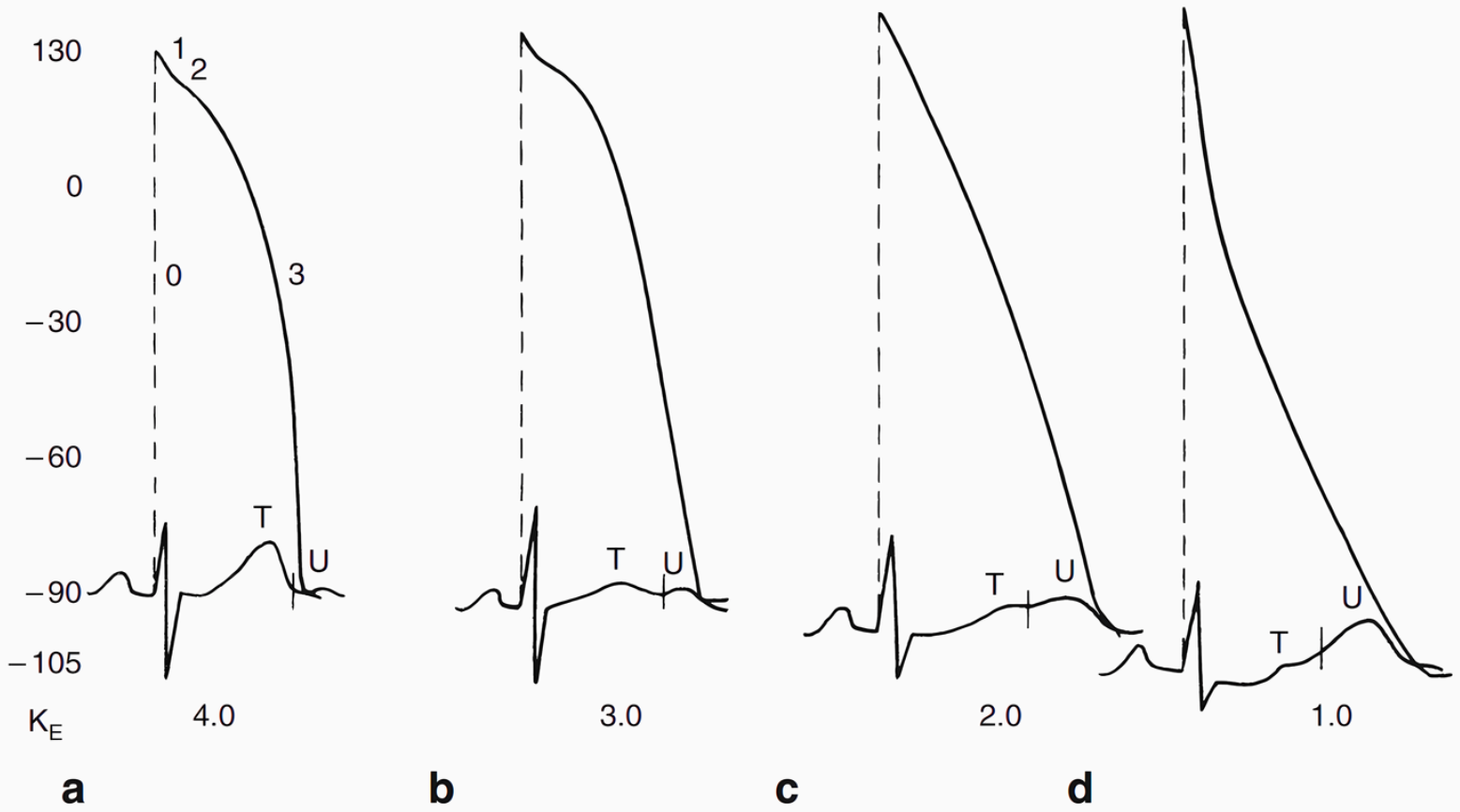


Figure 17.20

Diagram of the ventricular AP superimposed on the electrocardiogram for extracellular concentration of potassium (KE) of (a) 4.0, (b) 3.0, (c) 2.0, and (d) 1.0 ml^{-1}. The numbers on the left designate the transmembrane potential in millivolts, as in Fig. 15.18. The RMP, the amplitude of the AP, and the overshoot in the control tracing (a) are the same as for the ventricular AP in Fig. 17.19. Note the progressively increasing velocity of phase 2, decreasing velocity of phase 3, and increasing duration of the AP. Furthermore, note the progressive increase in the RMP (more negative), and the increasing amplitude of the AP. The electrocardiographic changes are described in the text. (After Surawicz [9]. © Mosby, St Louis, Missouri. Reproduced with permission.)

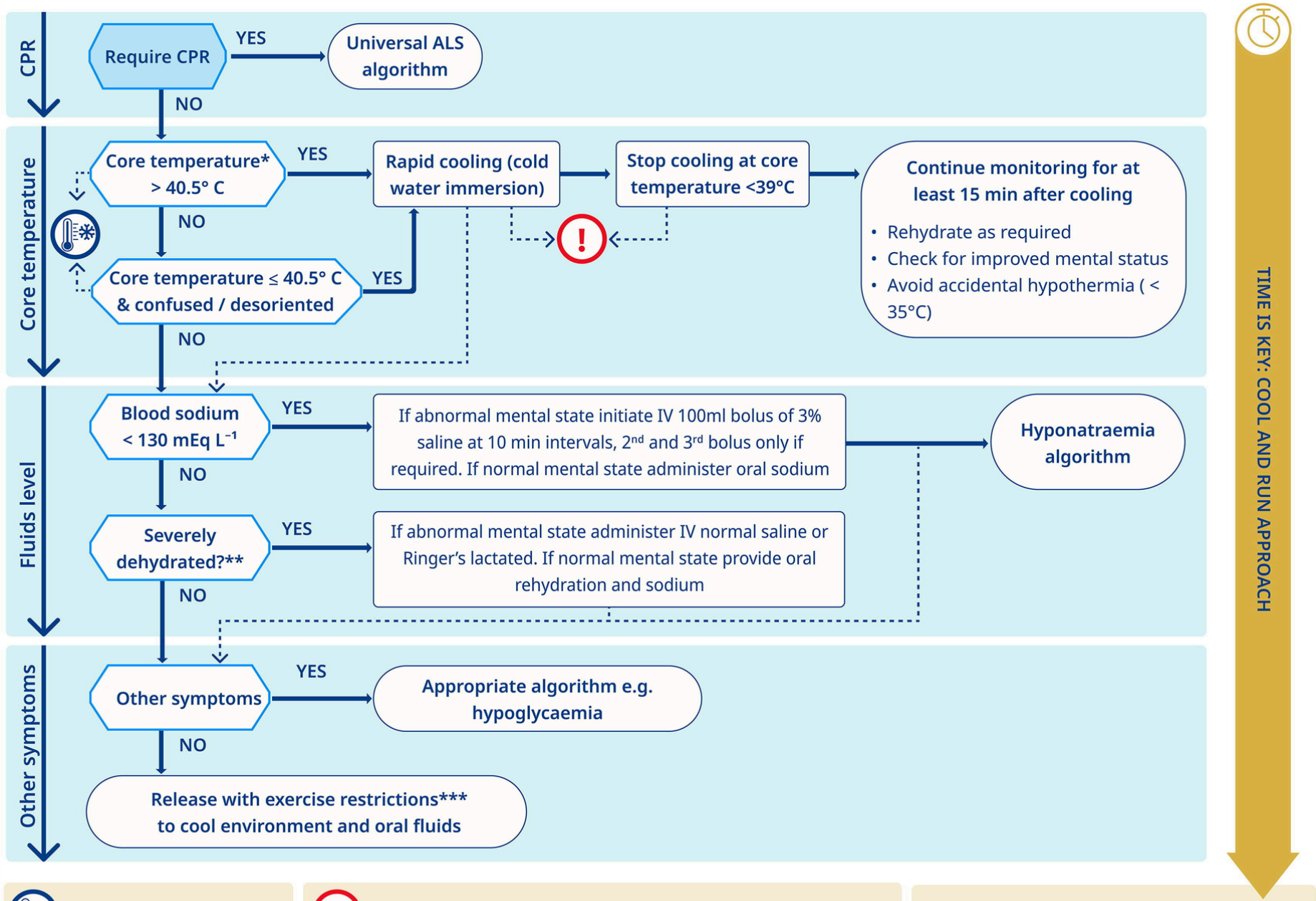
Macfarlane PW, Oosterom A van, Pahlm O, Kligfield P, Janse M, Camm J, eds. *Comprehensive Electrocardiology*. Springer; 2010.

ECG abnormalities in hypokalemia

	Abnormalities
P and QRS waves	Increased QRS wave amplitude Increased QRS wave duration Increased P wave amplitude Increased P wave duration
ST segment, T and U waves	Prominent U wave Flat or inverted T wave ST segment depression
Conduction and impulse formation	Atrial premature contractions Atrial tachycardia Supraventricular tachycardia AV junctional rhythm AV dissociation First degree AV block Ventricular premature contractions Bidirectional ventricular tachycardia Ventricular fibrillation

Macfarlane PW, Oosterom A van, Pahlm O, Kligfield P, Janse M, Camm J, eds. *Comprehensive Electrocardiology*. Springer; 2010.

Hyperthermia, malignant hyperthermia and toxin-induced hyperthermia



Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Hyperthermia and Malignant Hyperthermia

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. Resuscitation. 2025;215.
doi:10.1016/j.resuscitation.2025.110753

- **Hyperthermia**

  Body temperature above normal due to failed thermoregulation

- **Risk Factors**

  Extremes of age and multimorbidity

- **Malignant Hyperthermia**

  Rare pharmacogenetic disorder of skeletal muscle calcium homeostasis

- **Triggers**

  Halogenated anesthetics, succinylcholine, neuroleptics, and rarely non-pharmacological

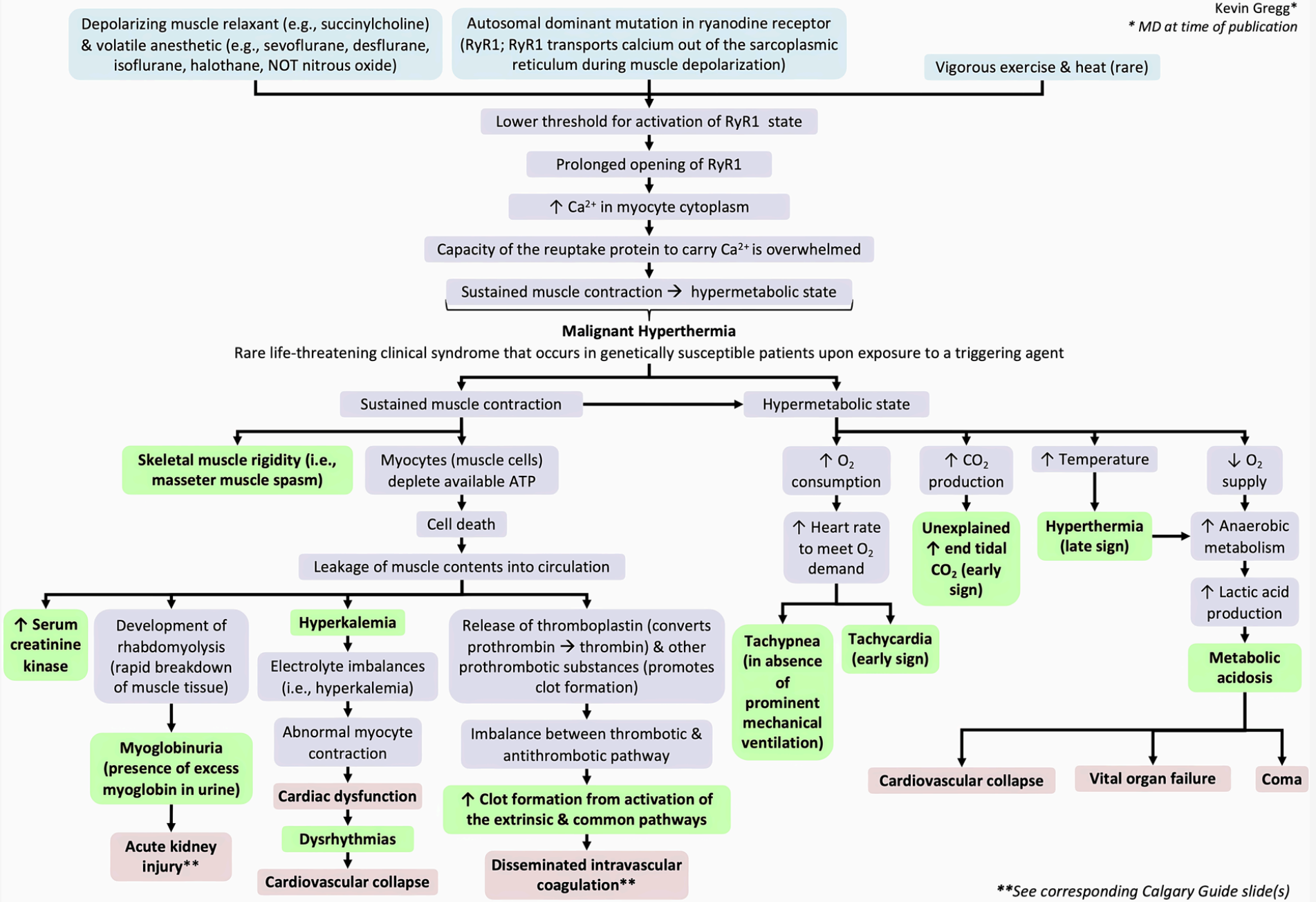
- **Clinical Signs**

  Hypercapnia, sinus tachycardia, hyperthermia, masseter spasm, muscle rigidity, acidosis, hyperkalaemia

salvatore.sardo@unica.it

Malignant Hyperthermia: *Pathogenesis & clinical findings*

Authors: Haotian Wang, Jen Guo
Reviewers: Julena Foglia, Priyanka Grewal, Luiza Radu
Kevin Gregg*
** MD at time of publication*



***See corresponding Calgary Guide slide(s)*

Published **Oct 25, 2015; updated Oct 2, 2024** on www.thecalgaryguid

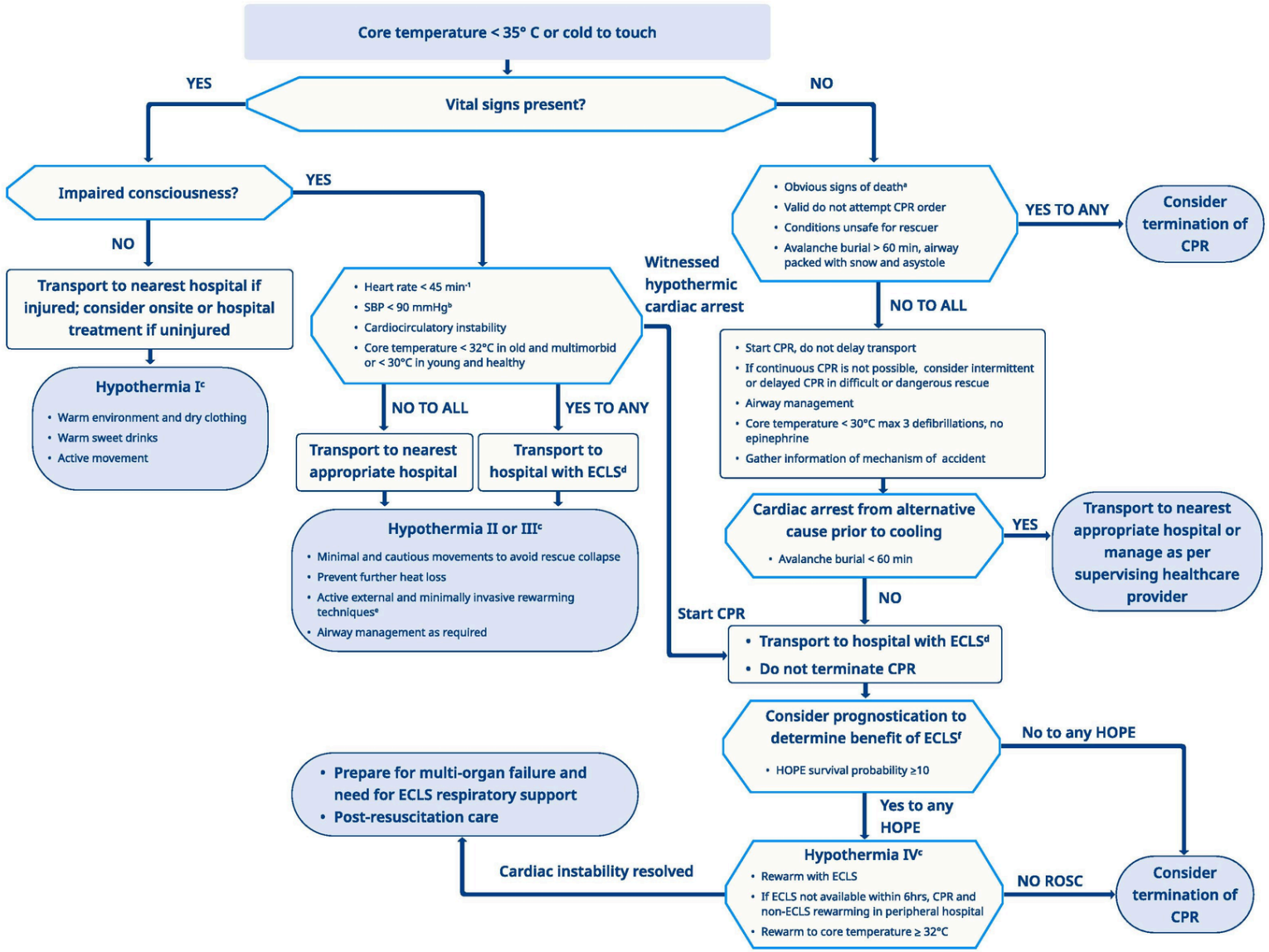
Category	Typical substances	Core mechanism	Key clinical features	Main diagnostic tests	Specific management
Antipsychotics	Risperidone Aripiprazole Haloperidol Olanzapine Quetiapine Clozapine Blonanserine	Dopamine receptor antagonism or acute dopamine agonist withdrawal leading to neuroleptic malignant syndrome	Hyperthermia muscle rigidity tremor autonomic instability altered mental state	Blood testing for antipsychotics	Benzodiazepines for agitation Bromocriptine for dopamine deficiency Dantrolene for muscle rigidity
Antidepressants	Lithium MAOI SSRI SNRI TCA	Excess serotonergic activity causing serotonin syndrome	Hyperthermia flushing shivering agitation akathisia mydriasis autonomic instability	Blood testing for antidepressants serum lithium level	Benzodiazepines for agitation Chlorpromazine may be considered Sodium bicarbonate for TCA toxicity
Recreational drugs	MDMA LSD	Central catecholamine release and reuptake inhibition with hypermetabolic state and muscle activation	Hyperthermia euphoria hallucinations agitation shivering mydriasis nausea	Urine drug screening	Benzodiazepines for agitation Dantrolene for hyperthermia Consider carvedilol in MDMA exposure
Methamphetamine	Methamphetamine	Marked sympathetic stimulation and hypermetabolic state	Hyperthermia hallucinations tremor agitation mydriasis autonomic instability	Urine or blood amphetamine screening	Urine acidification with ammonium chloride Activated charcoal if early

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Category	Typical substances	Core mechanism	Key clinical features	Main diagnostic tests	Specific management
Cocaine	Cocaine	Monoamine reuptake inhibition sodium channel blockade coronary vasoconstriction	Hyperthermia agitation hallucinations tremor mydriasis arrhythmias chest pain	Urine and blood toxicology	Sodium bicarbonate for wide complex arrhythmias Avoid beta blockers
Anticholinergics	Atropine Scopolamine Belladonna Brugmansia Amanita	Muscarinic receptor blockade causing anticholinergic hyperthermic syndrome	Hyperthermia tachycardia dry skin dry mucosa flushing mydriasis delirium	Urine and serum toxicology	Benzodiazepines for agitation Central acting cholinesterase inhibitors Activated charcoal early
Sympathomimetics	Ephedrine Pseudoephedrine	Sympathetic activation with increased metabolic rate and vasoconstriction	Hyperthermia tachycardia hypertension arrhythmias mydriasis muscle twitching	Urine screening serum toxicology	Benzodiazepines for agitation
Salicylates	Aspirin Methyl salicylate	Uncoupling of oxidative phosphorylation with increased heat production	Mild hyperthermia nausea tachypnoea tinnitus confusion	Serum salicylate level	Urine alkalinisation with sodium bicarbonate Prevent hypoglycaemia Dialysis if severe
Miscellaneous	Dinitrophenol	Uncoupling of oxidative phosphorylation with uncontrolled heat production	Severe hyperthermia sweating flushing agitation tachycardia tachypnoea	Serum toxicology for dinitrophenol	Supportive care Consider dantrolene

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Hypothermia



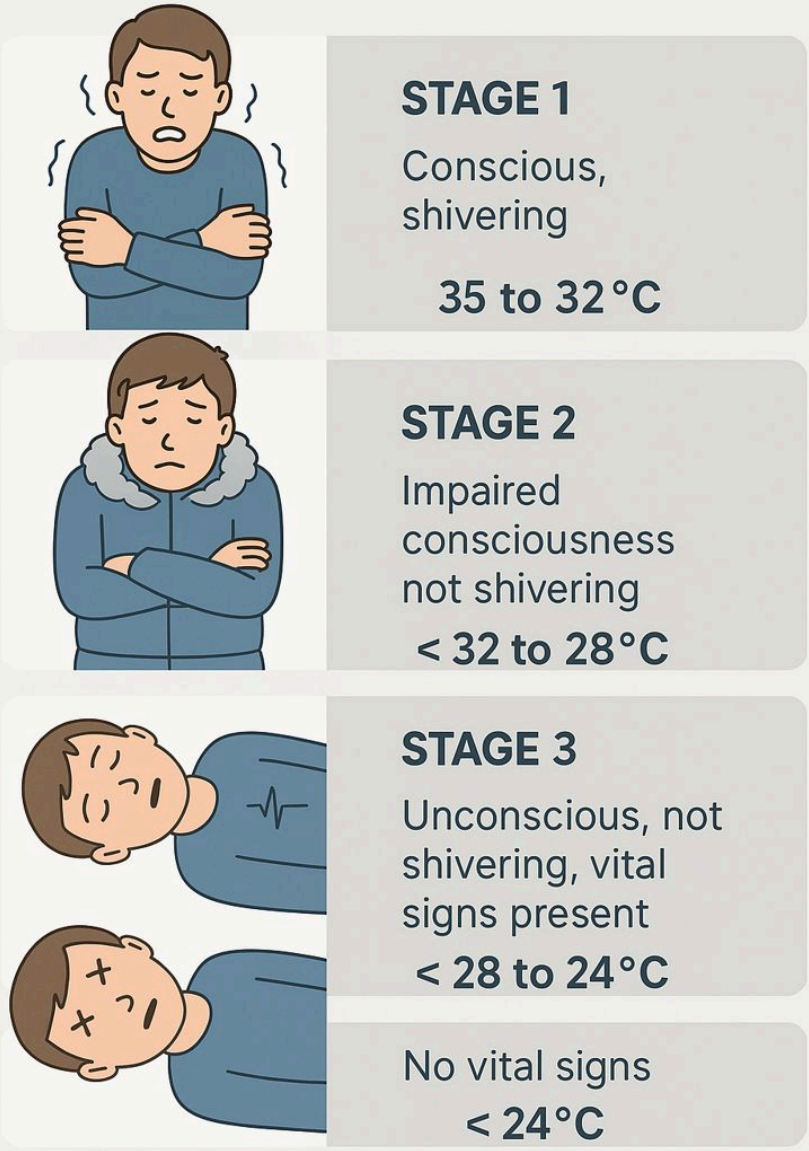
Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Hypothermia

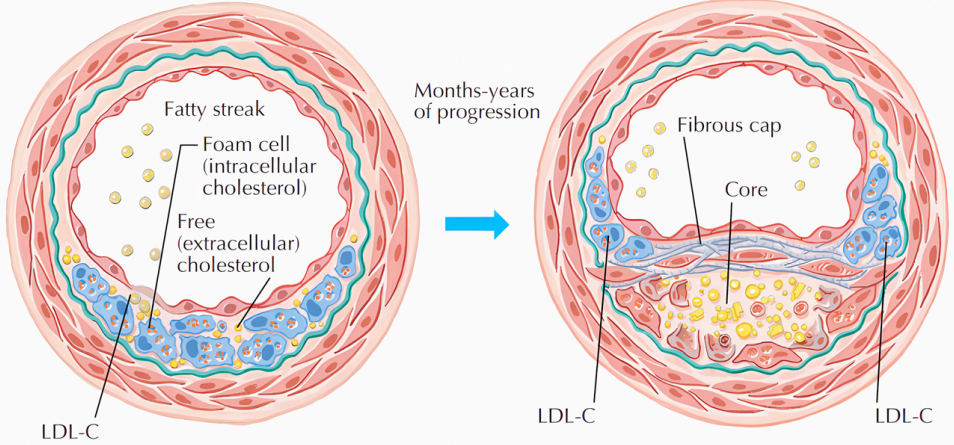
- Measure core temperature with a low reading thermometer to diagnose accidental hypothermia.
- Use the Swiss Staging System if core temperature cannot be measured.
- Transfer hypothermic patients with risk factors for imminent cardiac arrest and those in cardiac arrest directly to an extracorporeal life support (ECPR) centre for rewarming
- Delay CPR or use intermittent CPR in hypothermic cardiac arrest patients with a core temperature below 28 °C when immediate or continuous CPR is not feasible.•Delay further defibrillation attempts if ventricular fibrillation (VF) persists after three shocks until core temperature is > 30 °C
- .Below 30 °C adrenaline will accumulate and may have more detrimental than beneficial effects. Give IV 1 mg adrenaline once to facilitate ROSC unless planning imminent initiation of ECPR. Increase administration intervals for adrenaline to 6–10 min if the core temperature is 30–35 °C.
- Consider use of a mechanical CPR device if transport is prolonged, or when there are difficulties with the terrain.
- Base in-hospital prognostication of successful rewarming on the Hypothermia Outcome Prediction after Extracorporeal Life Support (HOPE) score.
- Rewarm hypothermic arrested patients with veno-arterial extra-corporeal membrane oxygenation (VA-ECMO).
- Initiate non-extracorporeal life support rewarming if an ECPR centre cannot be reached within a reasonable time (e.g. 6 h).

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

SWISS HYPOTHERMIA STAGING



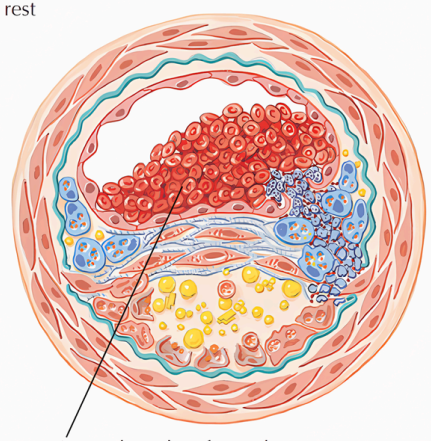
Thrombosis

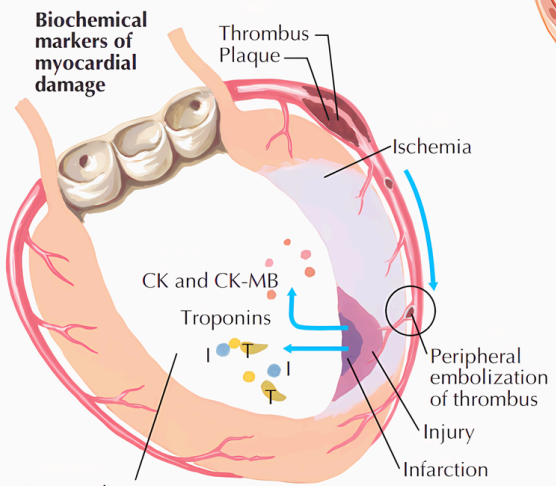


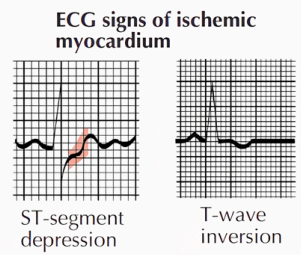
Most common etiology of acute coronary syndrome is the slow development of atherosclerotic coronary artery plaque, which presents acutely by thrombus formation on preexisting plaque

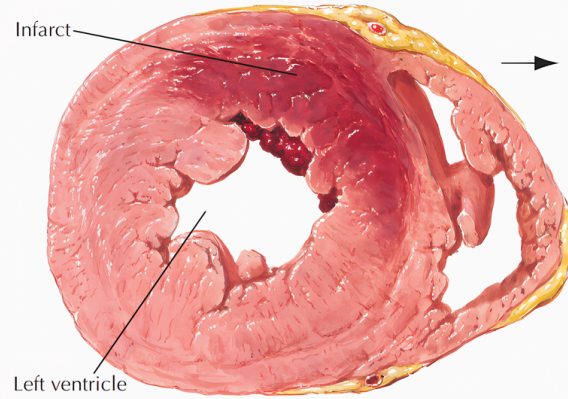
Patient may note abrupt change in pattern and severity of symptoms, often symptomatic at rest

Acute onset hours-days



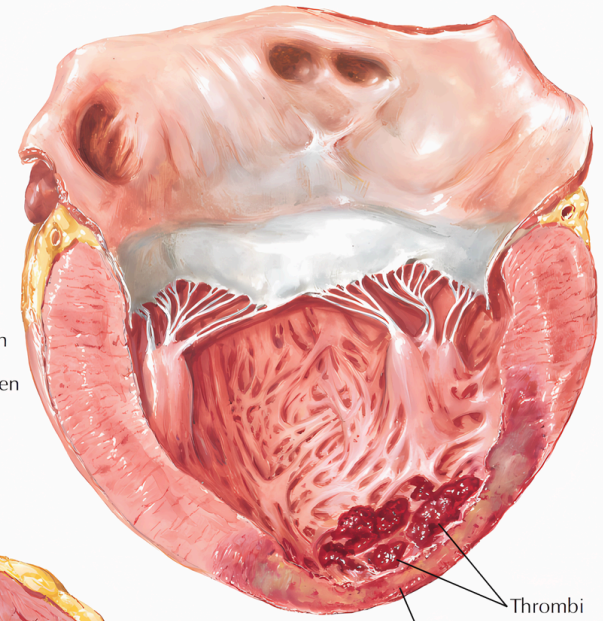




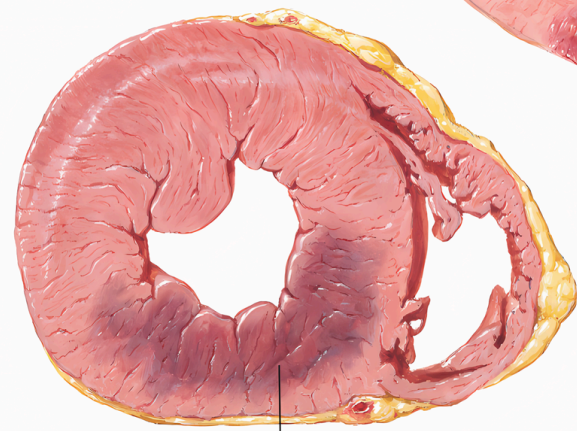


Acute anteroseptal infarct. With right bundle branch block

Acute anteroseptal, transmural infarct. Complicated by right bundle branch block, seen in coronal section of heart

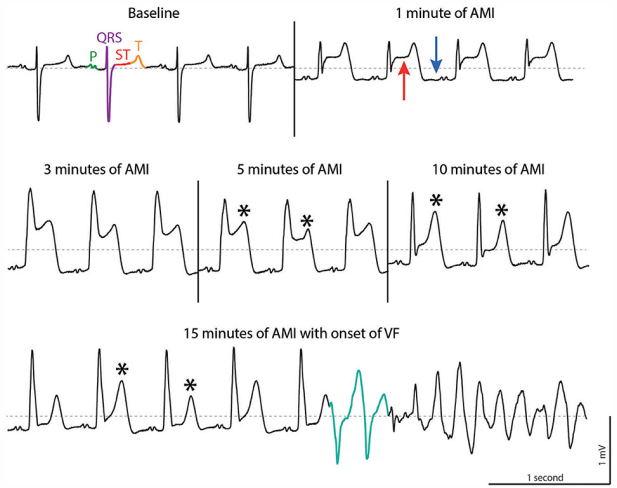


Subacute apical infarct. With resorption of muscle (thin wall) and mural thrombi. Seen in longitudinal section of heart

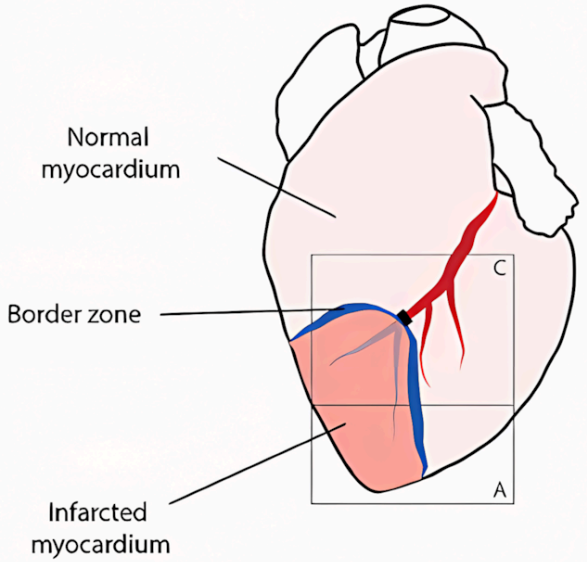


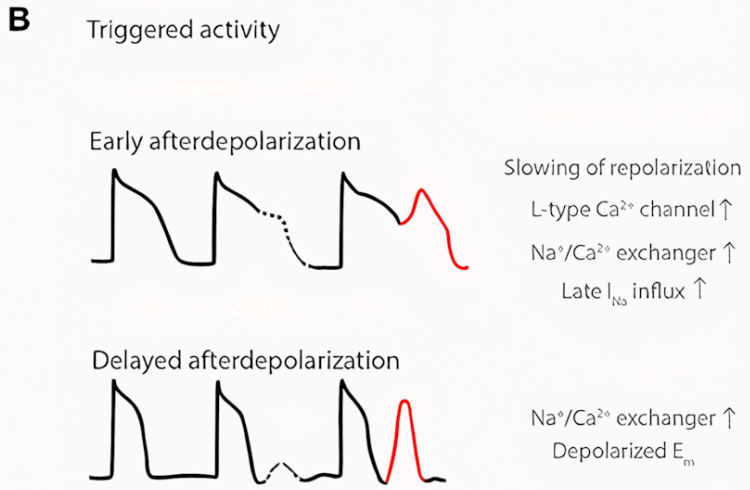
Acute posterior subendocardial infarct. Seen in coronal section of heart

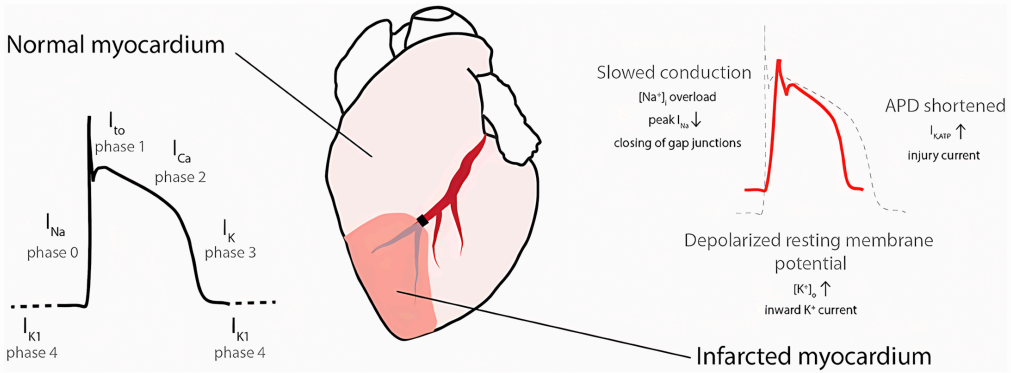
Conti JB. *The Netter Collection of Medical Illustrations: Volume 8*. 2nd ed. Elsevier - Health Sciences Division; 2014.

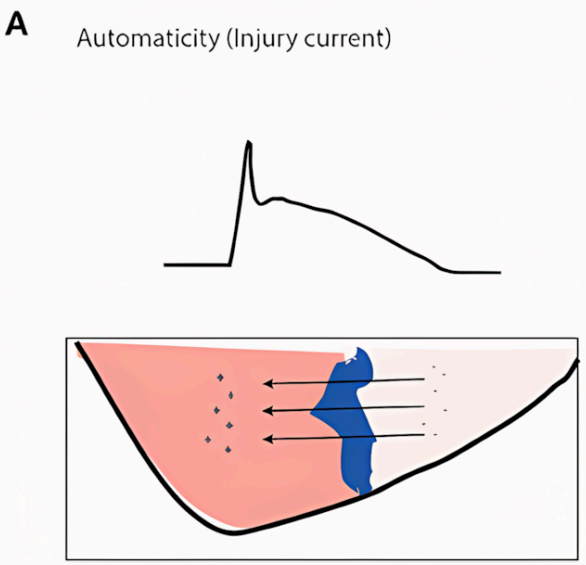


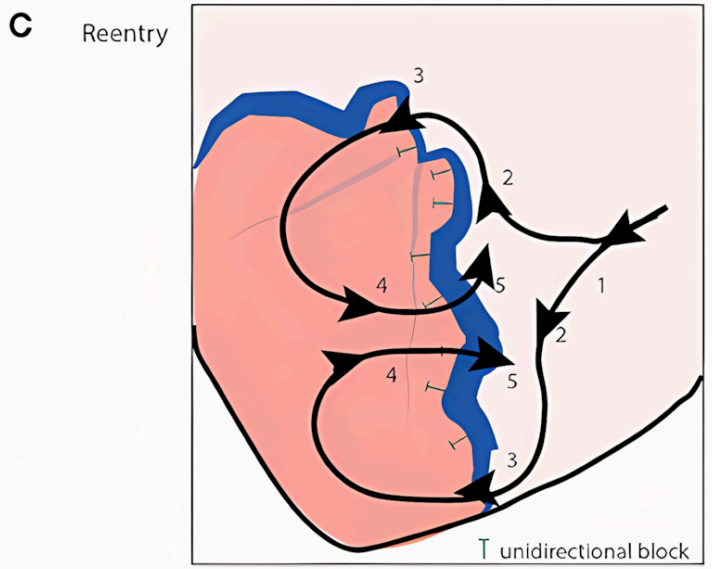
Sattler SM, Skibsbye L, Linz D, Lubberding AF, Tfelt-Hansen J, Jespersen T. Ventricular arrhythmias in first acute myocardial infarction: epidemiology, mechanisms, and interventions in large animal models. *Front Cardiovasc Med*. 2019;6. doi:10.3389/fcvm.2019.00158

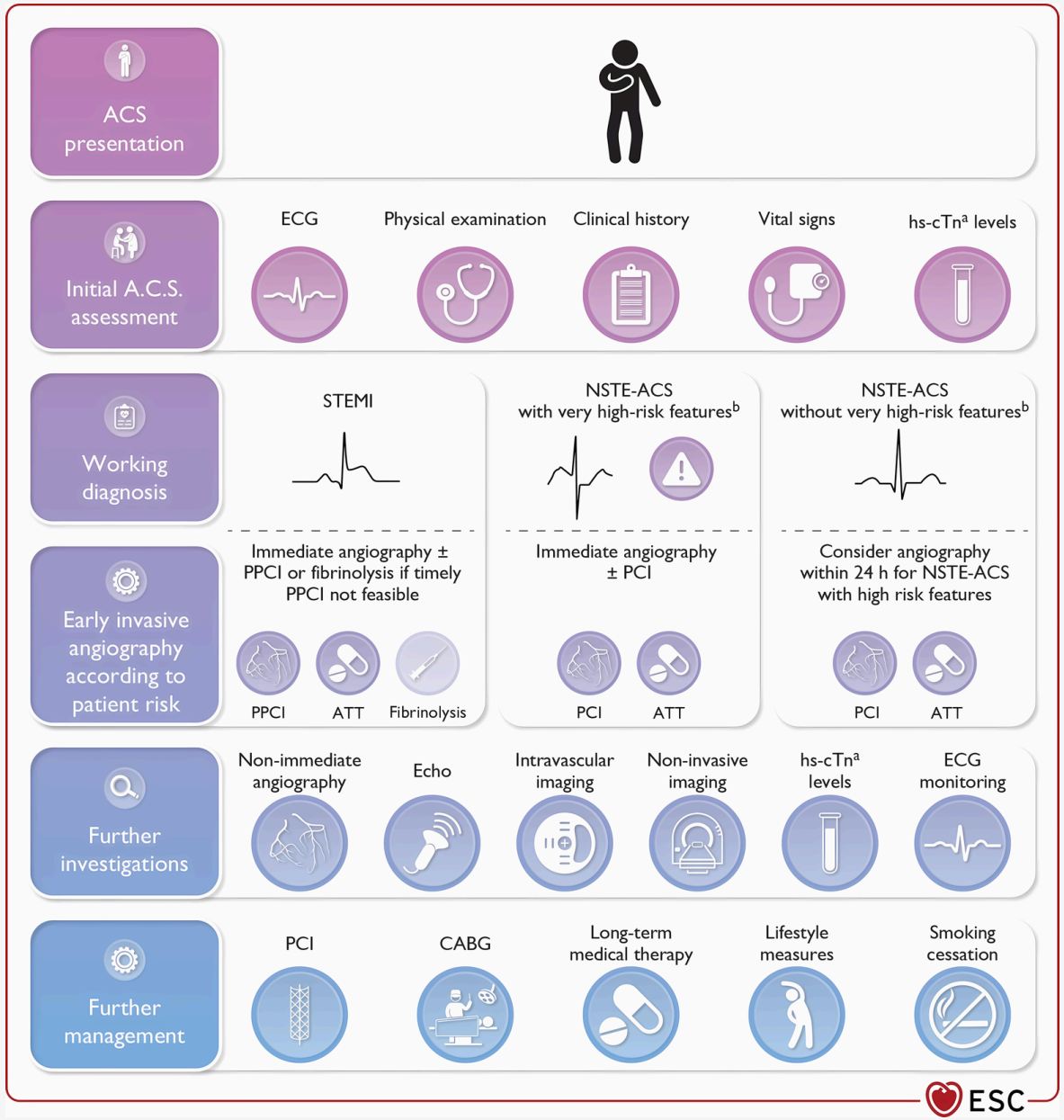












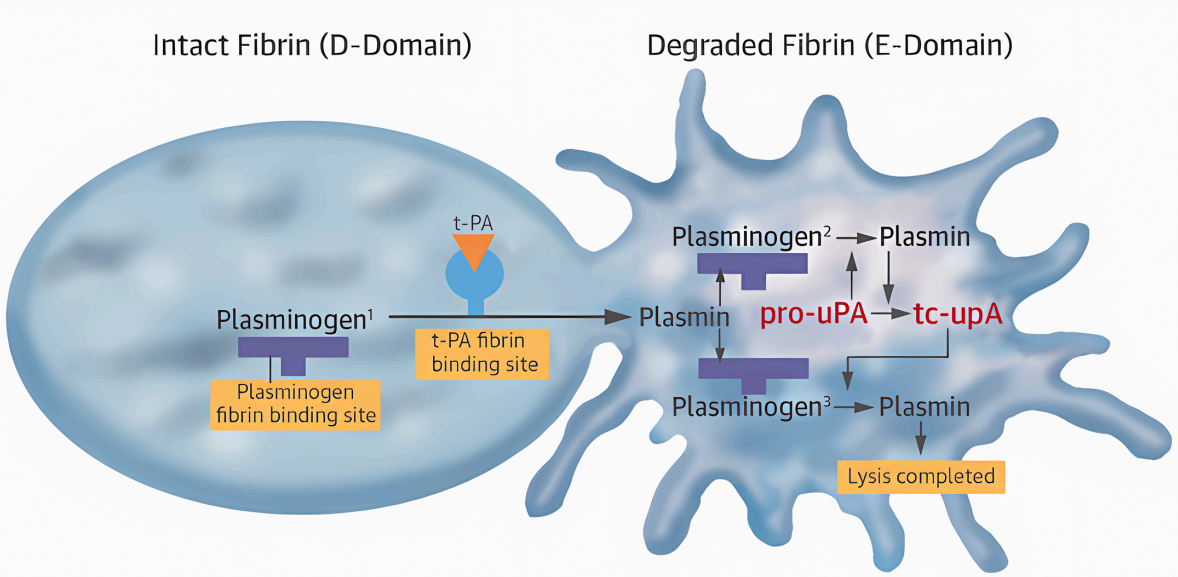
Byrne RA, Rossello X, Coughlan JJ, et al. 2023 ESC guidelines for the management of acute coronary syndromes. *Eur Heart J*. 2023;44(38):3720-3826. doi:10.1093/eurheartj/ehad191

Transfer the patient to a centre with PCI capability and activate existing STEMI networks in case of ST-elevation or suspected ongoing ischaemia.

In patients with sustained ROSC and ST-elevation on ECG:

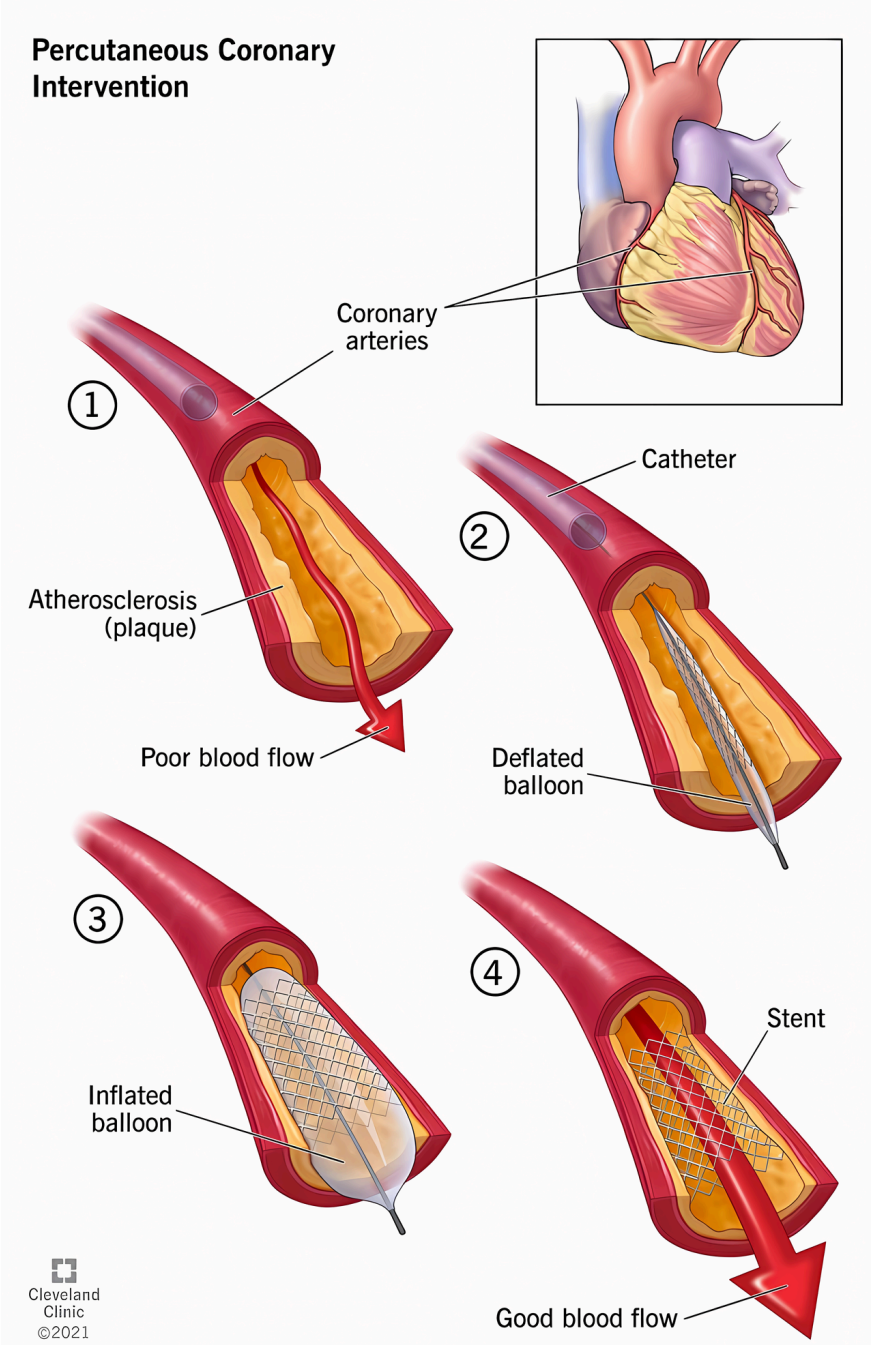
- Perform immediate coronary angiography (and PCI if required) within 120 min of diagnosis.
- Consider fibrinolysis in pre-hospital and non-PCI-capable settings if greater delay is expected.

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753



Gurewich V. Therapeutic fibrinolysis. *JACC*. 2016;68(19):2099-2106. doi:10.1016/j.jacc.2016.07.780





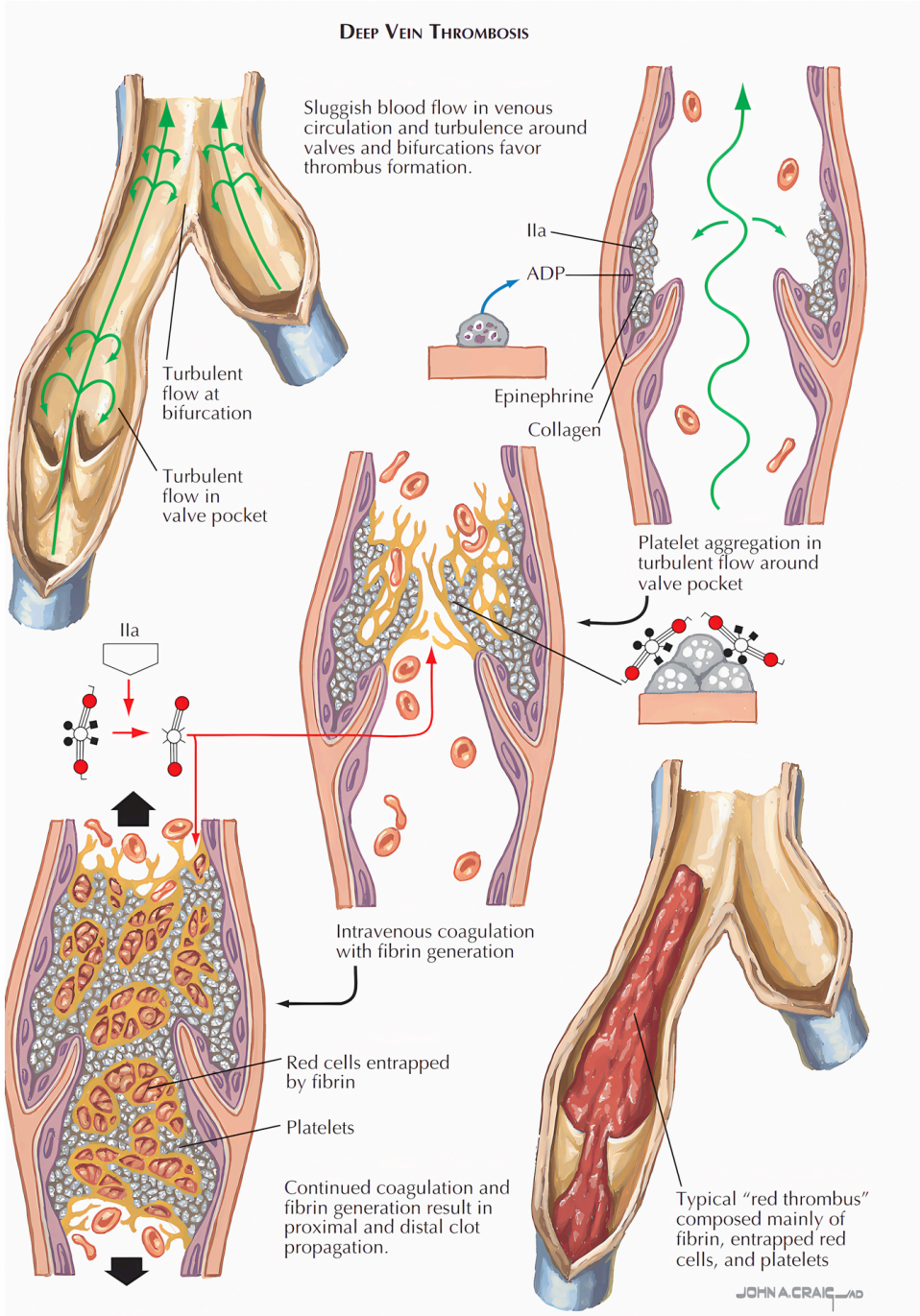
https://my.clevelandclinic.org/health/treatments/22066-percutaneous-coronary-intervention

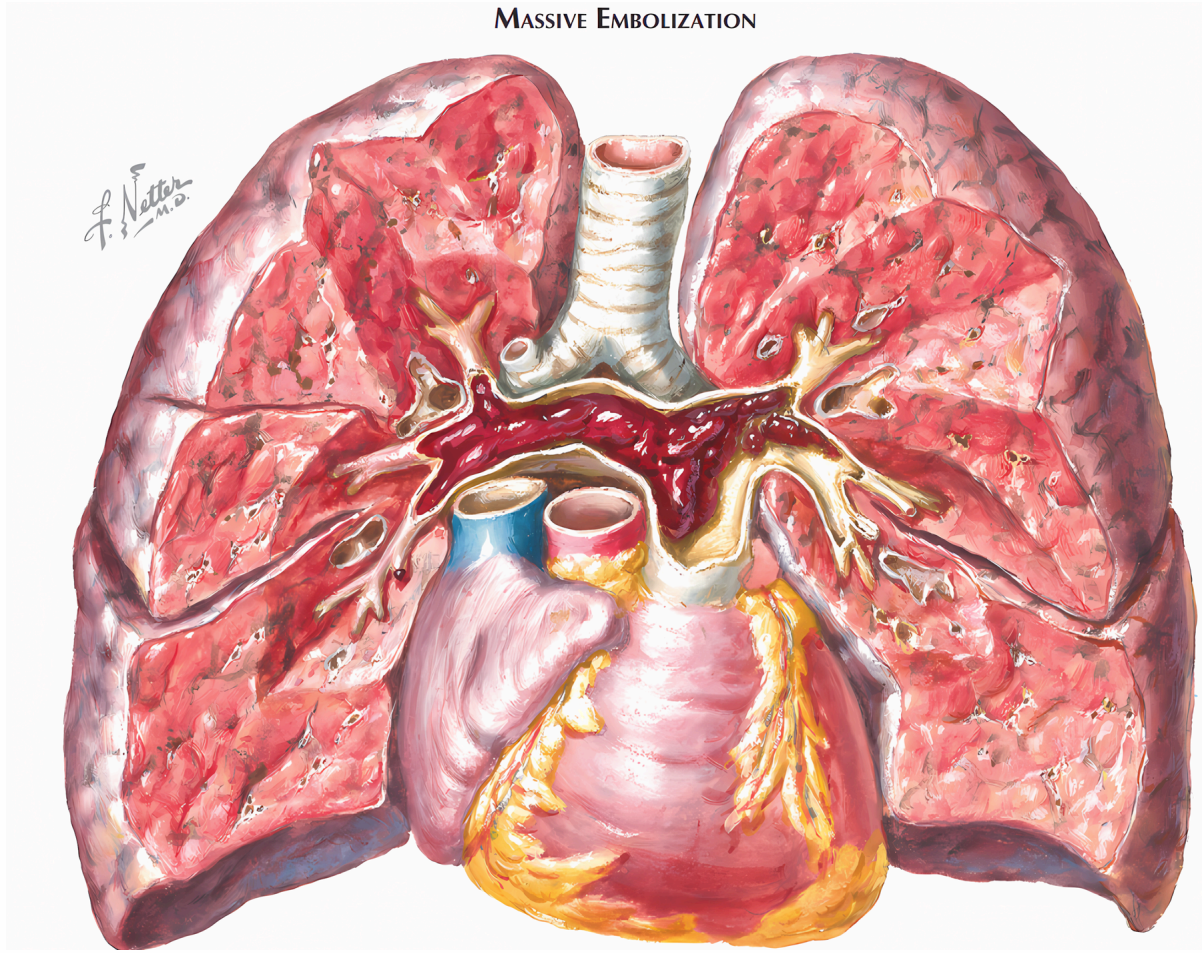
In patients with sustained ROSC and no ST-elevation on ECG:

- Consider immediate coronary angiography (and PCI if required) if the patient is haemodynamically unstable or shows signs of ongoing ischaemia.
- In stable patients without ischaemic signs, emergent cardiac catheterisation laboratory evaluation should not be systematic and can be delayed if there is no estimated high probability of acute coronary occlusion.
- Assess for non-coronary-causes if the clinical context suggests an alternative aetiology of the arrest.
- Unless on-going resuscitation is considered futile, transfer patients without sustained ROSC with ongoing CPR, to a PCI centre for consideration for angiography or ECPR depending on available resources and team expertise.

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Pulmonary Embolism





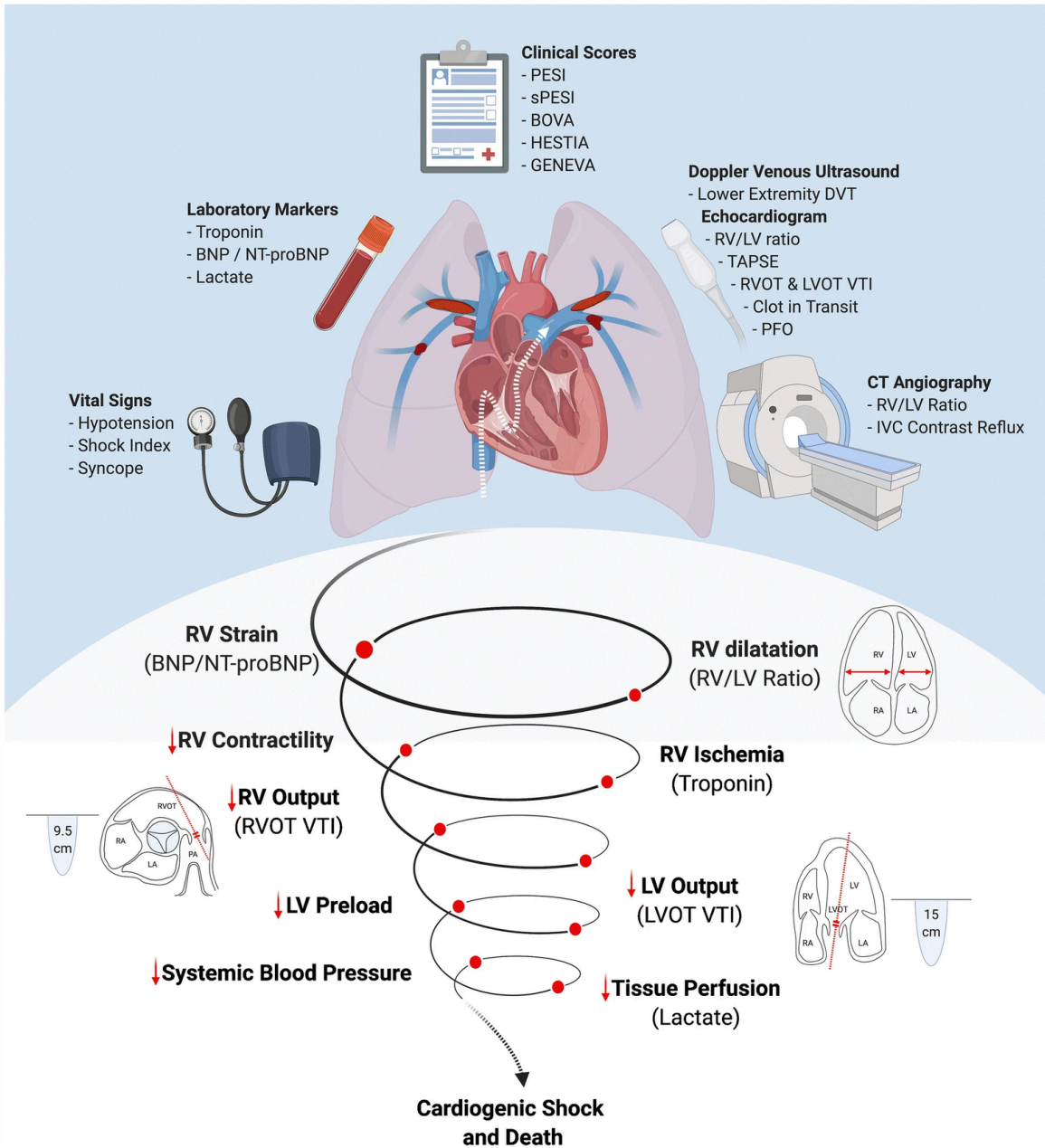
Conti JB. *The Netter Collection of Medical Illustrations: Volume 8*. 2nd ed. Elsevier - Health Sciences Division; 2014.

Modified Geneva Score Risk Factors and Points

Risk Factor	Points
Age >65 years	1
Previous deep venous thrombosis or pulmonary embolism	3
Surgery under general anaesthesia or fracture of the lower limbs within one month	2
Active malignancy (solid or hematologic; currently active or cured within the last year)	2
Unilateral lower-limb pain	3
Haemoptysis	2
Heart rate 75 to 94 beats per minute	3
≥95 beats per minute	5
Pain in lower limb deep venous palpation and unilateral oedema	4



salvatore.sardo@unica.it



Brailovsky Y, Allen S, Masic D, Lakhter D, Sethi SS, Darki A. Risk stratification of acute pulmonary embolism. *Curr Treat Options Cardiovasc Med*. 2021;23(7):48. doi:10.1007/s11936-021-00923-4


YouTube

Pulmonary embolism

- Consider pulmonary embolism in all patients with sudden onset of progressive dyspnoea and absence of known heart or pulmonary disease.
- Obtain 12-lead ECG (exclude acute coronary syndrome, look for right ventricular strain).
- Identify haemodynamic instability and high-risk pulmonary embolism.
- Perform bedside echocardiography.
- Initiate anticoagulation therapy (heparin 80 IU/kg IV) during diagnostic process, unless signs of bleeding or absolute contraindications.
- Confirm diagnosis with computed tomographic pulmonary angiography.
- Consider surgical embolectomy or catheter-directed treatment as alternative to rescue fibrinolytic therapy in rapidly deteriorating patients.

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation.* 2025;215. doi:10.1016/j.resuscitation.2025.110753

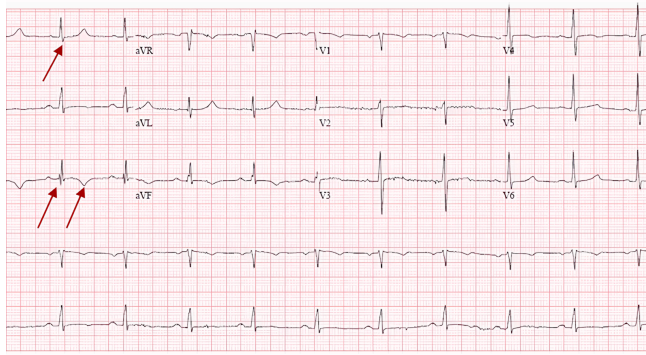
Parameter	Original version[226]	Simplified version[229]
Age	Age in years	1 point (if age >80 years)
Male sex	+10 points	–
Cancer	+30 points	1 point
Chronic heart failure	+10 points	1 point
Chronic pulmonary disease	+10 points	
Pulse rate ≥110 b.p.m.	+20 points	1 point
Systolic BP <100 mmHg	+30 points	1 point
Respiratory rate >30 breaths per min	+20 points	–
Temperature <36°C	+20 points	–
Altered mental status	+60 points	–
Arterial oxyhaemoglobin saturation <90%	+20 points	1 point
	Risk strata[a]	
	Class I: ≤65 points very low 30 day mortality risk (0–1.6%) **Class II: 66–85 points** low mortality risk (1.7–3.5%)	**0 points =** 30 day mortality risk 1.0% (95% CI 0.0–2.1%)
	Class III: 86–105 points moderate mortality risk (3.2–7.1%) **Class IV: 106–125 points** high mortality risk (4.0–11.4%) **Class V: >125 points** very high mortality risk (10.0–24.5%)	**≥1 point(s) =** 30 day mortality risk 10.9% (95% CI 8.5–13.2%)

BP

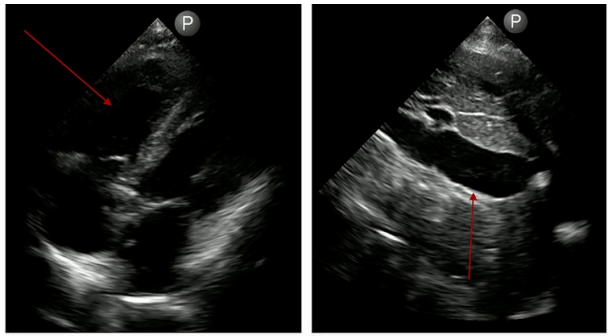
[a]Based on the sum of points.

Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the european respiratory society (ERS). *Eur Heart J.* 2020;41(4):543-603. doi:10.1093/eurheartj/ehz405

© ESC 2019



Skipina TM, Petty SA, Kelly CT. A near miss: subclinical saddle pulmonary embolism diagnosed by handheld ultrasound. *Oxf Med Case Rep.* 2021;2021(4):omab011. doi:10.1093/omcr/omab011



a) b)

Pulmonary embolism

- Low $ETCO_2$ values (<1.7 kPa/13 mmHg) while performing high quality chest compressions may support a diagnosis of pulmonary embolism, although it is a non-specific sign.
- Use fibrinolytic drugs for cardiac arrest when pulmonary embolism is the suspected cause of cardiac arrest.
- Use fibrinolytic drugs or surgical embolectomy or percutaneous mechanical thrombectomy for cardiac arrest when pulmonary embolism is the known cause of cardiac arrest.
- Consider ECPR as a rescue therapy for selected patients with cardiac arrest when conventional CPR is failing in settings in which it is implemented.
- Set-up a multidisciplinary team for making decisions on management of high-risk pulmonary embolism depending on local resources.

Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Molecule	Regimen	Contraindications to fibrinolysis
rtPA	100 mg over 2 h	**Absolute**
	0.6 mg/kg over 15 min (maximum dose 50 mg)[a]	History of haemorrhagic stroke or stroke of unknown origin
Streptokinase	250 000 IU as a loading dose over 30 min, followed by 100 000 IU/h over 12–24 h	Ischaemic stroke in previous 6 months
	Accelerated regimen: 1.5 million IU over 2 h	Central nervous system neoplasm
Urokinase	4400 IU/kg as a loading dose over 10 min, followed by 4400 IU/kg/h over 12–24 h	Major trauma, surgery, or head injury in previous 3 weeks
	Accelerated regimen: 3 million IU over 2 h	Bleeding diathesis
		Active bleeding
		Relative
		Transient ischaemic attack in previous 6 months
		Oral anticoagulation
		Pregnancy or first post-partum week
		Non-compressible puncture sites
		Traumatic resuscitation
		Refractory hypertension (systolic BP >180 mmHg)
		Advanced liver disease
		Infective endocarditis
		Active peptic ulcer

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Konstantinides SV, Meyer G, Becattini C, et al. 2019 ESC guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the european respiratory society (ERS). *Eur Heart J*. 2020;41(4):543-603. doi:10.1093/eurheartj/ehz405



Tension pneumothorax

Traumatic pneumothorax outside the hospital often results from

- penetrating injury,
- blunt trauma,
- rib fractures,
- or pulmonary decompression sickness.

In the hospital, traumatic pneumothorax may occur as a complication of procedures.

Procedural causes include:

- Central venous catheterization of the subclavian or internal jugular vein
- Lung biopsy
- Barotrauma from positive pressure ventilation
- Percutaneous tracheostomy
- Thoracentesis
- Pacemaker insertion
- Bronchoscopy
- Cardiopulmonary resuscitation
- Intercostal nerve block

Atraumatic pneumothorax may be primary with no identifiable cause or secondary to underlying lung disease

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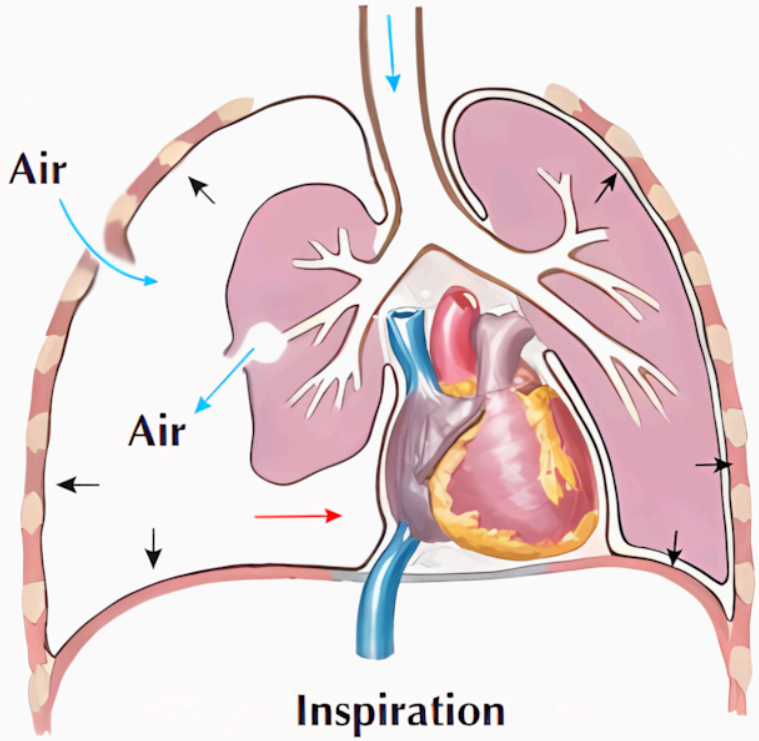
Sahota RJ, Sayad E. Tension pneumothorax. In: StatPearls. StatPearls Publishing; 2025. Accessed November 20, 2025. http://www.ncbi.nlm.nih.gov/books/NBK559090/



Hamidi H. Tension spontaneous pneumothorax. In: Radiopaedia.Org. Radiopaedia.org; 2020. doi:10.53347/rID-74883

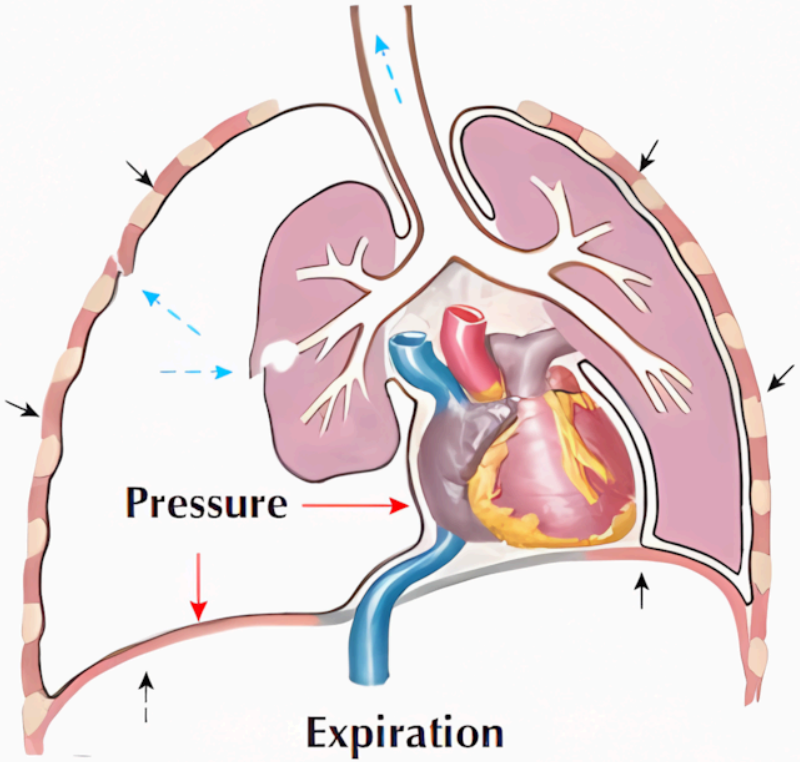
Tension Pneumothorax

Pathophysiology



Inspiration

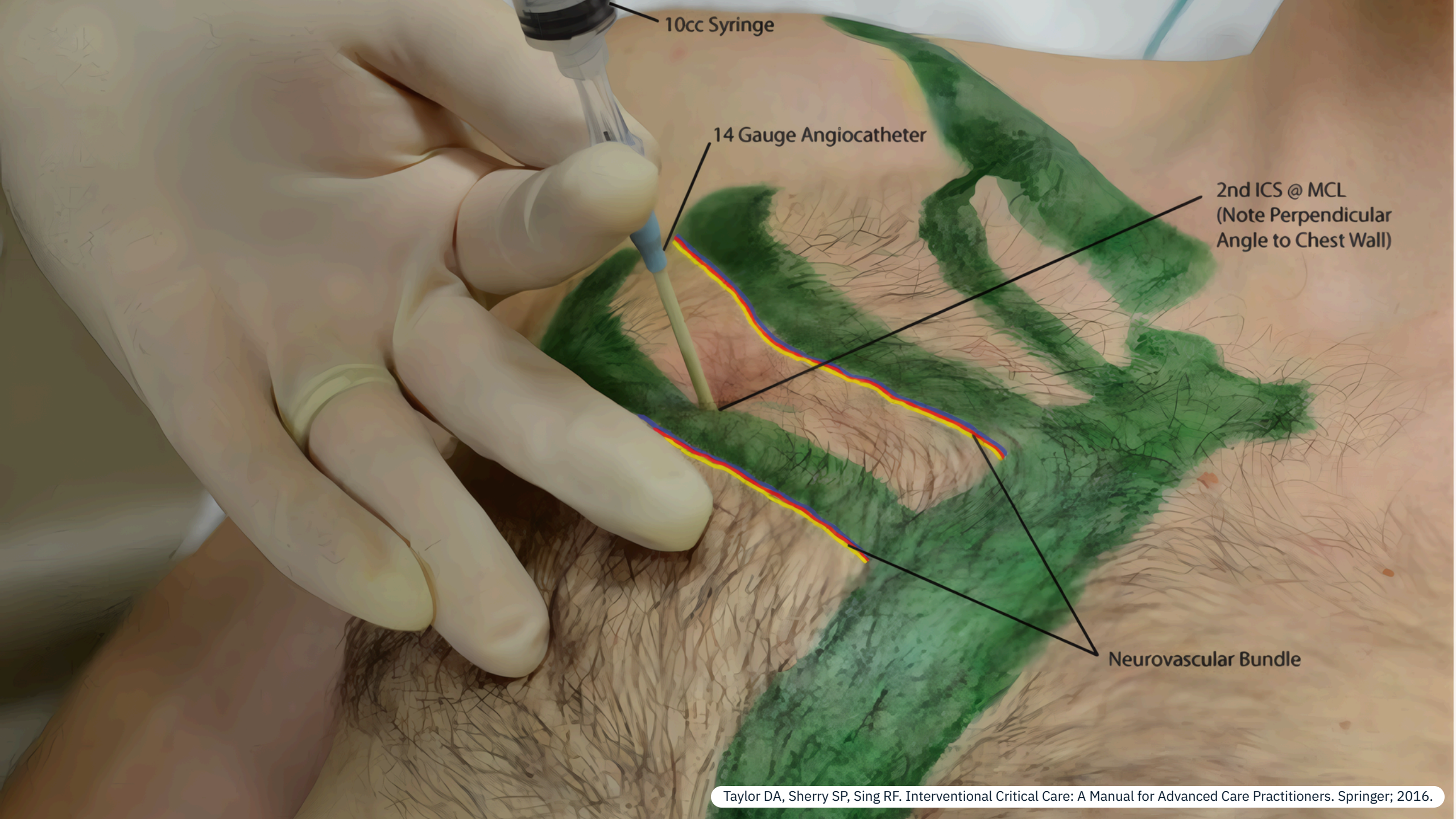
Air enters pleural cavity through lung wound or ruptured bleb (or occasionally via penetrating chest wound) with valvelike opening. Ipsilateral lung collapses, and mediastinum shifts to opposite side, compressing contralateral lung and impairing its ventilating capacity



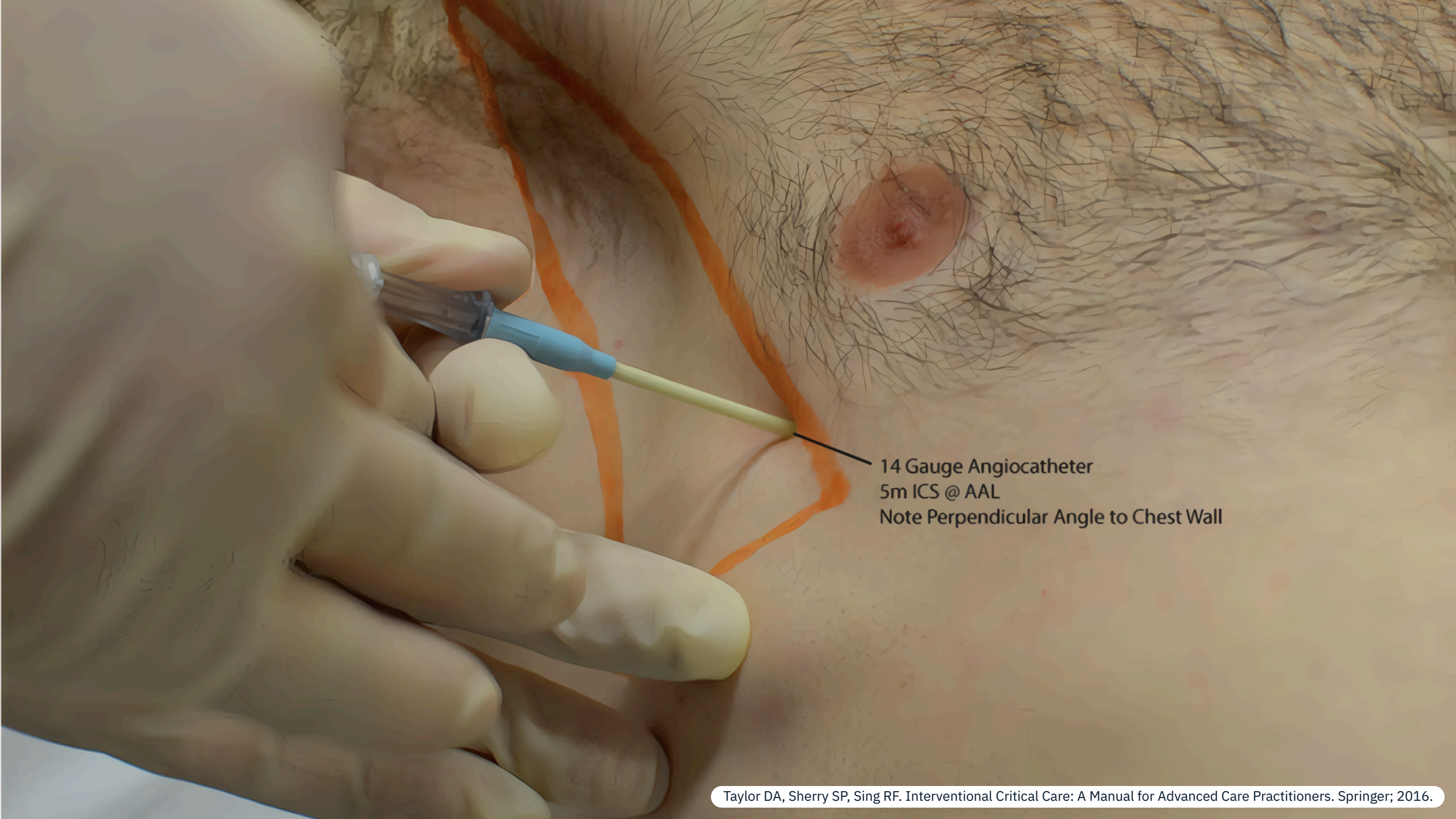
Expiration

Intrapleural pressure rises, closing valvelike opening, preventing escape of pleural air. Pressure is thus progressively increased with each breath. Mediastinal and tracheal shifts are augmented, diaphragm is depressed, and venous return is impaired by increased pressure and vena caval distortion

Smith RP. Netter Collection of Medical Illustrations: Netter Collection of Medical Illustrations: Reproductive System. 2nd ed. Elsevier - Health Sciences Division; 2011.

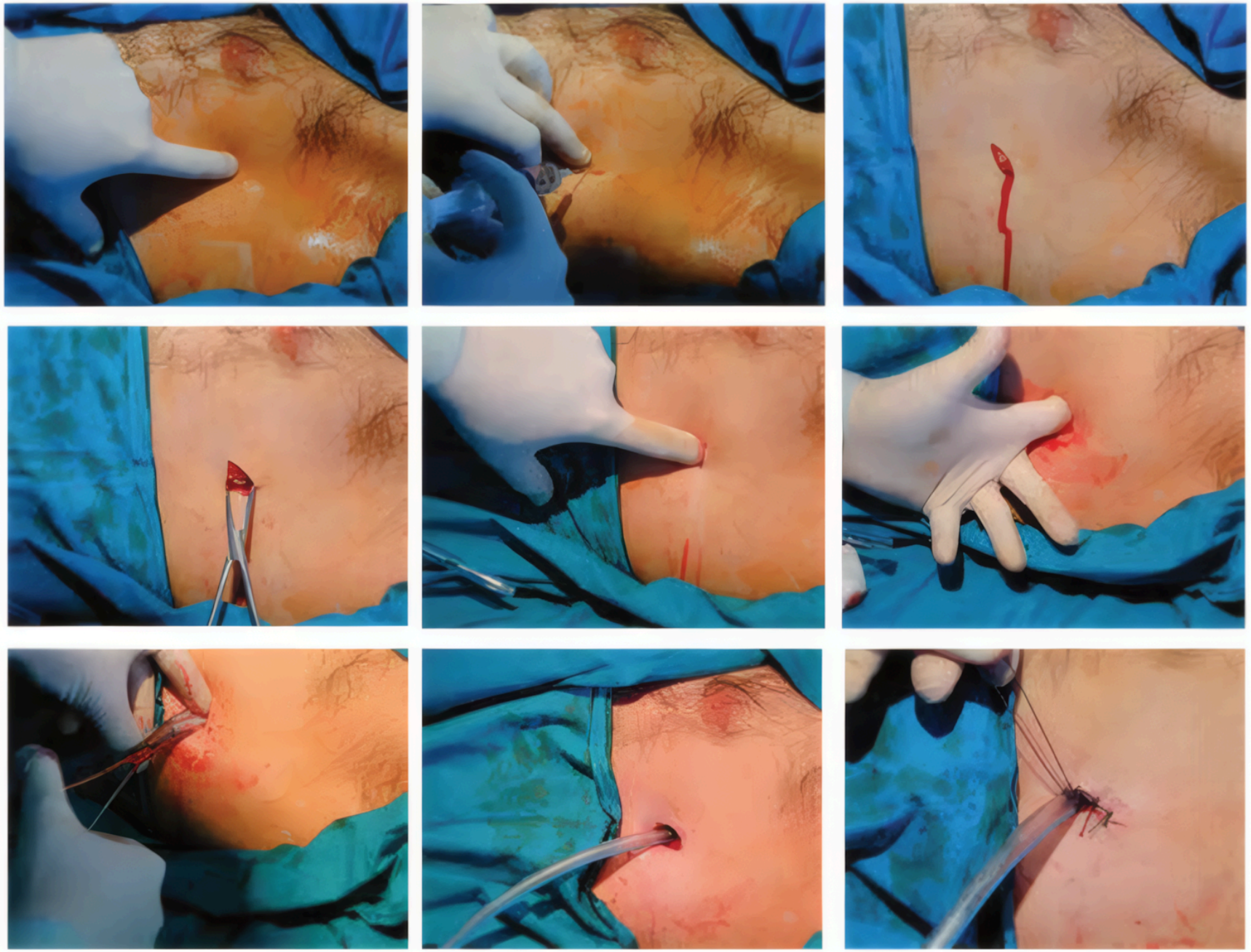


Taylor DA, Sherry SP, Sing RF. Interventional Critical Care: A Manual for Advanced Care Practitioners. Springer; 2016.



Taylor DA, Sherry SP, Sing RF. Interventional Critical Care: A Manual for Advanced Care Practitioners. Springer; 2016.


YouTube



Thies KC, Mountain A, Goode P, eds. The European Trauma Course Manual. 4.1 ed. European Resuscitation Counci

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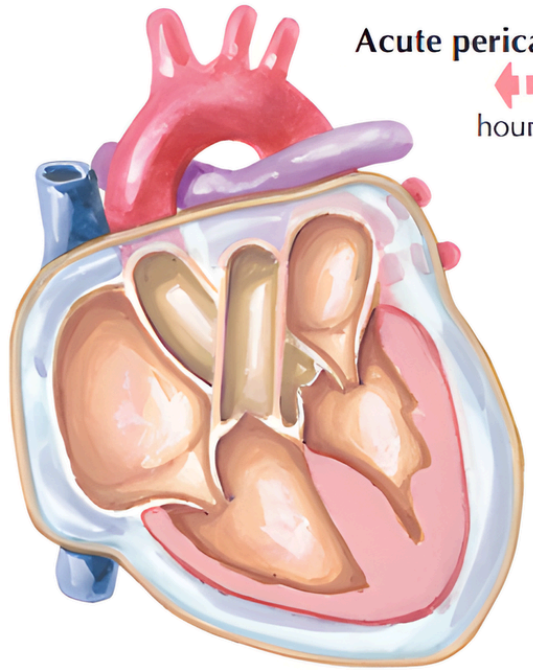
Thoracentesis

J. Terrill Huggins, Steven A. Sahn,
and Peter Doelken

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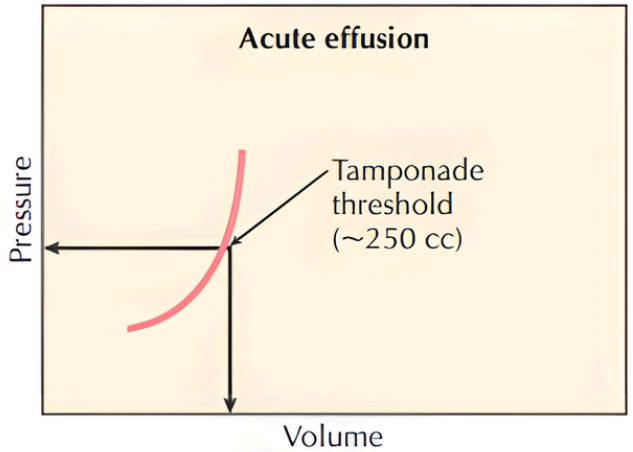

YouTube

Cardiac Tamponade



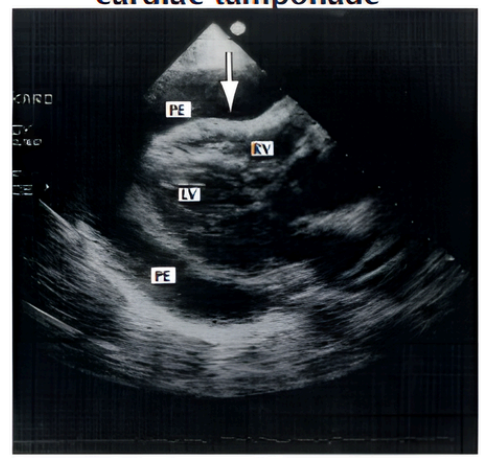
Acute pericardial effusion

hours/days



Normal (unstretched) pericardium is able to accommodate acute fluid accumulation up to ~250 cc, beyond which additional volume markedly increases intrapericardial pressure.

Echocardiographic findings in cardiac tamponade

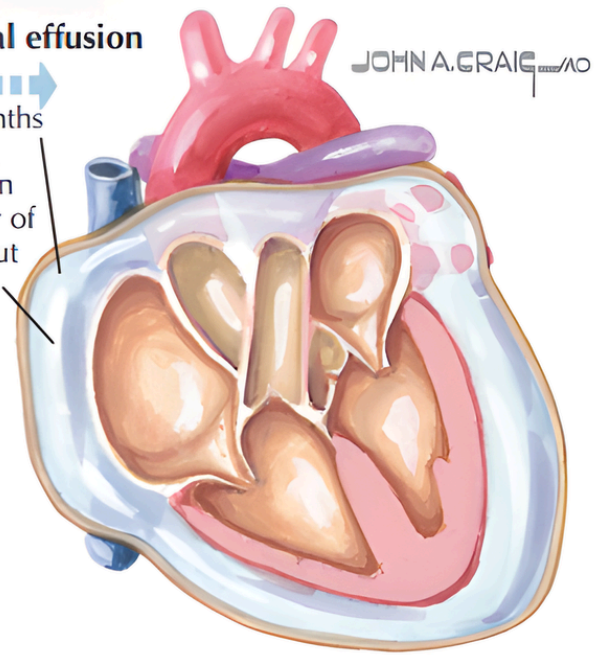


Long-axis view shows RV collapse due to large PE.

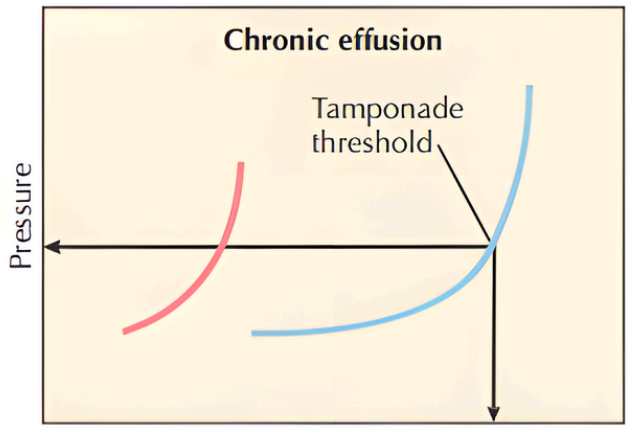
Chronic pericardial effusion

weeks/months

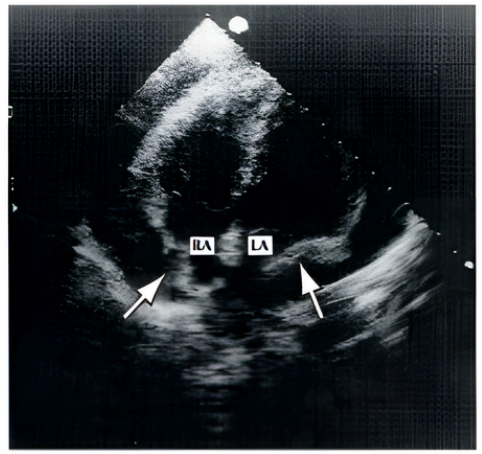
May contain up to a liter of fluid without tamponade



JOHN A. CRAIG MD

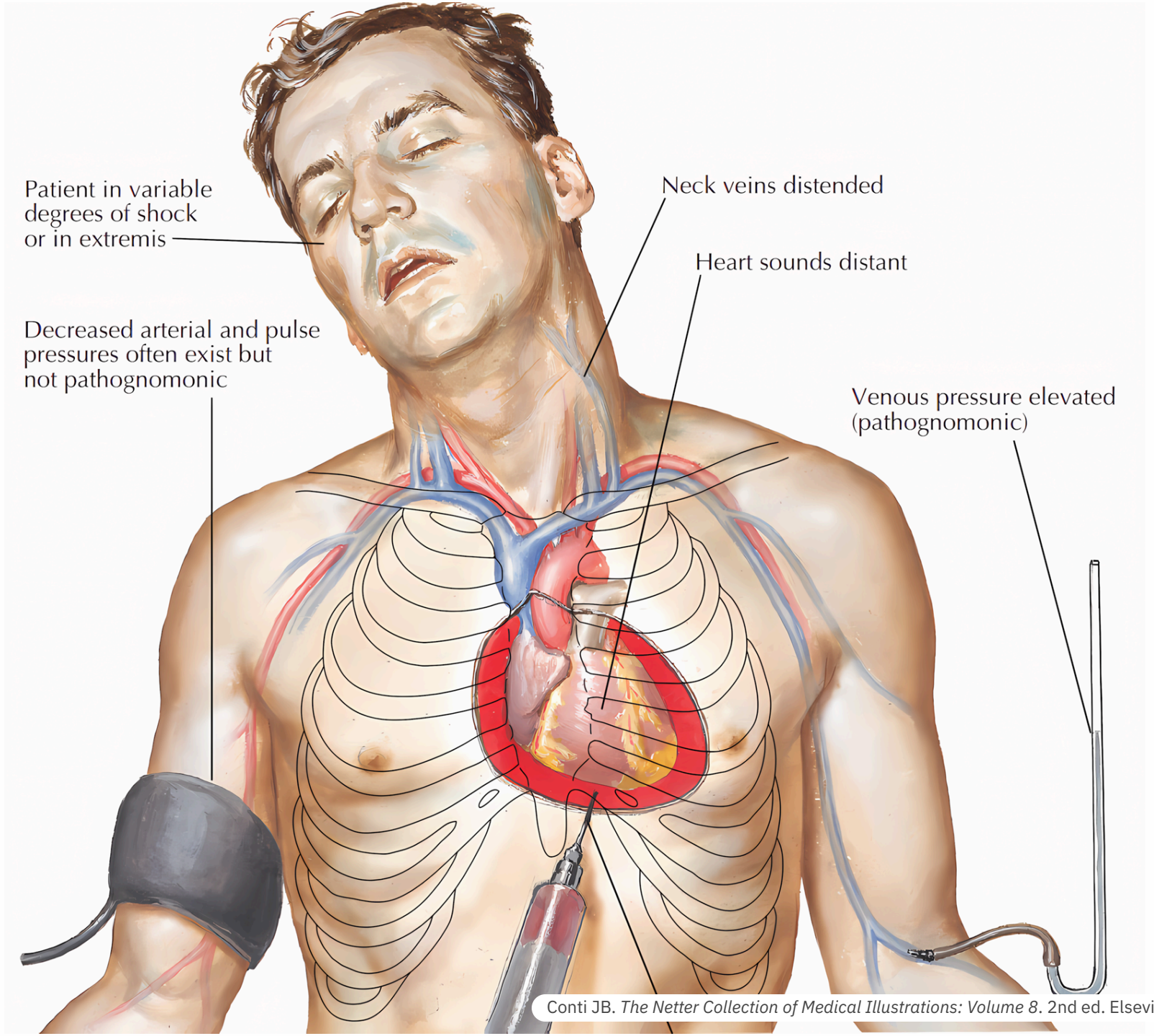


Pericardium that has been stretched over time by long-standing effusion is more distensible and is able to accommodate large fluid volume without critical increase in intrapericardial pressure.



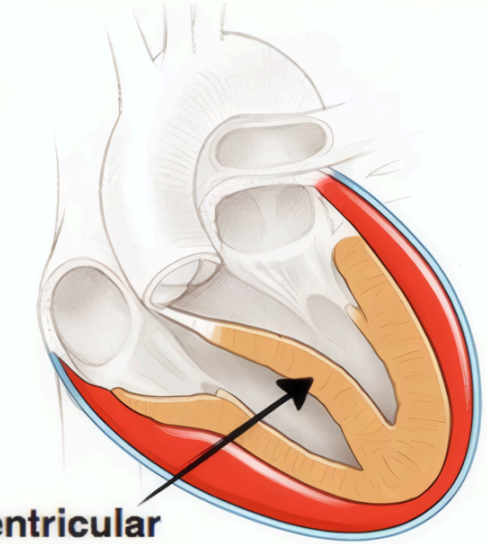
Four-chamber view demonstrates collapse of both RA and LA due to tamponade.

Stouffer GA, Runge MS, Patterson C, Rossi JS, Netter FH, eds. *Netter's Cardiology*. 3rd edition. Elsevier; 2019.



Conti JB. *The Netter Collection of Medical Illustrations: Volume 8*. 2nd ed. Elsevier - Health Sciences Division; 2014.

Cardiac Tamponade



Ventricular wall collapse

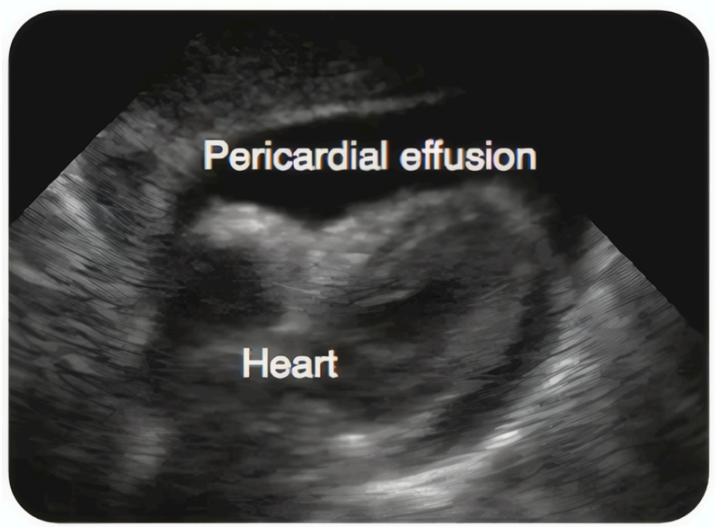
Beck's Triad

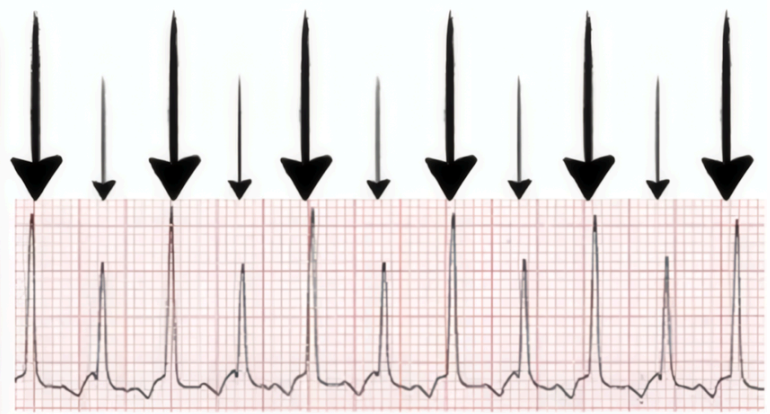
1. Hypotension
2. Jugular venous distension
3. Muffled heart sounds

Don't mix up with:

Tension pneumothorax

1. Hypotension
2. Jugular venous distension
3. **Absent breath sounds**





Electrical Alternans

https://www.grepmed.com/images/5140/tamponade-pericardial-cardiac-cardiology-triad

ELSEVIER

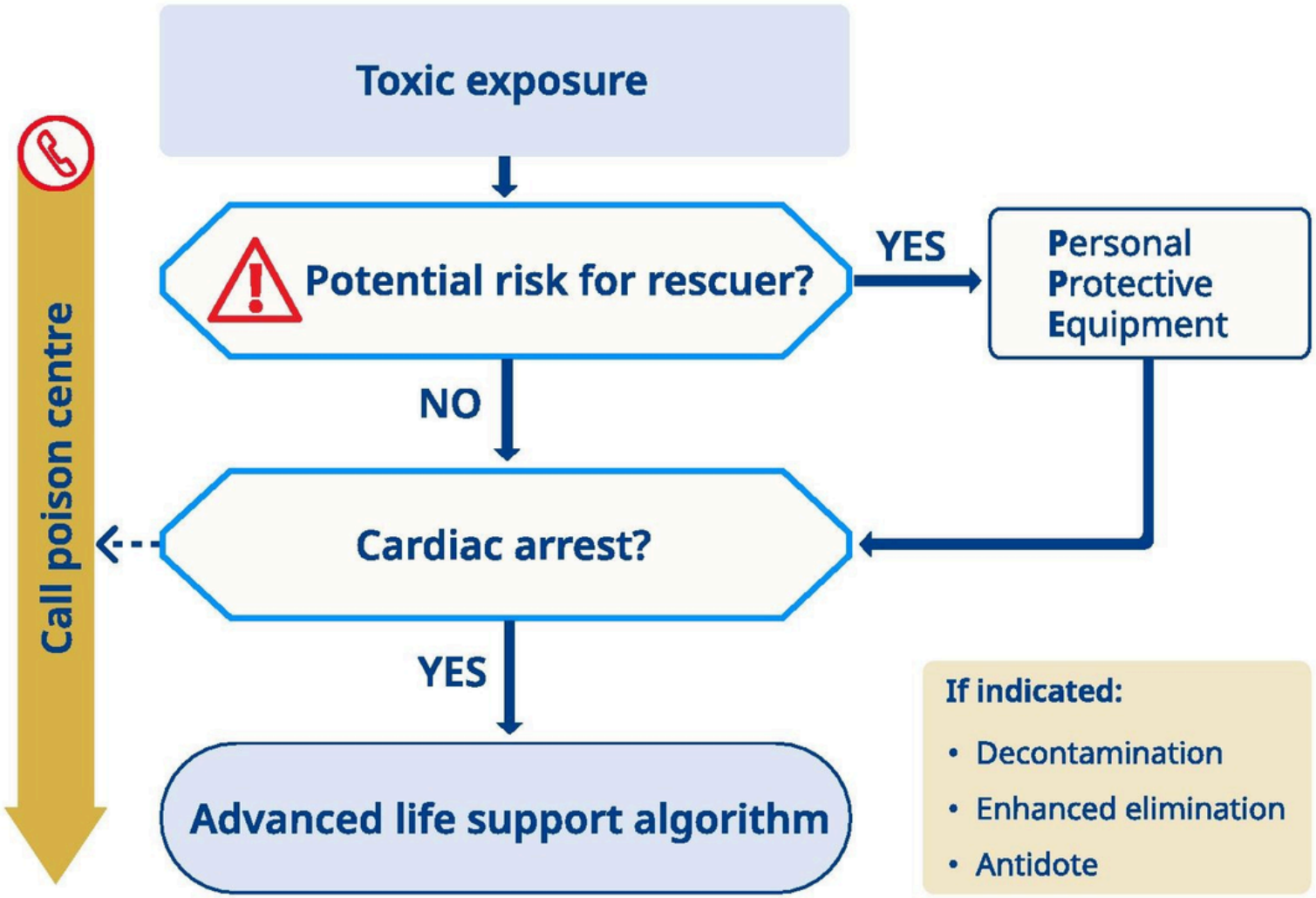
Pericardiocentesis

Stefano Maggiolini, Giovanni Vitale,
and Felice Achilli

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Toxic agents

GUIDELINES 2025
EUROPEAN RESUSCITATION COUNCIL©



Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753

Organophosphate poisoning

Source

Chemical warfare agents include sarin, soman, tabun, and VX.

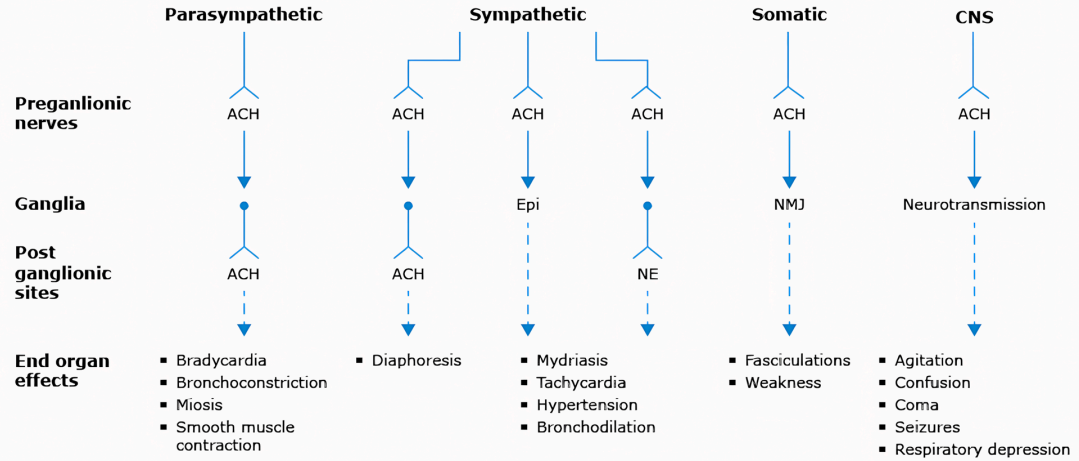
Insecticides include diazinon, dichlorvos, ethion, fenthion, malathion, and parathion.

Mechanism

Organophosphates inhibit acetylcholinesterase in peripheral and central synapses and in red blood cells. Aging of the enzyme produces irreversible inhibition.

Kloss BT, ed. *Toxicology in a Box*. McGraw-Hill Education; 2014.

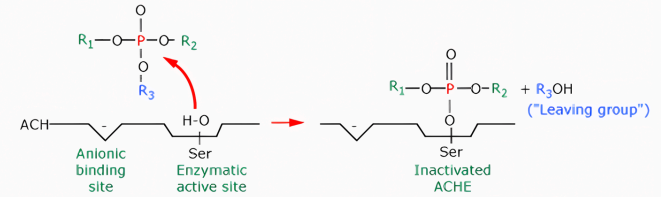
Neurologic effects of organophosphate and carbamate agents

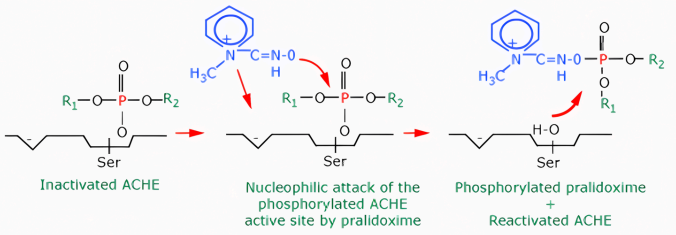


ACH: acetylcholine; CNS: central nervous system; Epi: epinephrine; NE: norepinephrine; NMJ: neuromuscular junction.

UpToDate®

Mechanism of action: Organophosphate and pralidoxime





UpToDate®

Organophosphate poisoning

Clinical features

Muscarinic signs predominate, including miosis, bradycardia, salivation, diaphoresis, bronchorrhea, bronchospasm, nausea, vomiting, diarrhea, lacrimation, urinary incontinence, confusion, lethargy, coma, seizure, death.

Nicotinic signs include muscle fasciculations and weakness.

Management

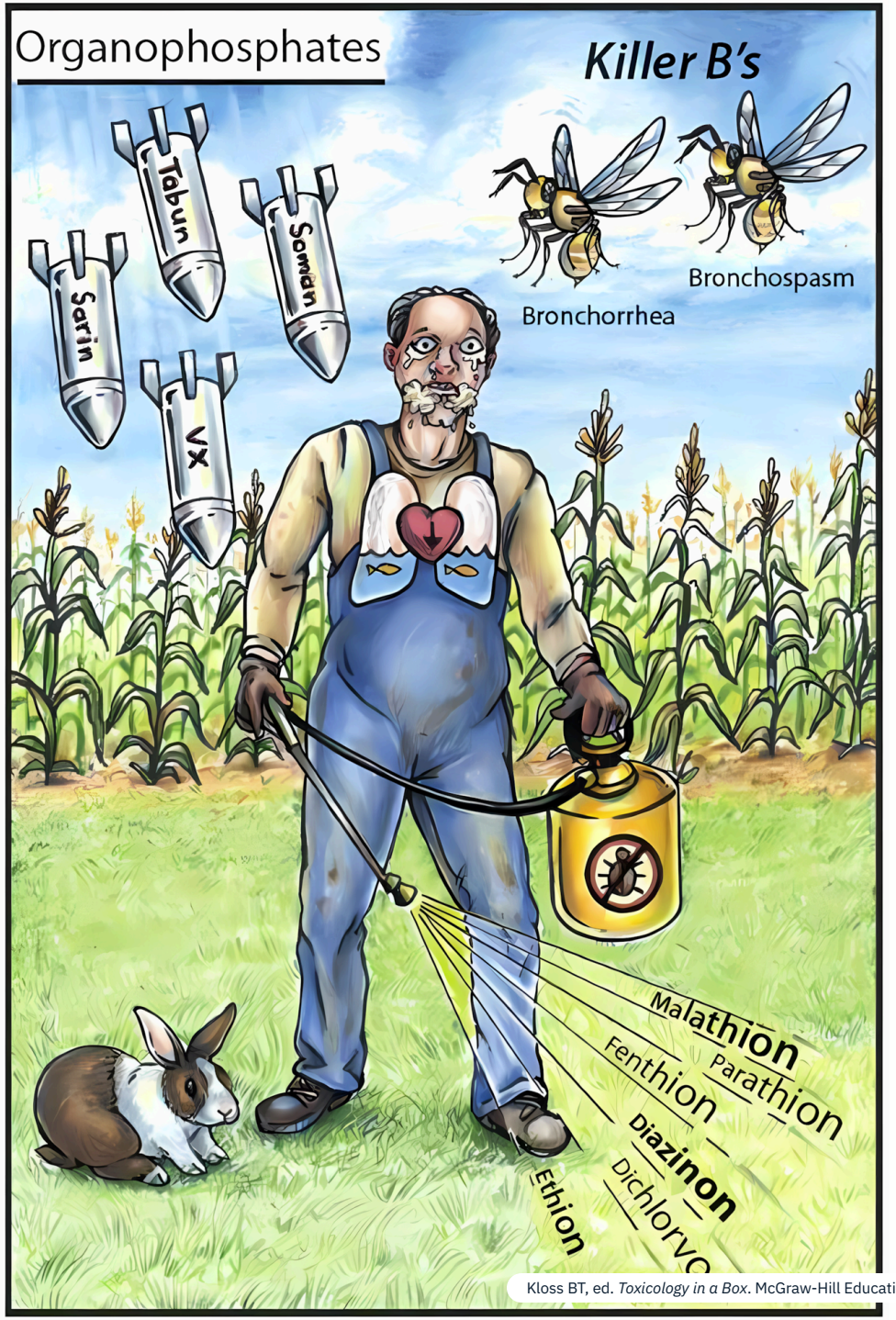
Decontamination and supportive care.

Atropine for respiratory secretions.
Initial dose 2 to 5 mg IV in adults or 0.05 mg per kg in children, doubled every 3 to 5 minutes until respiratory improvement.

Pralidoxime for neuromuscular effects.
Dose 1 to 2 g IV over 30 minutes in adults or 20 to 50 mg per kg in children.

Benzodiazepines for muscle spasm, sedation, seizure control. Diazepam preferred.

Autoinjectors with atropine and pralidoxime for prehospital or military use.



Kloss BT, ed. *Toxicology in a Box*. McGraw-Hill Education; 2014.

LAST

MECHANISMS OF LAST

NYSORA Learning System

LAs are generally safe and effective in therapeutic doses for tissue infiltration, fascial planes, or near a nerve/plexus of nerves. However, supratherapeutic plasma levels of LAs can result in **Local Anesthetic Systemic Toxicity (LAST)**.

PLASMA CONCENTRATION FACTORS

High plasma concentration of LAs:

- Partial venous/arterial injection
- Intravascular injection
- Rapid vascular absorption from the highly vascularized injection site

Plasma levels of LA: Proportional to the rate of systemic absorption from the site of therapy.

Rate of absorption:

- Varies among tissues
- Depends on the size of the absorptive surface
- Depends on vascularization of the tissue planes where the injection is made
- ↑ Doses = ↑ plasma levels of LAs, independent of the injection site

LA: MECHANISMS ON THE CELLULAR LEVEL

- **Inhibitory action** on nerve conduction by inhibiting the movement of ions through voltage-gated ionotropic channels at the level of the cell membrane
- **Primary therapeutic target** = voltage-gated sodium channel where inhibition alters the transmission of sensory and motor signals in axons
- **ALSO:** LAs inhibit voltage-gated Ca2+ channels, K+ channels, the Na-K ATPase, and other channels and enzymes
- **Non-ionized vs Ionized molecules:** The inhibition occurs from the intracellular side and requires LAs to cross the lipid bilayer first as unbound, non-ionized free molecules
- **At lower concentrations:** LAs block protein kinase signaling induced by tumor necrosis factor α (TNF-α)
- **At higher concentrations:** LAs can inhibit other channels, enzymes, and receptors, including the carnitine-acylcarnitine translocase in the mitochondria

LA & CARDIAC TOXICITY

Cardiovascular toxicity: Caused by the combination of electrophysiologic and contractile dysfunction

CAUTION: Bupivacaine = lipophilic and greater affinity for the voltage-gated sodium channels, resulting in its uniquely high cardiotoxic profile

More on bupivacaine: Cardiac toxicity can occur at lower serum concentrations because it can accumulate in the mitochondria and cardiac tissue at a ratio of about 6:1 (or greater) relative to plasma

https://www.nysora.com/topics/complications/local-anesthetic-systemic-toxicity/

LAST: SYMPTOMS, DIAGNOSIS & TREATMENT

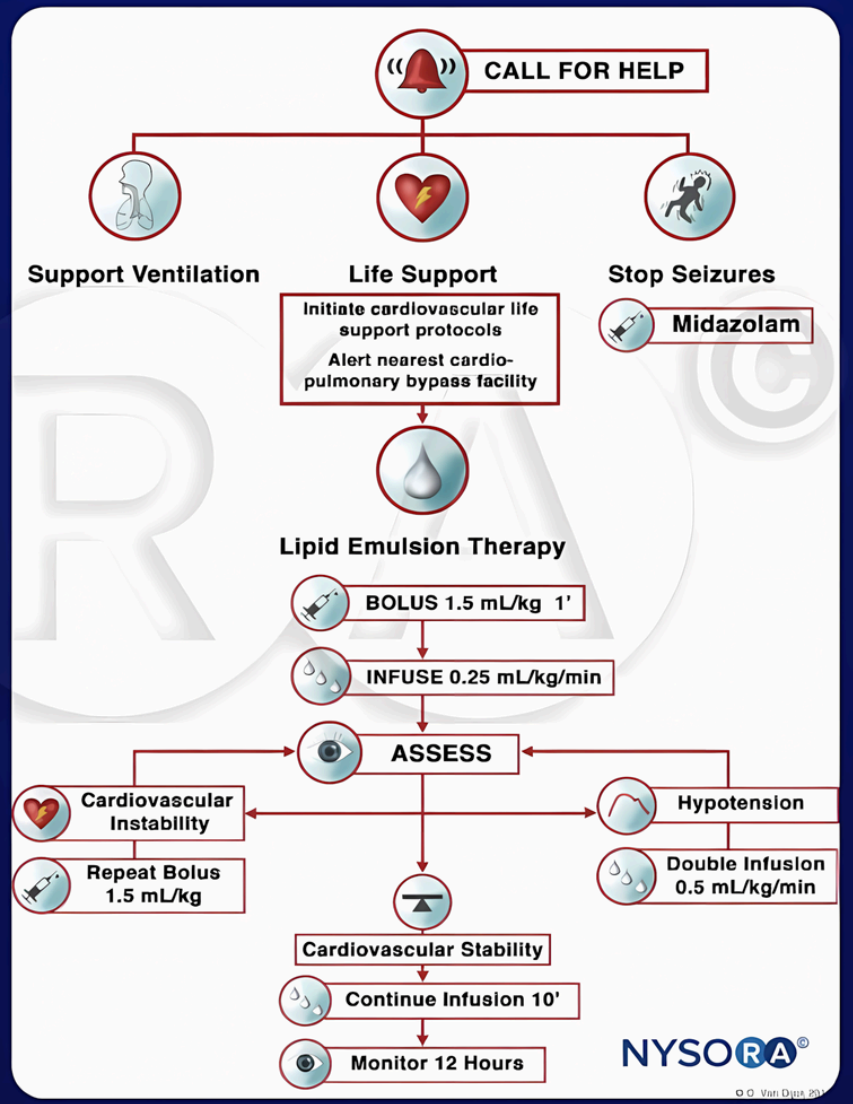
NYSORA Learning System

SYMPTOMS OF LAST (LOCAL ANESTHETIC SYSTEMIC TOXICITY)

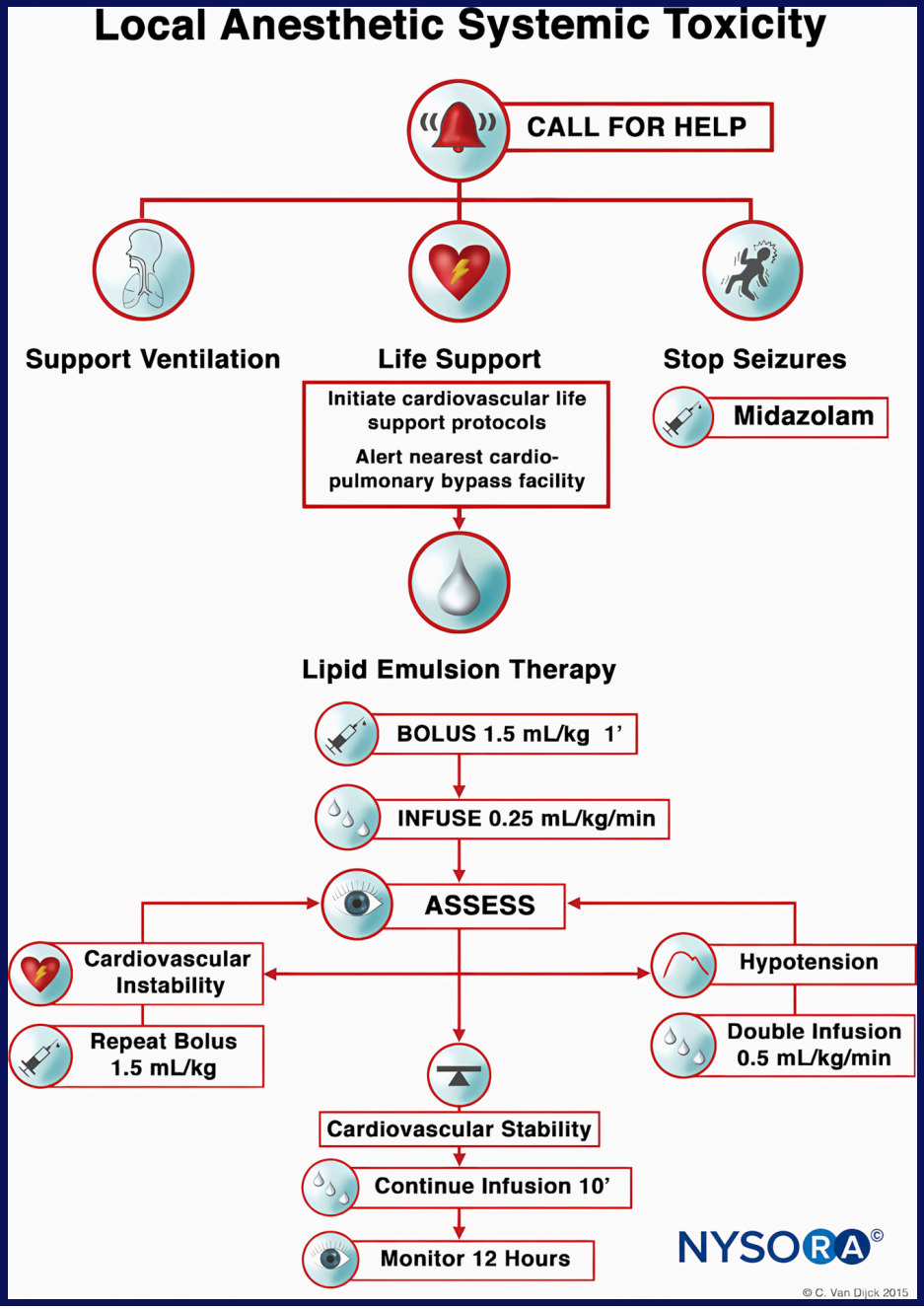
- CNS more sensitive to LA toxicity than CVS → CNS symptoms precede CVS symptoms
- LAs affect the balance between inhibitory and excitatory pathways in the CNS
- Neurological signs/symptoms range between excitation (early) and depression (late)
- Blockade of the voltage-gated sodium channels of inhibitory cortical neurons in the CNS occurs first → initially LAST = excitatory manifestations
- **Neurological symptoms:** Seizures *(70%)*, agitation *(10%)*, or loss of consciousness *(7%)*
- **Prodromal (EARLY) symptoms:** Perioral paresthesia, metallic taste, and tinnitus
- **± 40% of LAST -** present as a sudden, rapid-onset seizure, progressing to cardiac arrest
- With high dose of LA or inadvertent IV injection, EARLY (prodromal) CNS symptoms may be absent and the first manifestation could be CVS toxicity (11%)
- **CVS toxicity:** Heart conduction anomalies, ↓ cardiac contractility, ↓ systemic vascular resistance
- **Early-onset ECG changes:** ↑ PR and ↑ QTc, QRS abnormalities (bundle branch blocks) and ↑ ST intervals with/without refractory brady/tachyarrhythmias
- Depression of spontaneous pacemaker activity can rapidly lead to high-degree AV blocks or **asystole**
- Cardiogenic shock and refractory hypotension may follow as a result of ↓ cardiac contractility and vasomotor control disturbances, caused by peripheral vascular ion-channel alterations

DIAGNOSIS

- LAST can occur immediately at the time of injection *(usually accidental intravascular injection)* or up to an hour after it *(due to delayed tissue absorption)*
- Continue monitoring for 30-45 min after injection of large volumes or toxic doses of LAs
- Monitor patients with **any signs of LAST** for 2-6 hrs because cardiovascular depression due to LAs can persist or recur after treatment



https://www.nysora.com/topics/complications/local-anesthetic-systemic-toxicity/

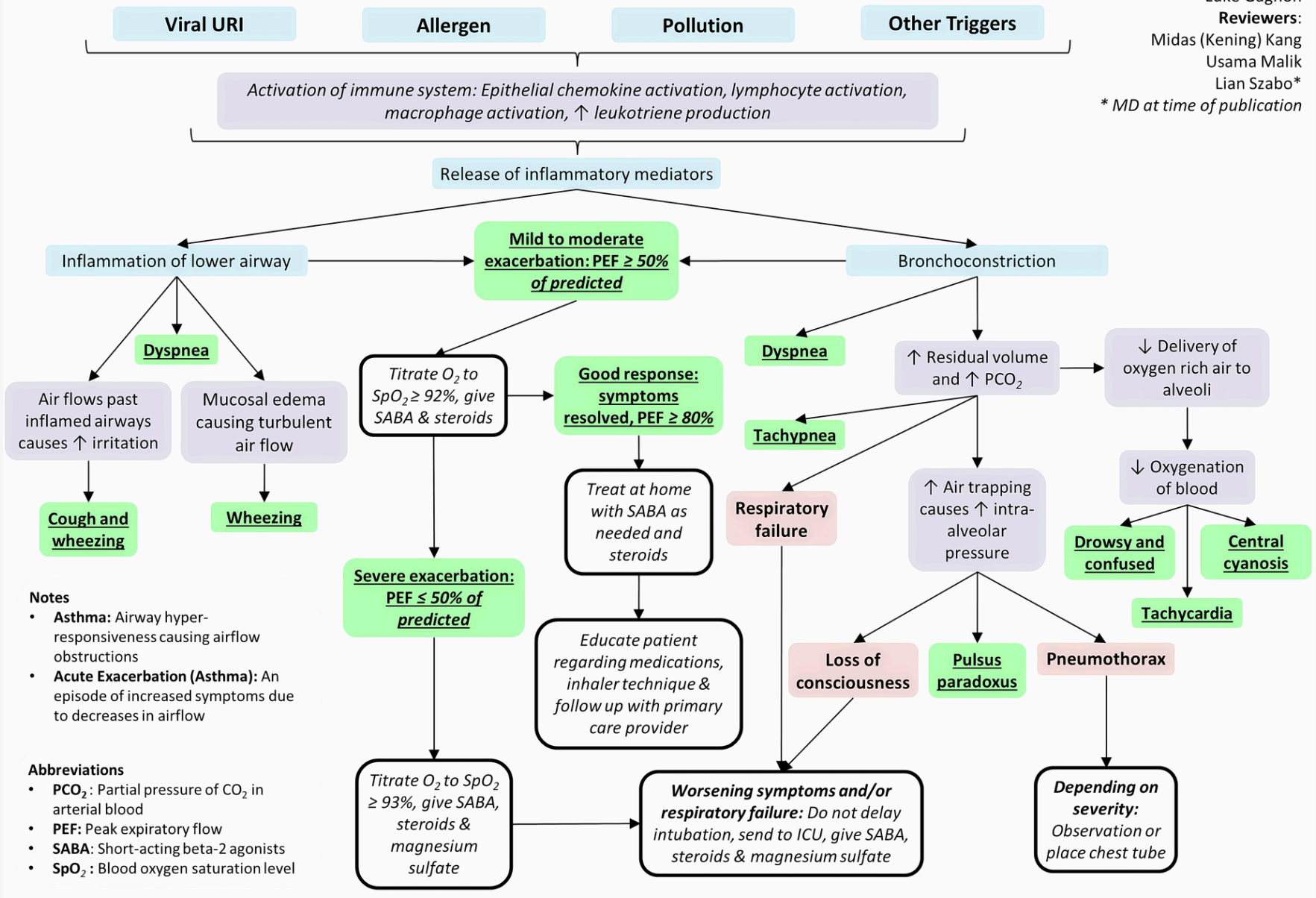


https://www.nysora.com/topics/complications/local-anesthetic-systemic-toxicity/

Asthma and chronic obstructive pulmonary disease

Asthma Acute Exacerbation: *Pathogenesis and Treatment*

Authors:
Luke Gagnon
Reviewers:
Midas (Kening) Kang
Usama Malik
Lian Szabo*
** MD at time of publication*



Notes

- **Asthma:** Airway hyper-responsiveness causing airflow obstructions
- **Acute Exacerbation (Asthma):** An episode of increased symptoms due to decreases in airflow

Abbreviations

- **PCO_2**: Partial pressure of CO_2 in arterial blood
- **PEF:** Peak expiratory flow
- **SABA**: Short-acting beta-2 agonists
- **SpO_2**: Blood oxygen saturation level

Legend: Pathophysiology | Mechanism | Sign/Symptom/Lab Finding | Complications

Published **February 06, 2018** on www.thecalgaryguide.com

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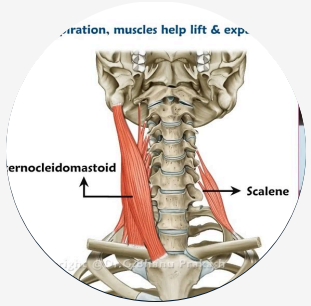
Simplified Acute Asthma Severity Scoring

Sign or Symptom	Mild	Moderate	Severe
Pulse rate, beats/min	< 90	91-119	> 120
Wheezing	Absent	Present	Present
Rales	Absent	Present	Present
Prolonged expirium	Absent	Present	Present
Oxygen saturation, %	95-100	90-94	< 89
Use of accessory muscles	Absent	Present	Present

Garner O, Ramey JS, Hanania NA. Management of life-threatening asthma. *Chest*. 2022;162(4):747-756. doi:10.1016/j.chest.2022.02.029

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Severe Asthma Clinical Findings



Use of accessory muscles of respiration

Brief, fragmented speech; inability to lie supine; profound diaphoresis; agitation; severe symptoms that fail to improve with initial emergency treatment

Life-threatening airway obstruction

Can still occur when the above signs are NOT present

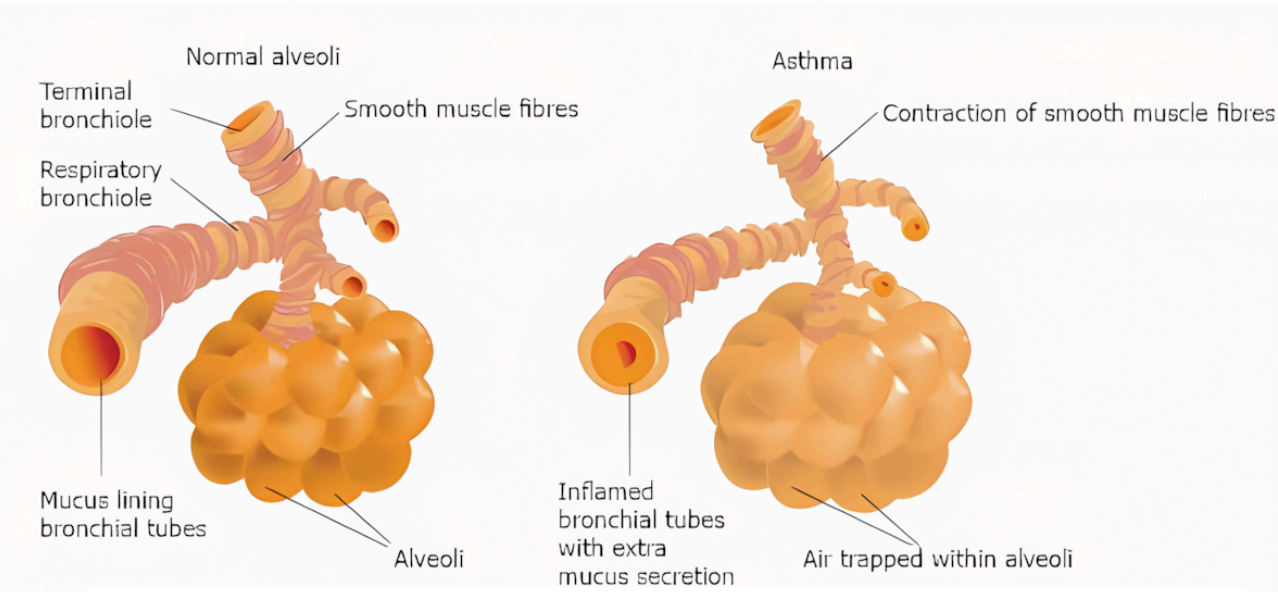
Signs of imminent respiratory arrest

Cyanosis, inability to maintain respiratory effort, and depressed mental status

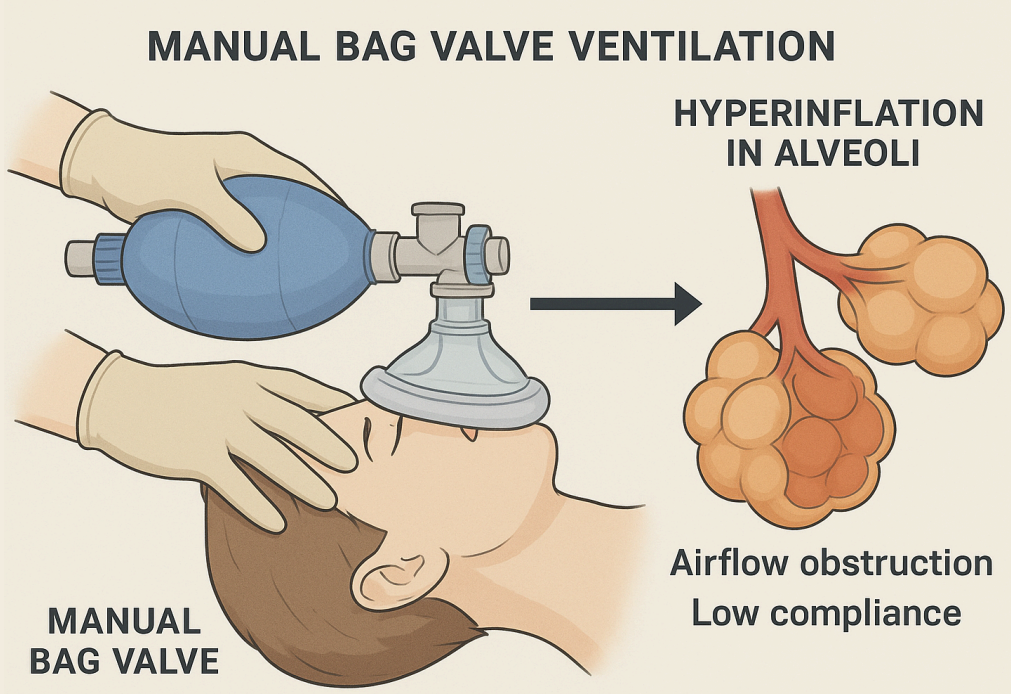
Recent case reports describe electromechanical dissociation triggered by positive pressure ventilation in patients with obstructive airway disease.

- In these cases, return of spontaneous circulation occurred after ventilation and resuscitative efforts were briefly stopped.
- During cardiopulmonary resuscitation, rapid manual ventilation without adequate exhalation can lead to dynamic hyperinflation in obstructive airway disease.
- The resulting auto PEEP increases intrathoracic pressure, reduces venous return, and lowers cardiac output even when an electrical rhythm is present.
- Short withdrawal of ventilation reduces dynamic hyperinflation, lowers intrathoracic pressure, and can restore circulation. Evidence remains limited and the true frequency of this mechanism is not known.

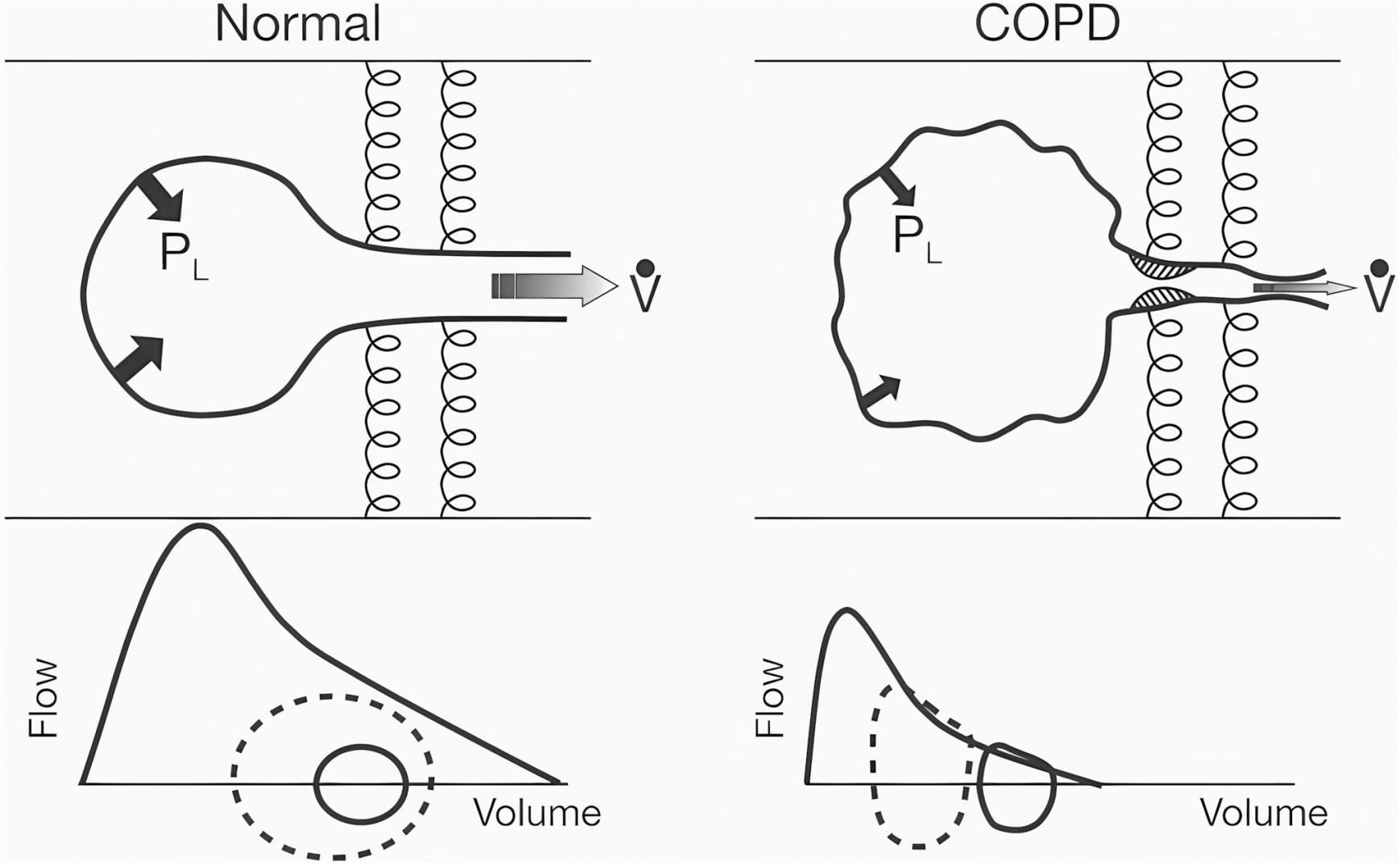
Lapinsky SE, Leung RS. Auto-PEEP and electromechanical dissociation. *N Engl J Med*. 1996;335(9):674-675. doi:10.1056/NEJM199608293350916



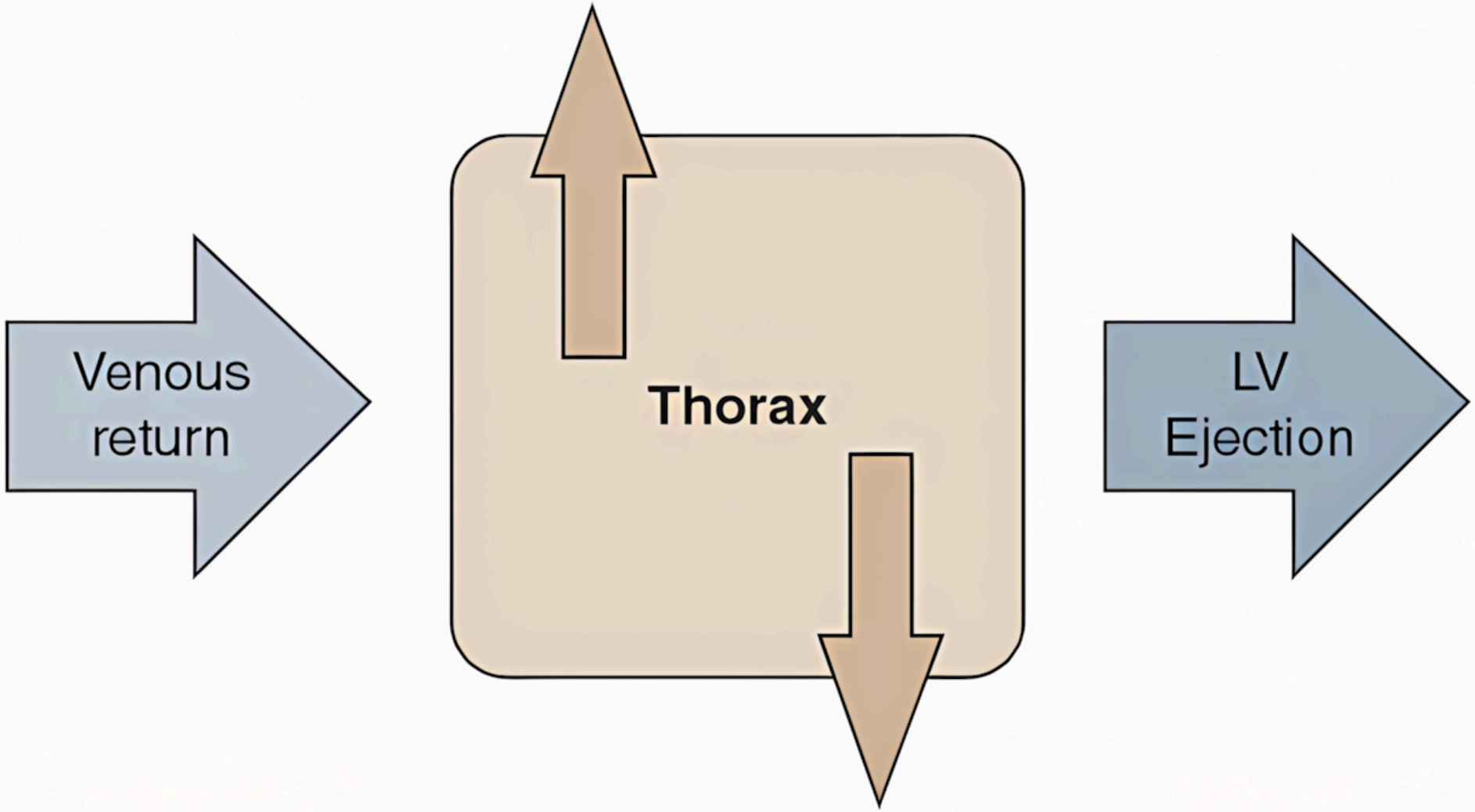
Gomes EL de FD, Costa D. Evaluation of functional, autonomic and inflammatory outcomes in children with asthma. *World J Clin Cases*. 2015;3(3):301-309. doi:10.12998/wjcc.v3.i3.301



$$\tau = C_L \times R_{\text{aw}}$$



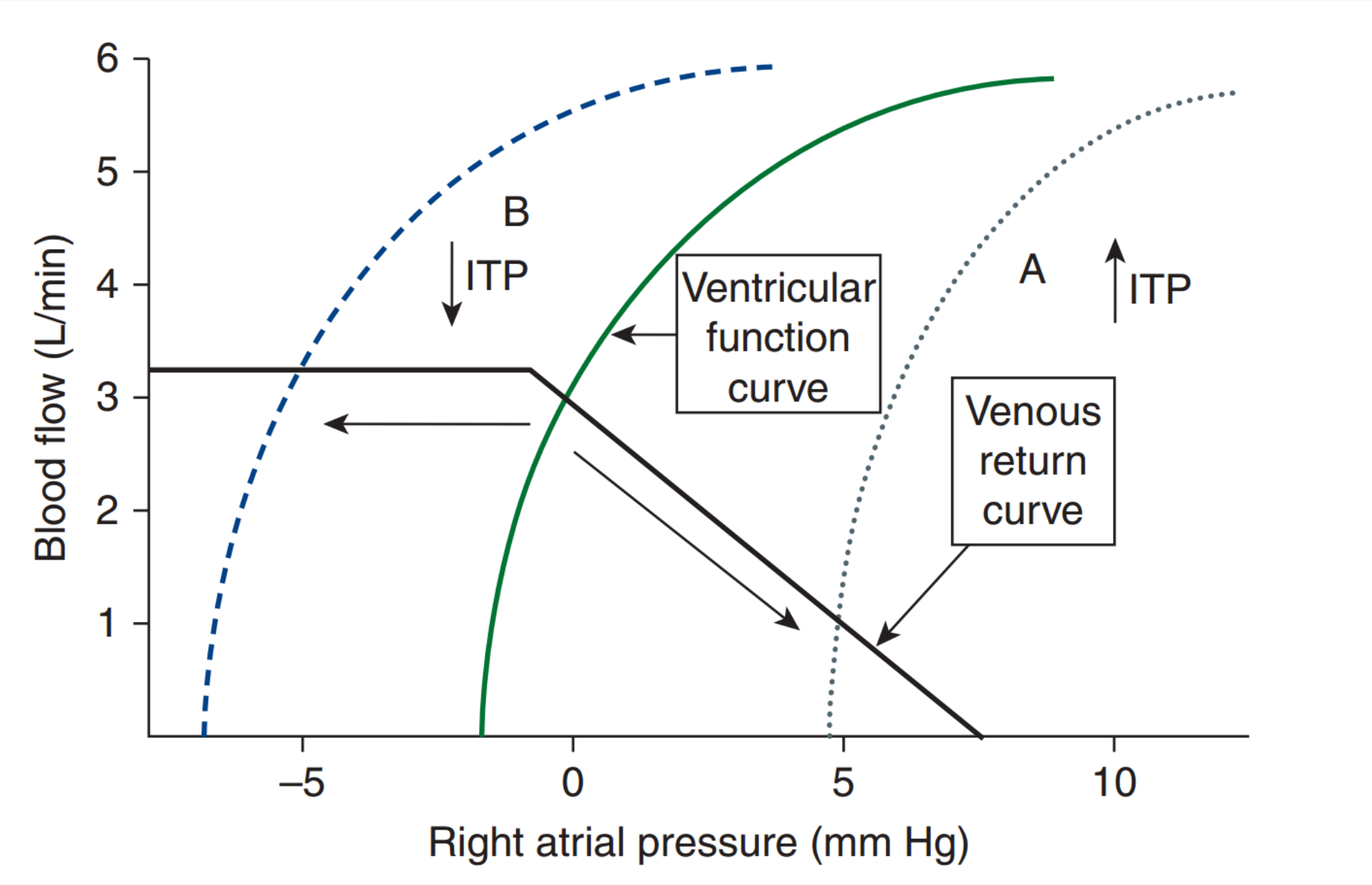
Thomas M, Decramer M, O'Donnell DE. No room to breathe: the importance of lung hyperinflation in COPD. *Prim Care Respir J*. 2013;22(1):101-111. doi:10.4104/pcrj.2013.00025



Increasing ITP
Decreases the pressure gradients for venous return and LV ejection

Decreasing ITP
Increases the pressure gradients for venous return and LV ejection

Tobin MJ. *Principles and Practice of Mechanical Ventilation*. 3rd ed. McGraw-Hill medical; 2013.

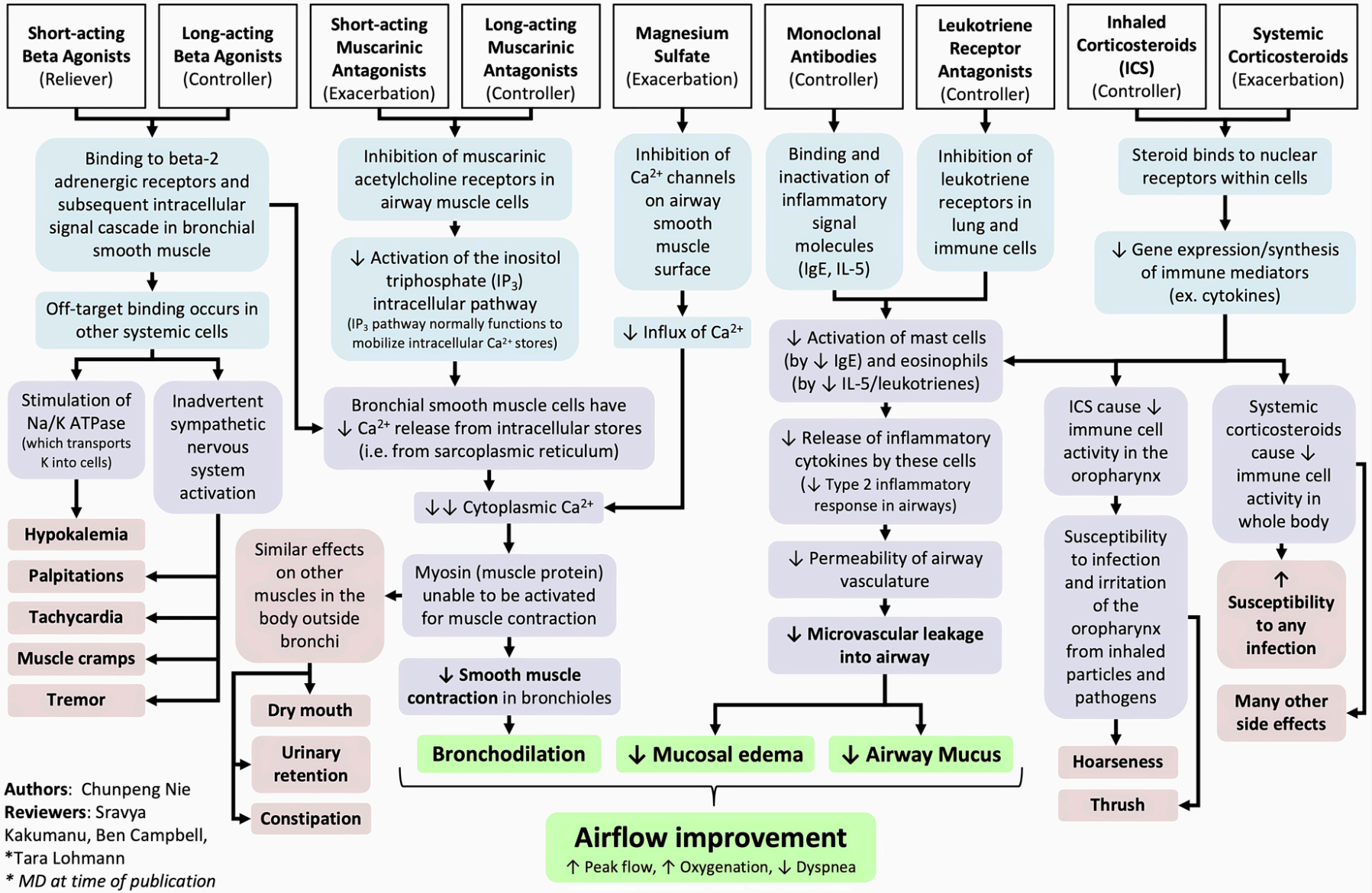


Tobin MJ. *Principles and Practice of Mechanical Ventilation*. 3rd ed. McGraw-Hill medical; 2013.

Asthma: *How Treatments Work and Common Side Effects*

- **Relievers:** Used as needed to ↓ symptoms during attacks
- **Controllers:** Used daily to ↓ frequency and severity of attacks
- **Exacerbation:** Used emergently in acute exacerbation

Note: Please see slide on *Asthma: Pathogenesis*



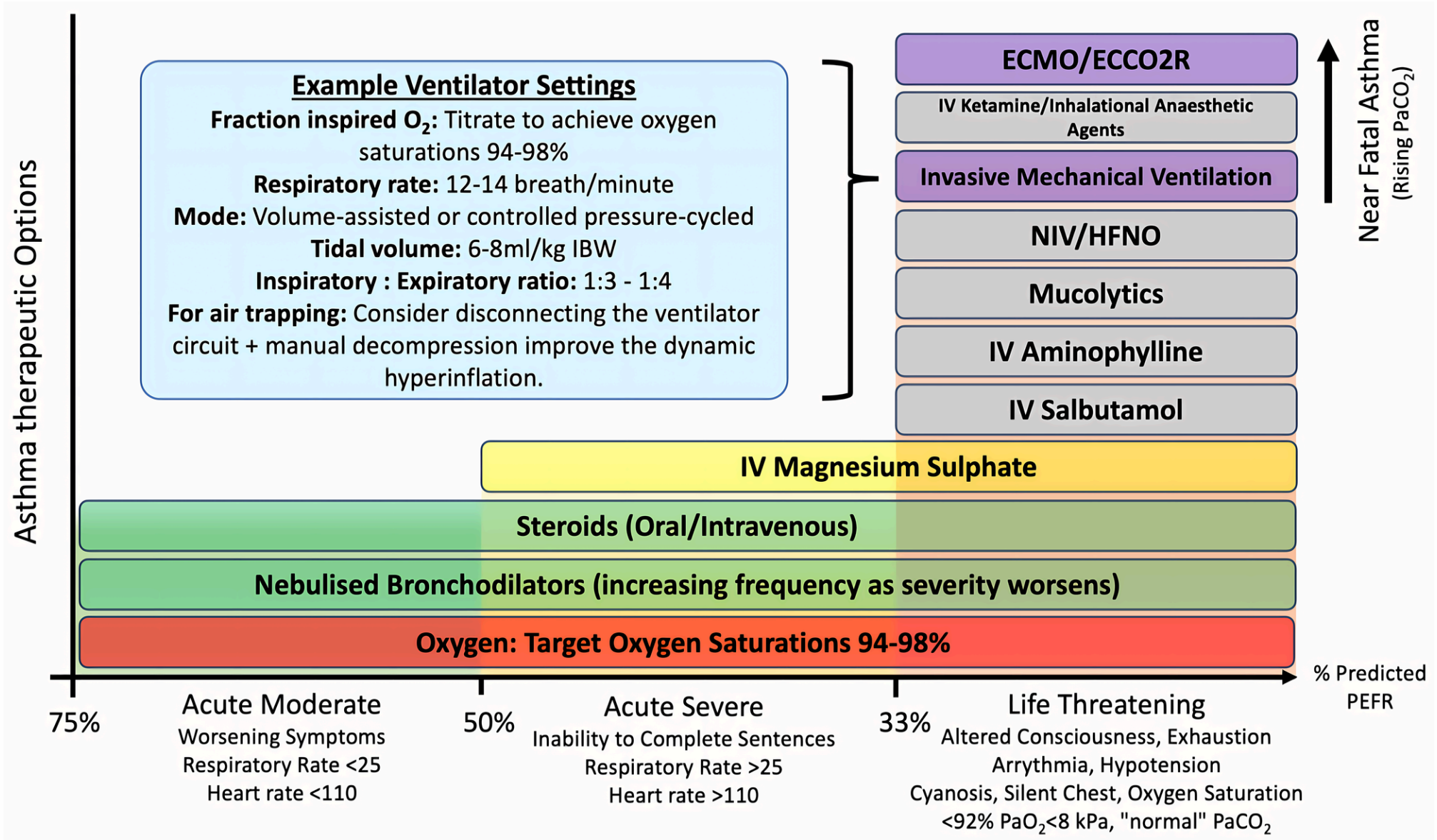
Authors: Chunpeng Nie
Reviewers: Sravya Kakumanu, Ben Campbell, *Tara Lohmann
** MD at time of publication*

Legend: Pathophysiology | Mechanism | Treatment Effect | Complications

Published **October 9, 2022** on www.thecalgaryguide.com

https://calgaryguide.ucalgary.ca/asthma-how-treatments-work-and-common-side-effects/

Anaphylaxis



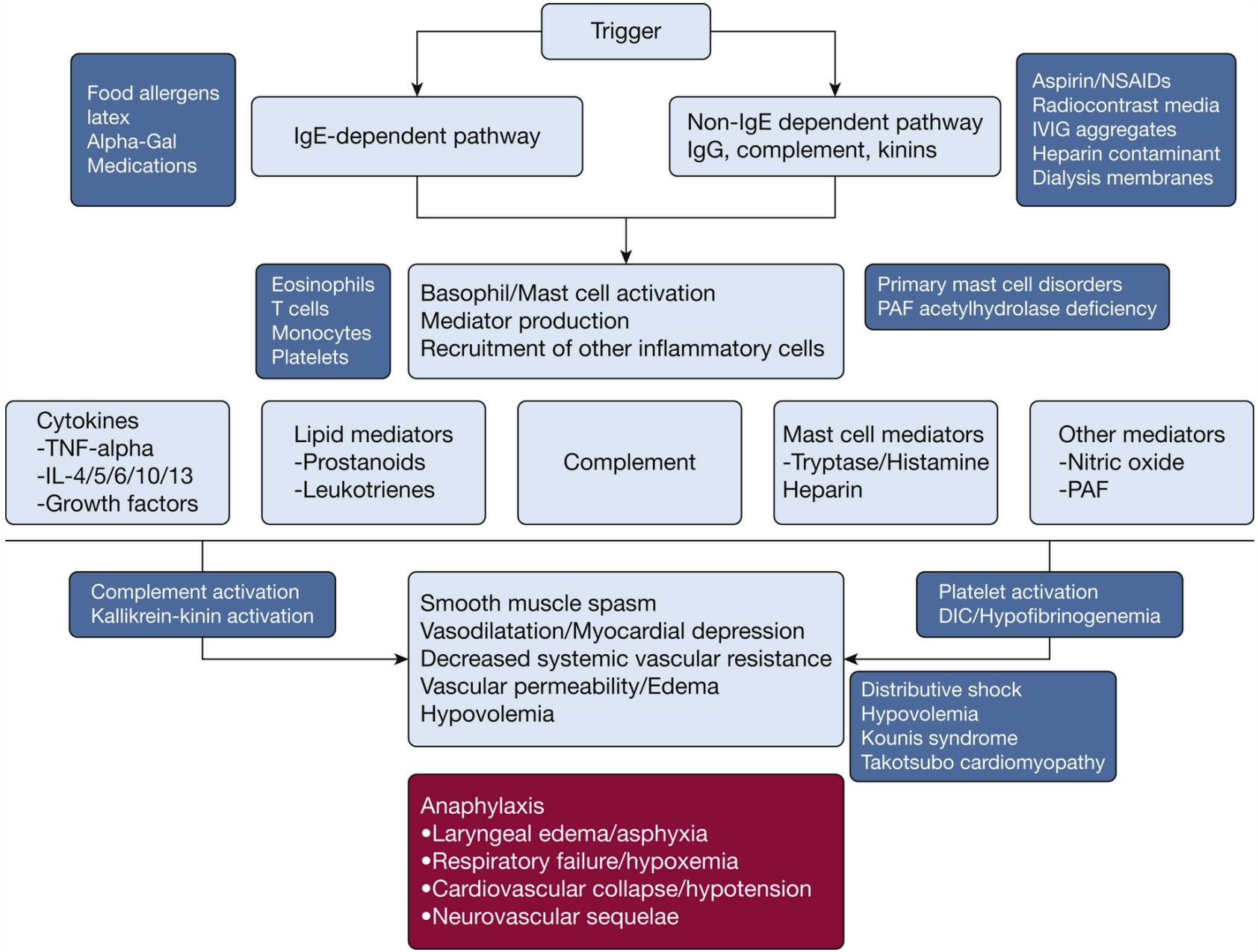
Talbot T, Roe T, Dushianthan A. Management of acute life-threatening asthma exacerbations in the intensive care unit. *Appl Sci*. 2024;14(2):693. doi:10.3390/app14020693

Relative and Immediate Indications for Mechanical Ventilation

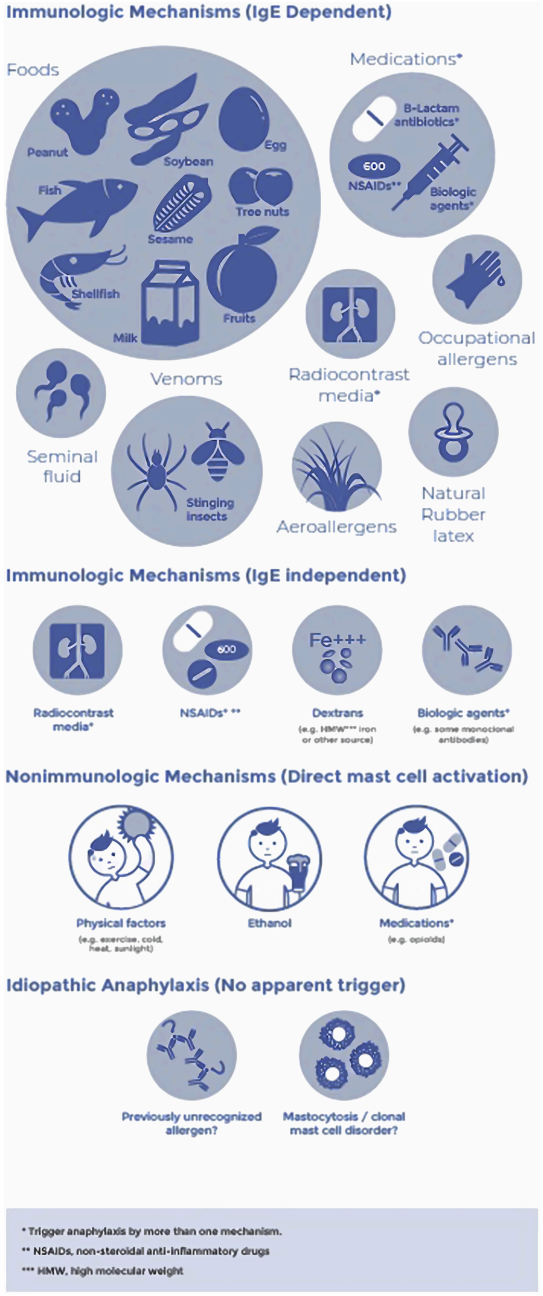
Relative Indications	Immediate Indications
Progressive exhaustion	Cardiac arrest
Increasing use of accessory muscles or change in rate/depth of respiration	PaO2 < 60 mmHg and/or PaCO2 > 45 mmHg
Change in posture or speech	Severe obtundation or coma
Failure to reverse severe respiratory acidosis despite intensive therapy	Impending respiratory failure with gasping or inability to speak
Altered sensorium	Respiratory arrest
Severe hypoxaemia with maximal oxygen delivery	Silent chest

Talbot T, Roe T, Dushianthan A. Management of acute life-threatening asthma exacerbations in the intensive care unit. *Appl Sci*. 2024;14(2):693. doi:10.3390/app14020693

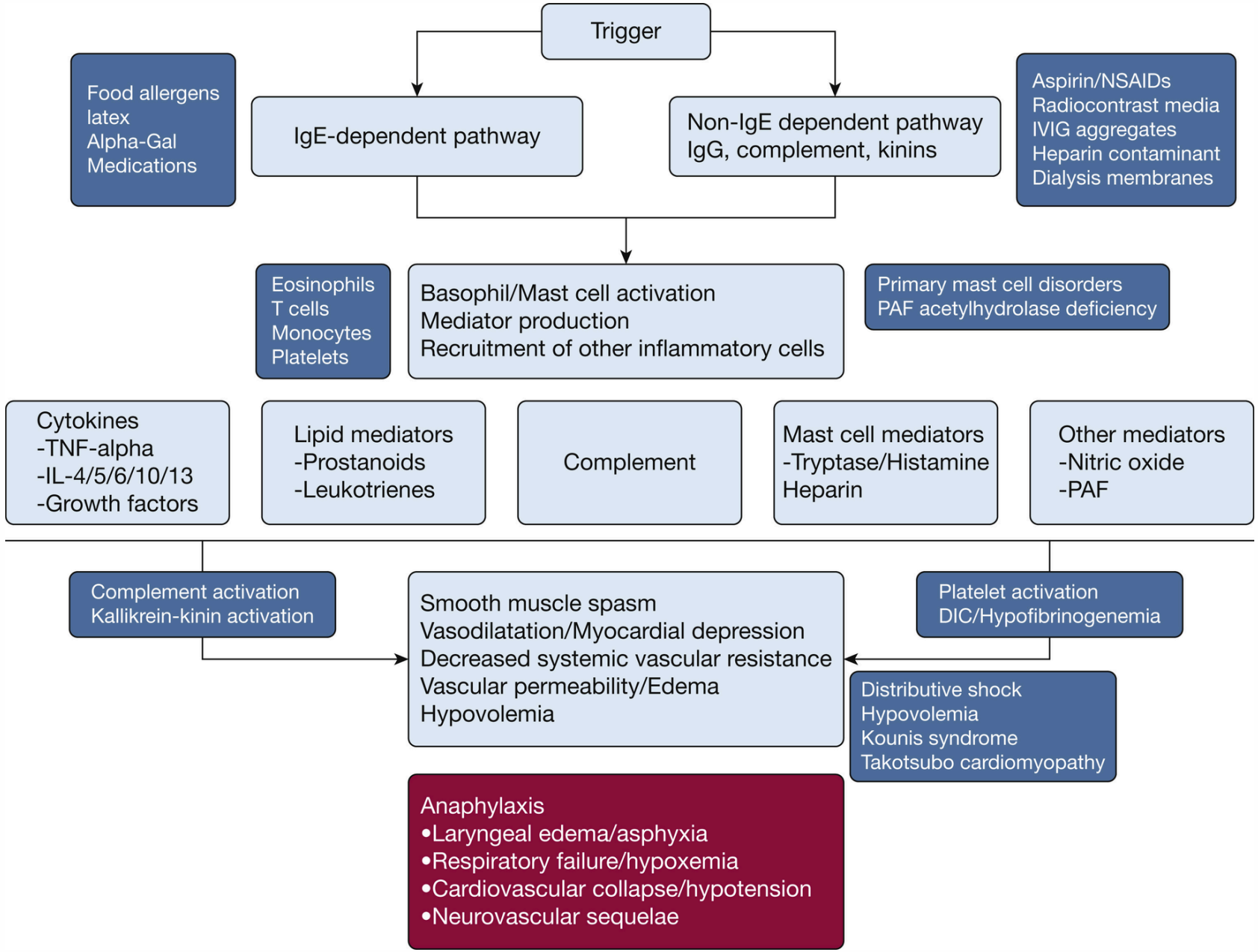
salvatore.sardo@unica.it



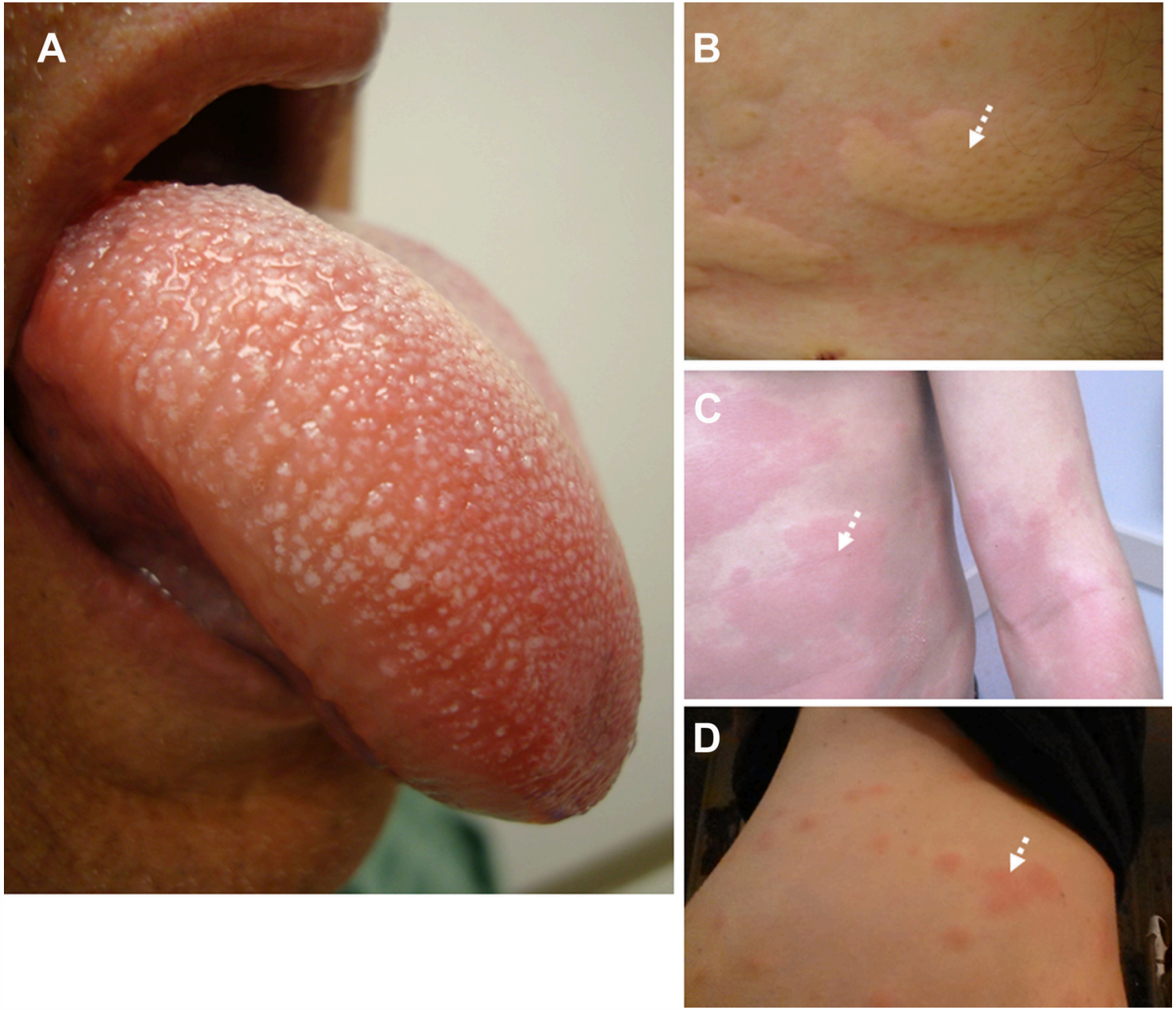
LoVerde D, Iweala OI, Eginli A, Krishnaswamy G. Anaphylaxis. *Chest*. 2018;153(2):528-543. doi:10.1016/j.chest.2017.07.033



Cardona V, Ansotegui IJ, Ebisawa M, et al. World allergy organization anaphylaxis guidance 2020. *World Allergy Organ J*. 2020;13(10):100472. doi:10.1016/j.waojou.2020.100472



LoVerde D, Iweala OI, Eginli A, Krishnaswamy G. Anaphylaxis. *Chest*. 2018;153(2):528-543. doi:10.1016/j.chest.2017.07.033



LoVerde D, Iweala OI, Eginli A, Krishnaswamy G. Anaphylaxis. *Chest*. 2018;153(2):528-543. doi:10.1016/j.chest.2017.07.033

Anaphylaxis is highly likely when any one of the following **two criteria is fulfilled**

1. Acute onset of an illness (minutes to several hours) with involvement of the skin, mucosal tissue, or both (e.g. generalized hives, pruritus or flushing, swollen lips-tongue-uvula)

and at least one of the following

A. Airway/Breathing: Respiratory compromise.
(e.g. dyspnea, wheeze-bronchospasm, stridor, reduced PEF, hypoxemia)

B. Circulation: Reduced BP or associated symptoms of end-organ dysfunction.
(e.g. hypotonia [collapse], syncope, incontinence)

C. Other: Severe gastrointestinal symptoms.
(e.g. severe crampy abdominal pain, repetitive vomiting), especially after exposure to non-food allergens)

2. Acute onset of **hypotension*** or **bronchospasm** or **laryngeal involvement†** after exposure to a known or highly probable allergen for that patient (minutes to several hours), **even in the absence of typical skin involvement.**

Infants and children: low systolic BP (age-specific) or greater then 30% decrease in systolic BP*

Adults: systolic BP of less than 90 mm Hg or greater than 30% drecrease from that person's baseline

or

Bronchospasm

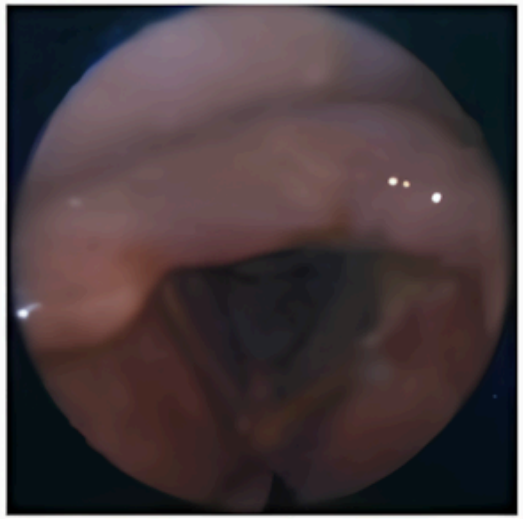
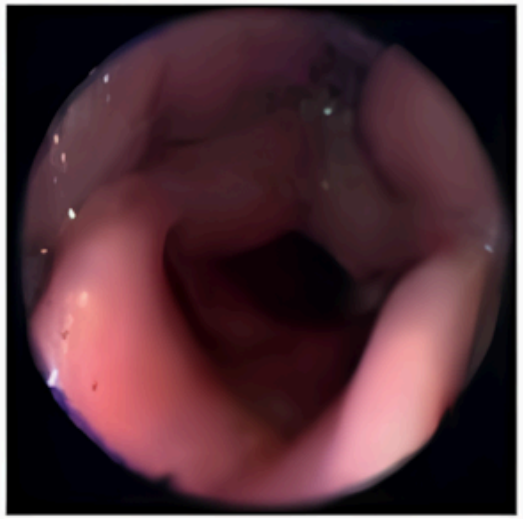
Laryngeal involvement

PEF, Peak expiratory flow; BP blood presure.

*Hypotension defined as a decrease in systolic BP greater than 30% from that person's baseline, OR
i. Infants and children under 10 years: systolic BP less than (70mmHg + [2 x age in years])
ii. Adults: systolic BP les than < 90 mmHg

† Laryngeal symptoms include: stridor, vocal changes, odynophagia.

Cardona V, Ansotegui IJ, Ebisawa M, et al. World allergy organization anaphylaxis guidance 2020. *World Allergy Organ J*. 2020;13(10):100472. doi:10.1016/j.waojou.2020.100472

			
25% obstruction of the supra-larynx - Few symptoms	50% obstruction of the supra-larynx - Moderate symptoms	75% obstruction of the supra-larynx - Severe symptoms	90% obstruction of the supra-larynx - Acute threat to life
LOC-1	LOC-2	LOC-3	LOC-4
- Monitoring	- Monitoring	- Tracheostomy if needed	- Securing of the airways - Nasal fiberoptic intubation - Tracheostomy under local anesthesia

Bas M. Evidence and evidence gaps of medical treatment of non-tumorous diseases of the head and neck. *GMS Curr Top Otorhinolaryngol - Head Neck Surg*. 2016;15:Doc02. doi:10.3205/cto000129

Trauma

Biomechanics of Trauma

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.

$$p = mv$$

- ## Newtoniam physics explains trauma

  Newtonian physics directly explains how forces, motion, and energy govern mechanical events and traumatic injuries.

- ## Newton's first law - inertia

  A body remains at rest or moves at constant velocity in a straight line unless acted on by an external force. This explains continued motion of the body when a vehicle suddenly stops.

- ## Momentum

  Momentum describes the state of motion of an object and depends on mass and velocity. In a motorcycle crash, the motorcycle stops abruptly but the rider continues moving until a force acts on the body.

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Biomechanics of Trauma

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.

$$E_k = \frac{1}{2}mv^2$$

$$F = ma$$

- **Kinetic energy**

  The energy of a moving object depends on its mass and the square of its velocity. Higher velocity results in a disproportionate increase in injury severity.

- **Newton's second law**

  Acceleration results from a net force and is inversely related to mass. Rapid deceleration over a short time produces large forces and tissue injury.

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Biomechanics of Trauma

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- ## Newton's second law

  Acceleration results from a net force and is inversely related to mass. Rapid deceleration over a short time produces large forces and tissue injury.

- ## Newton's third law

  For every action, there is an equal and opposite reaction. In collisions, interacting bodies exert equal and opposite forces on each other.

- ## Energy transfer and work

  Traumatic impacts are usually inelastic. Momentum is conserved, but kinetic energy is converted into work that causes deformation of tissues and structures, producing injury.

- ## Key injury principles

  Injury severity increases with mass and especially with velocity. Energy cannot be destroyed but is transferred or transformed into tissue damage. All injuries result from tensile, compressive, shear, or overpressure strain.

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Biomechanics of Trauma

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.

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$$\mathbf{F}_{12} = -\mathbf{F}_{21}$$

$$m_1\mathbf{v}_1 + m_2\mathbf{v}_2 = m_1\mathbf{v}'_1 + m_2\mathbf{v}'_2$$

$$W = \Delta E_k$$

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Stress, strain, and biomechanics

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.

- ## Stress and strain

  Stress is the force applied per unit area and produces deformation of a material.
  Strain describes the relative deformation caused by stress and reflects how much a structure changes shape.

$$\sigma = \frac{F}{A}$$

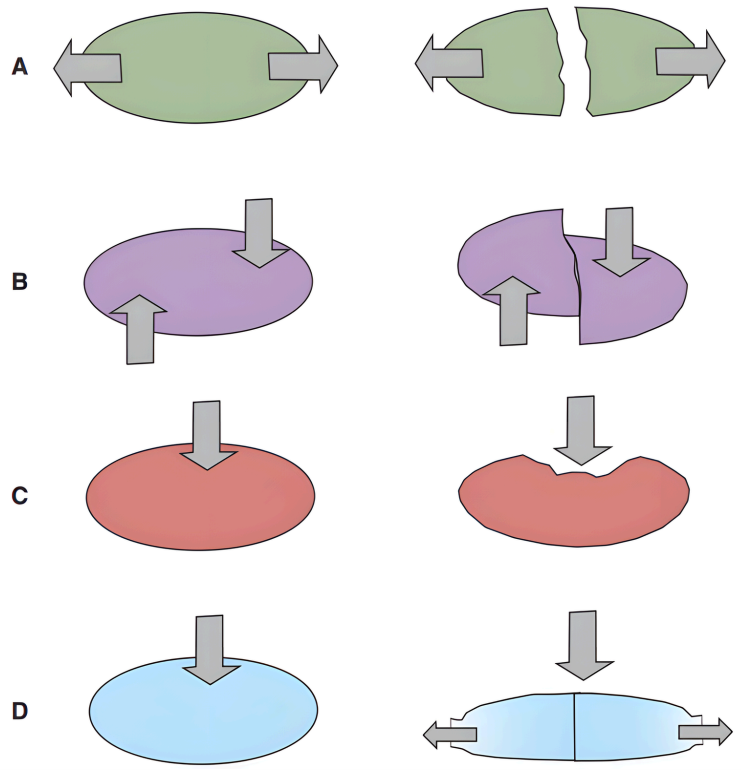
$$\varepsilon = \frac{\Delta L}{L_0}$$

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Stress, strain, and biomechanics

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.

- **Tensile strain.**
  Opposing forces act on the same structure and pull it apart, leading to rupture.
- **Shear strain.**
  Opposing forces act at different points, or different parts of a structure move at different velocities, causing tearing.
- **Compressive strain.**
  Direct impact causes crushing deformation and structural failure.
- **Overpressure.**
  Compression of gas or fluid filled structures leads to increased internal pressure and rupture.



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Stress, strain, and biomechanics

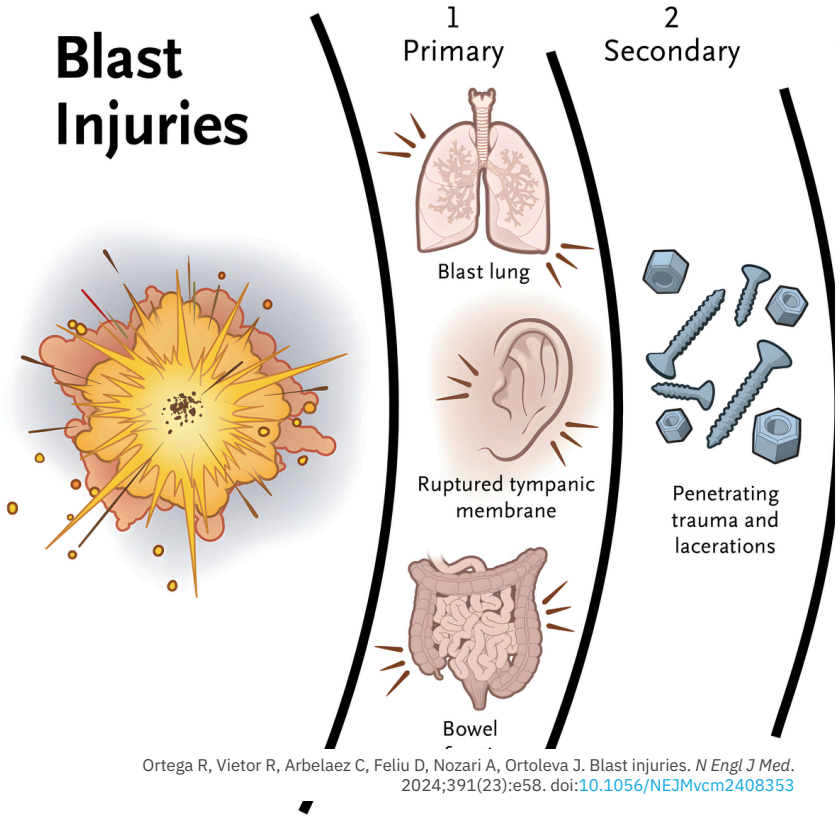
Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.

- **Tissue specific response to injury.**

  Energy transfer depends on tissue density and elasticity.
  Low density, elastic tissues such as lung dissipate energy more easily.
  High density tissues such as liver, spleen, and bone absorb energy and sustain greater structural damage.

- **Air filled organs.**

  Air filled organs are particularly vulnerable to blast injuries due to rapid pressure changes.



Ortega R, Vietor R, Arbelaez C, Feliu D, Nozari A, Ortoleva J. Blast injuries. *N Engl J Med*. 2024;391(23):e58. doi:10.1056/NEJMvcm2408353

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Blunt trauma

Understanding Blunt Trauma



Energy Transfer

Kinetic energy is transferred to the body, leading to injury



Direction of Force

The direction of the force applied to the body impacts injury severity



Energy Dissipation

How the energy is dispersed through tissues affects injury outcome



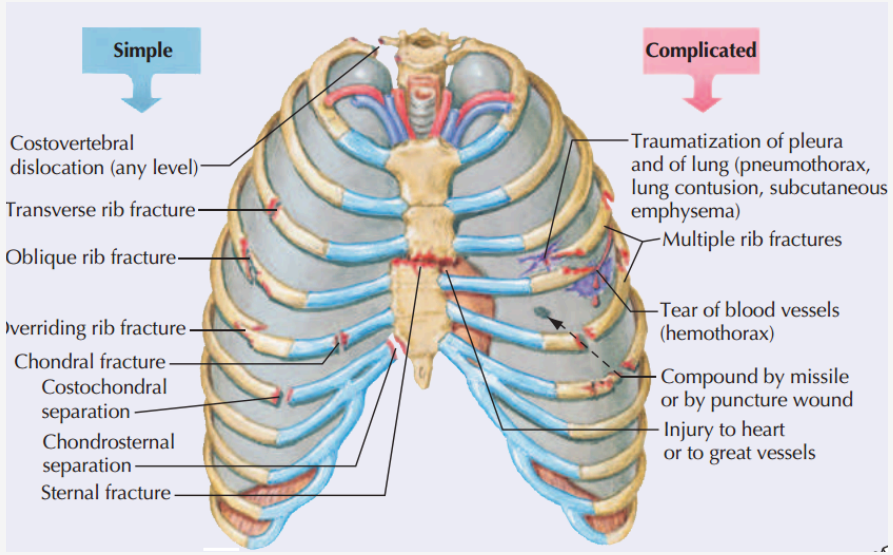
Common Mechanisms

Motor vehicle crashes, pedestrian strikes, and high falls are frequent causes of blunt trauma

Blunt trauma is a complex phenomenon, with injury severity dependent on the magnitude, direction, and dissipation of the applied forces.

Thoracic Blunt Trauma

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.



Hansen JT, Netter FH. *Netter's Clinical Anatomy*. 4th ed. Elsevier; 2019.

- ## Chest wall and ribs

  Blunt force deforms the sternum and compresses the rib cage. Rib fractures occur due to compression of the outer cortex with tensile stress on the inner cortex. Indirect fractures commonly involve lateral and posterolateral rib angles where stress concentrates.

- ## Overpressure injuries

  Chest compression increases intrapulmonary pressure beyond alveolar and pleural tolerance, leading to pneumothorax. Compression of the heart, a blood filled organ, increases intracardiac pressure and may cause blunt cardiac rupture, most often affecting the right atrium.

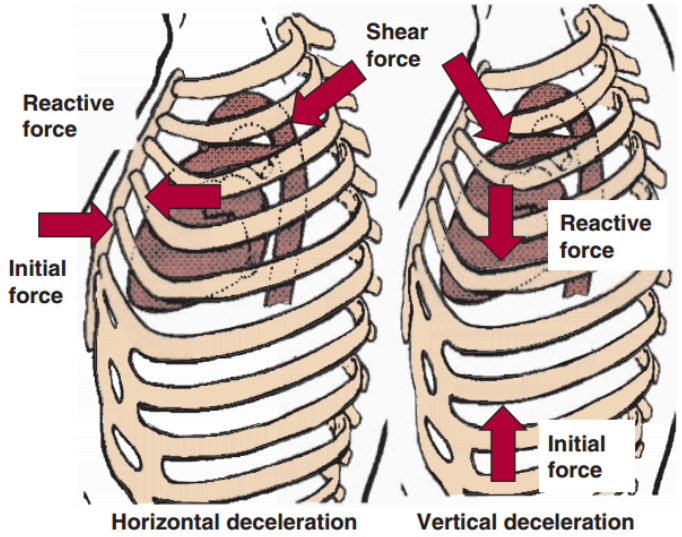


FIGURE 1-3 Various mechanisms of injury to the thoracic aorta. In a horizontal deceleration, the heart and arch move horizontally away from the descending aorta, causing shear strain and tearing at the ligamentum arteriosum. A vertical deceleration causes caudad movement of the heart, causing a strain at the root of the ascending aorta.

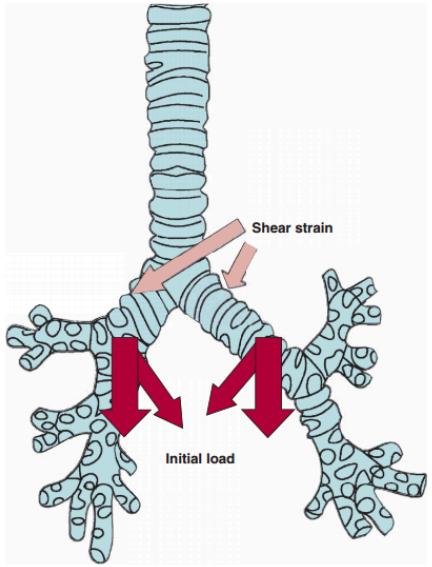


FIGURE 1-4 Mechanisms of injury for bronchial injury. The carina is tethered to the mediastinum and spinal complex, whereas the lungs are extremely mobile, setting up shear strain in the main stem bronchus upon horizontal or vertical deceleration.

Deceleration and shear injuries

Internal thoracic injuries reflect differences in mobility between fixed and mobile structures. Differential momentum generates compressive, tensile, and shear forces.

Aortic injury

In frontal or lateral impacts, the heart moves relative to the descending thoracic aorta, which is fixed to the spine. Shear stress develops at the ligamentum arteriosum. Vertical deceleration, such as falls from height, produces tensile strain at the aortic root.

Bronchial injury

Mobile lungs move relative to the tethered trachea and carina during deceleration. Shear forces occur at the main bronchi. Most blunt bronchial injuries occur within 2 cm of the carina.

In motor vehicle collisions and similar events, injury occurs through sequential impacts.

1. Vehicle collision, kinetic energy is absorbed by vehicle deformation.
2. Body collision, the occupant strikes the vehicle interior or restraint system.
3. Organ collision, internal organs continue moving and strike rigid structures or are stressed at ligamentous attachments.

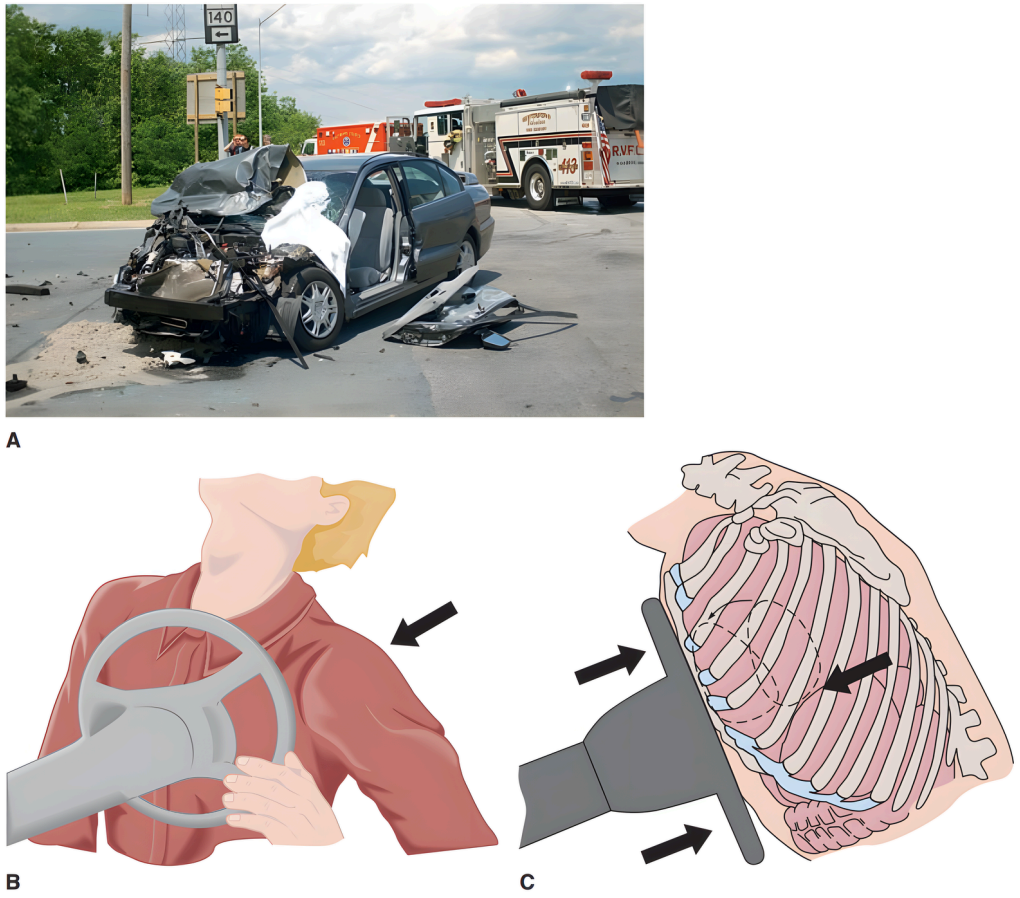


Figure 1-2 The three collisions of a motor vehicle crash. A. Vehicle collision. B. Body collision. C. Organ collision. *(Photo copyright Mark C. Ide)*

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

Frontal Impact Crash Injury Patterns

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.



- Traumatic Brain Injury
- Cervical Spine Fracture
- Facial Injuries
- Myocardial Contusion
- Pneumothorax/Haemothorax
- Aortic Disruption
- Spleen or Liver Laceration
- Posterior Hip Dislocation

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Lateral (T-Bone) Collision Injuries

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

- Contralateral Neck Sprain
- Cervical Spine Fracture
- Pneumothorax
- Lung Contusion
- Laceration of Spleen, Liver, Kidney
- Pelvic Fracture
- Extremity Injuries



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FOR HIGHWAY SAFETY
CS16009A

Rear impact Injuries

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

- Cervical spine injury



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to a mobile

Pedestrian versus car

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

- Head injury
- Abdominal visceral injuries
- Fracture of the lower extremities and pelvis



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CTS

Tractor accidents

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

- Crush injury
- Thermal injuries



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TUESDAY

Small-vehicle crashes

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

- Traumatic brain injury
- Facial fractures
- Pneumothorax/haemothorax
- Extremity and pelvic fractures
- Spinal fractures
- Degloving injuries
- Clothesline injuries with airway compromise
- Rectal and vaginal trauma



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Airbags and Injury Prevention in MVCs

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.



Air bag and three-point restraint prevents collisions 2 and 3.

- **Function**
  - Airbags inflate during frontal deceleration to protect front seat occupants. Designed to work with seat belts, not as a substitute.
- **Protection limits**
  - Effective for a single impact and deflate immediately. Do not prevent down and under movement. Tall drivers may still strike the dashboard.
- **Injury associations**
  - Small adults and infants in front facing seats face increased risk during deployment. Typical injuries include abrasions and superficial burns from airbag materials.

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Airbags and Injury Prevention in MVCs

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

- Responder safety
  - Undeployed airbags pose a hazard during rescue.
- Maintain minimum safety distances, known as the 5 to 10 to 20 rule.
  - Five inches from side curtain airbags.
  - Ten inches from steering wheel airbags.
  - Twenty inches from passenger side airbags.
- Additional features
  - Modern vehicles may include side airbags and inflatable air curtains.
- Key message
  Airbags reduce injury risk in collisions but have defined limits and require strict safety precautions during rescue operations.



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Falls from Height

1.Alson RL. International Trauma Life Support for Emergency Care Providers. Eighth edition. (Campbell JE, ed.). Pearson; 2016. 2.Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.



Mechanism and Energy Transfer

Injury results from vertical deceleration. Kinetic energy at impact depends mainly on fall height, since mass and gravity remain constant.



Surface Elasticity and Body Position

Injury severity depends on surface elasticity and body position at impact.

2

Epidemiology

Most urban falls occur from heights of 6 metres or less. Falls are the second leading cause of unintentional injury death worldwide. Highest risk groups are children and older adults.

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Fall-Related Injury Patterns and Risks

1.Alson RL. International Trauma Life Support for Emergency Care Providers. Eighth edition. (Campbell JE, ed.). Pearson; 2016. 2.Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.

- **Fracture Patterns**

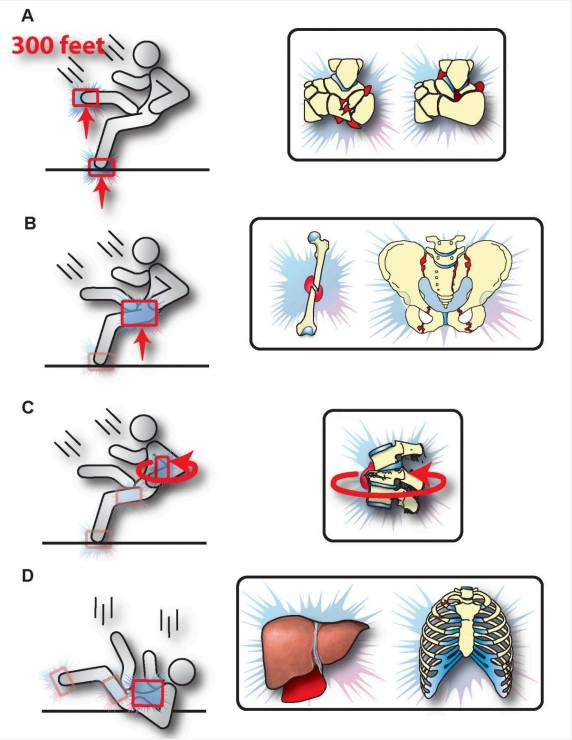
  Fractures occur in about three quarters of victims.

- **Spinal Injuries**

  Spinal column injuries occur in about one quarter, with neurological deficit in a small proportion.

- **Internal Injuries**

  Intra abdominal injuries are uncommon. Typical injuries include head trauma, spinal axial loading or flexion extension injuries, extremity fractures, and hip or pelvic injuries.



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Fall-Related Injury Patterns and Risks

1.Alson RL. International Trauma Life Support for Emergency Care Providers. Eighth edition. (Campbell JE, ed.). Pearson; 2016. 2.Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.



- ## Fall Height Risk

  Falls greater than 3 metres are associated with spinal or intracranial injury in about one in four patients. Serious intra abdominal injury occurs in about one in twenty patients. Severe injury can also occur after short distance or ground level falls.

- ## Older Adult Risks

  Older adults have increased risk due to impaired balance, vision, mobility, bone fragility, and anticoagulant use. Ground level falls in older adults may cause intracranial haemorrhage or hip fracture with high morbidity and mortality. Injury severity is often underestimated in this group.

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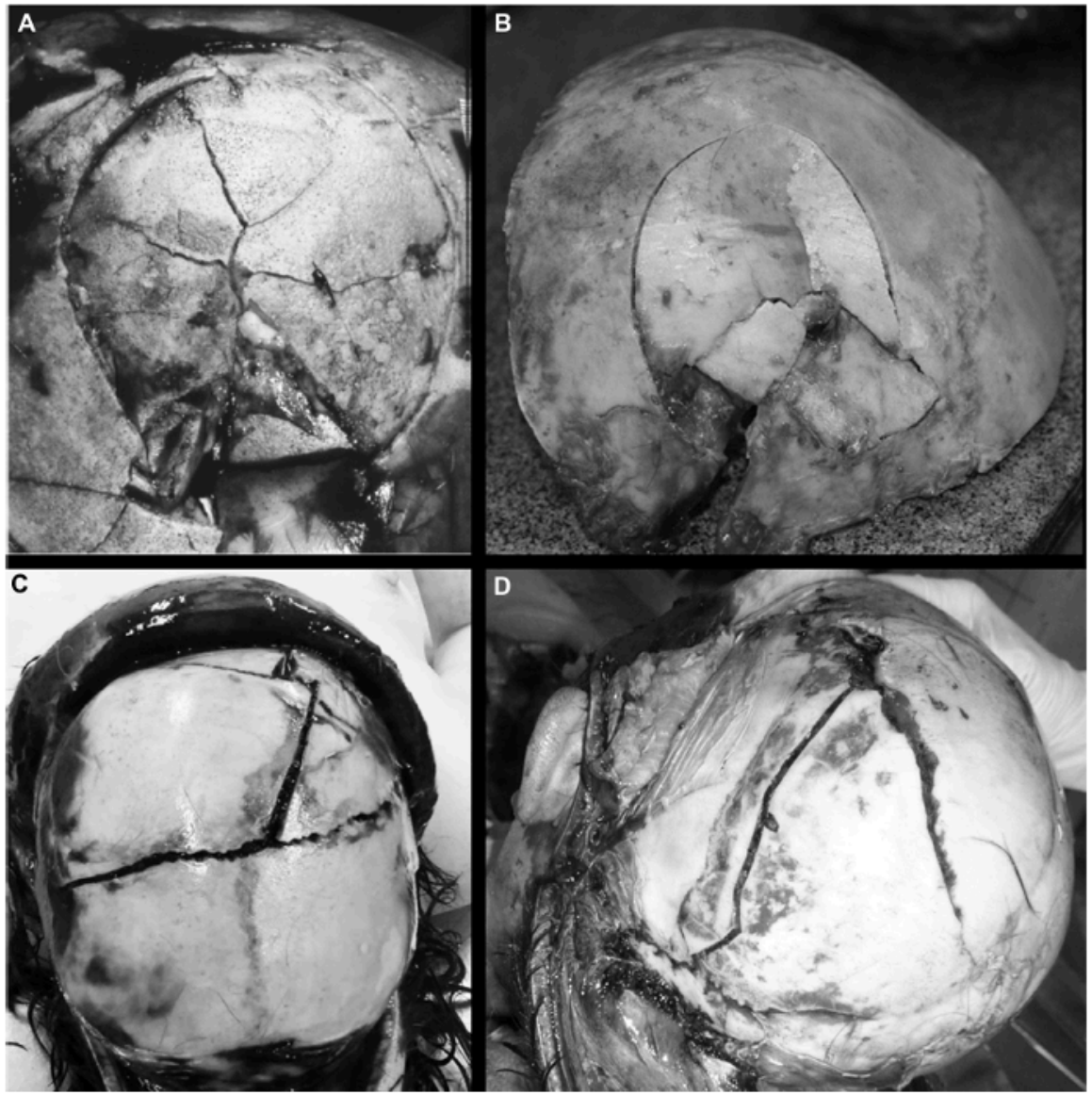


Figure 4 Examples of cranial blunt force trauma.

Notes: (**A**) Fall from height landing on the vault. Copyright © 2001 from Forensic *Pathology*. 2nd ed. by Di Maio VJM, Di Maio D.[18] Reproduced by permission of Taylor and Francis Group, LLC, a division of Informa plc. (**B**) Fall from the first floor (approximately 7 m height) landing on the edge of a step. (**C**) Fall after striking an obstacle. (**D**) Fall and compression fracture between metallic object and ground. (**B**–**D**) Source: Department of Forensic Sciences, University of Crete.

Kranioti E. Forensic investigation of cranial injuries due to blunt force trauma: current best practice. *Res Rep Forensic Med Sci*. Published online October 2015:25-38. doi:10.2147/RRFMS.S70423



Figure (4): Depressed comminuted fracture with radiating multiple fissure fractures in head first impact.



Figure (5): Massive scalp contusions associated with fissure fractures in head first impact.

Ramadan A, Soliman E, Abo El-Noor M, Shahin M. Patterns of injuries in fatal fall from height cases in gharbia governorate: autopsy study. *Egypt J Forensic Sci Appl Toxicol*. 2020;20(4):27-42. doi:10.21608/ejfsat.2020.40323.1165



Figure (6): Extensive subdural haemorrhage and brain contusions in head first impact.

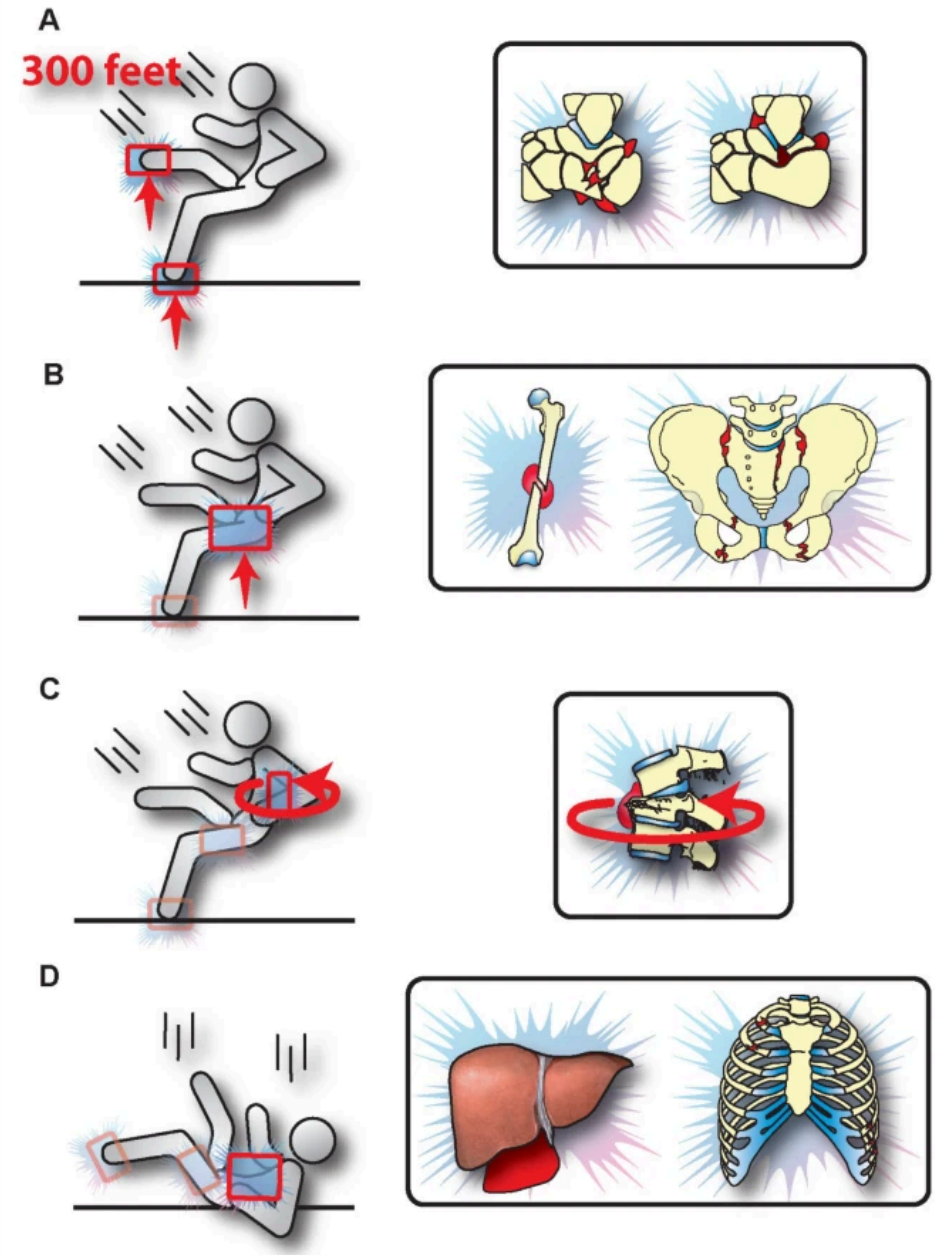


Figure (7): Fracture Tibia and Fibula in Feet first impact.

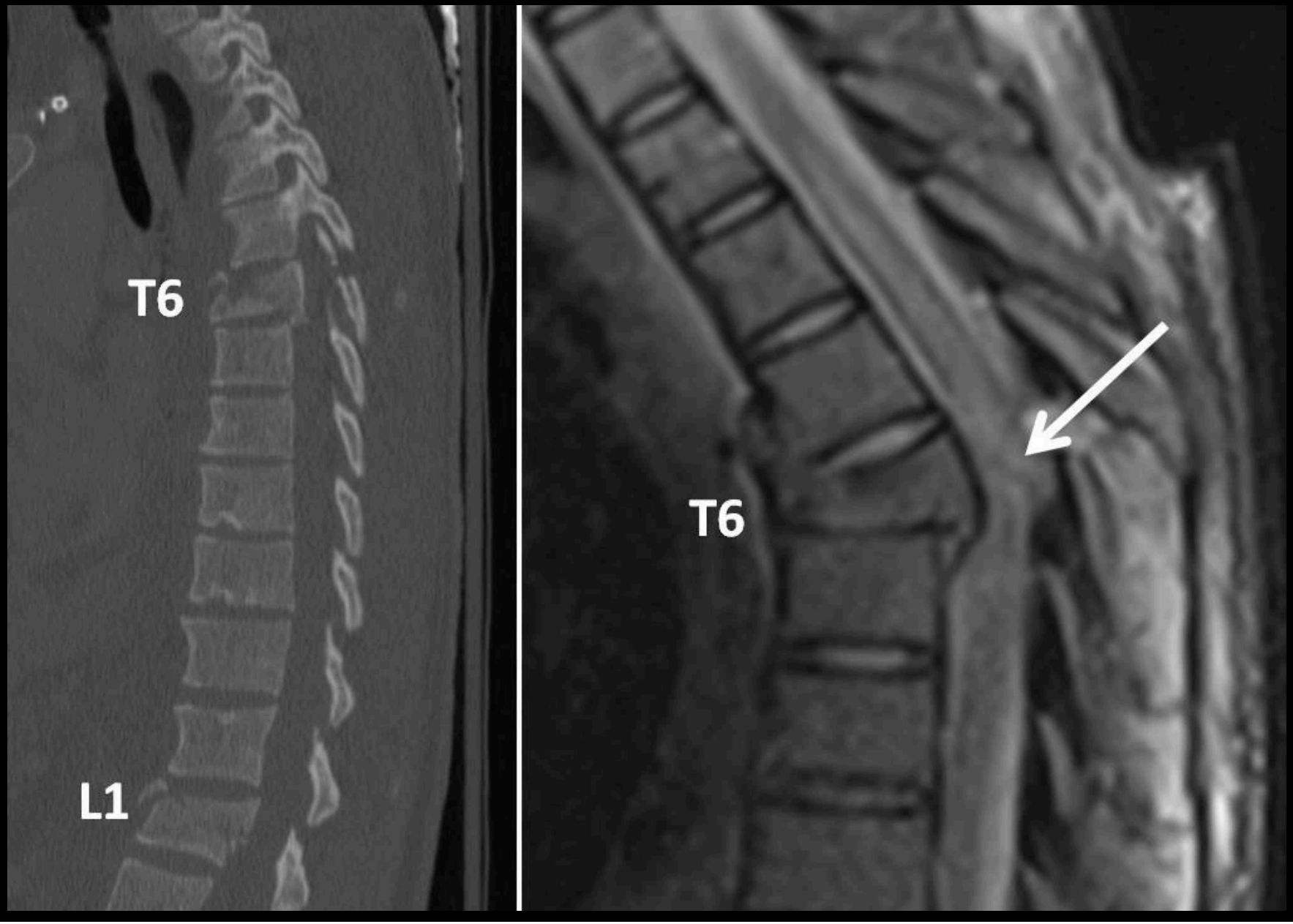


Figure (8): Lung contusions and lacerations in fatal fall from height.

Ramadan A, Soliman E, Abo El-Noor M, Shahin M. Patterns of injuries in fatal fall from height cases in gharbia governorate: autopsy study. *Egypt J Forensic Sci Appl Toxicol*. 2020;20(4):27-42. doi:10.21608/ejfsat.2020.40323.1165



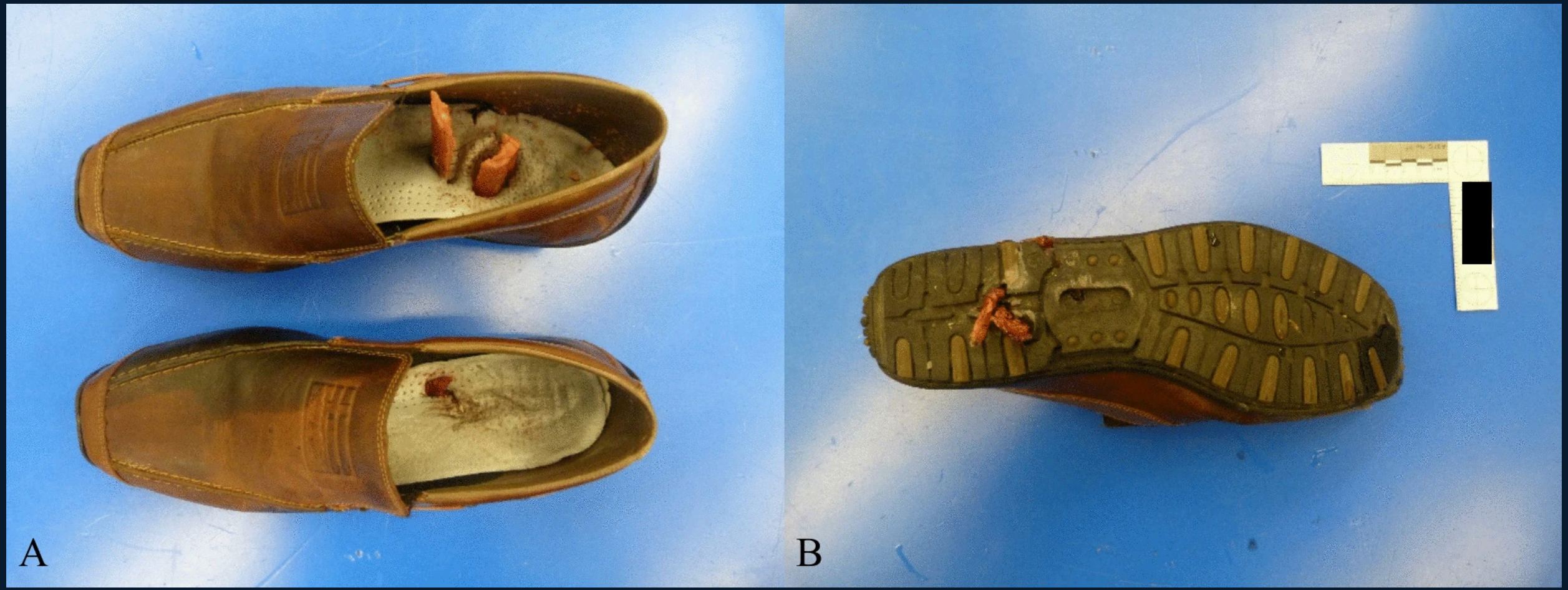
Weckbach S, Flierl MA, Blei M, Burlew CC, Moore EE, Stahel PF. Survival following a vertical free fall from 300 feet: the crucial role of body position to impact surface. *Scand J Trauma Resusc Emerg Med*. 2011;19(1):63. doi:10.1186/1757-7241-19-63



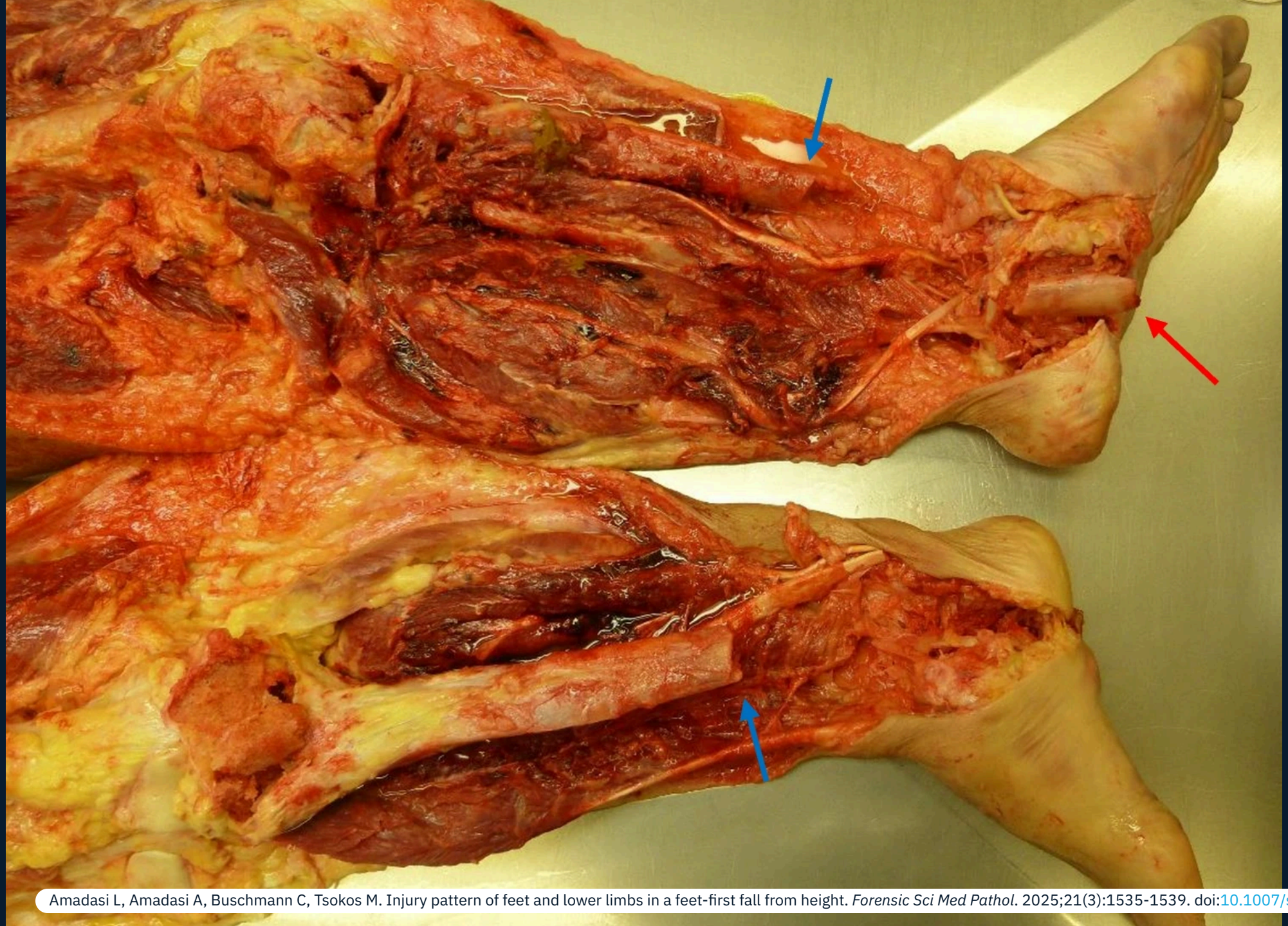
Weckbach S, Flierl MA, Blei M, Burlew CC, Moore EE, Stahel PF. Survival following a vertical free fall from 300 feet: the crucial role of body position to impact surface. *Scand J Trauma Resusc Emerg Med*. 2011;19(1):63. doi:10.1186/1757-7241-19-63



Amadasi L, Amadasi A, Buschmann C, Tsokos M. Injury pattern of feet and lower limbs in a feet-first fall from height. *Forensic Sci Med Pathol*. 2025;21(3):1535-1539. doi:10.1007/s12024-025-00939-3



Amadasi L, Amadasi A, Buschmann C, Tsokos M. Injury pattern of feet and lower limbs in a feet-first fall from height. *Forensic Sci Med Pathol*. 2025;21(3):1535-1539. doi:10.1007/s12024-025-00939-3



Amadasi L, Amadasi A, Buschmann C, Tsokos M. Injury pattern of feet and lower limbs in a feet-first fall from height. *Forensic Sci Med Pathol*. 2025;21(3):1535-1539. doi:10.1007/s12024-025-00939-3

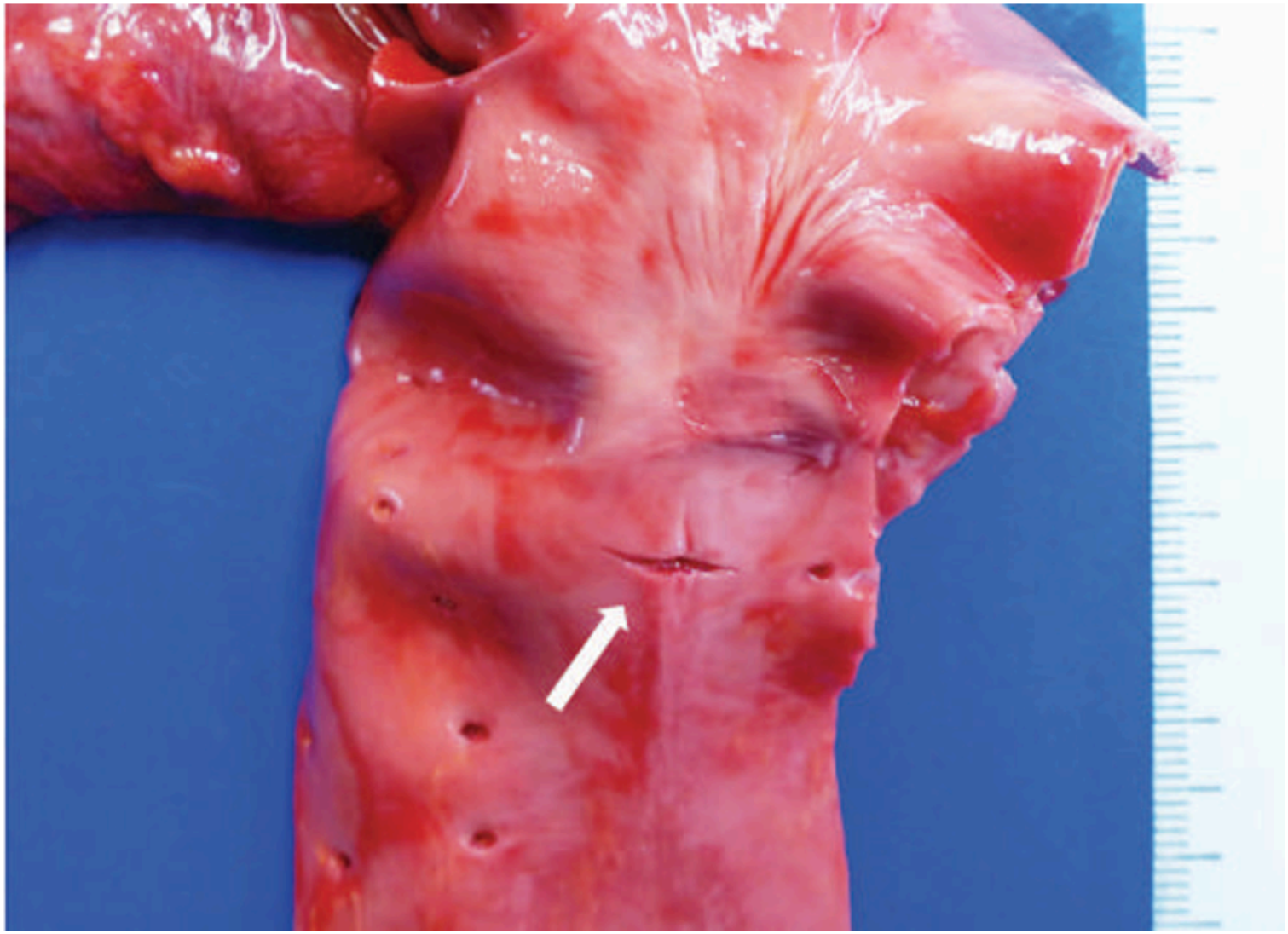


Figure 9. Small horizontal laceration of the intima (white arch) of the thoracic aorta.

Doberentz E, Geile J, Madea B. Fatal free falls from very great heights. *Rom J Legal Med*. 2019;27(4):354-360. doi:10.4323/rjlm.2019.354

Overview of Ballistics

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.



Ballistics

Studying the motion and impact of projectiles



Internal Ballistics

Analyzing projectile properties at launch



External Ballistics

Examining gravity, drag, and environmental factors



Terminal Ballistics

Investigating interaction with target and tissue injury

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Kinetic energy and tissue damage

- Projectile kinetic energy depends on mass and velocity.
- Tissue injury results from transfer of kinetic energy as projectile momentum is lost.
- Higher velocity projectiles cause greater energy transfer and tissue disruption.

Drag and ballistic coefficient

- Resistance depends on projectile shape, cross sectional area, velocity, and medium density.
- Ballistic coefficient reflects sectional density.
- High sectional density leads to lower drag and deeper penetration.
- Low sectional density leads to higher drag and greater local energy transfer.

Yaw, deformation, and fragmentation

- Yaw is deviation of the projectile tip from its flight axis.
- Increased yaw raises drag and energy release into tissue.
- Deforming or expanding bullets increase tissue damage.
- Fragmentation creates multiple injury tracks and amplifies tissue destruction.

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.

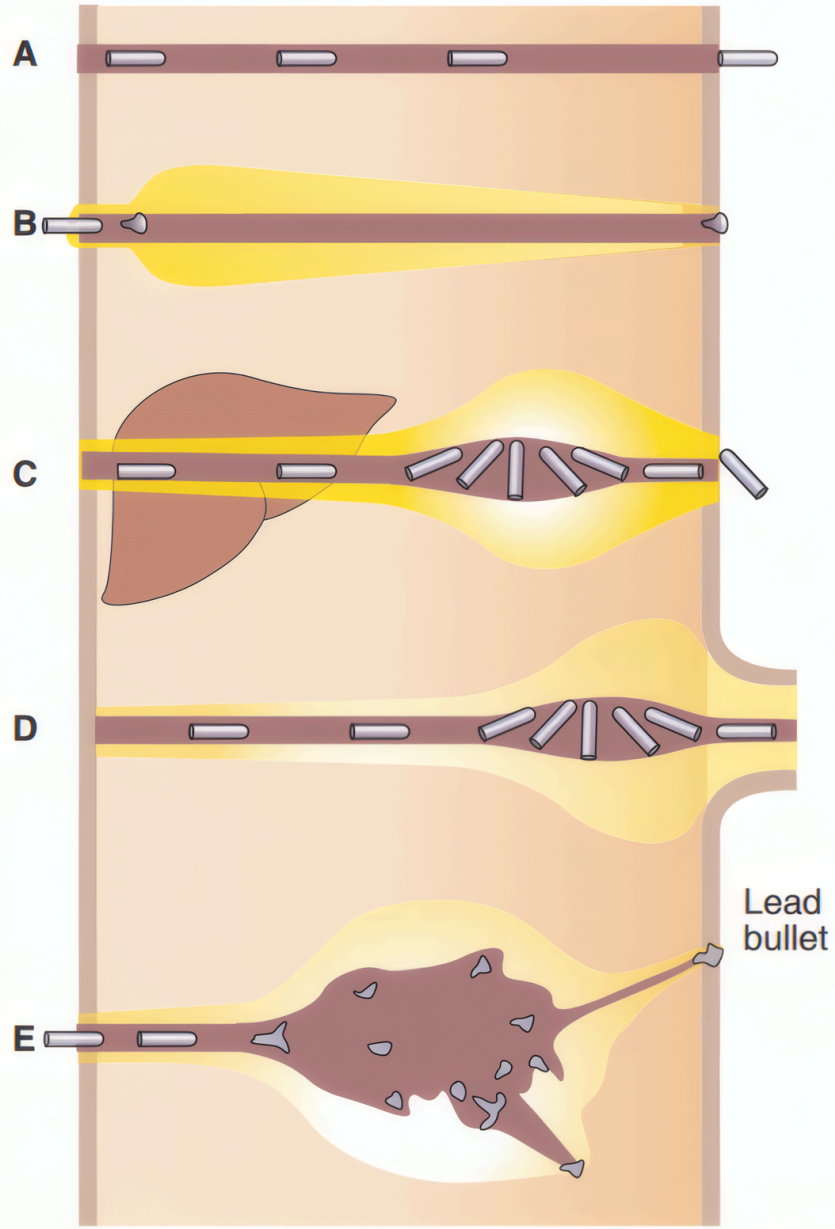


FIGURE 1-6 Wound profile of (**A**) nonfragmenting bullet through tissue with minimal yaw, (**B**) mushrooming bullet, (**C**) bullet tumbling through solid organ, (**D**) bullet tumbling with large exit skin flaps due to hydrostatic pressure wave, and (**E**) bullet fragmenting.

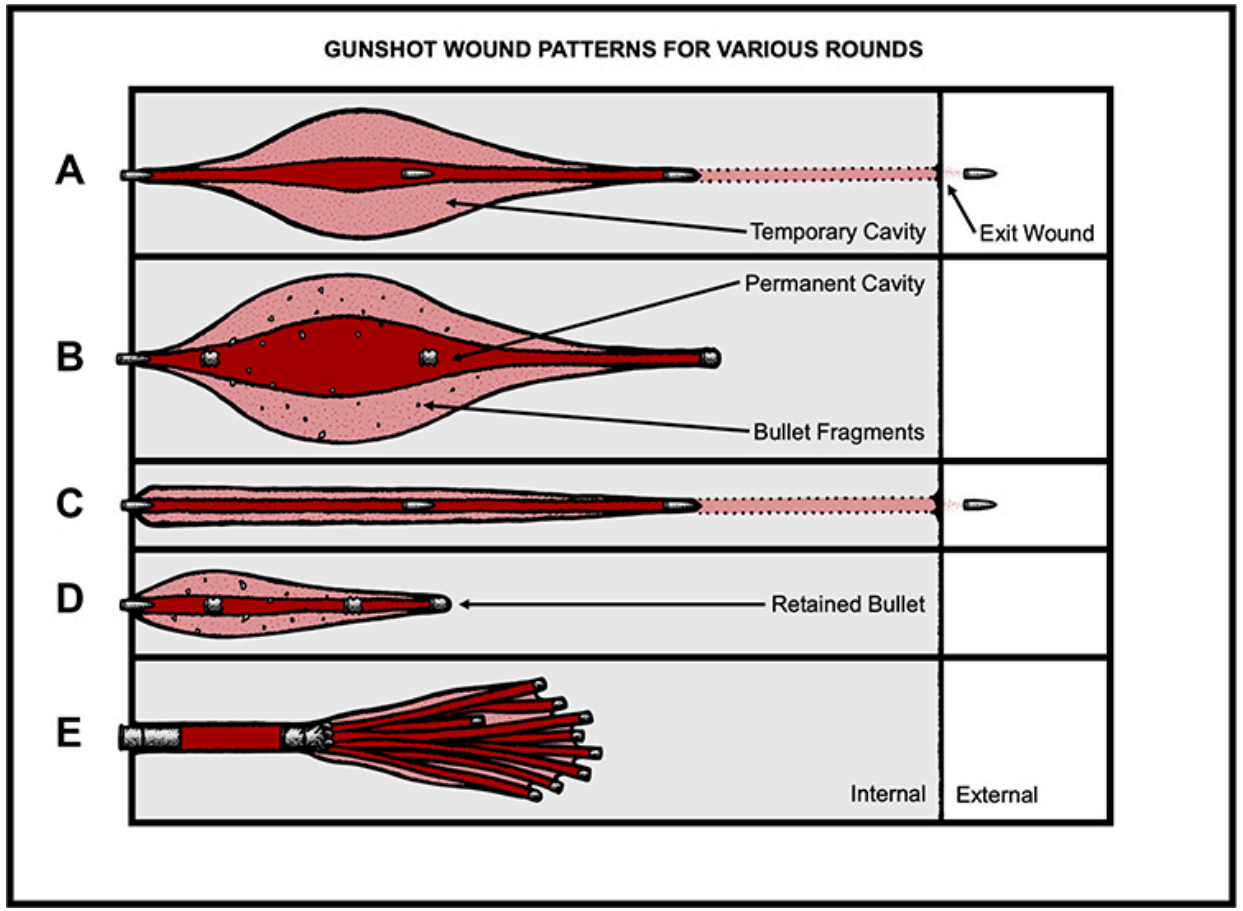
Cavitation

- Permanent cavity results from direct tissue crushing along the track.
- Temporary cavity results from pressure waves that stretch surrounding tissues.
- Damage severity depends on tissue elasticity and resistance.

Tissue specific effects

- Low density tissues such as lung and fat offer limited resistance.
- Dense and inelastic tissues such as liver and kidney sustain greater damage.

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.



Baum GR, Baum JT, Hayward D, MacKay BJ. Gunshot wounds: ballistics, pathology, and treatment recommendations, with a focus on retained bullets. *Orthop Res Rev*. 2022;14:293-317. doi:10.2147/ORR.S378278

Blast Injury Classification

Feliciano DV, Mattox KL, Moore EE. Trauma, Ninth Edition. McGraw Hill / Medical; 2021.

Primary Blast Injury

Injuries caused by the shock wave of the explosion itself, such as organ damage and internal bleeding.

Secondary Blast Injury

Injuries caused by flying debris and fragments, such as lacerations, penetrating wounds, and blunt trauma.

Tertiary Blast Injury

Injuries caused by the displacement of the body and impact with objects, such as fractures, traumatic amputations, and brain injuries.

Quaternary Blast Injury

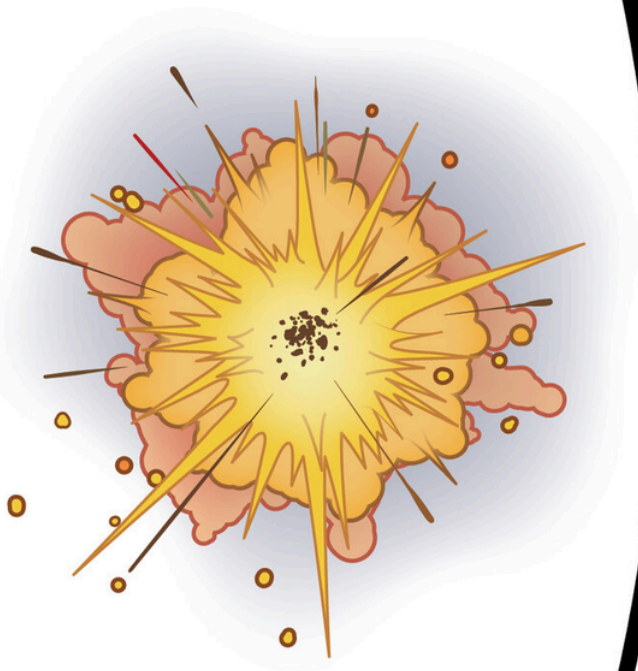
Injuries caused by burns, inhalation of toxic gases, and radiation exposure related to the explosive device.

Quinary Blast Injury

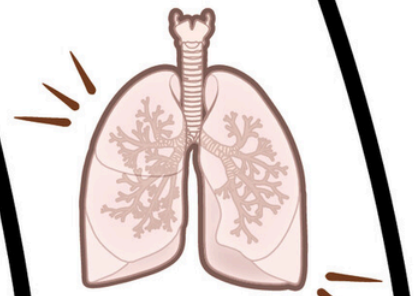
Additional systemic or toxic effects related to the explosive, such as chemical exposure or environmental contamination.

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Blast Injuries



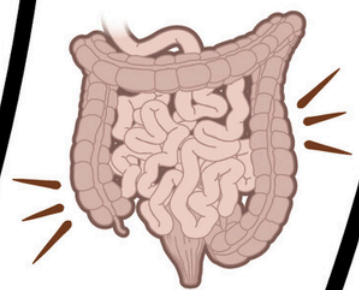
1 Primary



Blast lung

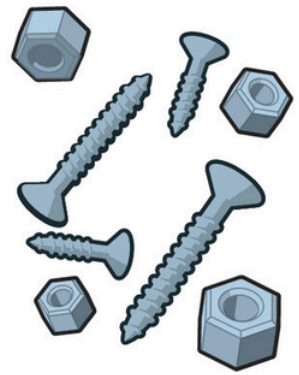


Ruptured tympanic membrane



Bowel perforations

2 Secondary



Penetrating trauma and lacerations

3 Tertiary



Blunt trauma and fractures

4 Quaternary



Burns, asphyxiation, and toxic substances

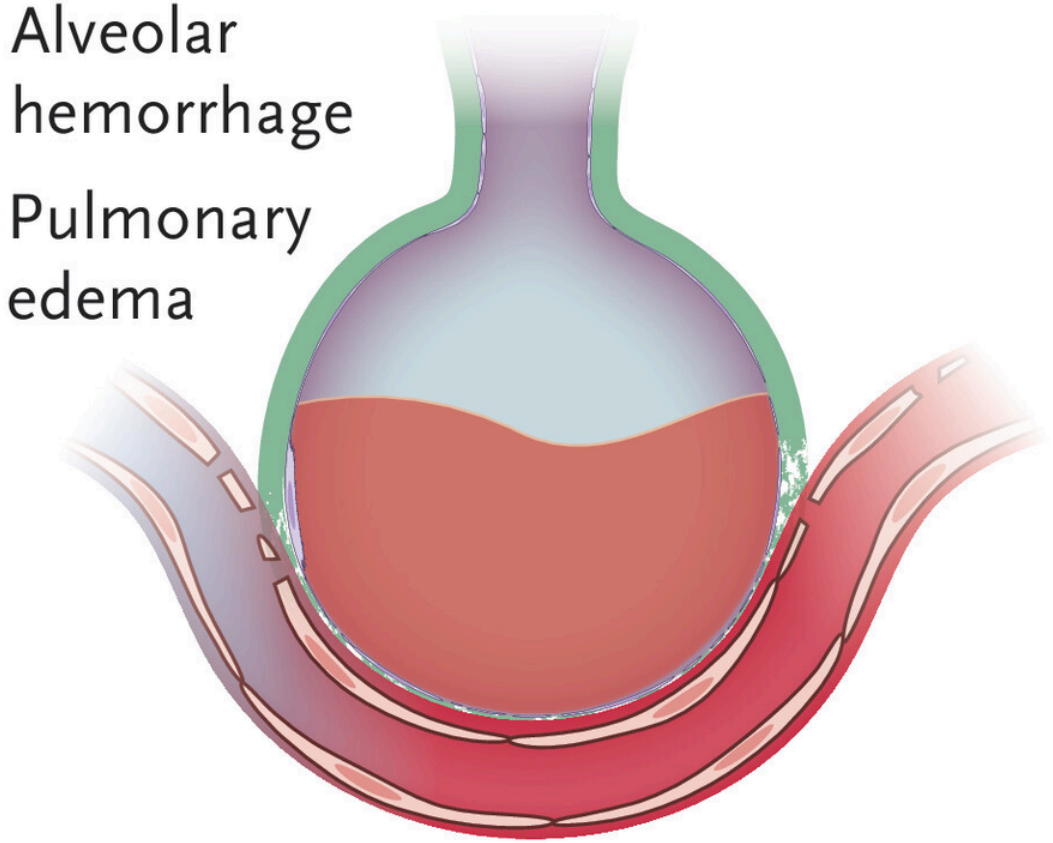
5 Quinary



Chemical burns, radiation, and infection

Ortega R, Vietor R, Arbelaez C, Feliu D, Nozari A, Ortoleva J. Blast injuries. *N Engl J Med*. 2024;391(23):e58. doi:10.1056/NEJMvcm2408353

Blast Lung Alveolar Disruption



Most lethal form of primary blast injury and leading cause of death from this mechanism.

High vulnerability due to large air tissue interface in the lungs.

- Blast wave disrupts alveoli and capillary walls.
- Blood and fluid are forced into alveoli, causing alveolar haemorrhage and pulmonary oedema.
- Key clinical features are dyspnoea, cough, and hypoxia.
- Air entry into pulmonary circulation can cause arterial air embolism, stroke, or myocardial infarction.
- Associated injuries include pneumothorax and haemothorax with impaired gas exchange.
- Bronchovascular disruption may cause bronchoalveolar fistulae and systemic air embolisation.

Ortega R, Vietor R, Arbelaez C, Feliu D, Nozari A, Ortoleva J. Blast injuries. *N Engl J Med*. 2024;391(23):e58. doi:10.1056/NEJMvcm2408353

Fundamentals concepts of Pre-hospital Trauma Management

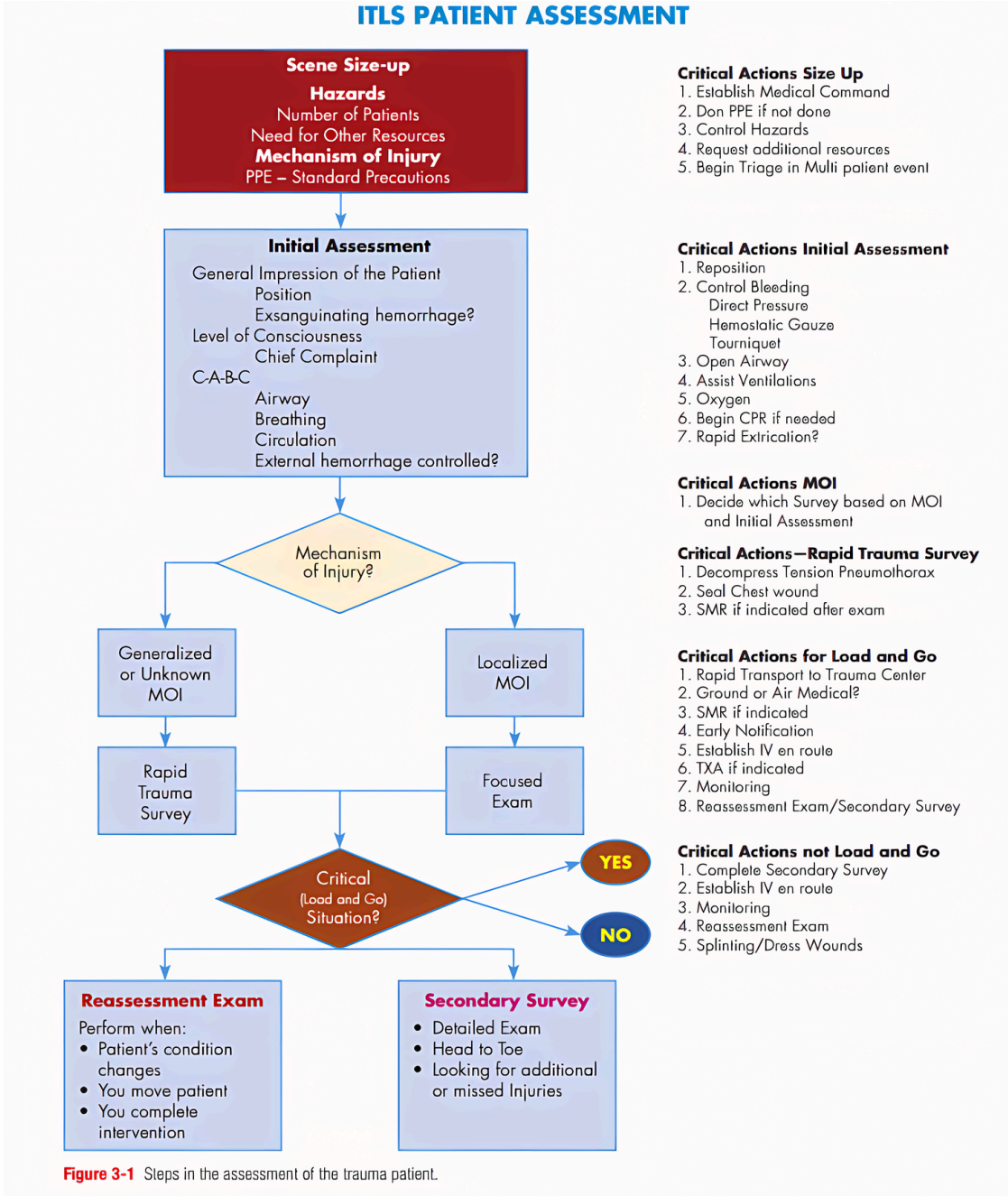


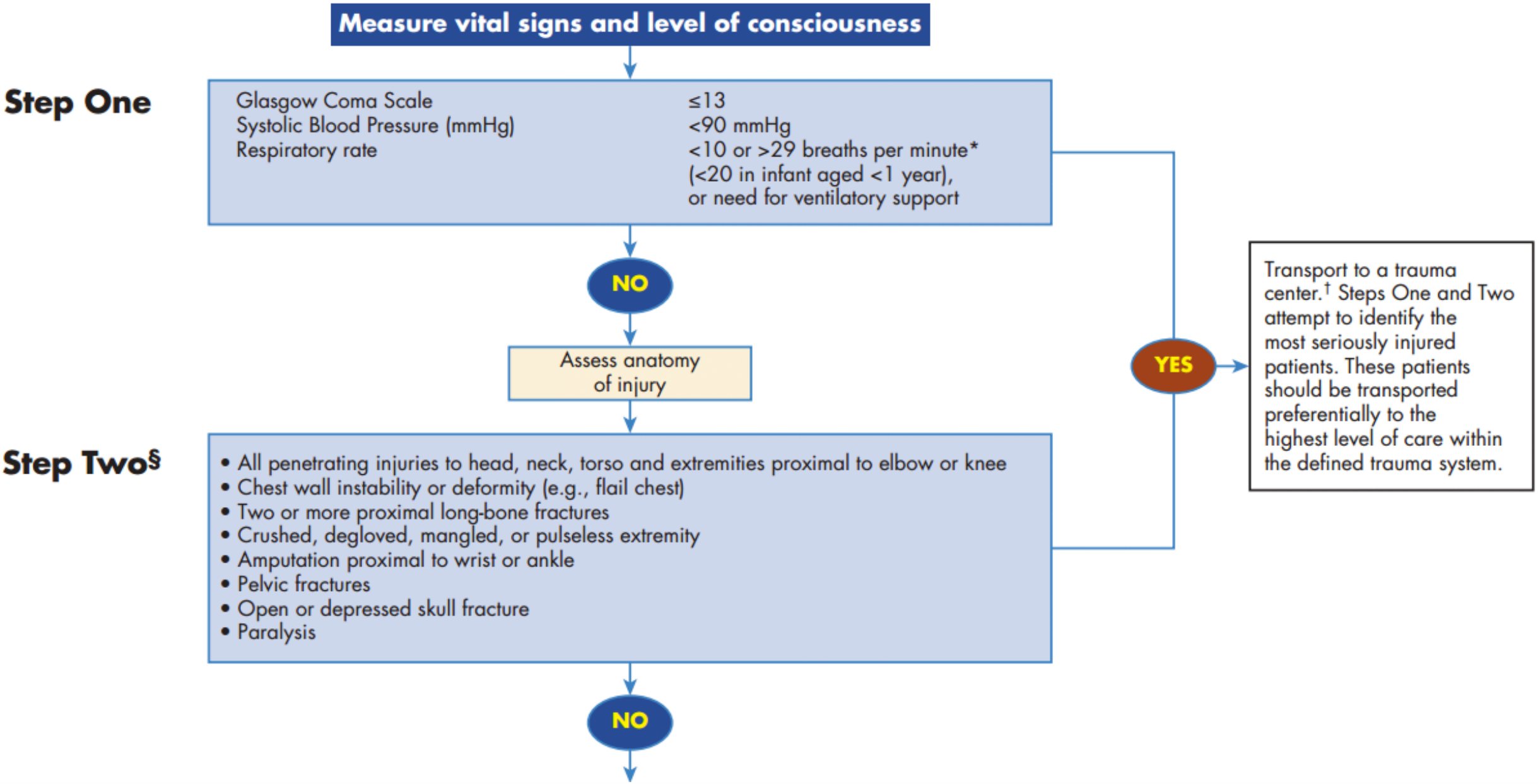
Figure 3-1 Steps in the assessment of the trauma patient.

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

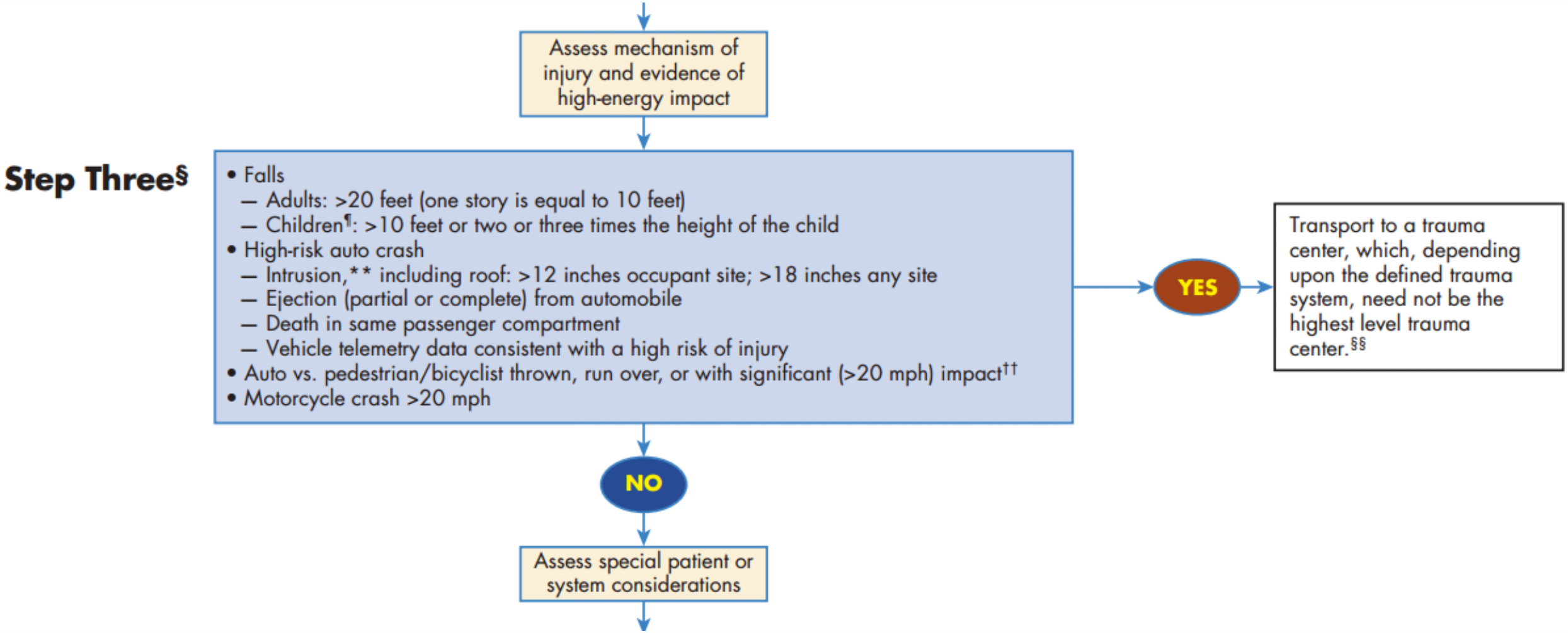
Table 1-4: Priorities of Care

XABC	Explanation/Adaptation to Civilian EMS	MARCHE (TCCC)
E**X**sanguinating hemorrhage	• Identify and treat hemorrhage; treatments including tourniquets, pressure dressings.	**M**assive hemorrhage control
Airway	• Open the airway and ensure adequate ventilation and oxygenation; importantly, this does not necessarily mean invasive airway management and BLS airway maneuvers such as jaw thrust, airway adjuncts (OPA/NPA), and/or BVM are often sufficient.	**A**irway control
Breathing	• Treat tension pneumothorax with needle decompression. • Beware of overventilation, which can decrease cardiac venous return and be harmful in traumatic brain injury.	**R**espiratory support
Circulation	• Assess and treat shock, manifested by hypotension, tachycardia, and/or low capnography. • With the exception of traumatic brain injury, hypotensive resuscitation is favored to reduce further hemorrhage and minimize crystalloid infusion. • Consider TXA and blood product transfusion, if available.	**C**irculation and shock management
	• Minimize hypothermia in order to prevent coagulopathy and increased hemorrhage. • Maintain CNS perfusion and oxygenation.	**H**ypothermia **H**ead injury
	• Environment (hyperthermia, hypothermia) • Eye Injury • Preparation for transport • Pain Control • Antibiotics if indicated	**E**verything else

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

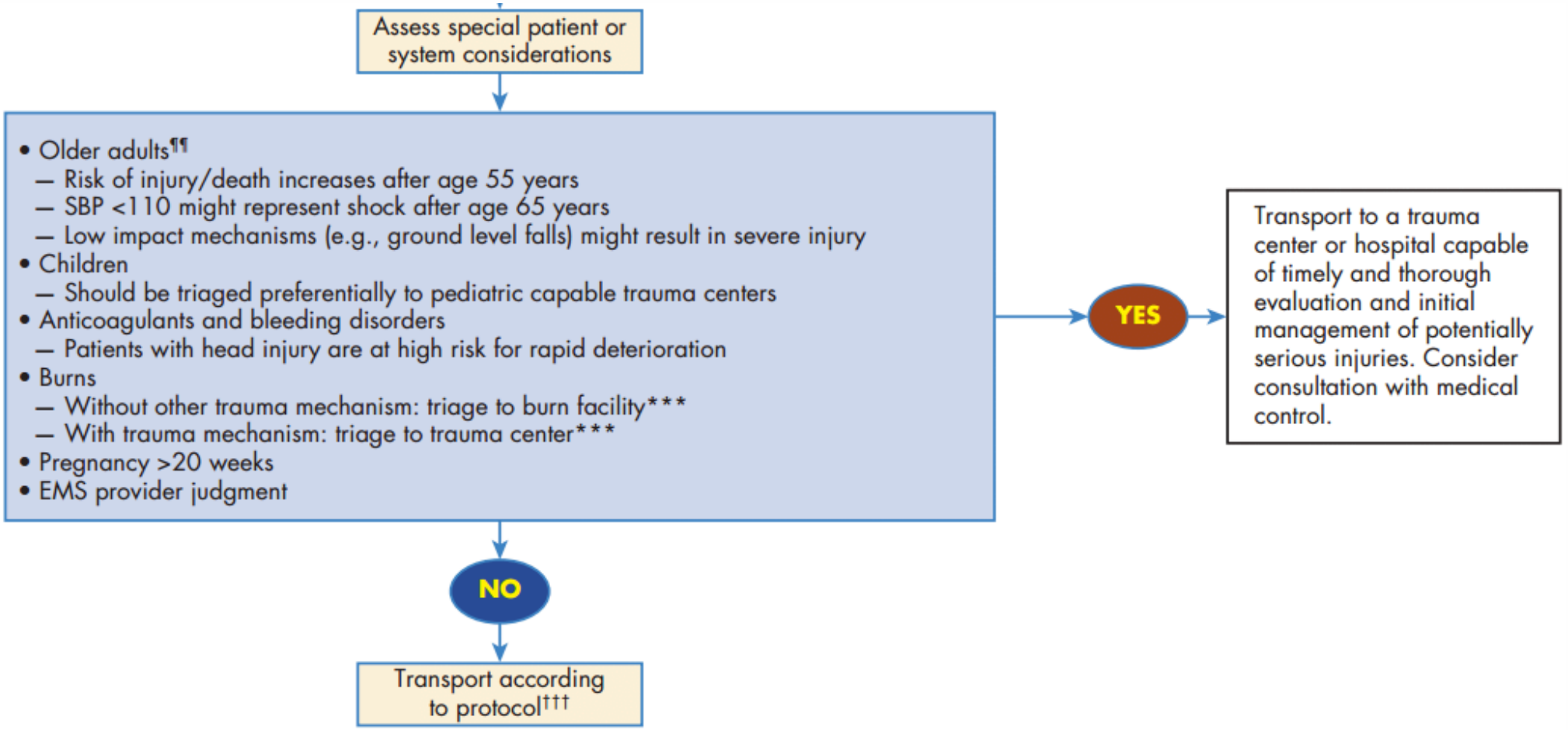


Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.



Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

Step Four



When in doubt, transport to a trauma center

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

Scottish Ambulance Service
Taking Care to the Patient

Adult Trauma Triage Tool ≥16

SCOTTISH TRAUMA NETWORK

Use this tool to Triage all Significantly Injured Patients
or Patients involved in a High Mechanism Incident

Clinical Judgement is important and valued.

If you are concerned that your patient's triage category does not reflect their needs, you require clinical or logistical advice please contact the **Trauma Desk** directly on

03333 990 211

or by airwave by placing a callback to your local area dispatcher who will arrange a callback from the Trauma Desk.

Triage Questions

Step 1 — Assess your Patient's Physiology

Does your Patient have any of the following:
- Systolic blood pressure <90 mmHg, or no radial pulse
- Glasgow Coma Scale < 14
- Respiratory Rate < 10 or > 29 breaths/min

Step 2 — Assess your Patient's Injuries

Does your Patient have any of the following:
- Penetrating injury to head, neck, torso or extremities proximal to elbow or knee
- Chest Wall instability or deformity
- Two or more proximal limb fractures
- Crushed, degloved, mangled or pulseless extremity
- Amputation proximal to wrist or ankle
- Suspected Pelvic Fracture
- Open or Depressed Skull Fracture
- Paralysis

Step 3 — Assess the Mechanism of Injury

Did any of the following occur:
- Fall > 20 Feet
- High Risk Vehicle Accident
  - With > 12" Intrusion
  - Ejection (partial or complete)
  - Death in same passenger compartment
- Vehicle Striking Pedestrian/Cyclist at > 20 mph
- Motorcycle accident at > 20 mph

Step 4 — Special Considerations

Are any of the following present:
- Age > 55 years
- Bleeding Disorder or Anticoagulant Treatment
- Isolated Burns (Liaise with Trauma Desk)
- Pregnancy > 20 weeks
- Morbid Obesity

YES / NO

Response Category

Should the airway become compromised and cannot be managed, consider conveying/diverting to the nearest locally designated Emergency Department

Major trauma centre care

Your Patient requires
Major Trauma Centre (MTC) Care
- If <45 minutes from MTC = convey to MTC
- If >45 minutes from MTC = contact Trauma Desk

If you do not think your patient requires MTC, contact Trauma Desk

Remember to pre-alert the receiving hospital via airwave if you are managing a patient triaged to MTC

Trauma unit care

Your Patient requires
Trauma Unit (TU) Care
- Convey to the nearest TU, or MTC if closer
- If >45 minutes from TU/MTC contact Trauma Desk

If you do not think your patient requires TU/MTC, contact Trauma Desk

Local

Convey your patient to the nearest Local Emergency Hospital

If you think your patient requires TU/MTC, contact Trauma Desk

VERSION 9 (DRAFT)

Scene Size-Up

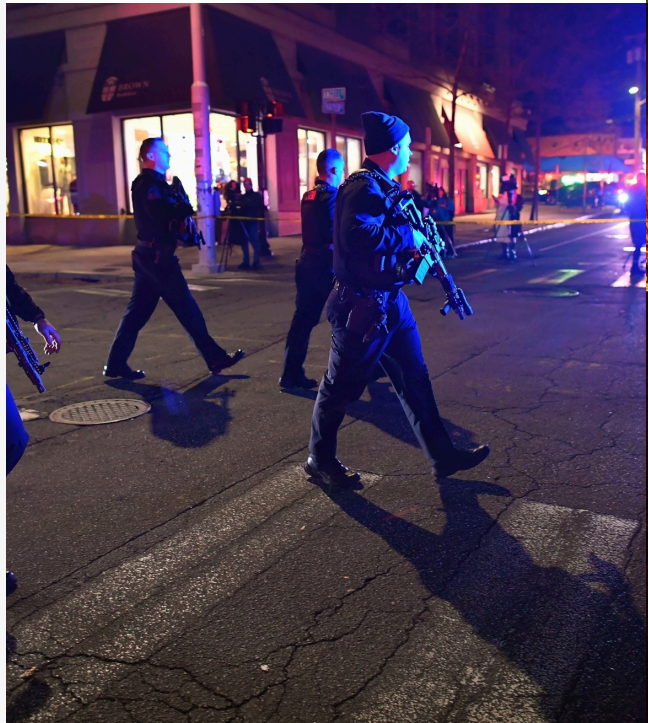
Early identification of hazards to protect responders, patients, and bystanders before patient contact.

Key hazards to assess.

- Crash and rescue scenes, risk of fire, toxic substances, electrocution, unstable surfaces or structures, low oxygen or toxic environments such as sewers, silos, or ship holds.
- Hazardous materials incidents, warning placards on vehicles or structures, require specialist resources and appropriate training.
- Farms, confined spaces such as silos, livestock, heavy machinery, manure pits or ponds.
- Crime scenes, potential presence of armed or fleeing individuals, risk to responders, preservation of evidence, entry only after clearance by law enforcement.
- Bystanders, risk of violence, intoxication, weapons, domestic violence, or dangerous animals.
- Blast scenes, risk of secondary devices, chemical or radiological hazards, broken glass zone, specialist assessment before entry.
- Mass shooting or active shooter events, ongoing threat, coordinated response with law enforcement, focus on rapid life saving interventions and evacuation.









Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.

Scene Size-Up

Standard precautions.

- Use personal protective equipment according to risk.
- Minimum protection includes gloves.
- Maximum protection may include chemical protective suits and self contained breathing apparatus.

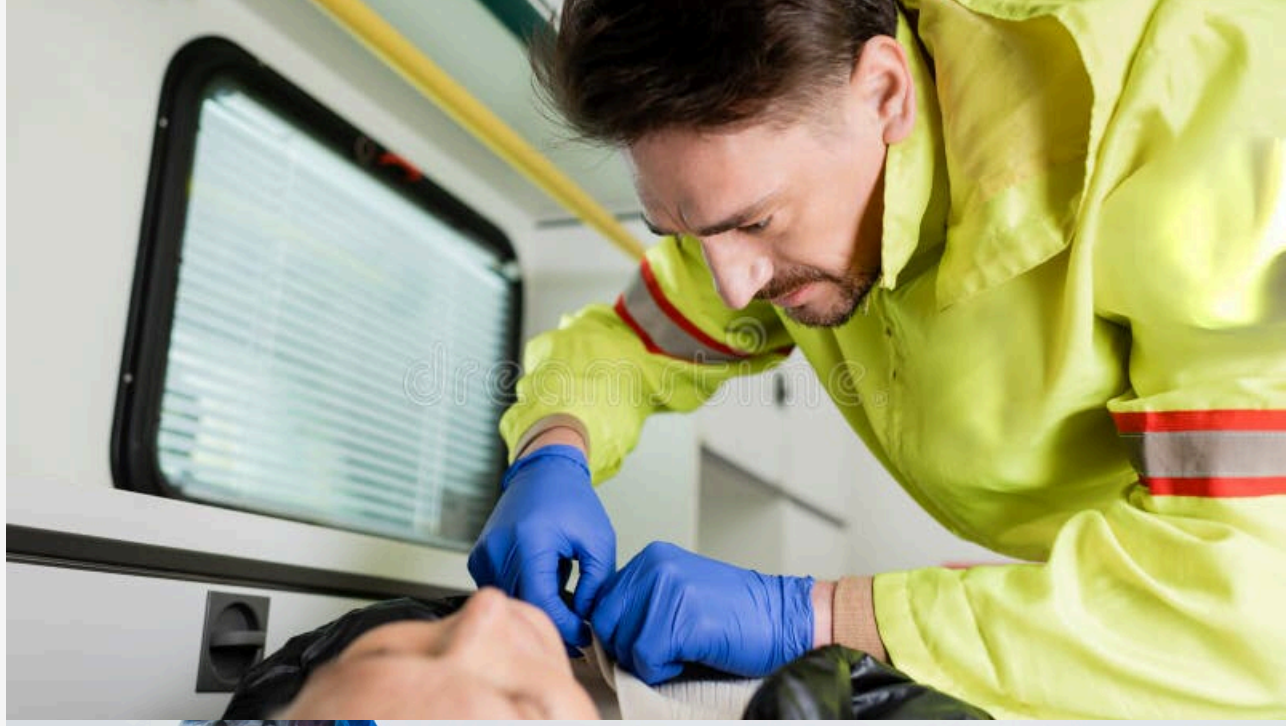
Steps of scene size up.

Standard precautions and personal protective equipment.

- Scene safety assessment.
- Need for additional help or equipment.
- Assessment of mechanism of injury.

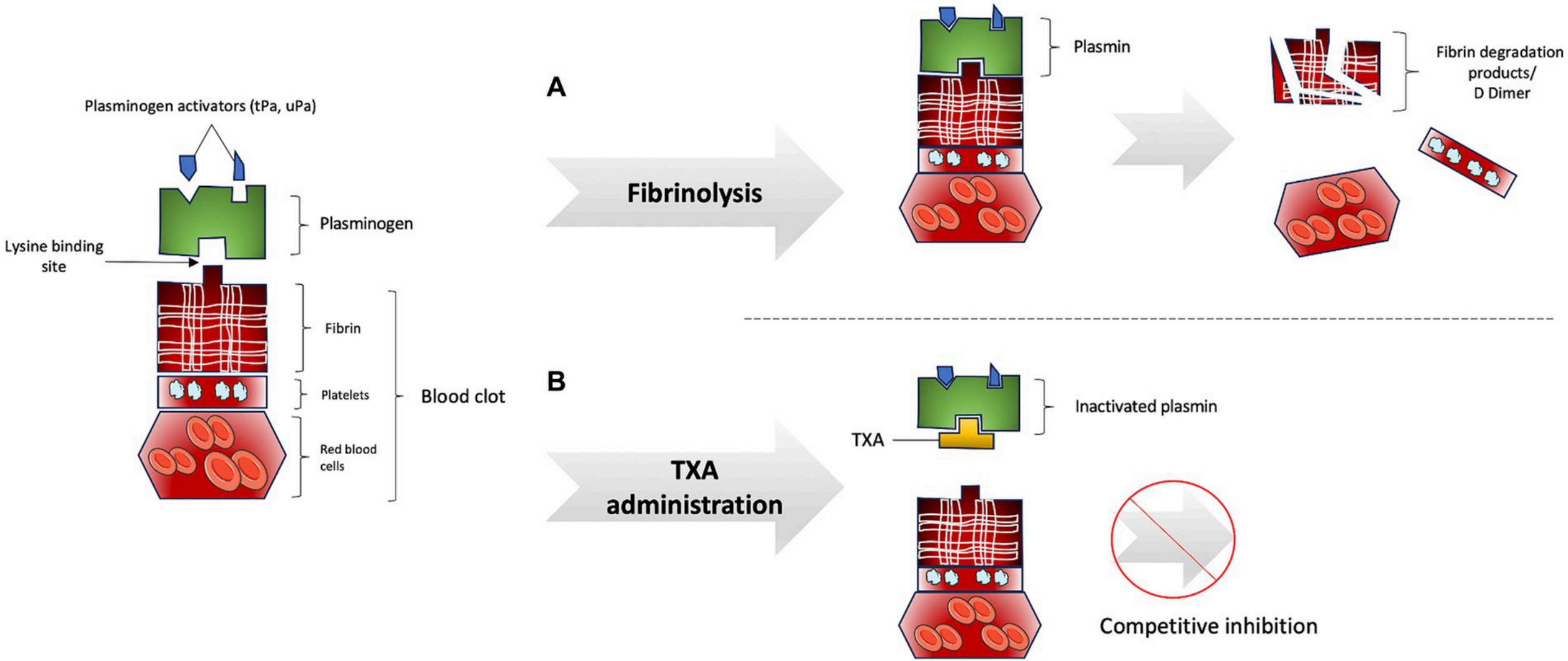
Operational principles.

- Do not enter unsafe scenes without training, equipment, and support.
- Request specialist resources early when hazards are suspected.
- Prioritise responder safety while enabling rapid, life saving care when conditions allow.





Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.



Bouras M, Bourdiol A, Rooze P, Hourmant Y, Caillard A, Roquilly A. Tranexamic acid: a narrative review of its current role in perioperative medicine and acute medical bleeding. *Front Med*. 2024;11. doi:10.3389/fmed.2024.1416998

Pre-hospital Spinal Motion Restriction

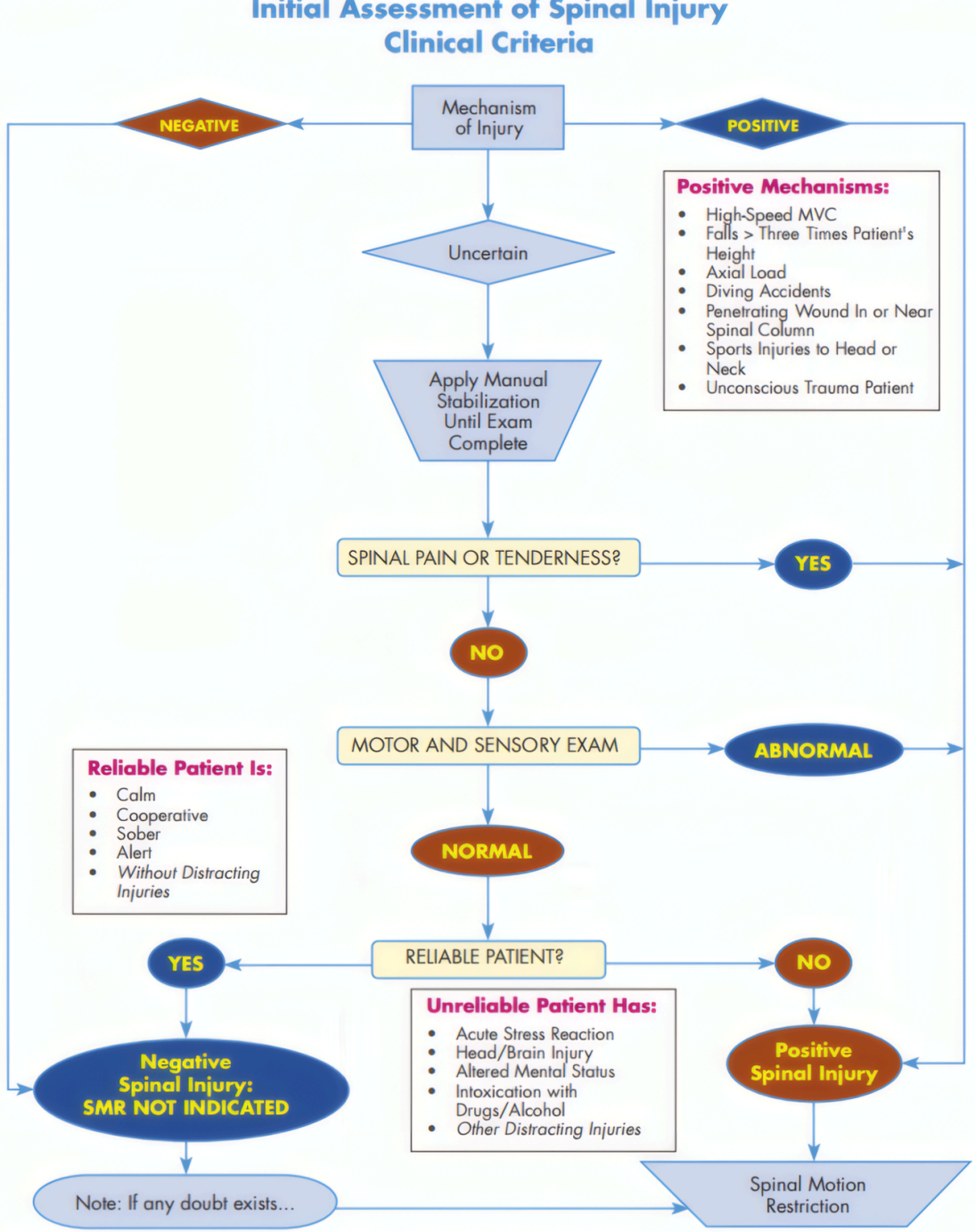


Figure 10-6 Decision tree for spinal motion restriction. *(Reprinted by permission of Peter Goth, MD)*

Alson RL, Han KH, Campbell JE. *International Trauma Life Support for Emergency Care Providers*. 9th ed. Pearson Education, Inc.; 2020.



Center for Prehospital Care

YouTube

Spinal Motion Restriction

Principles of Acute Injury Management



Treat the greatest threat to life first

Focus on the most urgent and life-threatening injuries before addressing less severe ones



Lack of definitive diagnosis should not delay urgent treatment

Start providing crucial treatment even if the exact diagnosis is unclear



Initial, detailed history is not essential to begin evaluation and treatment

Gather necessary information later, but do not wait to start caring for the patient

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

Effective Teamwork & Communication in Trauma Resuscitation



Teamwork & Communication

Effective teamwork and clear communication are critical during trauma resuscitation to prevent adverse events



Structured Communication Points

Prearrival huddles, systematic handovers, and regular reassessments promote shared situational awareness



Defined Roles & Responsibilities

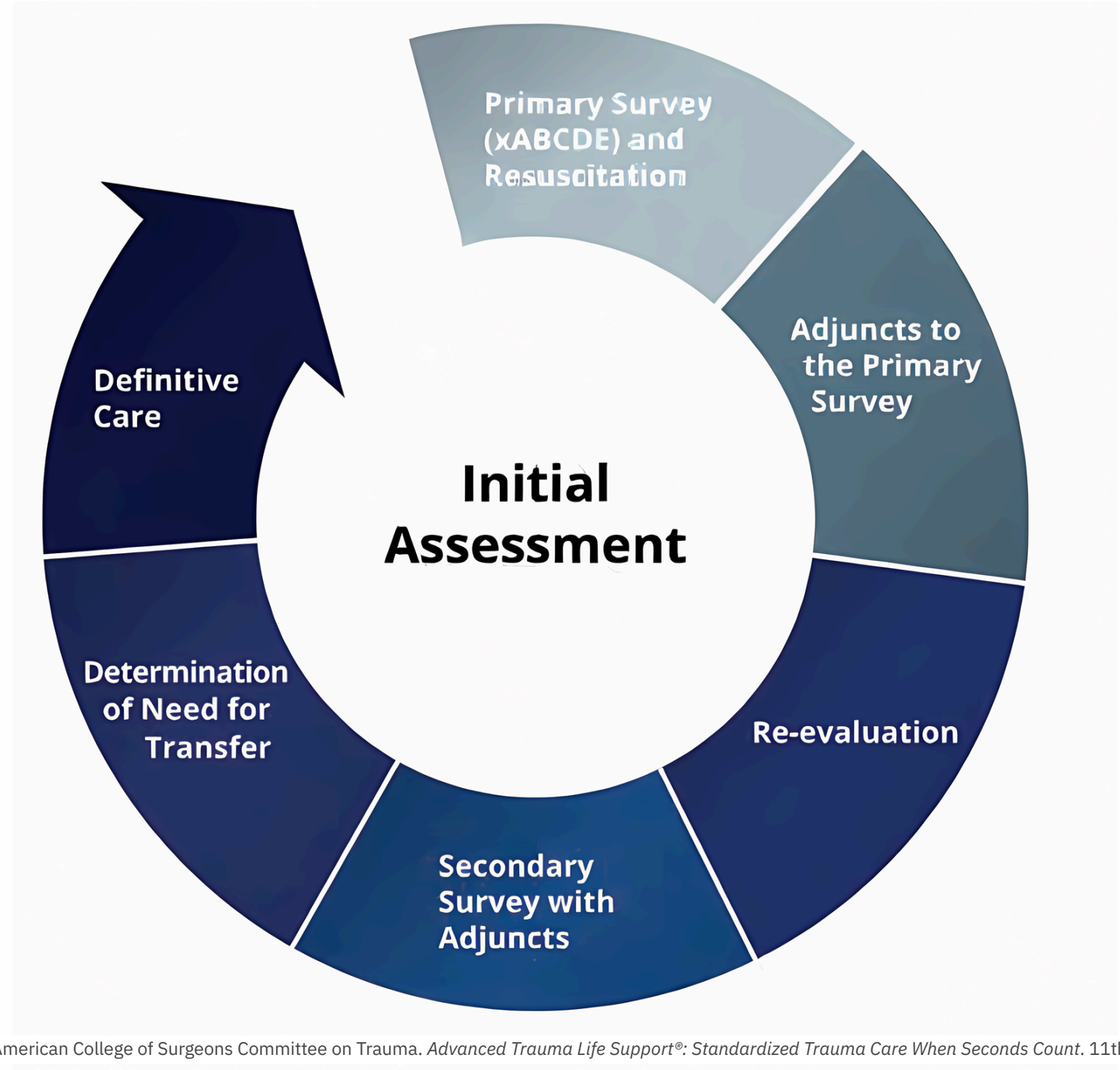
All team members should have clearly assigned roles and be able to share information without fear of judgement



Adapt to Unplanned Arrivals

When no prearrival notice is given, the team leader must rapidly assign roles and request clear reporting

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



National Guideline for the Field Triage of Injured Patients

RED CRITERIA
High Risk for Serious Injury

Injury Patterns

- Penetrating injuries to head, neck, torso, and proximal extremities
- Skull deformity, suspected skull fracture
- Suspected spinal injury with new motor or sensory loss
- Chest wall instability, deformity, or suspected flail chest
- Suspected pelvic fracture
- Suspected fracture of two or more proximal long bones
- Crushed, degloved, mangled, or pulseless extremity
- Amputation proximal to wrist or ankle
- Active bleeding requiring a tourniquet or wound packing with continuous pressure

Mental Status & Vital Signs

All Patients

- Unable to follow commands (motor GCS score <6)
- RR <10 or >29 breaths/min
- Respiratory distress or need for respiratory support
- Room-air pulse oximetry < 90%

Ages 0-9 years

- SBP < 70 mm Hg + (2 x age in years)

Ages 10-64 years

- SBP < 90 mm Hg or
- HR > SBP

Patients meeting any one of the above RED criteria should be transported to the highest-level trauma center available within the geographic constraints of the regional trauma system.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

YELLOW CRITERIA
Moderate Risk for Serious Injury

Mechanism of Injury

- High-Risk Auto Crash
  - — Partial or complete ejection
  - — Significant intrusion (including roof)
    - >12 inches occupant site OR
    - >18 inches any site OR
    - Need for extrication for entrapped patient
  - — Death in passenger compartment
  - — Child (age 0-9 years) unrestrained or in unsecured child safety seat
  - — Vehicle telemetry data consistent with severe injury
- Rider separated from transport vehicle with significant impact (e.g., motorcycle, ATV, horse, etc.)
- Pedestrian/bicycle rider thrown, run over, or with significant impact
- Fall from height >10 feet (all ages)

EMS Judgment

Consider risk factors, including:

- Low-level falls in young children (age ≤5 years) or older adults (age ≥65 years) with significant head impact
- Anticoagulant use
- Suspicion of child abuse
- Special, high-resource healthcare needs
- Pregnancy >20 weeks
- Burns in conjunction with trauma
- Children should be triaged preferentially to pediatric capable centers

If concerned, take to a trauma center

Patients meeting any one of the YELLOW CRITERIA WHO DO NOT MEET RED CRITERIA should be preferentially transported to a trauma center, as available within the geographic constraints of the regional trauma system (need not be the highest-level trauma center).

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

S-xABCDE-BAR Communication Tool

Acronym	Intent	Information
S-xABCDE	**Situation-x-Airway-Breathing-Circulation-Disability-Exposure/Environment**	• Referring clinician and facility • Name, age, gender, mechanism of injury • Indication for transfer • **x**-emergency hemorrhage control interventions (e.g. pressure, packing, or tourniquet) • **Airway:** assessment, intervention • **Breathing:** assessment, intervention • **Circulation:** assessment, intervention • **Disability**: assessment, intervention • **Environment/Exposure:** assessment, intervention
B	**Background**	• AMPLE history • Fluid/blood administered • Medications (date and time) • Images • Reduction/splints
A	**Assessment**	**Current Status:** • Physiology • Response to interventions • Likely injuries
R	**Recommendation**	• Transport mode and capability • Transport interventions • Other necessary interventions

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

The Primary Survey

& SIMULTANEOUS RESUSCITATION

		Key Causes and Issues
X	e**X**sanguinating e**X**ternal hemorrhage	Massive bleeding from open extremity and other wounds
A	**A**irway	Inability to maintain the airway patency due to direct injury, altered mental status, shock
B	**B**reathing	Compromise of oxygenation and/or ventilation due to direct injury, shock
C	**C**irculation	1. Presence of shock from hemorrhagic, neurogenic, cardiogenic, or other sources 2. Hemorrhage control 3. Restoration and maintenance of end-organ perfusion
D	**D**isability	Injury to the central nervous system • Traumatic brain injury • Spinal cord injury
E	**E**xposure/ **E**nvironment	1. Exposure to prevent missed injuries while maintaining patient dignity 2. Maneuvers to prevent hypothermia

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

Trauma Bleeding Control



Preventable Cause of Trauma Death

External haemorrhage is the most preventable cause of early trauma death.



Prioritise Bleeding Control

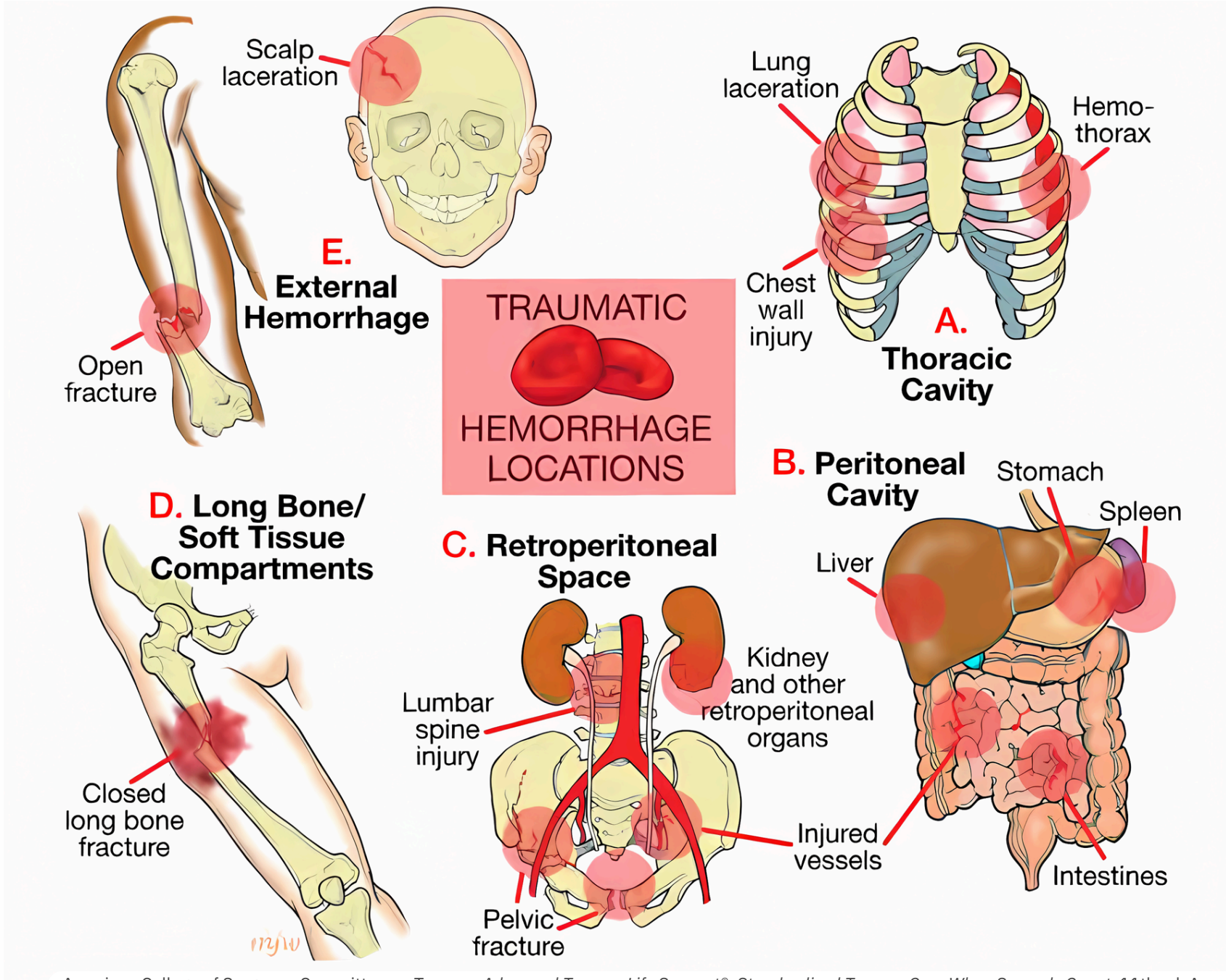
Prioritisation before airway interventions when severe bleeding is evident.



Immediate Interventions

Immediate interventions must not be delayed by diagnostic activities.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



Traumatic Haemorrhage: Compressible vs Noncompressible

- ## Compressible Haemorrhage

  Bleeding that **is visible** and potentially controllable **without making an incision**, using methods like pressure, packing, and tourniquet application.

- ## Noncompressible Haemorrhage

  Bleeding that **is not visible or controllable without making an incision**.

- ## Visible Bleeding Assessment

  Clinician visually inspects patient, clothing, and stretcher for continuous large-volume bleeding, pooling of blood, and spurting arterial haemorrhage.

- ## Non-life-threatening External Hemorrhage

  When external bleeding is not significant in volume, not spurting from a larger artery, or is adequately controlled during prehospital management, the clinician proceeds to the airway assessment and management.

IDENTIFICATION OF LIFE-THREATENING HEMORRHAGE

There is **PULSATILE** or **STEADY BLEEDING** from the wound





Traumatic **AMPUTATION** of the **arm** or **leg**



BRIGHT RED BLOOD is pooling on the ground

Overlying clothing becoming **SOAKED WITH BLOOD**



Bandages or makeshift bandages used to cover the wound are **INEFFECTIVE** and steadily becoming **SOAKED WITH BLOOD**

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TACTICAL COMBAT CASUALTY CARE

TCCC

HIGHLIGHTS

MASSIVE HEMORRHAGE CONTROL IN TACTICAL FIELD CARE

Committee on Tactical Combat Casualty Care (CoTCCC)

INITIAL DIRECT PRESSURE BEFORE INTERVENTION

DIRECT PRESSURE can and **should be used** as a temporary measure **until** a **tourniquet** or **dressing** is in place



It is hard to use direct pressure alone to control significant bleeding or while moving the casualty

Direct pressure can be **used** if a treatment no longer maintains control of the bleeding **while a new treatment is started**

⚠ **REMEMBER** to ask other first responders to assist as needed

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CPP TCCC

Module 6: Massive Hemorrhage Control

DELIBERATE TOURNIQUETS IN TACTICAL FIELD CARE



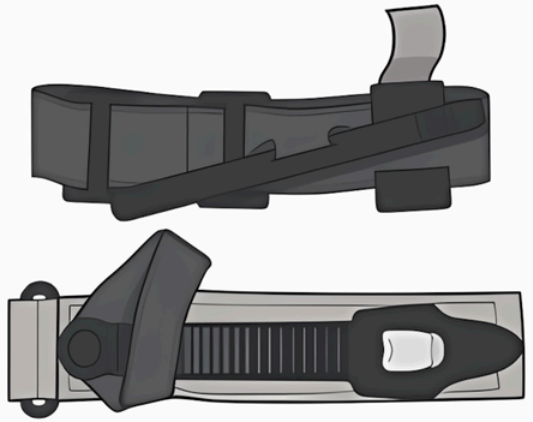
In TFC the **source** can be **identified** to ensure deliberate TQ placement

Deliberate TQs (in TFC) are applied **2-3 INCHES ABOVE THE WOUND**, directly on the skin (**not over clothing or on a joint**)



Ensure **all the slack** of the **TQ band** is pulled **tightly as possible**

If bleeding is **NOT** controlled with the first TQ, apply a second side-by-side with the first



Use a TQ to control life-threatening external hemorrhage that is anatomically amenable to TQ use or for **ANY traumatic amputation**



TQs need to be applied rapidly. The bleeding should be stopped **WITHIN ONE MINUTE** and the TQ fully secured within 3 minutes

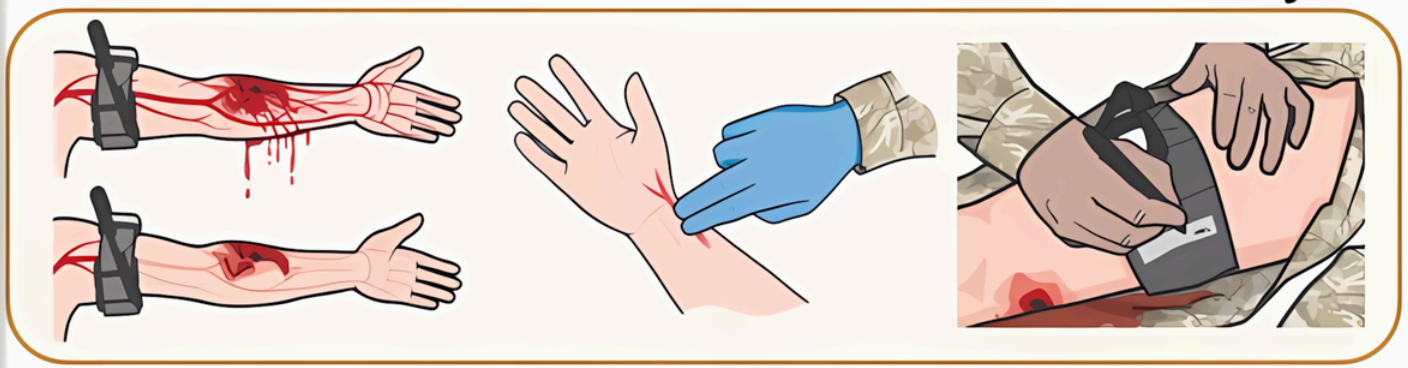
The time of tourniquet placement should be documented on the tourniquet itself and on the DD Form 1380 TCCC Casualty Card in TFC

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TOURNIQUET EFFECTIVENESS AND DOCUMENTATION

Is there a role for intermittent tourniquet loosening in TFC?	
Answer	**Rationale**
NO	Periodic loosening of tourniquets for the purpose of reperfusing the limb has resulted in incremental exsanguination and has no role on the battlefield. Additionally, periodic reperfusion of the ischemic limb may increase the amount of damage to the limb by worsening of the ischemia-reperfusion injury.

TQs can be assessed for effectiveness by:



Ensuring that the **BLEEDING** has Stopped

Checking a pulse distally on the limb where the TQ is applied to ensure there is **NO PULSE**

Time of TQ that is placed should be **documented** during the TFC and **NOT** the CUF phase

TQ application time is **important** in overall casualty care

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COMMON TOURNIQUET ERRORS

- **NOT** using one when you should or waiting too long to put it on
- **NOT** pulling all the slack out before tightening
- **NOT** making it tight enough – the TQ should stop the bleeding **AND** eliminate the distal pulse
- **NOT** using a second TQ, if needed
- Using a TQ for minimal bleeding *(However, **when in doubt**, apply a TQ)*
- Putting it on too proximally if the bleeding site is clearly visible
- Loosening TQs for a period to allow recirculation of a limb
- Taking a TQ off **prematurely** when it is still needed for hemorrhage control
- **DON'T** put TQs over joints!



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Immediate Lower Extremity Tourniquet Application to Delay Onset of Reperfusion Injury after Prolonged Crush Injury

Daniel S. Schwartz, MD, Zach Weisner, EMT-P, Jehangir Badar, MD

Schwartz DS, Weisner Z, Badar J. Immediate Lower Extremity Tourniquet Application to Delay Onset of Reperfusion Injury after Prolonged Crush Injury. *Prehospital Emergency Care*. 2015;19(4):544-547.
doi:10.3109/10903127.2015.1005264

Abstract

Reperfusion after severe crush injury is an infrequent, but life-threatening condition. It is a unique aspect of prehospital medicine that occurs in the presence of emergency responders attempting to extricate and treat patients who have suffered a crushing injury. These events are unlikely to occur in the hospital setting and, as a result, remain poorly studied. Some evidence exists regarding prophylaxis, but the efficacy of these treatments has not been clearly established. The use of commercial tourniquets to delay the onset of reperfusion injury has previously been described in theory. Extensive literature now exists supporting the safety of tourniquet use in limb trauma and this potential life-saving measure requires further study in patients with crush injury. We present a case of prehospital tourniquet application to delay reperfusion injury after crush injury that resulted in a reduction in morbidity and complete limb salvage. **Key words:** crush injury, prehospital, reperfusion injury, tourniquet

PREHOSPITAL EMERGENCY CARE 2015;Early Online:1–4



FIGURE 1. Immediate application of bilateral lower extremity tourniquet after removal of crushing weight. (Photo courtesy of Monroeville (PA) Police Department.) Photo used with permission of patient.

Schwartz DS, Weisner Z, Badar J. Immediate Lower Extremity Tourniquet Application to Delay Onset of Reperfusion Injury after Prolonged Crush Injury. *Prehospital Emergency Care*. 2015;19(4):544-547.
doi:10.3109/10903127.2015.1005264

JOINT TRAUMA SYSTEM CLINICAL PRACTICE GUIDELINE (JTS CPG)

Crush Syndrome - Prolonged Field Care

This Role 1 prolonged field care (PFC) guideline is meant to provide medical professionals who encounter crush syndrome in austere environments with evidence-based guidance for how to manage the various aspects of crush injury care and monitoring.

Contributors

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Publication Date: 28 December 2016

TOURNIQUETS FOR MANAGEMENT OF CRUSH

Tourniquets may delay the life-threating complications of a reperfusion injury if immediate fluid resuscitation or monitoring is not initially available. Consider tourniquet placement for crush injury before extrication if the length of entrapment exceeds 2 hours and crush injury protocol cannot be initiated immediately.[22–24]

Goal: Delay acute toxicity until after fluid resuscitation and monitoring are available.

- **Best:** Apply two tourniquets side by side and proximal to the injury immediately before extrication
- **Minimum:** Apply two tourniquets side-by-side proximal to the injury immediately after extrication
- Initiate crush injury protocol before loosening tourniquet, and then only if the patient meets criteria for tourniquet conversion or removal given in the TCCC guideline

A limb that is cool, insensate, tensely swollen, and pulseless is likely dead. Patient may develop shock and kidney damage and may die. Consider fasciotomy. If no improvement, place two tourniquets side by side and proximal to the injury and do not remove. Amputation anticipated.

HEMOSTATIC DRESSINGS

INDICATIONS for **Hemostatic Dressing** use are compressible (external) hemorrhage not amenable to limb tourniquet use or as an adjunct to tourniquet removal

- **Hemostatic dressing** is safe and contains active ingredients that assist with blood-clotting at the bleeding site
- **Hemostatic dressings** can be used with or without a pressure bandage
- JFAK contains **one hemostatic** dressing and **one dry sterile gauze**

⚠ **DO NOT** pack hemostatic dressings into chest wounds

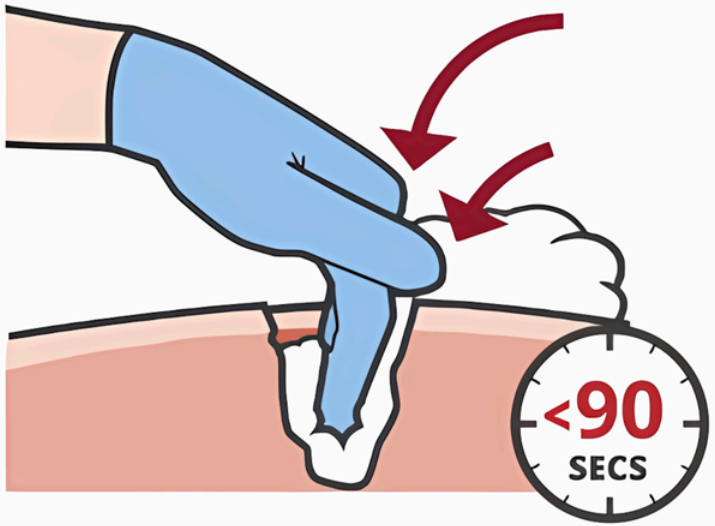
CoTCCC-Recommended Hemostatic Dressings:

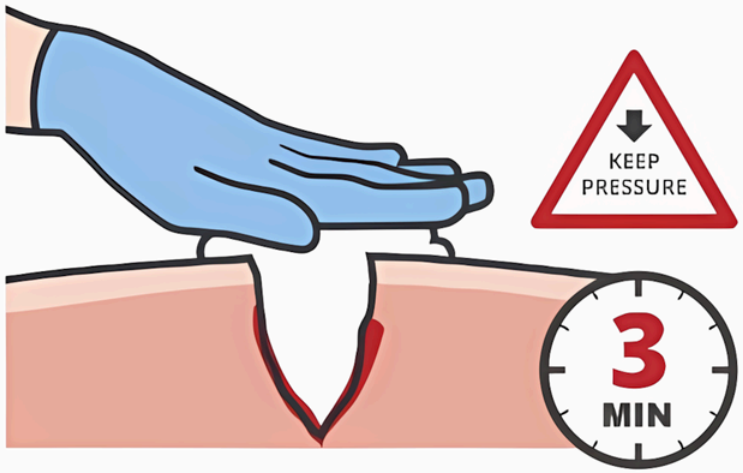
- **Combat Gauze**

Alternative hemostatic adjuncts:

- **Celox Gauze**
- **ChitoGauze**
- **XStat** (best for deep, narrow-tract junctional wounds)
- **iTClamp** (may be used alone or in conjunction with hemostatic dressing or XStat)

WOUND PACKING PRINCIPLES







- Identify **exact source** of bleeding and **APPLY** **direct pressure** **UNTIL** dressing or gauze is placed
- Pack the wound **maintaining** **CONSTANT** direct pressure within **90 SECONDS** to be effective
- Fill and pack the wound tightly, ensuring dressing or gauze extends 1-2 inches above the skin
- **HOLD** direct pressure for at least **3 MINS** (***this is necessary***, *even with the active ingredient in hemostatic dressing)*
- When packing a large wound, more than one hemostatic gauze and/or **additional** gauze may be **needed**
- Carefully **observe** to determine if bleeding has been **controlled**
- Once you are sure the bleeding has **stopped**, apply a pressure bandage

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PRESSURE BANDAGE PRINCIPLES

ALL dressings for **significant** bleeding **should be secured** with pressure bandages



Place the bandage pad **directly** on the dressing, **continuing to apply direct pressure**

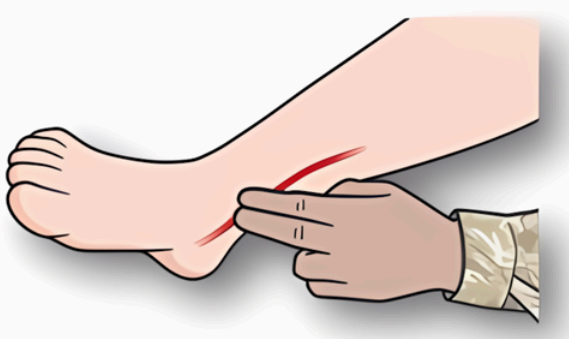
Wrap the pressure/elastic bandage **tightly**, focusing pressure over the wound



SECURE the hooking **ends** of the hook and loop or closure bar onto the last wrap of the bandage

Level of Evidence: C-LD

Check **circulation BELOW** the pressure bandage (**feeling for distal pulse)**





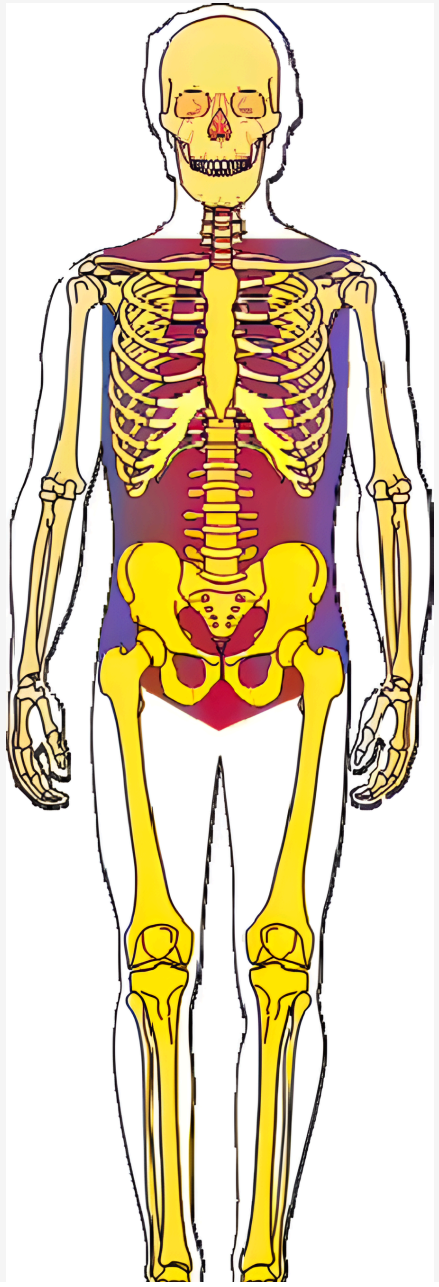
Assess **skin BELOW** the pressure bandage for **coolness** to touch, **bluish** hue, or **numbness**

If the pressure bandage is acting as a tourniquet, then you should **loosen** and resecure the bandage

Dressings and bandages should be **reassessed** and checked frequently and/or **EVERY TIME a casualty is moved**

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Managing Non-Compressible Torso Hemorrhage

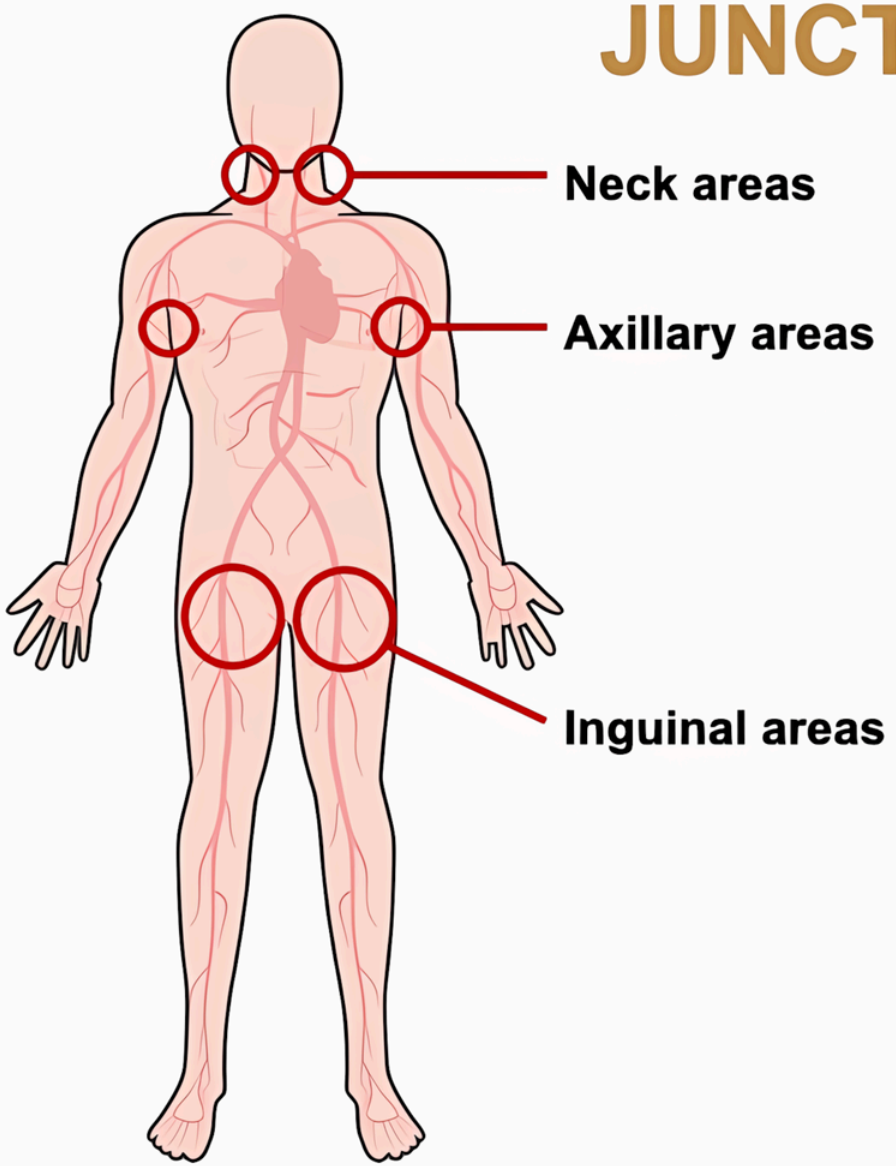


- **Early recognition of non-compressible torso haemorrhage**
  Quickly identify severe bleeding that cannot be controlled by direct pressure, such as from a pelvic or junctional injury
- **Rapid activation of massive transfusion protocol**
  Immediately call for a coordinated, pre-planned response to provide large volumes of blood products to restore circulating volume
- **Expedite transfer to operative or interventional control**
  Urgently transport the patient to the operating room or interventional radiology suite to surgically or radiologically control the bleeding source

Blackbourne LH, Czarnik J, Mabry R, et al. Decreasing killed in action and died of wounds rates in combat wounded. *J Trauma Acute Care Surg*. 2010;69(1):S1. doi:10.1097/TA.0b013e3181e4206f

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

JUNCTIONAL ANATOMY

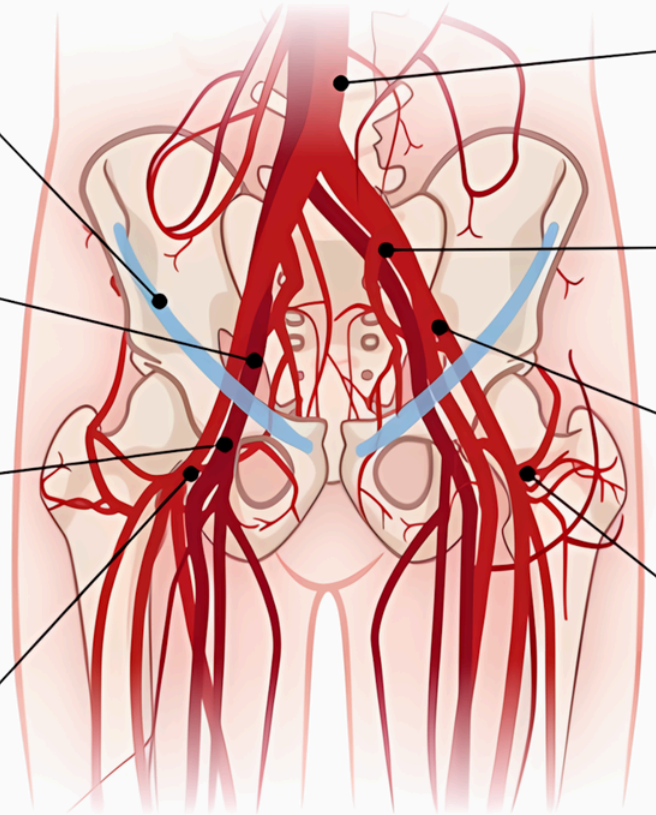


- Junctional areas are located at the **junctions of the extremities** and **neck** with the torso
- Junctional hemorrhage occurs with injury to the large blood vessels that pass through the junctional areas
- Junctional hemorrhage can also occur on the extremities if the **injury** is **TOO CLOSE** **to the torso** for a limb tourniquet to be applied



Blood vessels at **junctional areas are LARGER** than in the limbs but **can still be COMPRESSED** with direct pressure

PELVIC AND INGUINAL ANATOMY



BOTTOM LINE:

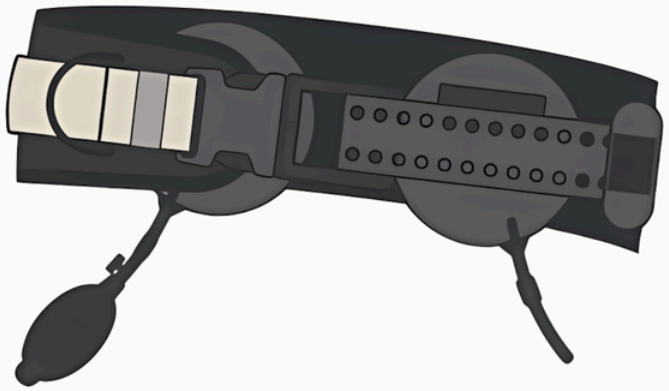
The site of lethal hemorrhage was **truncal (67.3%)**, followed by **junctional (19.2%)** and **peripheral-extremity (13.5%)** hemorrhage*

For a piece of shrapnel, the high thigh and groin are **Target Rich Environments** not covered by body armor

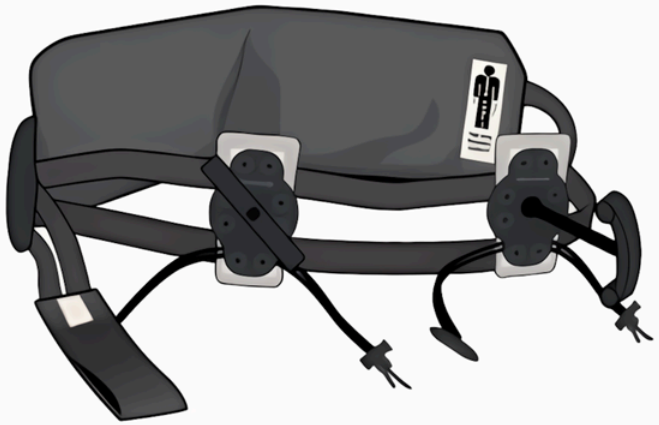
Level of Evidence: C-LD

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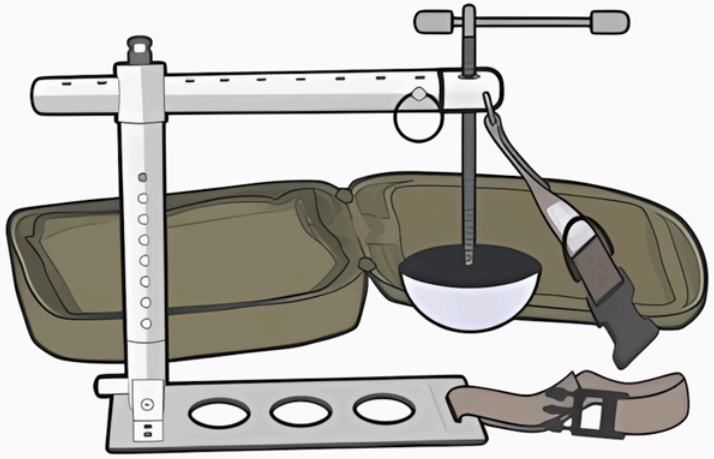
JUNCTIONAL TOURNIQUETS



SAM JUNCTIONAL TOURNIQUET



JETT JUNCTIONAL TOURNIQUET



CROC JUNCTIONAL TOURNIQUET

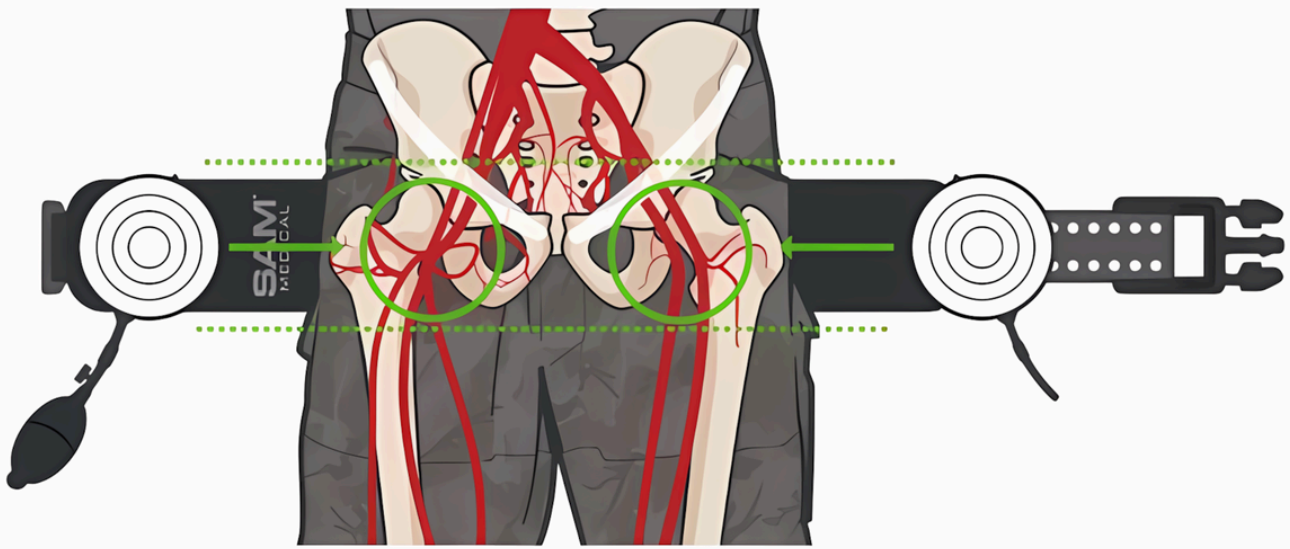
Level of Evidence: C-LD

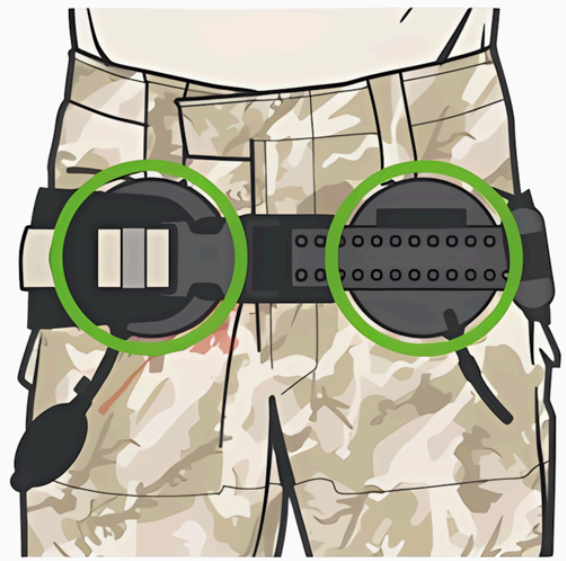


REMEMBER Apply direct pressure and/or pack any open wounds with hemostatic dressing while preparing the junctional tourniquet

SAM JUNCTIONAL TOURNIQUET

⚠ Targeted compression devices (TCDs) **must** be positioned appropriately to compress the large vessels and control bleeding



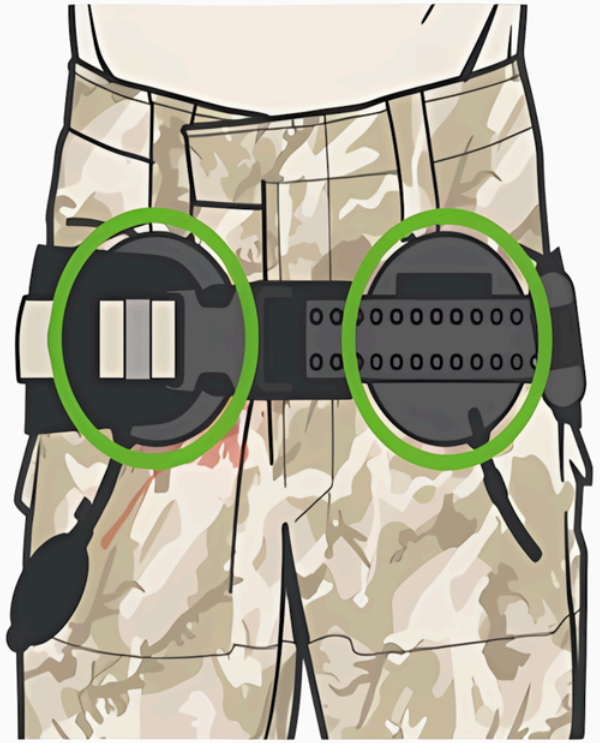


For inguinal junctional hemorrhage, the TCD(s) should be positioned over the femoral artery just below the inguinal ligament

For effective hemorrhage control, an audible click should be heard when the belt and buckle are appropriately secured; all slack must be removed from the belt before TCD inflation

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SAM JUNCTIONAL TOURNIQUET PLACEMENT



- **Inflate TCD(s)** until the hemorrhage stops and distal pulse is not longer present
- The bleeding should be stopped **WITHIN 90 SECONDS**
- When treating bilateral junctional injuries, use a second TCD following the same procedure
- **DOCUMENT** time of junctional TQ(s) placement tourniquet itself and on the DD Form 1380 TCCC Casualty Card



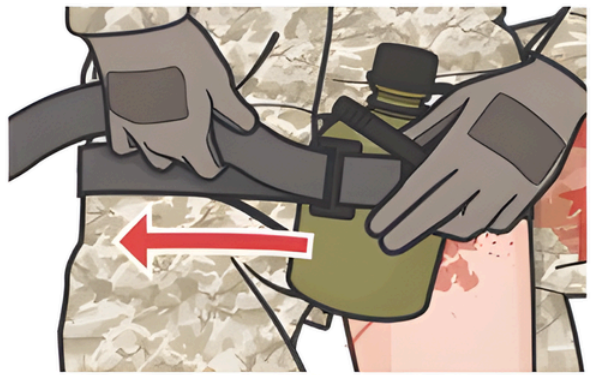
REMEMBER Monitor for hemorrhage control and adjust device as necessary especially after any casualty movement

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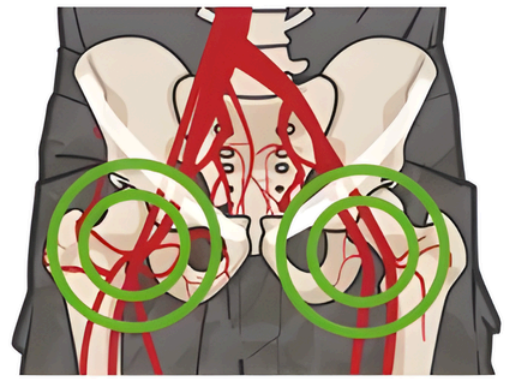
JUNCTIONAL HEMORRHAGE CONTROL WITH IMPROVISED PRESSURE DELIVERY DEVICE



A **Pressure Delivery Device (PDD)** can be made by using materials such as a **shoe/boot, full water bottle, or canteen**



Pack groin injuries with hemostatic dressing and then use an **improvised PDD** for additional targeted, sustained pressure



The PDD is placed in the inguinal gutter while **MAINTAINING CONSTANT pressure** on the dressing-packed wound



The PDD is then **secured** with a tourniquet* and **tightened** to add **ADDITIONAL** pressure

* ***Two TQs*** *may need to be joined* ***TOGETHER*** *when securing an improvised PDD*

REMEMBER Monitor for hemorrhage control and adjust device as necessary especially after any casualty movement

DOCUMENT time of Improvised PDD placement tourniquet on the casualty and on the DD Form 1380 TCCC Casualty Card

https://books.allogy.com/web/tenant/8/books/3f274639-3bbc-4530-8ed0-569fb9e7721f/

NECK JUNCTIONAL HEMORRHAGE CONTROL



Pack the wound with hemostatic dressing until the wound cavity is filled



Apply firm, manual pressure for **3 MINS**



Secure with bandage
While maintaining pressure, wrap the pressure bandage diagonally across the chest under the opposite axilla



Swath the arm on the injured side

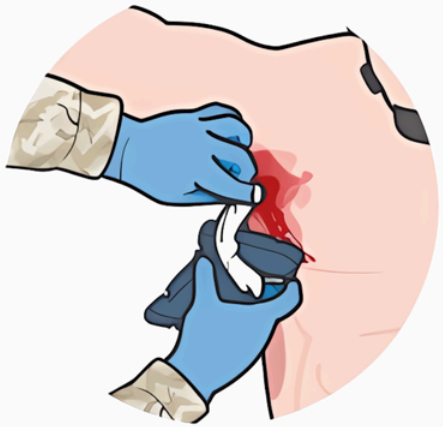
REMEMBER Monitor for hemorrhage control and adjust device as needed, especially after any casualty movement; **DO NOT FORGET** to ask other first responders to assist as needed

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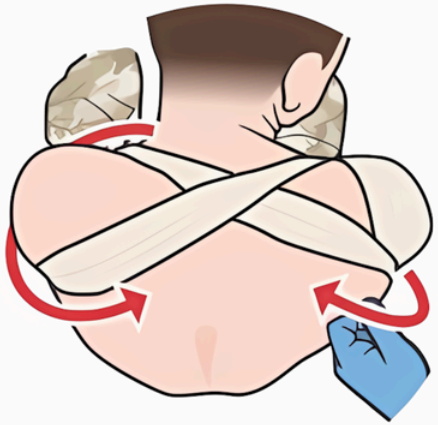
AXILLARY JUNCTIONAL HEMORRHAGE CONTROL



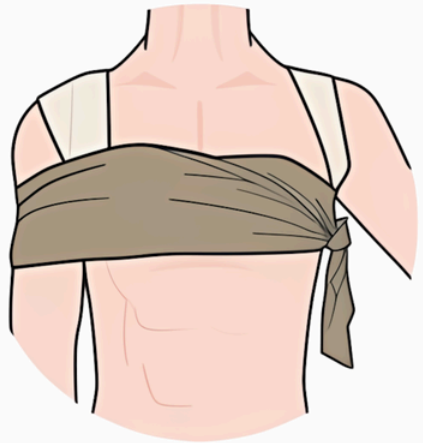
Lift the arm to expose the wound and assess the bleeding source



Pack the wound tightly with hemostatic gauze



Wrap elastic bandage across, back, and under opposite axilla, anchoring around opposite shoulder



Swath the upper arm on injured side to the chest using a **cravat**

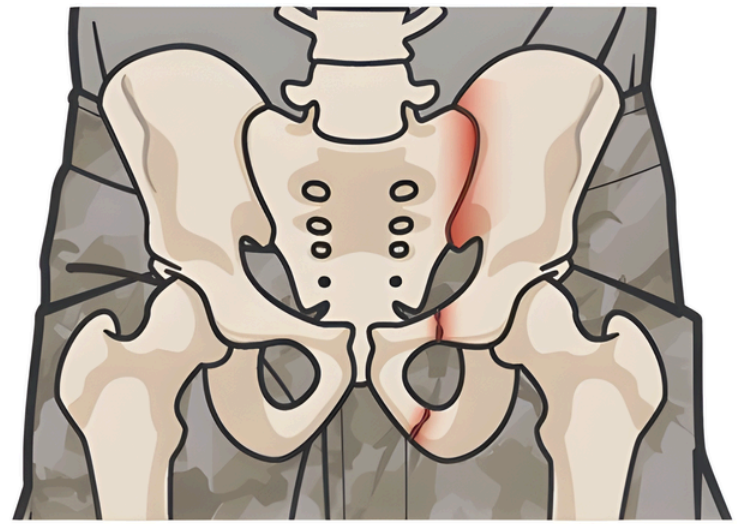
REMEMBER Monitor for hemorrhage control and adjust device as needed, especially after any casualty movement; **DO NOT FORGET** to ask other first responders to assist as needed

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SIGNS & SYMPTOMS OF PELVIC FRACTURES

Pelvic fracture may be **suspected** if the casualty's injuries are a result of blunt force or blast with **ONE OR MORE** of the following physical signs suggesting a pelvic fracture:

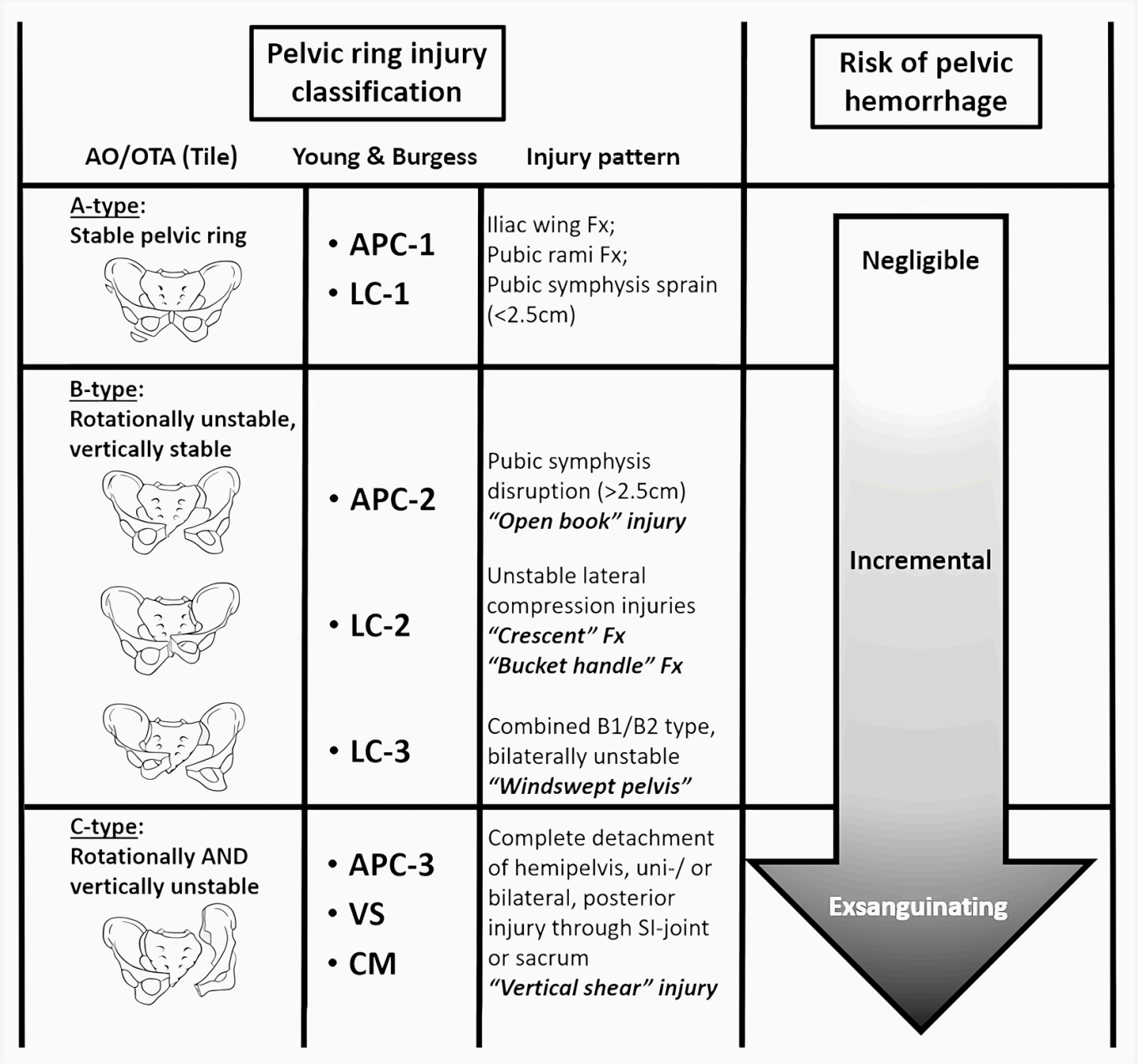
- **Pelvic pain**
- Any Major lower limb **amputation** or **near amputation**
- **Physical exam findings** suggestive of a pelvic fracture e.g., deformities, penetrating injuries, bruising near the pelvis
- **Pelvic instability, crepitus** or unequal leg lengths
- **Unconsciousness** or **Shock**



⚠ Bleeding pelvic fractures with hemodynamic instability have up to a **40% mortality**

MARCH

https://books.allogy.com/web/tenant/8/books/ebec0331-f93c-42cf-9fe5-c3ab2476fd93/



Stahel PF, Ziran N. The pathophysiology of pelvic ring injuries: a review. *Patient Saf Surg*. 2024;18(1):16. doi:10.1186/s13037-024-00396-x

Pelvic ring injuries usually result from high energy blunt trauma with rapid deceleration or axial loading.

Common mechanisms include road traffic collisions and falls from height.

The magnitude and direction of the applied force determine the pattern of pelvic disruption and the risk of haemorrhage.

Pelvic ring stability depends on the pubic symphysis anteriorly and the posterior ligamentous complex, particularly the iliosacral ligaments.

Increasing force causes progressive ligament disruption.

Posterior ligament failure allows pelvic volume expansion and uncontrolled retroperitoneal bleeding.

Antero posterior compression forces cause widening of the pubic symphysis with external rotation of the hemipelvis. This open book deformity places tensile stress on the posterior ligaments and increases pelvic volume, promoting venous and arterial bleeding.

Lateral compression forces produce internal rotation of the hemipelvis with compressive failure of the posterior ligaments.

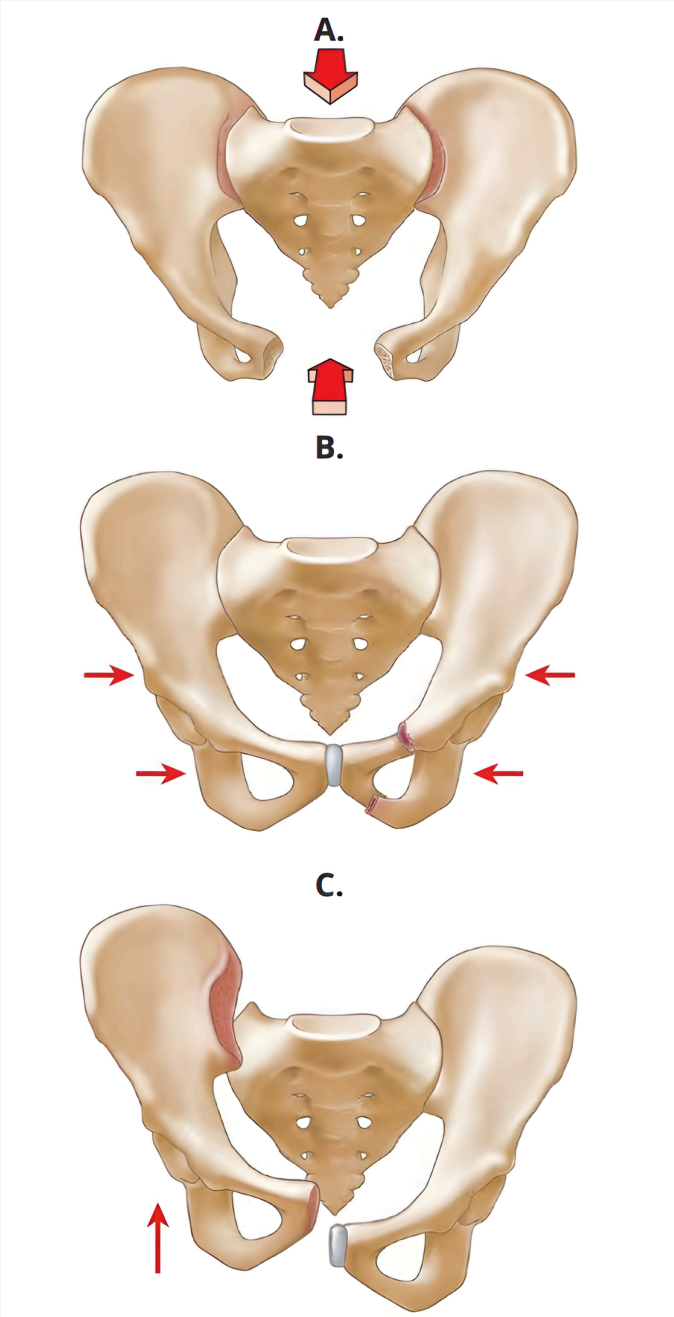
Bleeding risk is variable but increases with ligamentous disruption.

Vertical shear and combined mechanisms result from massive axial loading. These forces cause complete pelvic ring disruption with vertical translation and rotational instability.

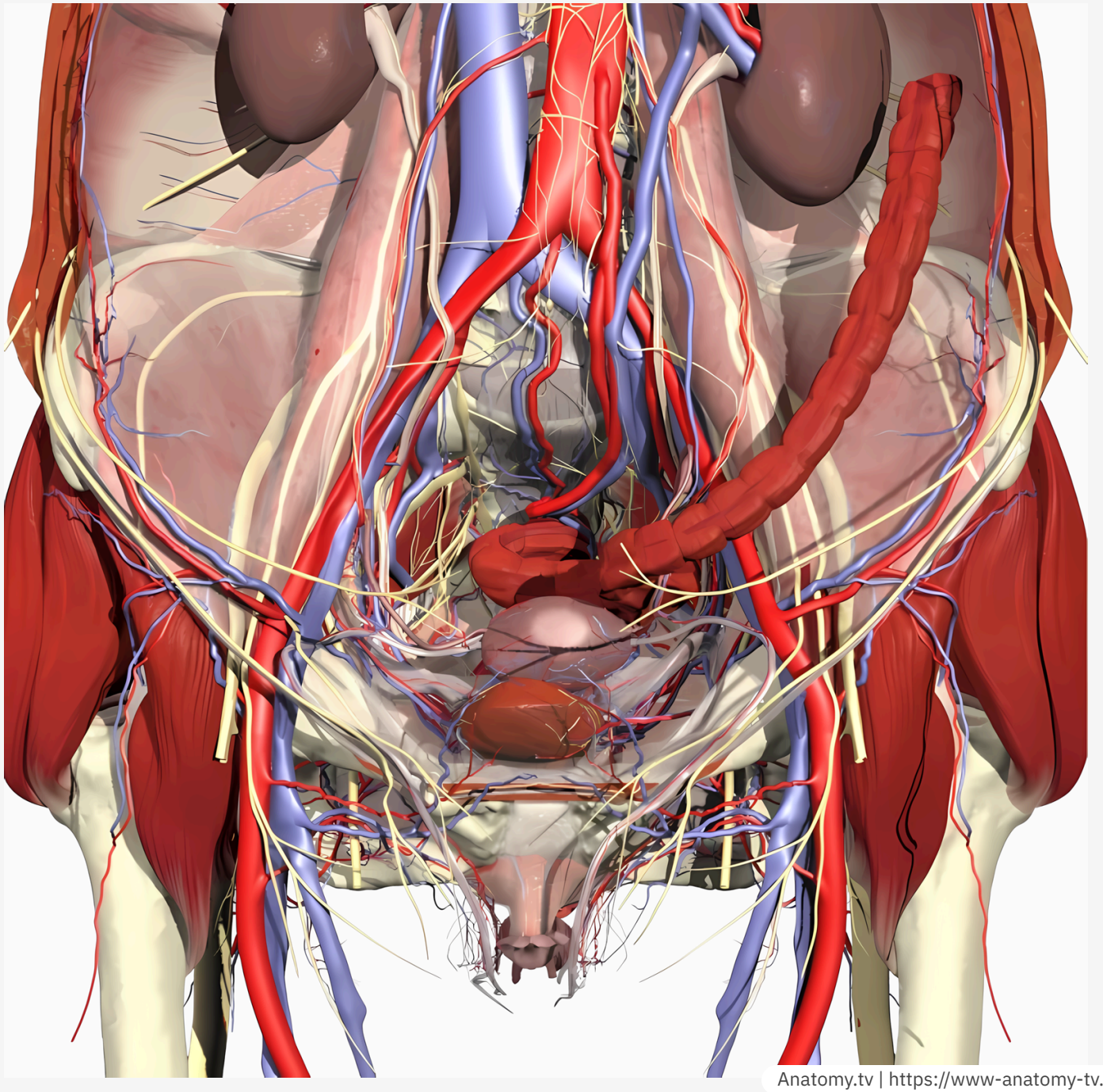
These patterns are strongly associated with rapid, life threatening haemorrhage.

Severe pelvic bleeding leads to haemorrhagic shock and triggers the lethal triad of metabolic acidosis, hypothermia, and coagulopathy. Early recognition of biomechanical instability and prompt control of bleeding are critical determinants of survival.

Stahel PF, Ziran N. The pathophysiology of pelvic ring injuries: a review. *Patient Saf Surg.* 2024;18(1):16. doi:10.1186/s13037-024-00396-x



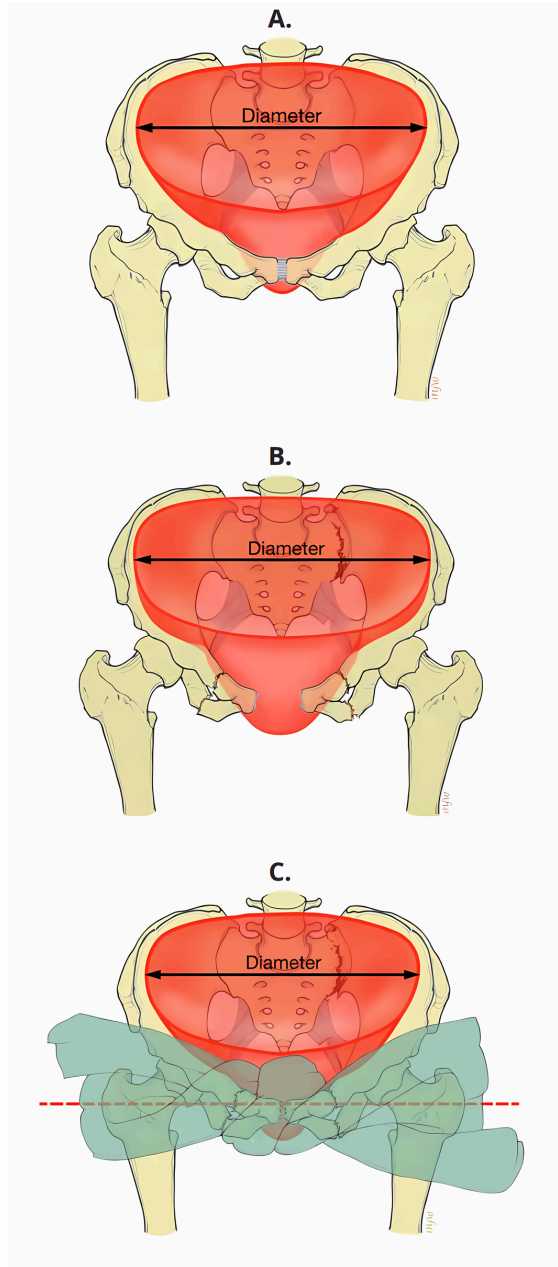
American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



Anatomy.tv | https://www-anatomy-tv.unica.idm.oclc.org/titles#AnatomyandPhysiology

Clinical Data Consistent with Pelvic Fracture

MECHANISM	Motor vehicle or motorcycle crash Fall from height Fall in frail patient Fall from animal transport Crush Bicycle crash
SYMPTOMS	Pelvic pain Low abdominal pain Hip pain Pain radiating in lower extremities Low back pain
SIGNS	Hypotension, tachycardia Pain on palpation of pelvis Lower abdominal tenderness Pain with hip movement External rotation lower extremities Sacral/coccyx tenderness Suprapubic widening Suprapubic, iliac ecchymosis Perineal ecchymosis Blood at urethral meatus Vaginal blood



American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

Pelvic binders are used as an early temporary measure to control haemorrhage in pelvic ring fractures, both prehospital and in the trauma bay. Their primary purpose is to stabilise the pelvis and reduce pelvic volume, particularly in open book fractures, which can accommodate large volumes of blood.

By approximating the pelvic ring, binders reduce pelvic volume and stabilise bony fragments, helping to limit ongoing bleeding. After application, a repeat pelvic radiograph is recommended to confirm reduction of an open book injury. Binders are radiolucent and do not interfere with imaging.

Binder use is appropriate in haemodynamically unstable patients with suspected or confirmed antero posterior compression injuries.

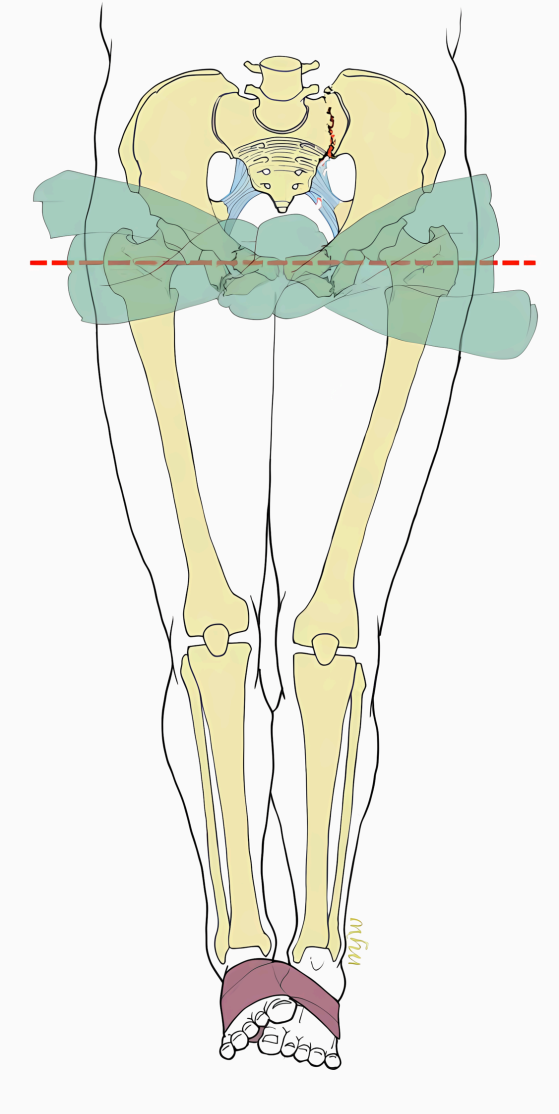
In lateral compression injuries, excessive binding may worsen bleeding and should be avoided.

Correct placement is essential. The binder must be centred over the greater trochanters, not over the iliac crests or abdomen. Excessive tightening should be avoided, especially before imaging confirmation, as overcompression can cause harm. Pelvic binders are a temporary and imperfect solution.

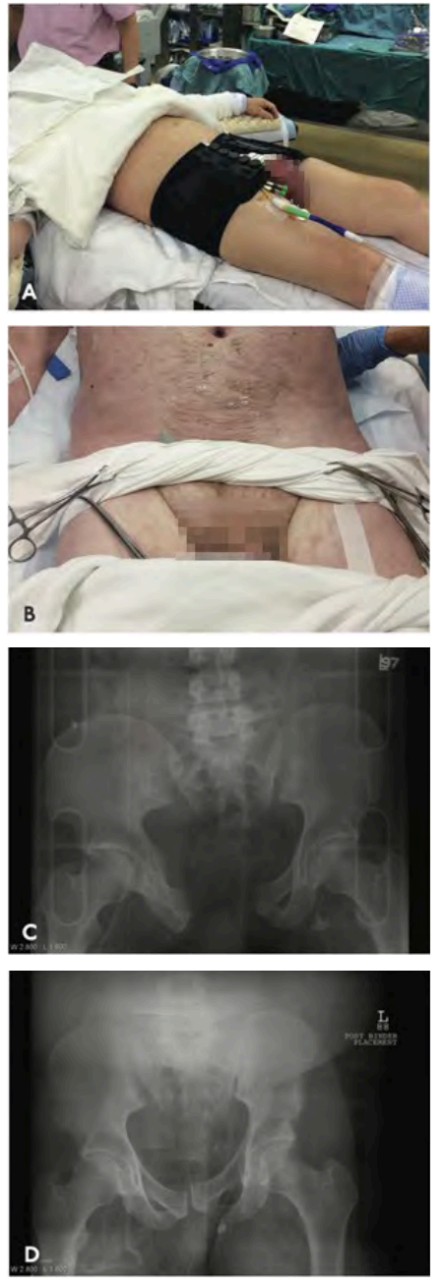
Prolonged application can cause skin, soft tissue, or muscle injury. Use should be limited in duration, ideally less than 24 hours, and definitive pelvic stabilisation and haemorrhage control should be planned as early as possible.

Improvised binders, such as a bed sheet tightened around the pelvis, can be effective when commercial devices are unavailable.

Feliciano DV, Mattox KL, Moore EE. *Trauma, Ninth Edition*. McGraw Hill / Medical; 2021.



American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

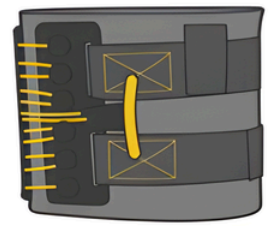




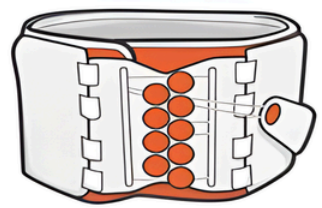
INDICATIONS AND APPLICATION METHODS OF PELVIC COMPRESSION DEVICES (PCDs)

APPLY PCD WHEN:

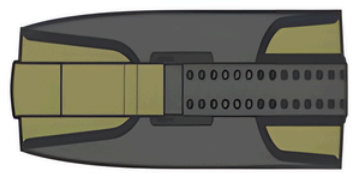
- Mechanism of injury includes **severe blunt force trauma** and/or **blast injury** and casualty has **one or more of the indications** previously mentioned
- **PLACE** at the level of greater trochanters, NOT the iliac crests
- **SECURE** knees and/or feet together to prevent external rotation of the legs



PelvicBinder®



T-POD™ Pelvic Stabilization Device



SAM® Pelvic Sling II

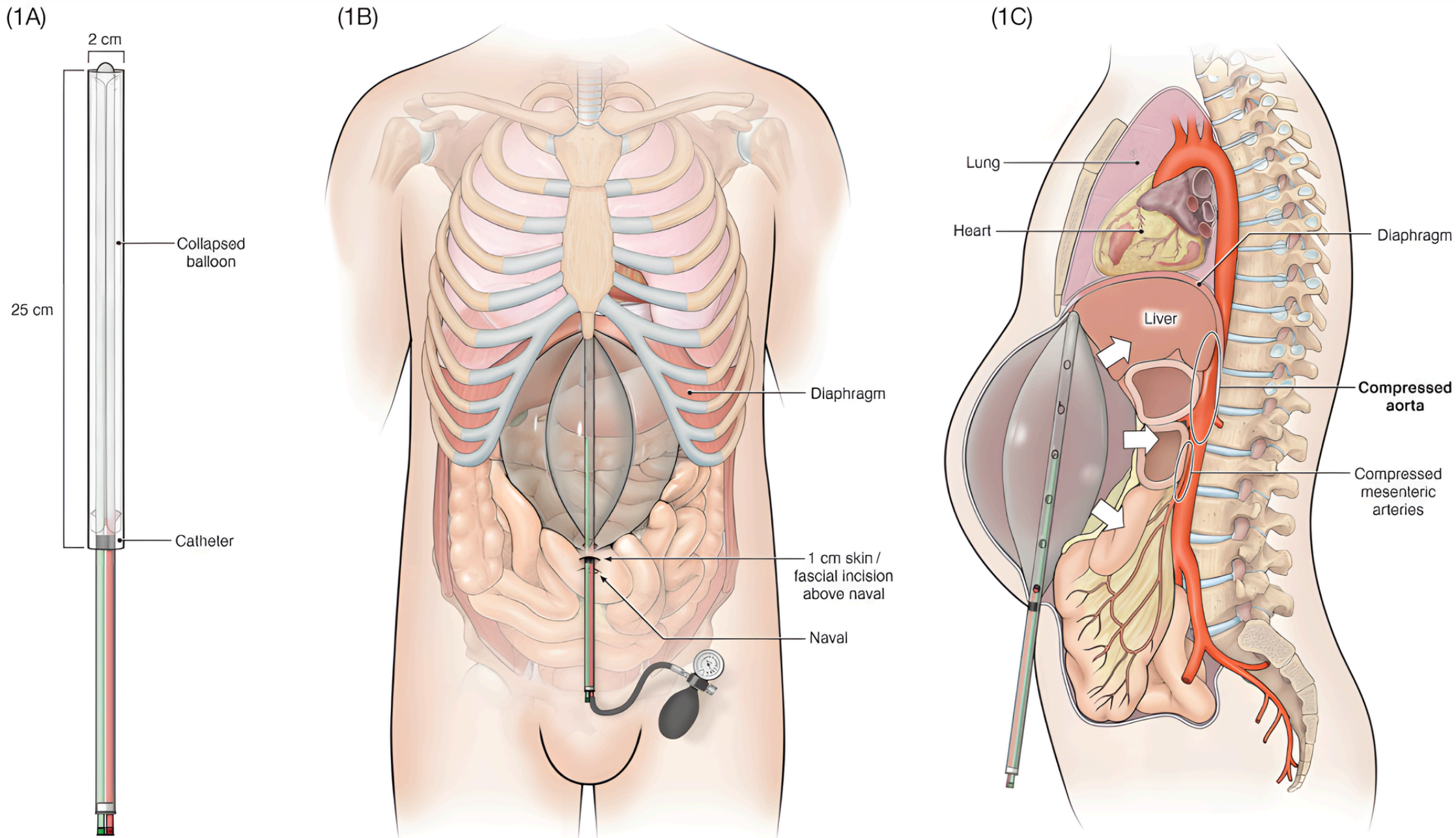
Contraindications for Pelvic Compression Device:

- Open Pelvic Fractures
- Perineal Lacerations
- Intraabdominal injuries requiring surgery
- Burns
- Severe associated pelvic soft tissue injuries

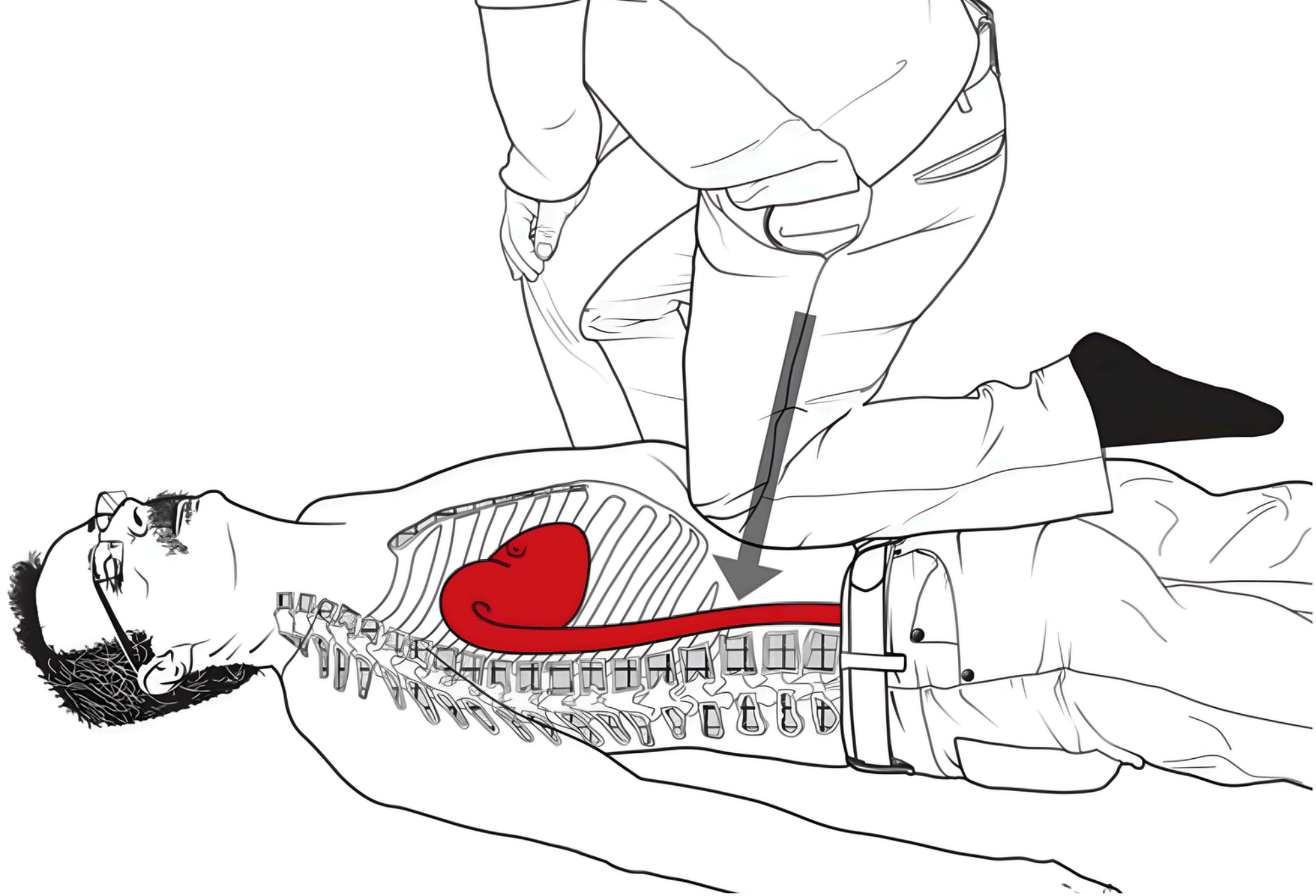
Level of Evidence: C-EO

MARCH

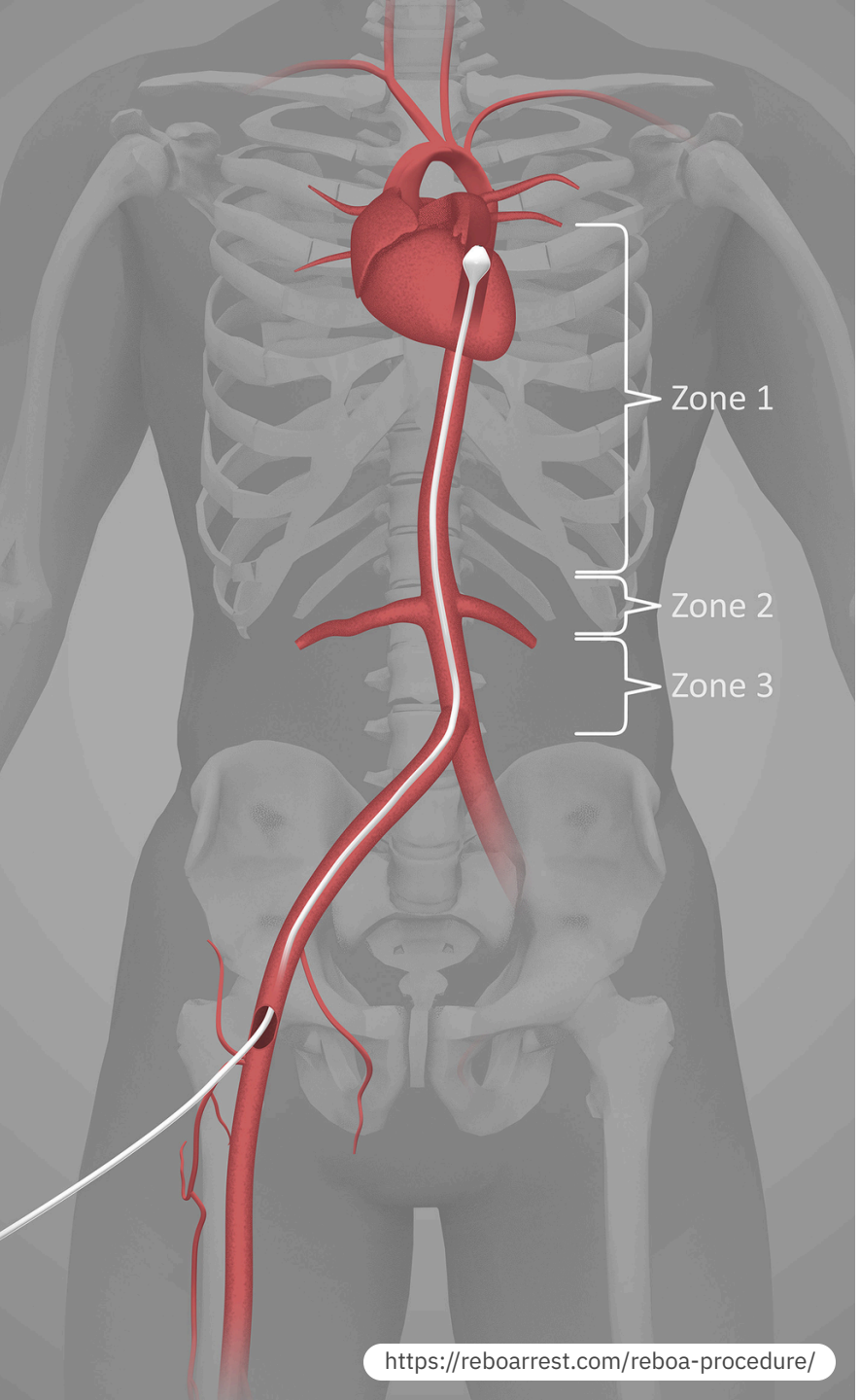
https://books.allogy.com/web/tenant/8/books/ebec0331-f93c-42cf-9fe5-c3ab2476fd93/



Leclerc S, Doshi S, Rezende-Neto JB. Perspectives on the management of non-compressible torso hemorrhage: a narrative review. *J Mil Veteran Fam Health*. 2022;8(s2):29-36. doi:10.3138/jmvfh-2021-0118



Paix BR, Tingey DJ, Copley G, et al. Prehospital external aortic compression for temporizing exsanguinating sub-diaphragmatic hemorrhage – a promising technique, but with challenges: four illustrative cases, including two survivors. *Prehospital Disaster Med*. 2020;35(1):115-118. doi:10.1017/S1049023X19005235

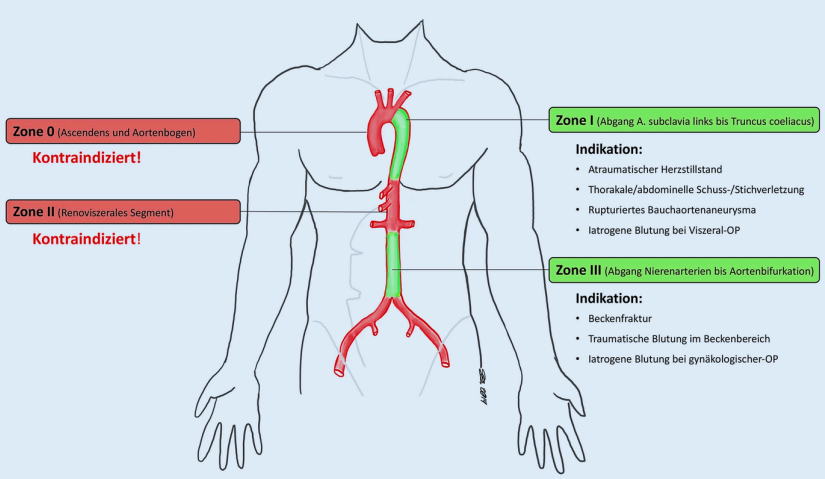


Resuscitative Endovascular Balloon Occlusion of the Aorta (REBOA)

Parsons HM, Wood A, Milne A. Resuscitative endovascular balloon occlusion of the aorta. BJA Educ. 2022;22(11):412-415. doi:10.1016/j.bjae.2022.07.004

- **Resuscitative endovascular balloon occlusion of the aorta (REBOA)**
  Reduces sub-diaphragmatic haemorrhage while increasing proximal perfusion in non-compressible truncal exsanguination
- **Catheter-borne balloon inflation in descending aorta**
  Aims to optimise coronary and cerebral perfusion, while minimising distal haemorrhage and ischaemia
- **Considered in blunt or penetrating sub-diaphragmatic injury with life-threatening bleeding or traumatic cardiac arrest**
  Requires an established, protocolised system for deployment
- **Lack of high-grade evidence demonstrating superiority over conventional manual aortic occlusion (AO) via thoracotomy**
  UK-REBOA trial is the first RCT to assess its efficacy
- **Used at an increasing number of international centres**
  Mostly deployed in major trauma, but also used in sub-diaphragmatic haemorrhage of other aetiologies and in non-traumatic cardiac arrest

Descending Aorta Zones and Balloon Inflation Strategies



- **Descending aorta zones**

  The descending aorta is divided into three zones: Zone 1 (origin of left subclavian artery to coeliac artery), Zone 2 (origin of coeliac artery to caudal renal artery), and Zone 3 (origin of caudal renal artery to aortic bifurcation).

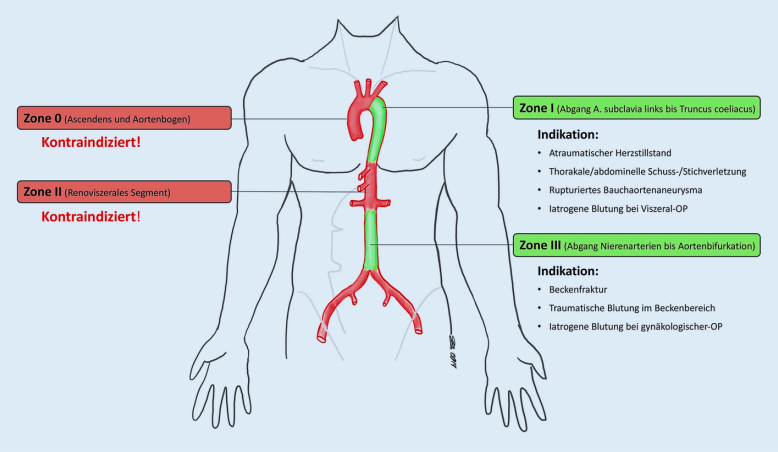
- **Zone 1 balloon inflation**

  Balloon inflation in Zone 1 reduces bleeding from sub-diaphragmatic sources and augments coronary perfusion pressure, thereby increasing the probability of return of spontaneous circulation after cardiac arrest.

Wortmann M, Engelhart M, Elias K, Popp E, Zerwes S, Hyhlik-Dürr A. „Resuscitative endovascular balloon occlusion of the aorta“ (REBOA). *Chirurg*. 2020;91(11):934-942. doi:10.1007/s00104-020-01180-0

Parsons HM, Wood A, Milne A. Resuscitative endovascular balloon occlusion of the aorta. *BJA Educ*. 2022;22(11):412-415. doi:10.1016/j.bjae.2022.07.004

Descending Aorta Zones and Balloon Inflation Strategies





Zone 3 balloon inflation

Zone 3 deployment reduces bleeding from pelvic or junctional sources (e.g. groin wounds).

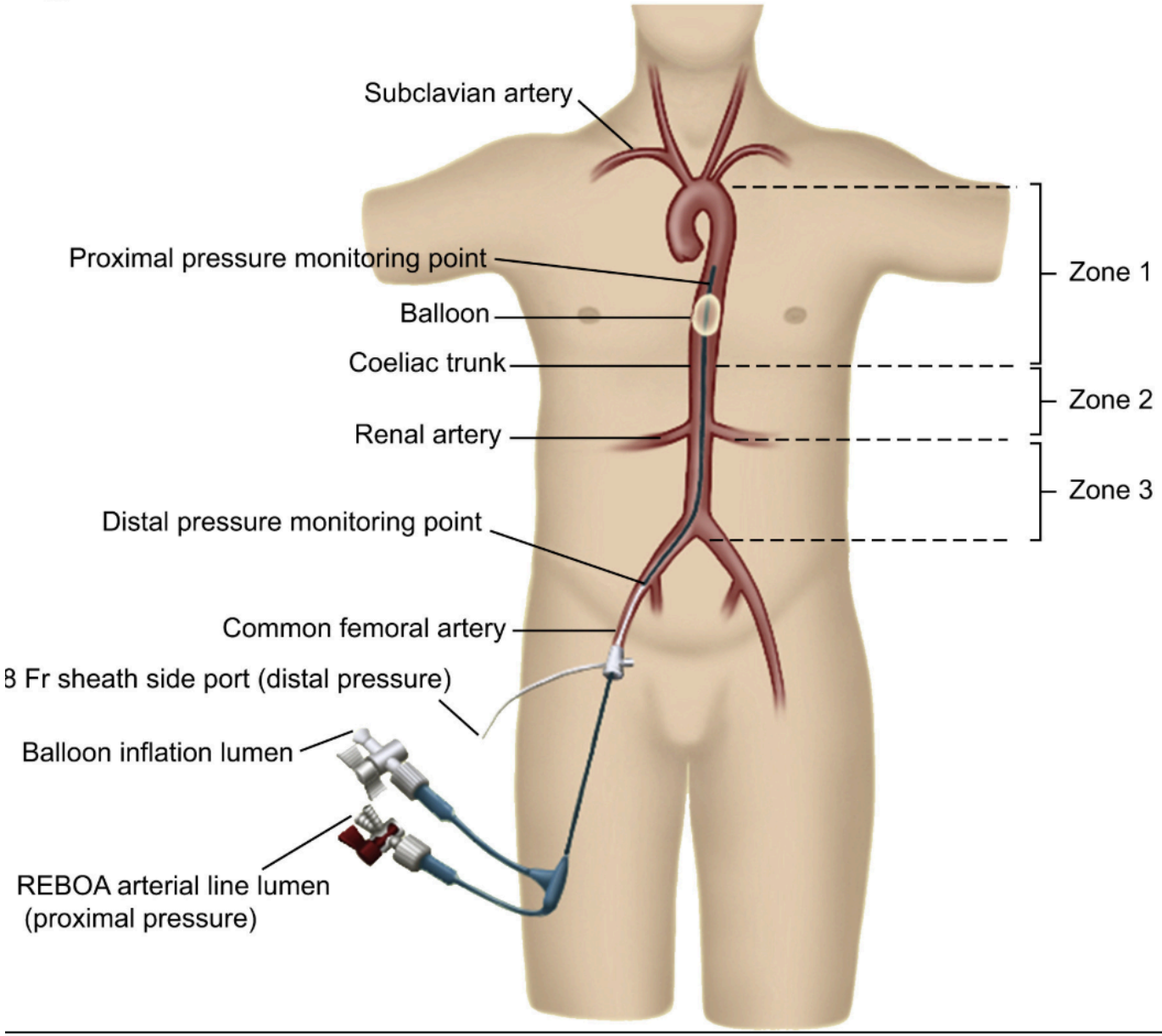


Avoid Zone 2

The balloon is never intentionally inflated in Zone 2.

Wortmann M, Engelhart M, Elias K, Popp E, Zerwes S, Hyhlik-Dürr A. „resuscitative endovascular balloon occlusion of the aorta“ (REBOA). *Chirurg*. 2020;91(11):934-942. doi:10.1007/s00104-020-01180-0

Parsons HM, Wood A, Milne A. Resuscitative endovascular balloon occlusion of the aorta. *BJA Educ*. 2022;22(11):412-415. doi:10.1016/j.bjae.2022.07.004



Parsons HM, Wood A, Milne A. Resuscitative endovascular balloon occlusion of the aorta. *BJA Educ*. 2022;22(11):412-415. doi:10.1016/j.bjae.2022.07.004

The descending aorta is divided into three zones:

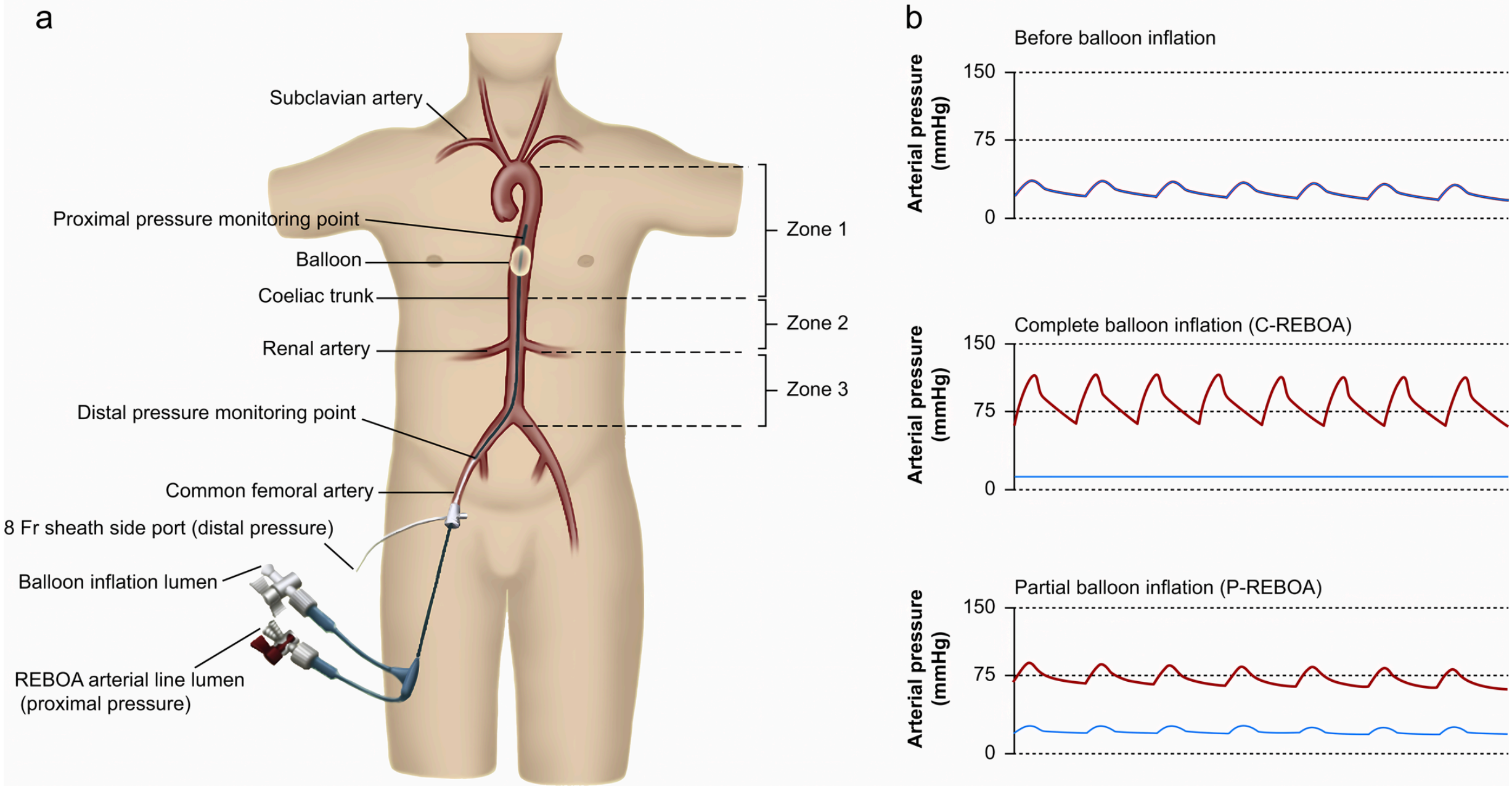
- Zone 1 (origin of left subclavian artery to coeliac artery),
- Zone 2 (origin of coeliac artery to caudal renal artery),
- Zone 3 (origin of caudal renal artery to aortic bifurcation).

Balloon inflation in Zone 1

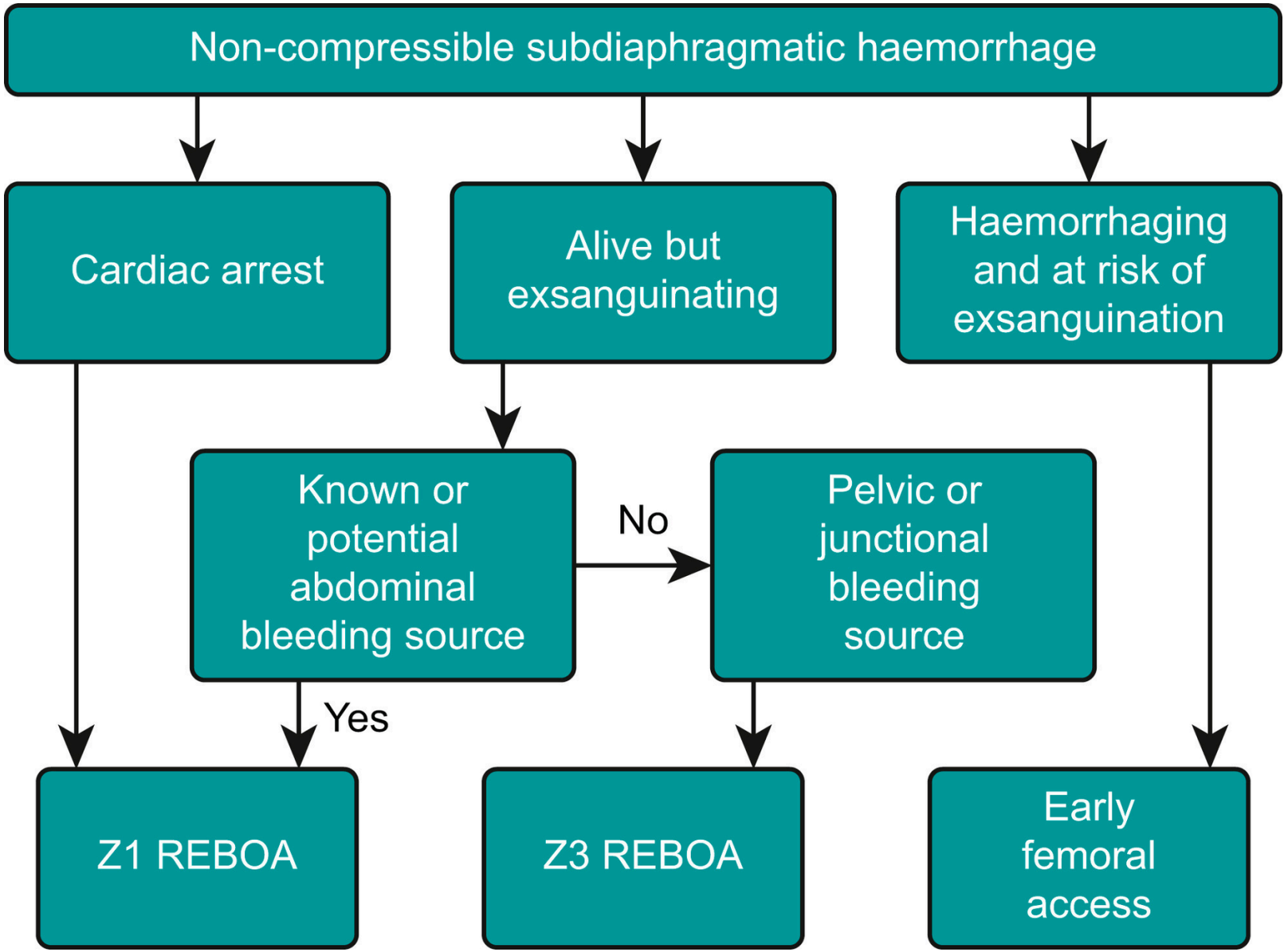
- reduces bleeding from sub-diaphragmatic sources
- augments coronary perfusion pressure, thereby increasing the probability of return of spontaneous circulation after cardiac arrest.

Zone 3 deployment reduces bleeding from pelvic or junctional sources (e.g. groin wounds).

The balloon is never intentionally inflated in Zone 2.



Parsons HM, Wood A, Milne A. Resuscitative endovascular balloon occlusion of the aorta. *BJA Educ*. 2022;22(11):412-415. doi:10.1016/j.bjae.2022.07.004



Parsons HM, Wood A, Milne A. Resuscitative endovascular balloon occlusion of the aorta. *BJA Educ*. 2022;22(11):412-415. doi:10.1016/j.bjae.2022.07.004

REBOA Deployment: Operator, Timing, and Technique

Parsons HM, Wood A, Milne A. Resuscitative endovascular balloon occlusion of the aorta. *BJA Educ*. 2022;22(11):412-415. doi:10.1016/j.bjae.2022.07.004

Operator Selection and Training

The primary REBOA operator is an experienced member of the trauma team who has undergone specific training to perform this procedure.

Therapeutic Window and Timing

The therapeutic window for REBOA is narrow. Too early deployment may expose patients who do not have the degree of haemorrhage to warrant it, while too late deployment delays AO, worsening blood loss and physiological derangement.

Early Femoral Access

To address the timing concerns, early femoral access is advocated in patients with sub-diaphragmatic haemorrhage who are deemed at risk of progression to exsanguination. The common femoral artery (CFA) is cannulated with a small (4 Fr) sheath using ultrasound guidance.

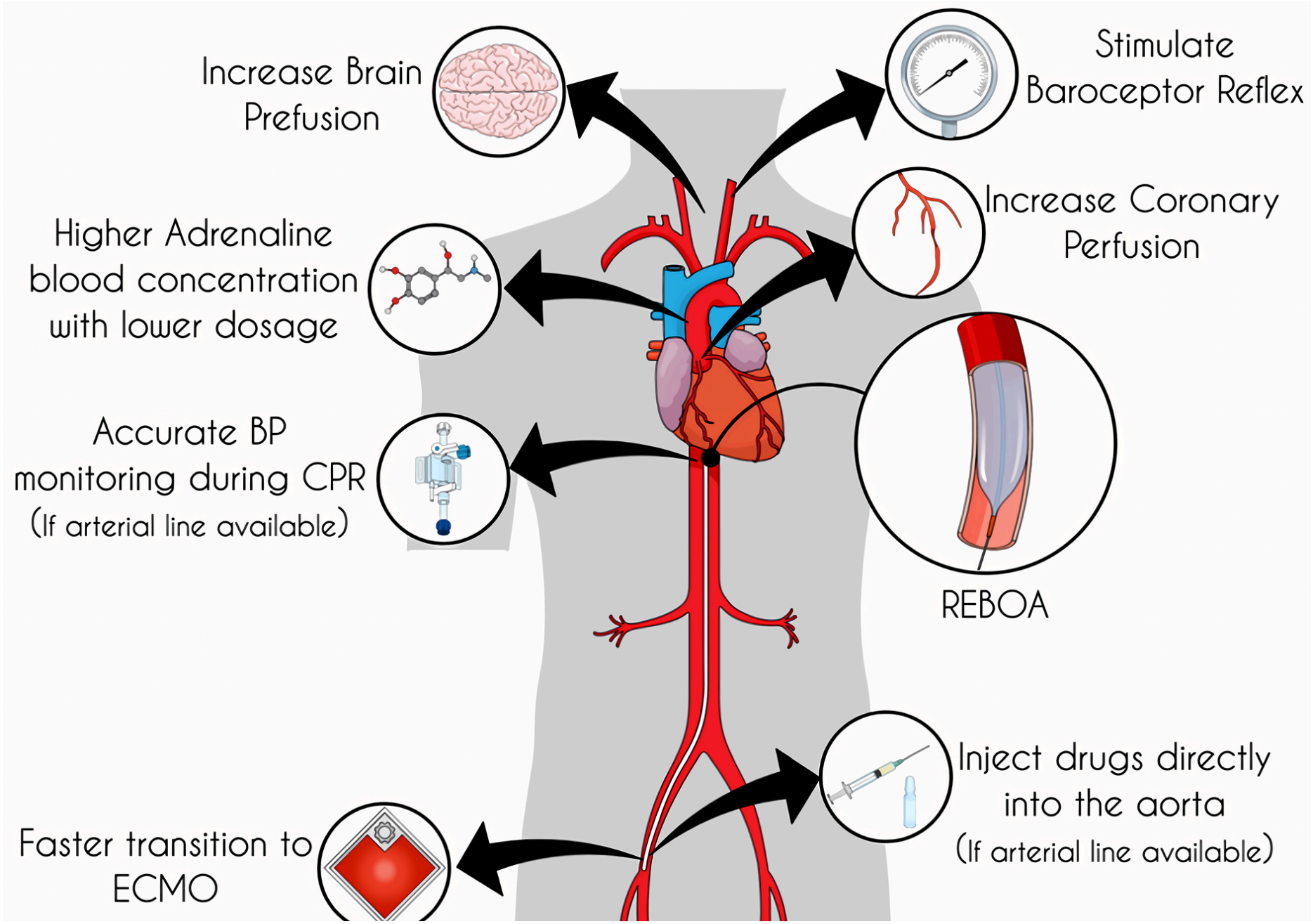
Complete REBOA (C-REBOA) Deployment

In patients with established exsanguination, an 8 Fr sheath is inserted into the CFA, either directly or upsized from a 4 Fr sheath. The 6 Fr ER-REBOA™ catheter is then inserted to the appropriate length, and the balloon is inflated with saline while observing the blood pressure (BP) response proximal and distal to the balloon. Inflation endpoint is improvement in proximal BP and distal waveform flatline.

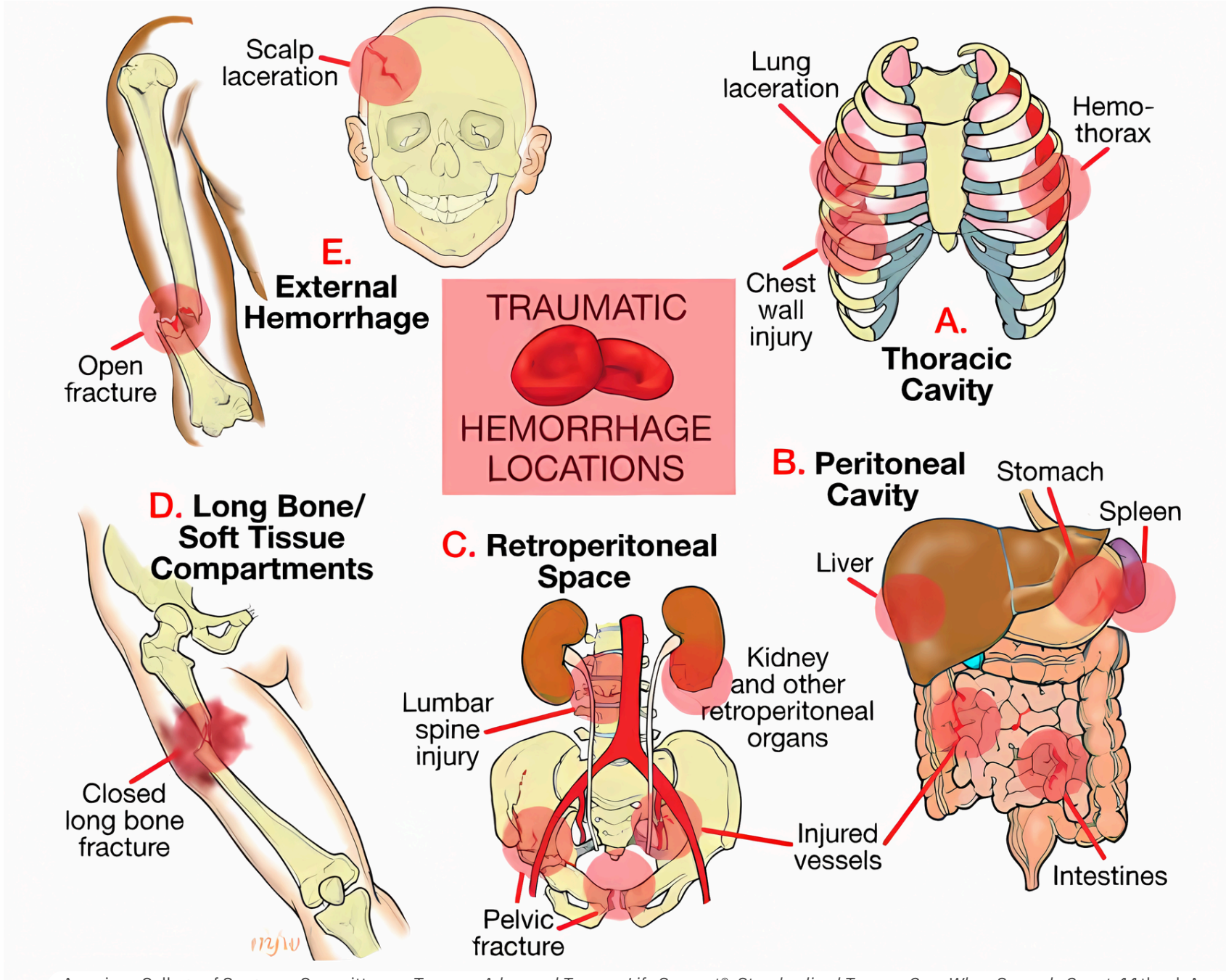
Partial REBOA (P-REBOA) Deployment

After inflation to C-REBOA (restricted to 15 or 20 min in Z1 or Z3, respectively), partial REBOA (P-REBOA) is targeted as soon as possible to minimise distal ischaemia. This procedure entails sequential balloon deflation until distal pulsatility is restored and distal MAP reaches 10 mmHg above that observed during C-REBOA.

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Mazzoli CA, Chiarini V, Coniglio C, et al. Resuscitative endovascular balloon occlusion of the aorta (REBOA) in non-traumatic cardiac arrest: a narrative review of known and potential physiological effects. *J Clin Med*. 2022;11(3):742. doi:10.3390/jcm11030742



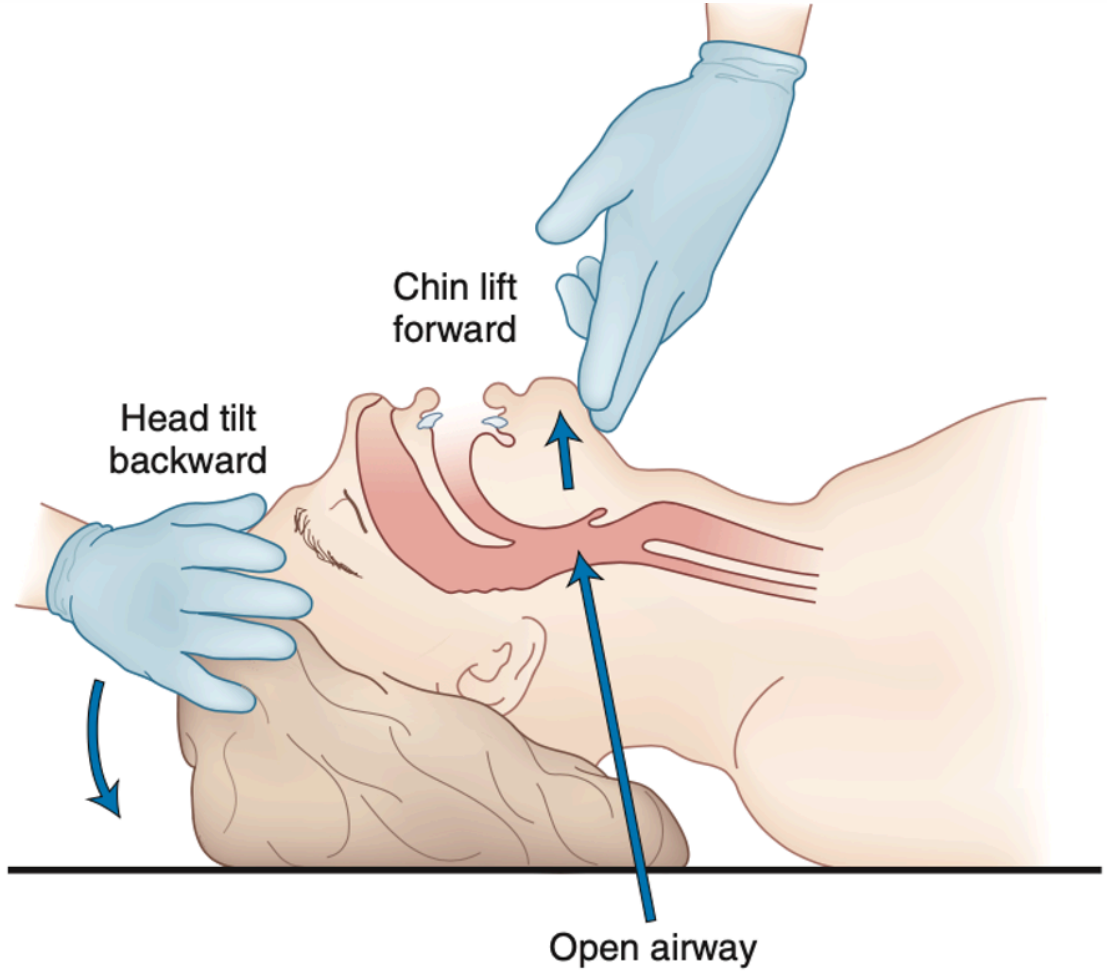
American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

Airway

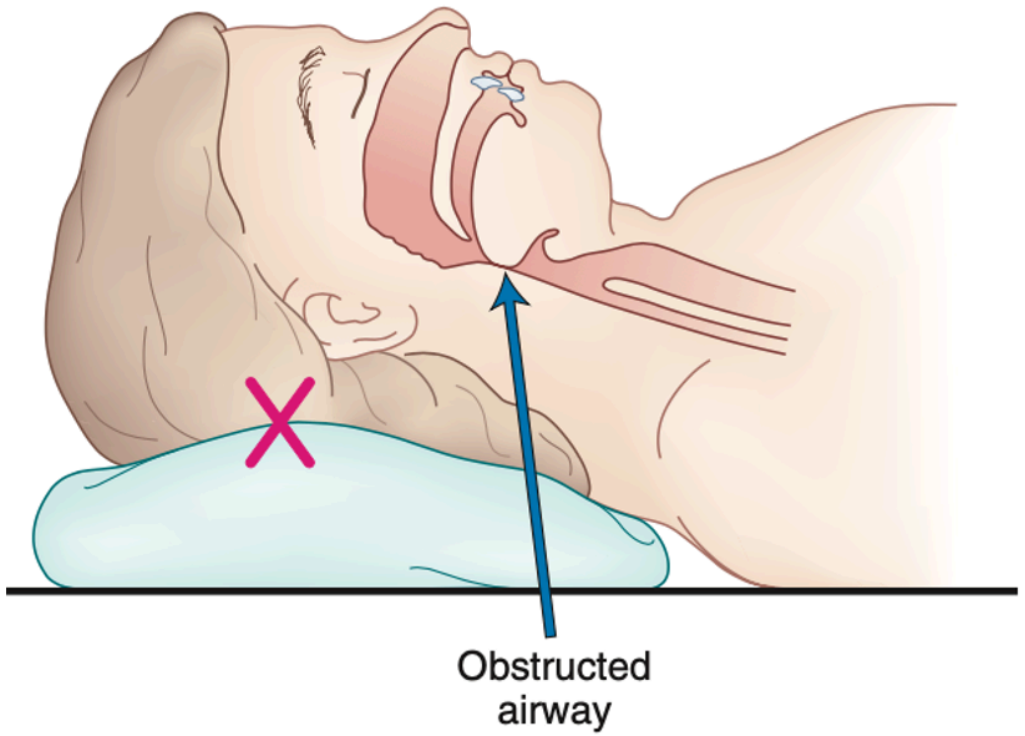
Airway	Partial or complete airway obstruction Anticipated or impending airway obstruction (e.g., direct trauma, hemorrhage, or burns)
Breathing	Severe chest injury with respiratory failure Significant hypoxia (SpO_2 <90%) despite high-flow oxygen, after exclusion of pneumothorax Hypercapnia from hypoventilation, without an immediately reversible cause (e.g. due to neurological or respiratory failure)
Circulation	Altered mental status due to shock
Disability	Severe TBI with GCS score 8 or lower
Exposure	High percentage body surface area burns

SpO_2: Pulse oximety. TBI: Traumatic brain injury. GCS: Glasgow coma scale

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

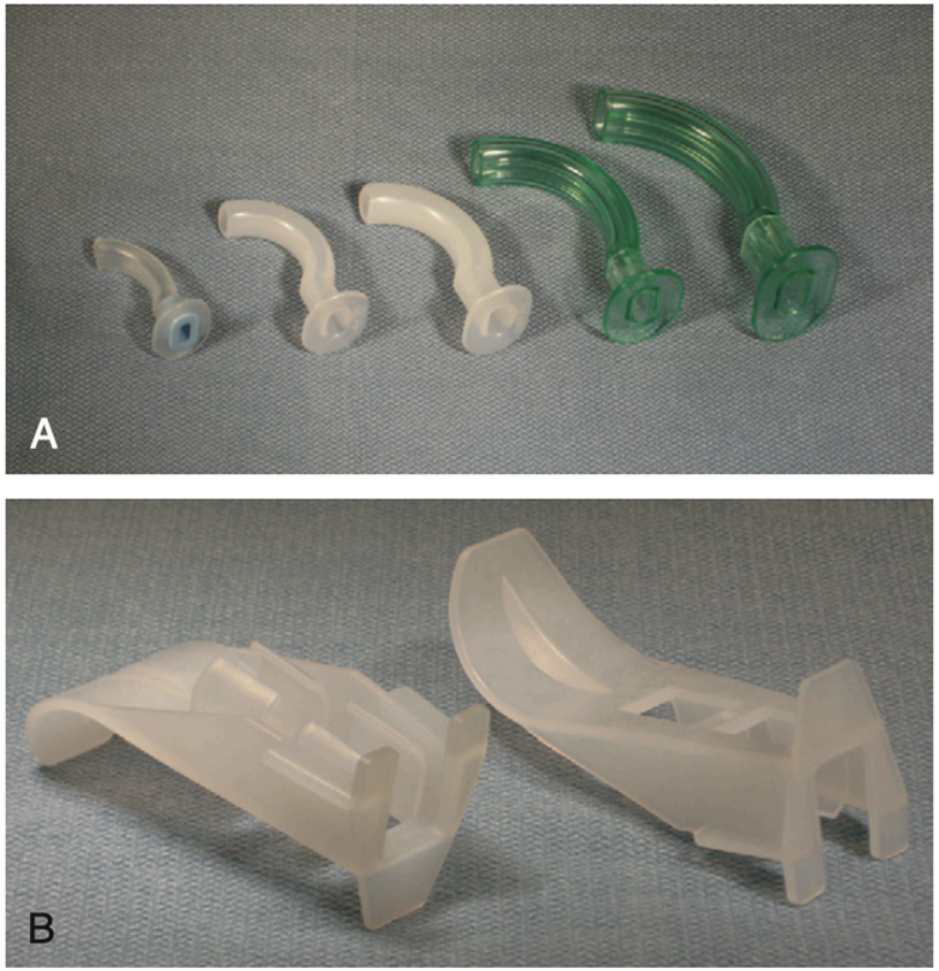


B **Head tilt and chin lift to obtain extended position**



Tongue in apposition to posterior pharyngeal wall

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.



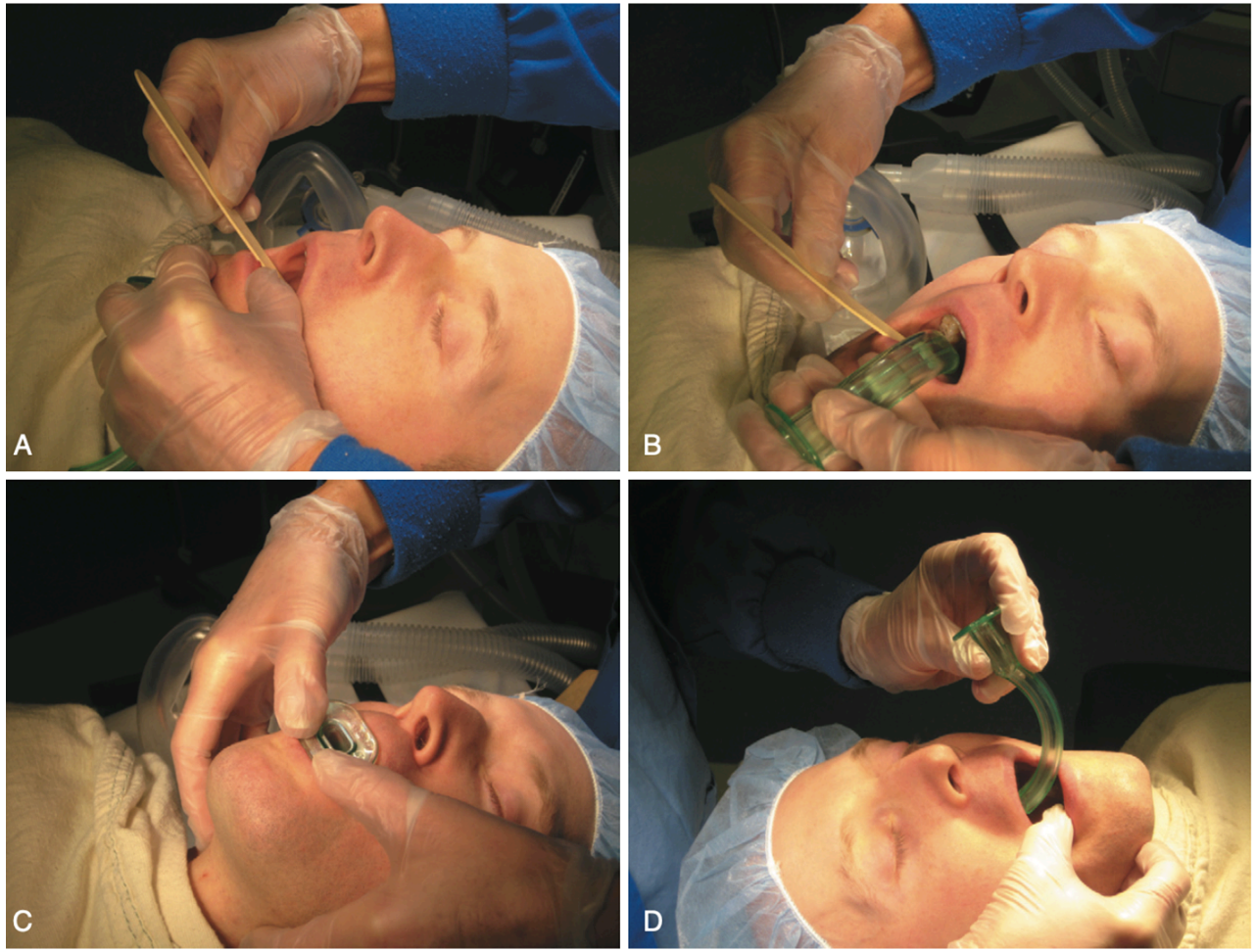


Figure 15-7 Oropharyngeal airways. **A,** Guedel Airways in sizes from neonatal to large adult. **B,** The Ovassapian Airway has a large anterior flange to control the tongue. The airway is open posteriorly (including no posterior flange) so that an endotracheal tube can be inserted with a flexible fiberoptic scope and the assembly later separated.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

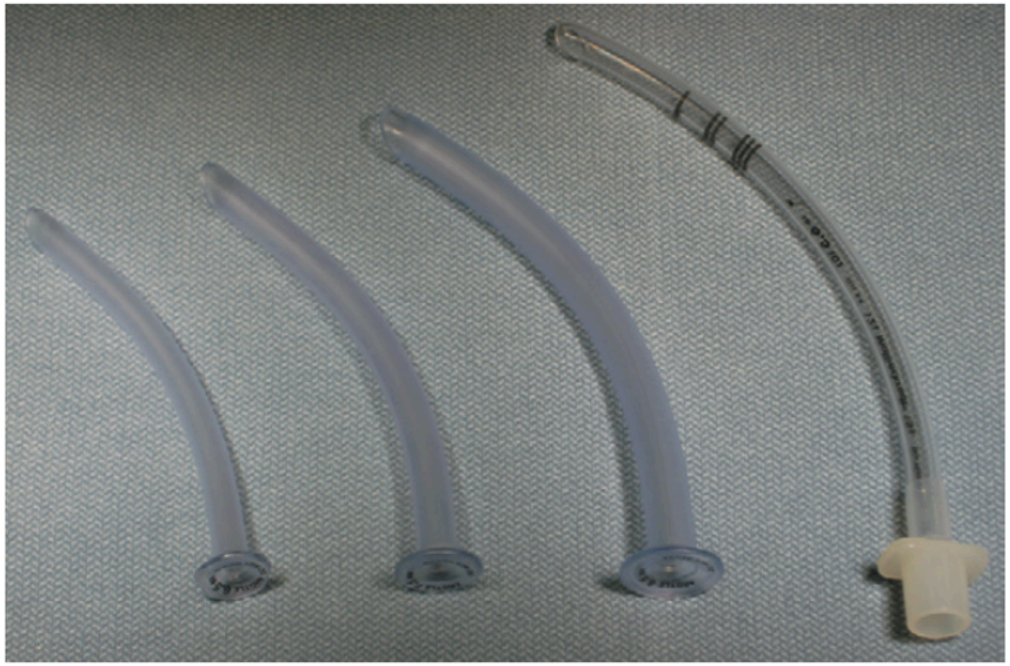


Figure 15-9 Nasopharyngeal airways. A flange prevents the outside end from passing beyond the nares, controlling the depth of insertion. Alternatively, an endotracheal tube may be cut down to provide a longer airway, with its 15-mm adapter reinserted in the cut end.



Figure 15-10 Insertion of a nasopharyngeal airway. The airway is oriented with its concave side toward the hard palate and inserted straight posteriorly. Gripping the airway near the top allows the tube to bend if there is resistance to passage. If it is gripped too close to the naris, the clinician can generate sufficient force to shear off a turbinate.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

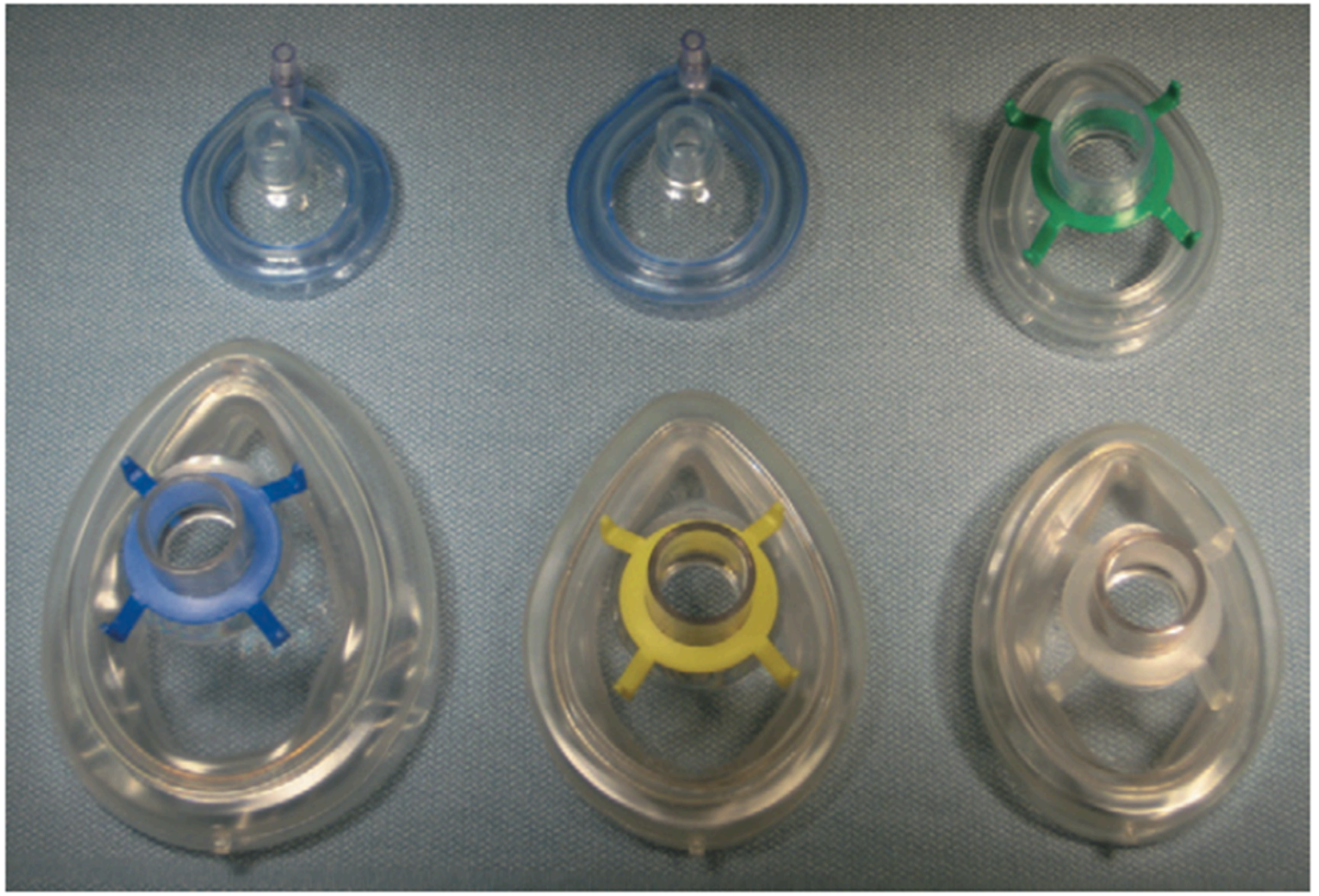


Figure 15-11 Assorted sizes of disposable, transparent face masks. The smallest masks have a 15-mm male adapter, and the larger sizes have a 22-mm female adapter to allow them to be connected to a standard breathing circuit or resuscitator bag.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

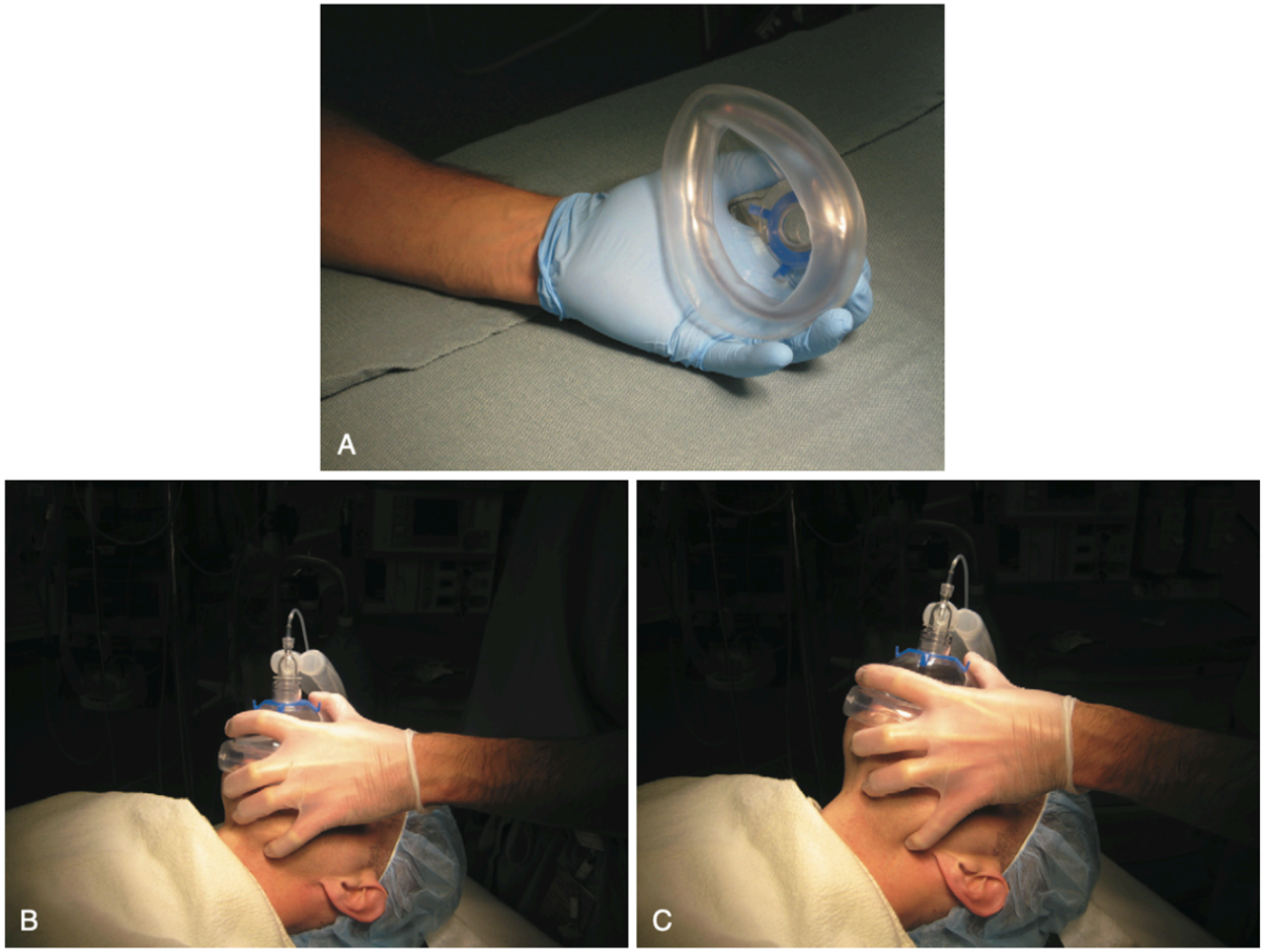


Figure 15-13 Suggested techniques for holding and supporting a face mask. **A,** In the proper hand grip of the face mask, the thumb and index finger encircle the collar while the hypothenar eminence extends below the left side of the mask. **B,** In the side view of the standard one-handed application of the face mask, the thumb and first finger (or first two) encircle the collar of the mask while the remaining fingers pull the mandible up into the mask while gently extending the head. **C,** During the one-handed mask grip with concurrent jaw thrust, notice how the little finger is located at the angle of the jaw, pulling backward and upward to maintain the jaw thrust (subluxation). Because of the increased span of the hand, only the first finger is on the mask while the middle and ring fingers pull the mandible up into the mask and extend the head.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

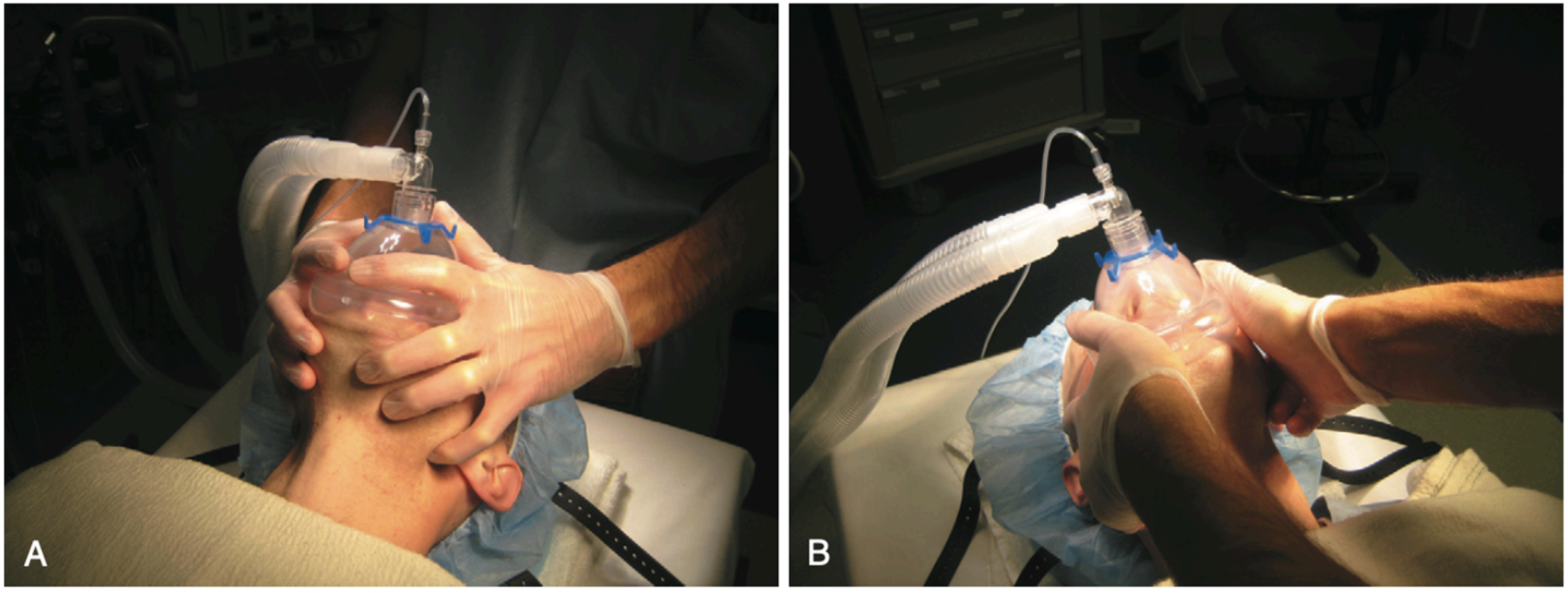


Figure 15-15 Two-hand control of a face mask. In both scenarios, a second provider must ventilate the patient. **A,** In the view of two-hand control of a face mask from above the patient, notice how the lower fingers on both hands apply a jaw thrust while the thumbs seal the mask to the face. **B,** In the view of two-hand control of a face mask from the side of the patient, the person ventilating the patient has improved access to the head as the airway is maintained from the patient's side. This arrangement is beneficial if the ventilating provider is preparing to perform laryngoscopy.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

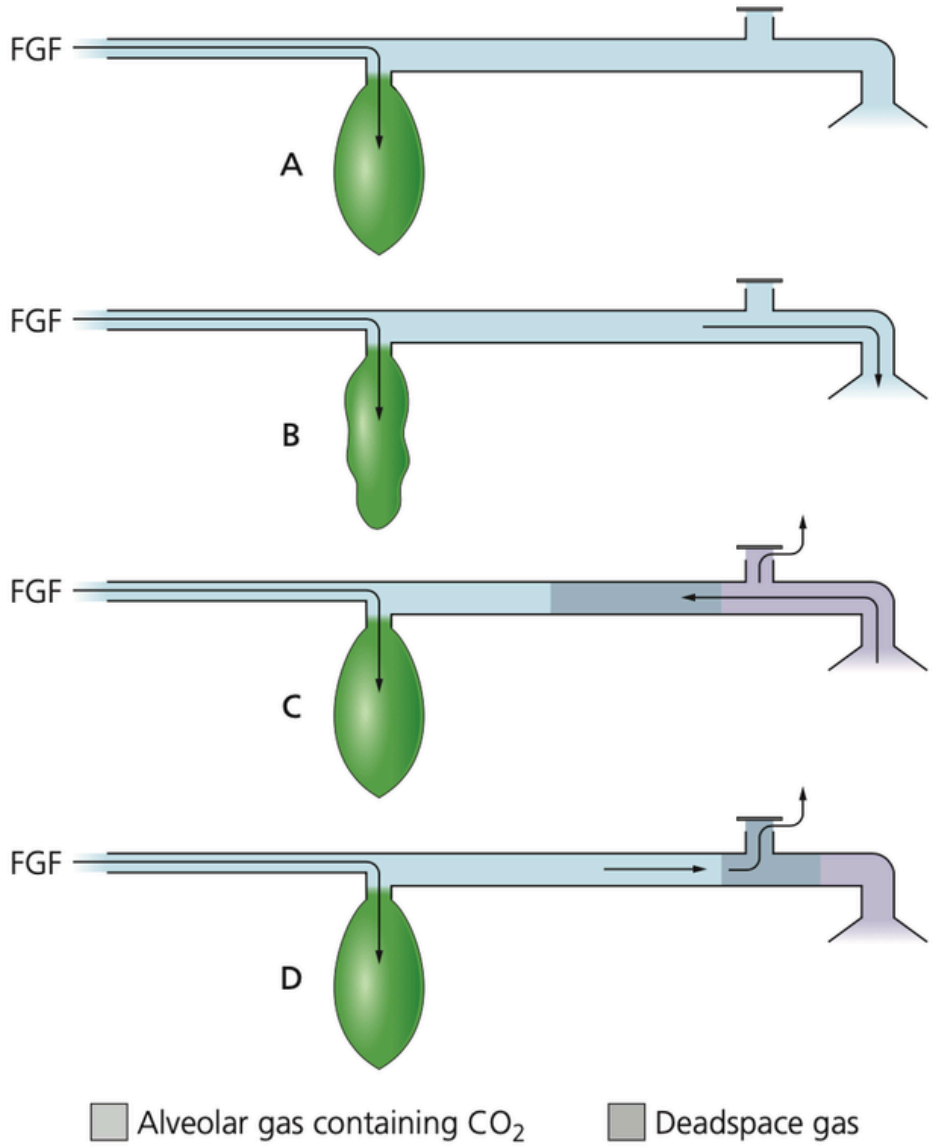


Figure 15-14 Adult and pediatric sizes of air-mask-bag unit (AMBU). The AMBU is a portable, self-inflating, easy-to-use system for the delivery of positive-pressure ventilation. It can be used with a face mask, laryngeal mask airway, or endotracheal tube.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.



Fig. 4.8 Intersurgical adult Mapleson C system.



Al-Shaikh B. *Essentials of Equipment in Anaesthesia, Critical Care, and Peri-Operative Medicine e-Book*. 5th ed. Elsevier; 2017.

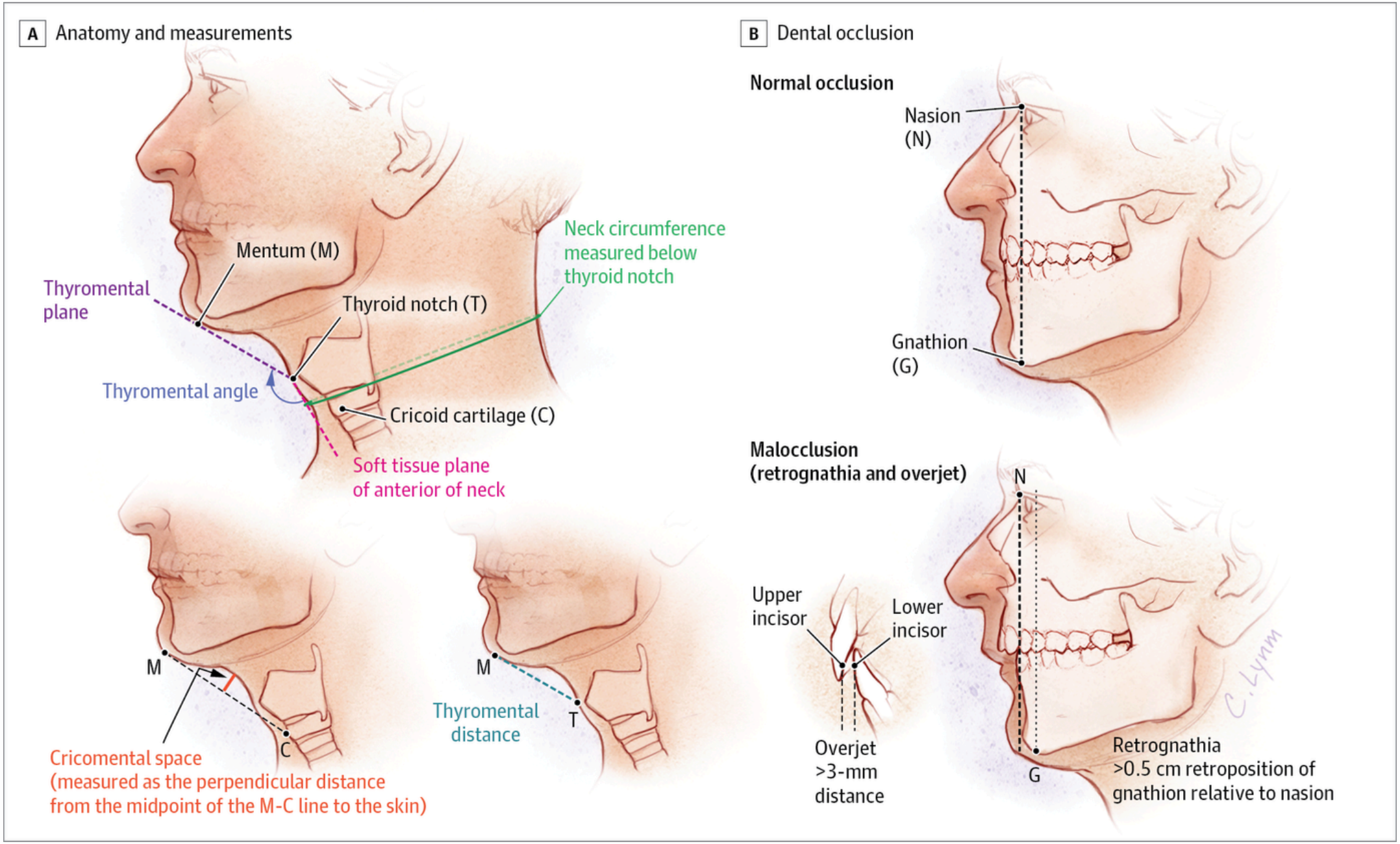
Airway assessment	Points
Look externally:	
Facial trauma	1
Large incisors	1
Beard or moustache	1
Large tongue	1
Evaluate the 3-3-2:	
Mouth opening <3 finger breadths	1
Hyomental distance <3 finger breadths	1
Thyrohyoid distance <2 finger breadths	1
Obstruction:	
Presence of an obstructed airway	1
Neck mobility:	
Presence of poor neck mobility	1
Total	**/9**



Athanassoglou V, Rogers A, Hofmeyr R. In-hospital management of the airway in trauma. *BJA Educ*. 2024;24(7):238-244. doi:10.1016/j.bjae.2024.03.004



Myers KA, Mrkobrada M, Simel DL. Does this patient have obstructive sleep apnea?: the rational clinical examination systematic review. *JAMA*. 2013;310(7):731-741. doi:10.1001/jama.2013.276185



Myers KA, Mrkobrada M, Simel DL. Does this patient have obstructive sleep apnea?: the rational clinical examination systematic review. *JAMA*. 2013;310(7):731-741. doi:10.1001/jama.2013.276185

Airway Management in Facial Fractures

- **Complex Airway Management**

  Facial fractures often present with active bleeding, airway obstruction, and difficulty with bag-mask ventilation, oxygenation, and intubation

- **Le Fort Fracture Classification**

  Describes three distinct patterns of maxillary fractures with varying levels of facial skeleton and cranium dissociation

- **Cervical Spine Precautions**

  Maintain neutral alignment to prevent further injury, use blocks, tape, and MILS during interventions

- **Airway Maintenance Strategies**

  Use airway adjuncts, allow upright positioning, manually displace unstable fracture segments, use facemasks to splint unstable structures

- **Intubation Considerations**

  Rapid sequence intubation may be high-risk, awake intubation or advanced techniques like retromolar, submental, or retrograde intubation may be safer options

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Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

Head and neck position and the axes of the head and neck upper airway

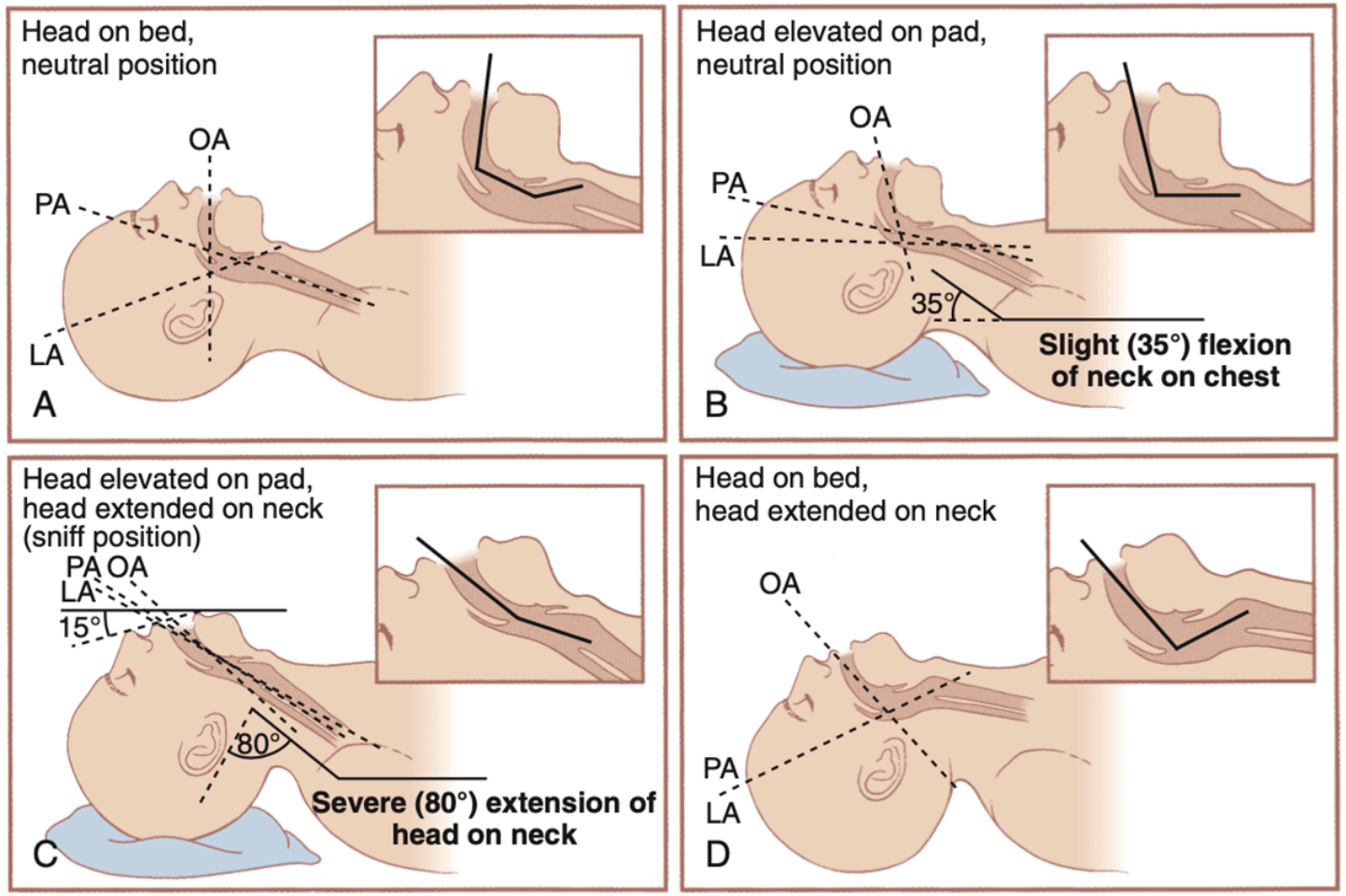


Figure 17-1 Schematic diagrams show the alignment of the oral axis *(OA),* pharyngeal axis *(PA),* and laryngeal axis *(LA)* in four different head positions. Each head position is accompanied by an *inset* that magnifies the upper airway (oral cavity, pharynx, and larynx) and superimposes *(bent bold line)* the continuity of these three axes within the upper airway. **A,** The head is in the neutral position with a marked degree of nonalignment of the LA, PA, and OA. **B,** The head is resting on a large pad that flexes the neck on the chest and aligns the LA with the PA. **C,** The head is resting on a pad (which flexes the neck on the chest). Concomitant extension of the head on the neck brings all three axes into alignment (sniffing position). **D,** Extension of the head on the neck without concomitant elevation of the head on a pad, which results in nonalignment of the PA and LA with the OA. (From Benumof JL, editor: *Airway management: principles and practice,* St. Louis, 1996, Mosby, p 263.)

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

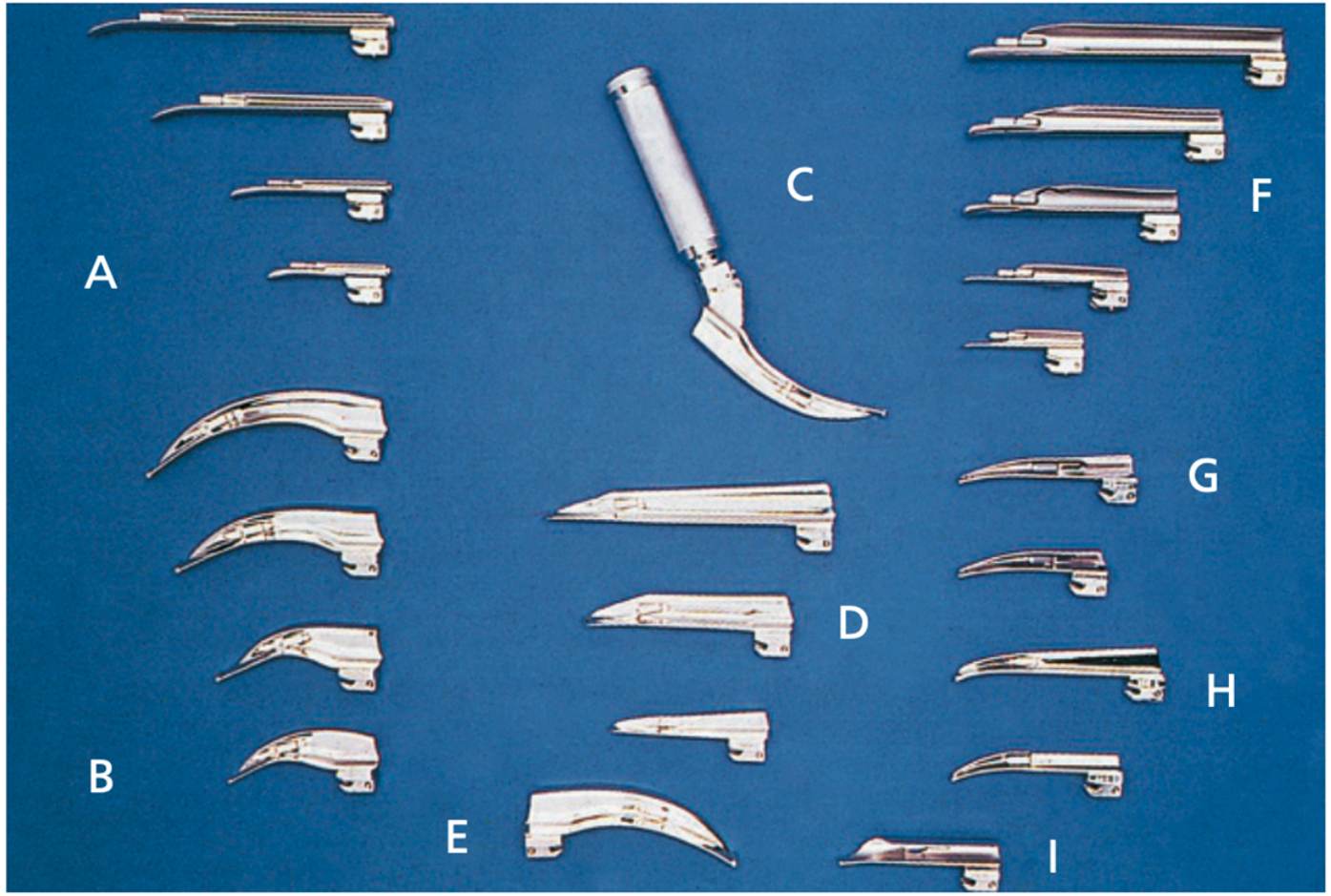
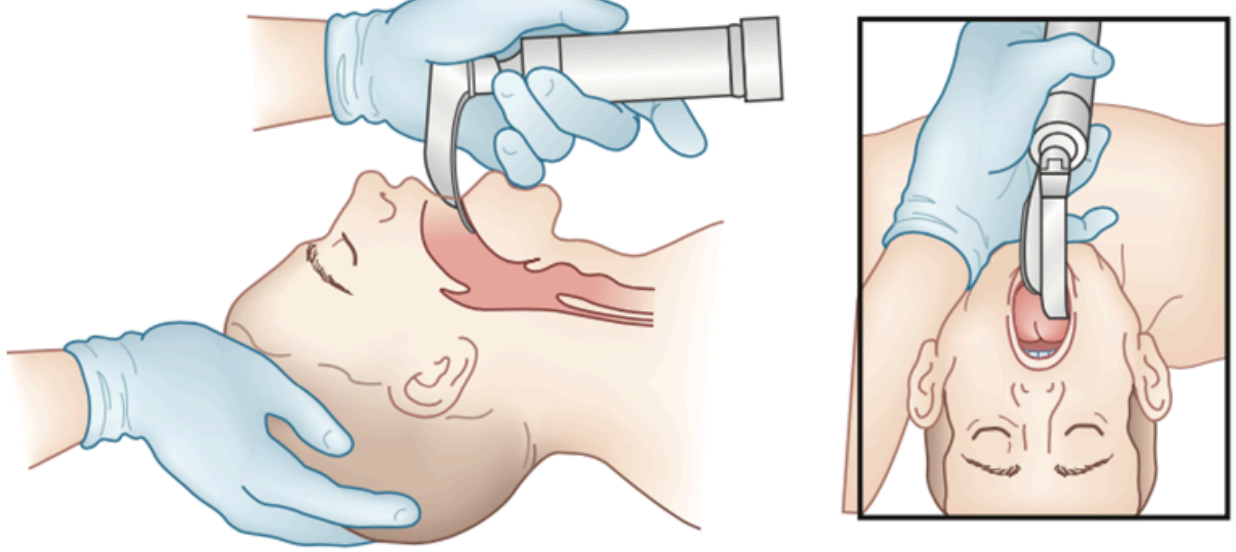
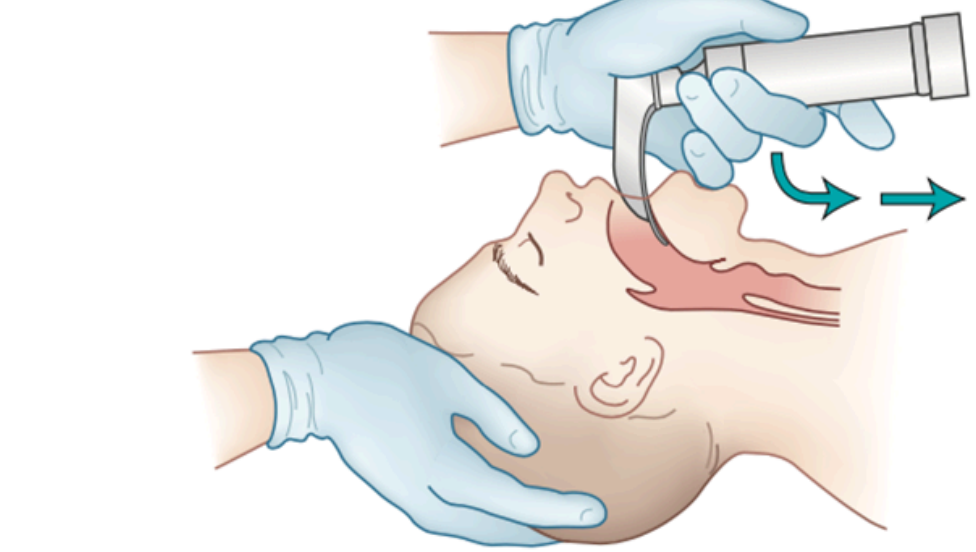


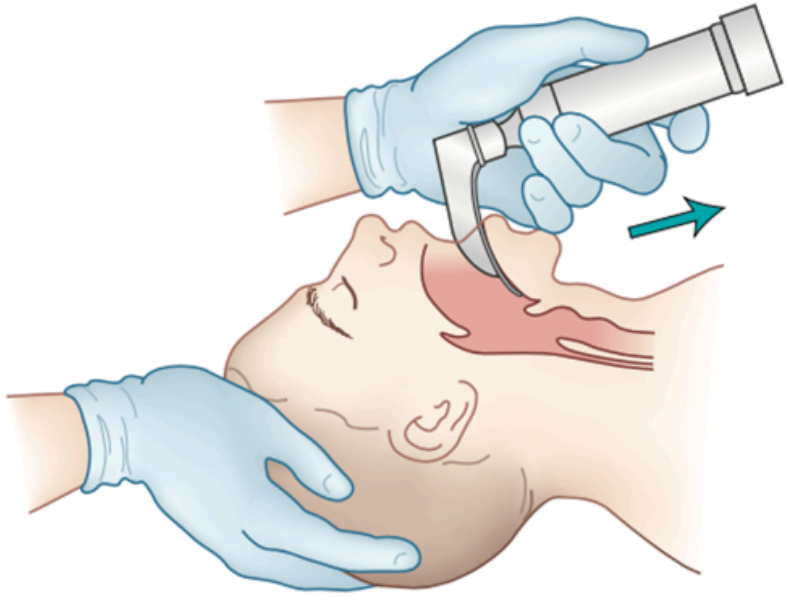
Fig. 7.2 A wide range of laryngoscope blades. (A) Miller blades (large, adult, infant, premature); (B) Macintosh blades (large, adult, child, baby); (C) Macintosh polio blade; (D) Soper blades (adult, child, baby); (E) left-handed Macintosh blade; (F) Wisconsin blades (large, adult, child, baby, neonate); (G) Robertshaw blades (infant, neonatal); (H) Seward blades (child, baby); (I) Oxford infant blade.

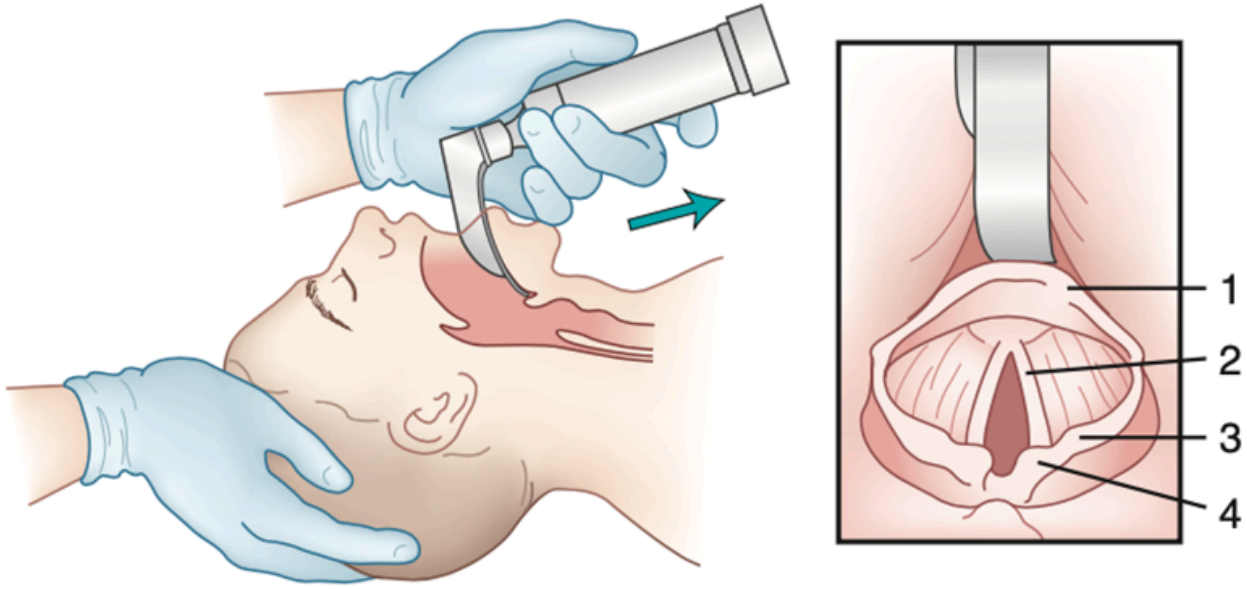


A Insert the laryngoscope blade into the right side of the mouth



B Advance the laryngoscope blade toward the midline of the base of the tongue by rotating wrist





Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.


YouTube

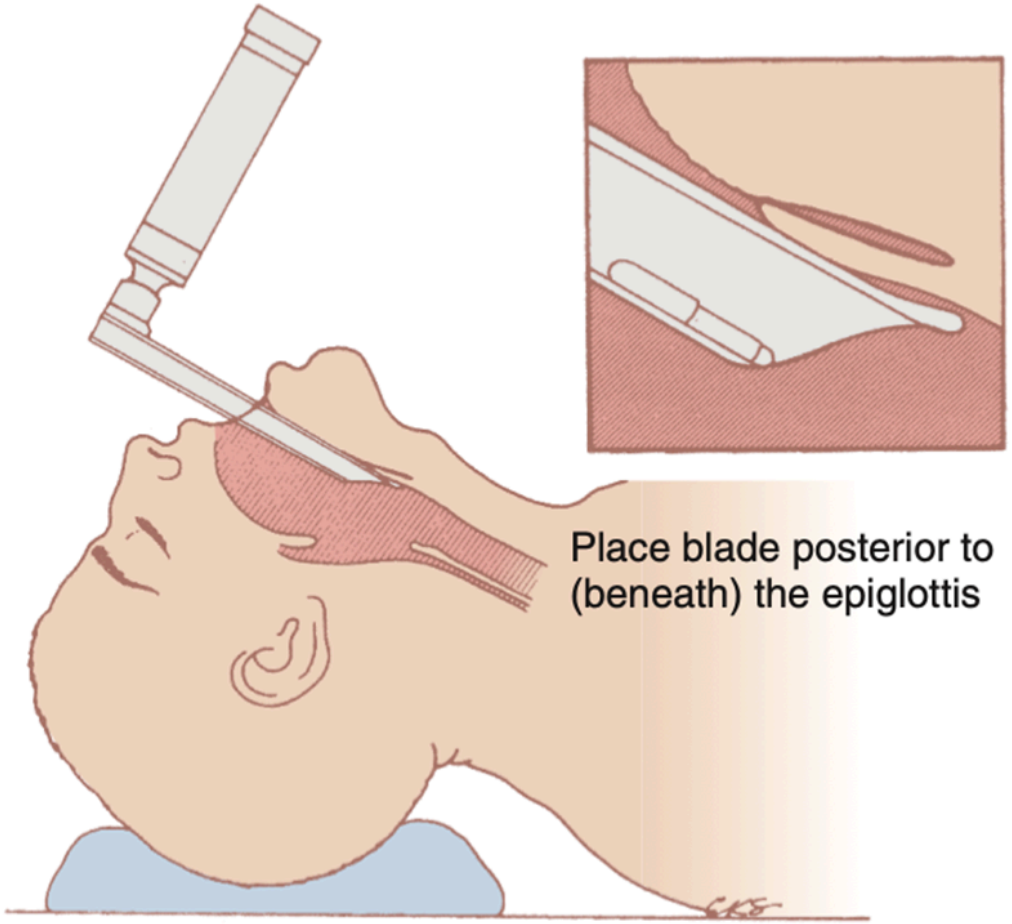


Figure 17-6 Conventional laryngoscopy with a straight blade. A straight laryngoscope blade (Miller blade) should be passed underneath the laryngeal surface of the epiglottis. The handle of the laryngoscope then should be elevated at a 45-degree angle, similar to the lifting that takes place with the use of a curved laryngoscope blade. (From Benumof JL, editor: *Airway management: Principles and practice,* St. Louis, 1996, Mosby, p 268.)

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

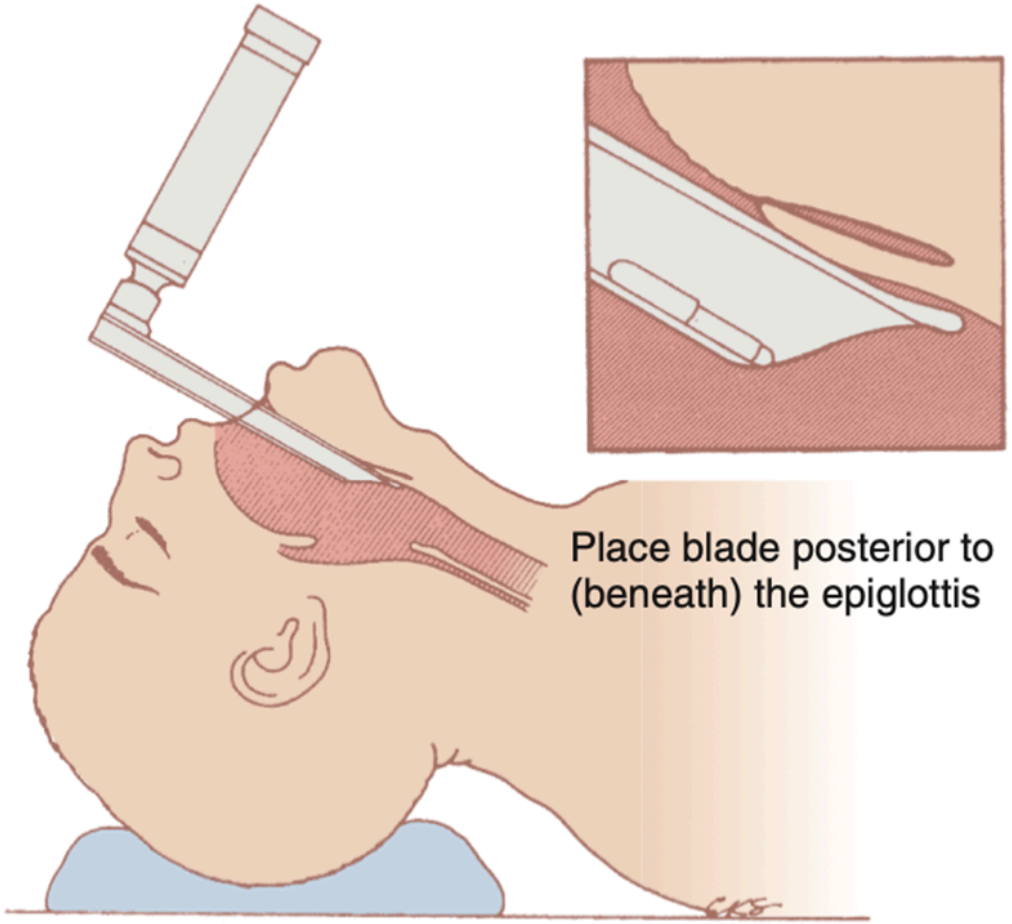


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Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

Guiding a nasotracheal tube into the larynx using a Magill forceps

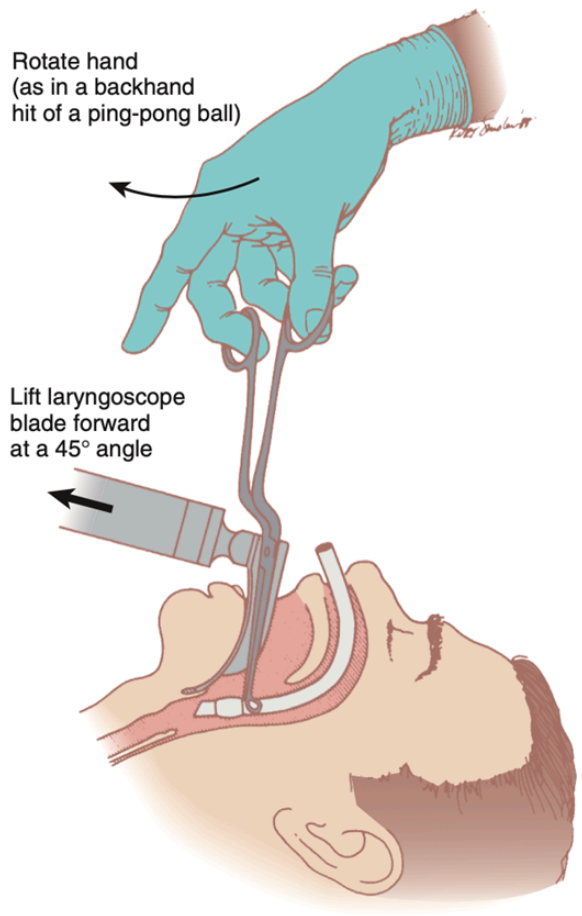


Figure 17-12 A nasotracheal tube can be guided under direct vision (laryngoscopic control) through the laryngeal aperture with a Magill forceps by rotating the hand as when using a backhand motion to hit a ping pong ball. The Magill forceps should grab the nasotracheal tube proximal to the cuff of the ETT. (From Benumof JL, editor: *Airway management: Principles and practice,* St. Louis, 1996, Mosby, p 275.)

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

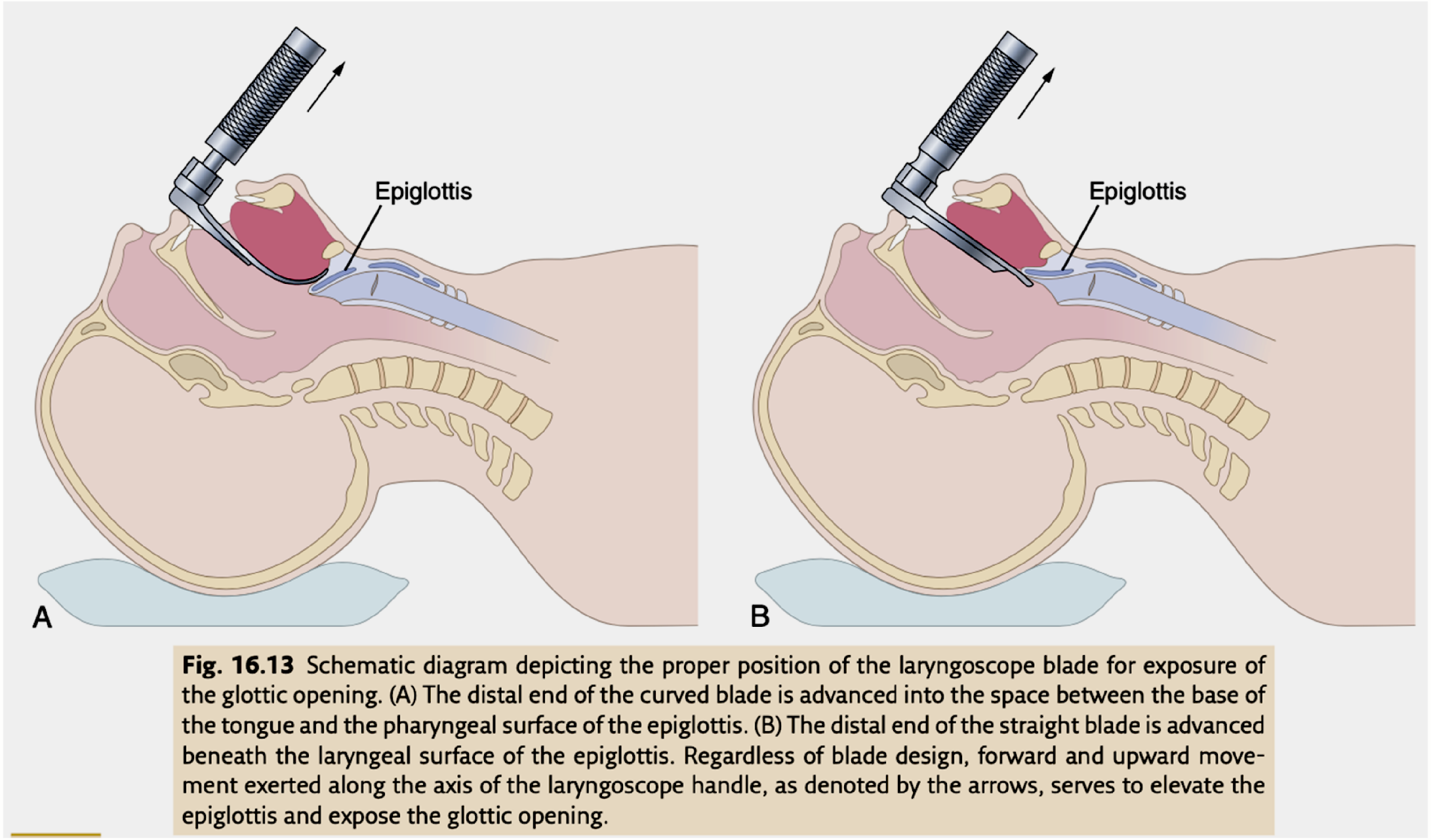


Fig. 16.13 Schematic diagram depicting the proper position of the laryngoscope blade for exposure of the glottic opening. (A) The distal end of the curved blade is advanced into the space between the base of the tongue and the pharyngeal surface of the epiglottis. (B) The distal end of the straight blade is advanced beneath the laryngeal surface of the epiglottis. Regardless of blade design, forward and upward movement exerted along the axis of the laryngoscope handle, as denoted by the arrows, serves to elevate the epiglottis and expose the glottic opening.

Pardo MC, Miller RD. *Basics of Anesthesia*. 7. edition. Elsevier; 2018.


STORZ
8401 ZX
C-MAC



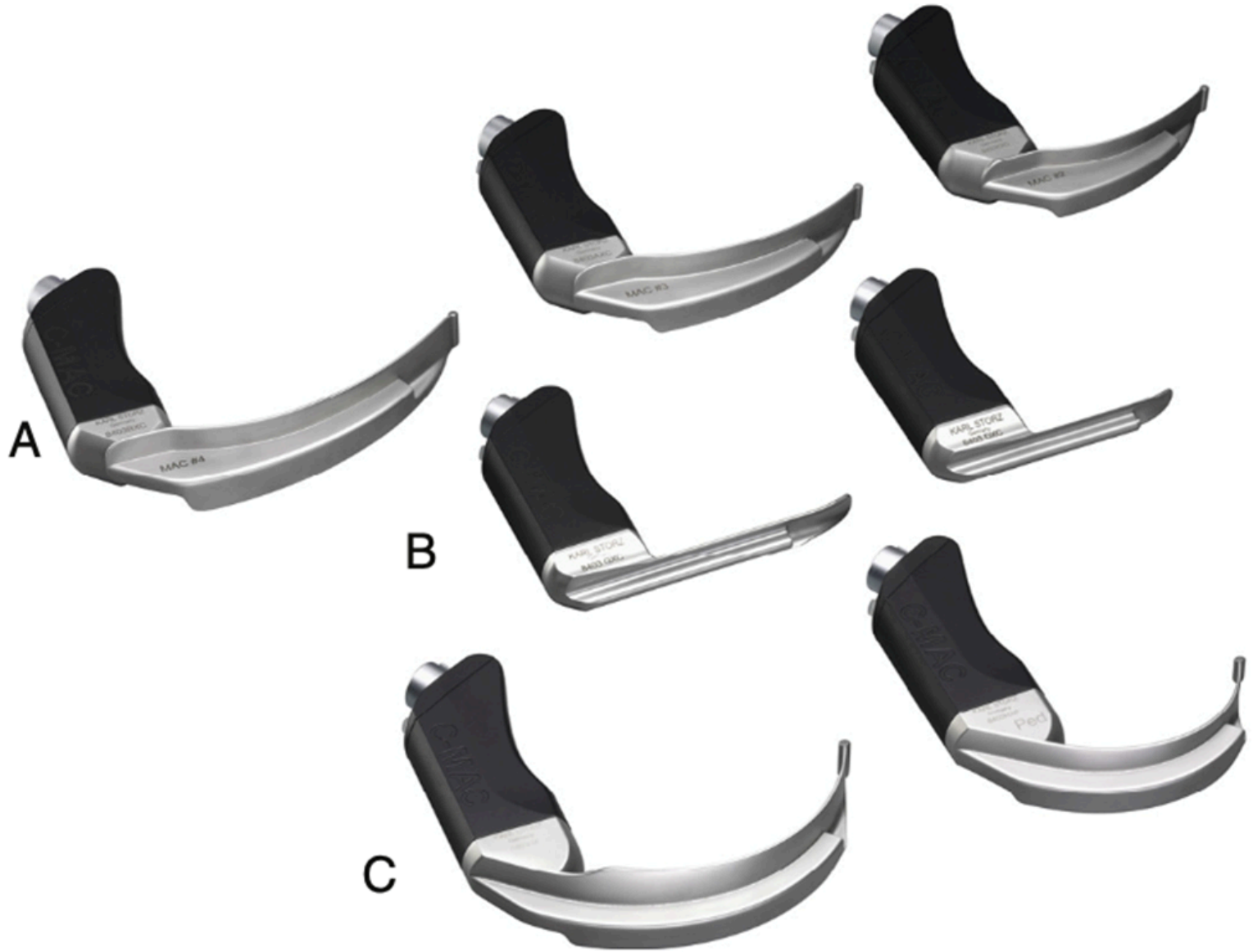


Fig. 16.16 Comparison of the different C-MAC blade types. (A) Macintosh style blade, (B) Miller style blade, and (C) D-blade. (Images courtesy of KARL STORZ Endoscopy, El Segundo, CA.)

Pardo MC, Miller RD. *Basics of Anesthesia*. 7. edition. Elsevier; 2018.

Box 16.2 Complications of Endotracheal Intubation

During Direct Laryngoscopy and Endotracheal Intubation

Dental and oral soft tissue trauma
Systemic hypertension and tachycardia
Cardiac dysrhythmias
Myocardial ischemia
Inhalation (aspiration) of gastric contents

While the Endotracheal Tube Is in Place

Endotracheal tube obstruction
Endobronchial intubation
Esophageal intubation
Endotracheal tube cuff leak
Pulmonary barotrauma
Nasogastric distention
Accidental disconnection from the anesthesia breathing circuit
Tracheal mucosa ischemia
Accidental extubation

Complications After Endotracheal Extubation

Laryngospasm
Inhalation (aspiration) of gastric contents
Pharyngitis (sore throat)
Laryngitis
Laryngeal or subglottic edema
Laryngeal ulceration with or without granuloma formation
Tracheitis
Tracheal stenosis
Vocal cord paralysis
Arytenoid cartilage dislocation

Pardo MC, Miller RD. *Basics of Anesthesia*. 7. edition. Elsevier; 2018.

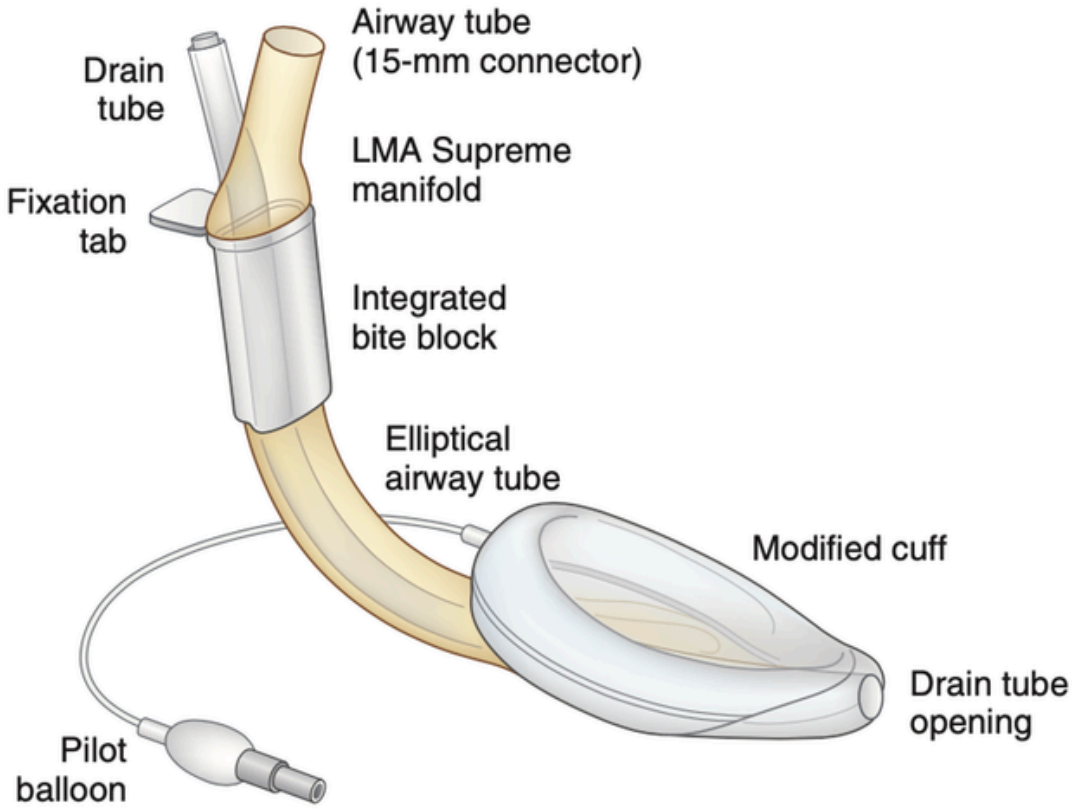


Figure 22-24 The LMA Supreme has a manifold with an integral bite block, an anatomically shaped airway tube enclosing a drain tube, a modified cuff through which the drain tube passes, and a cuff inflation line with pilot balloon.

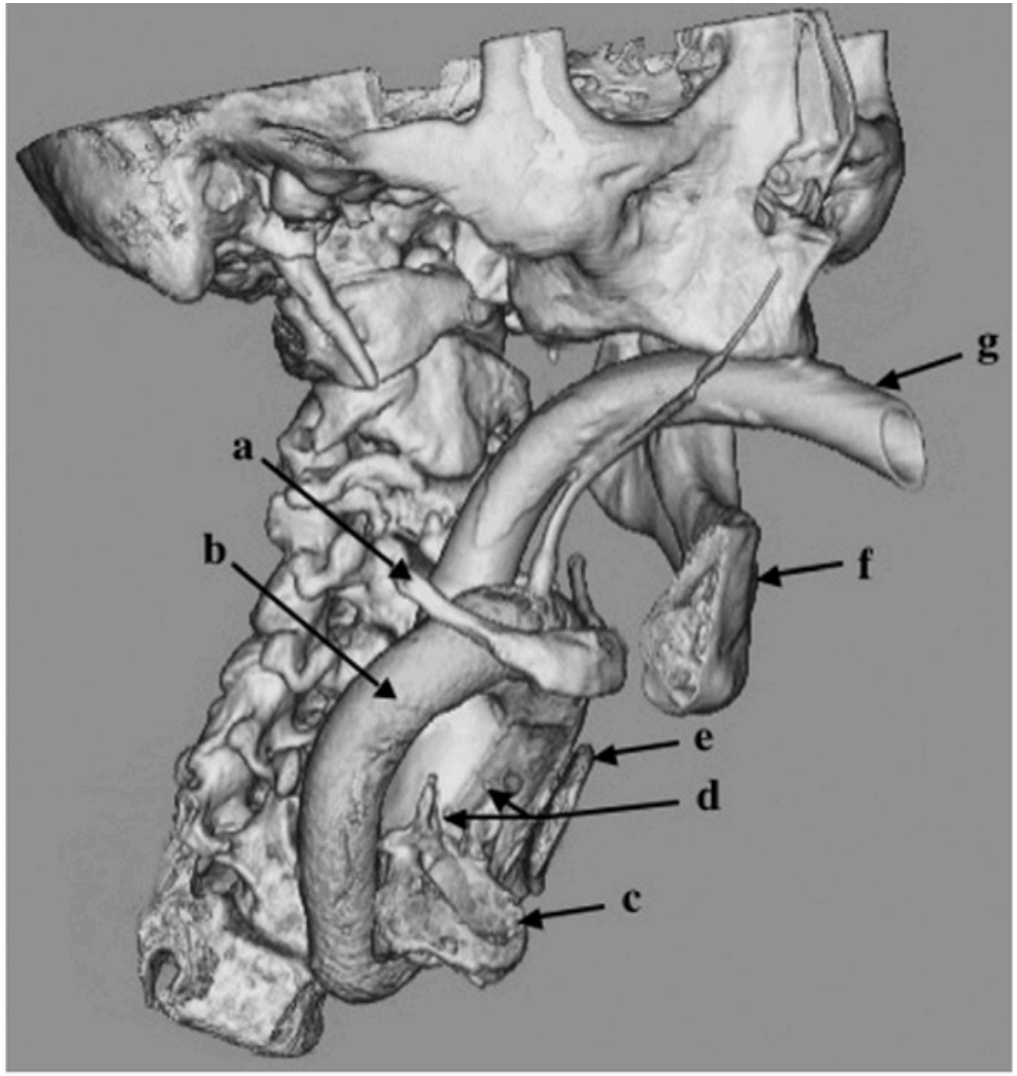


Figure 22-1 Three-dimensional radiologic reconstruction of the human airway with the laryngeal mask airway (LMA) in situ: hyoid bone *(a)*; LMA's cuff *(b)*; cricoid ring *(c)*; arytenoid cartilages *(d)*; thyroid cartilage *(e)*, which is digitally partially removed to demonstrate the position of the LMA; mandible *(f)*, which is digitally partially removed to demonstrate the position of the LMA; and the LMA's shaft *(g)*. The LMA's cuff forms a seal with the periglottic tissues and provides a continuous connection between the natural airway and the device.

Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

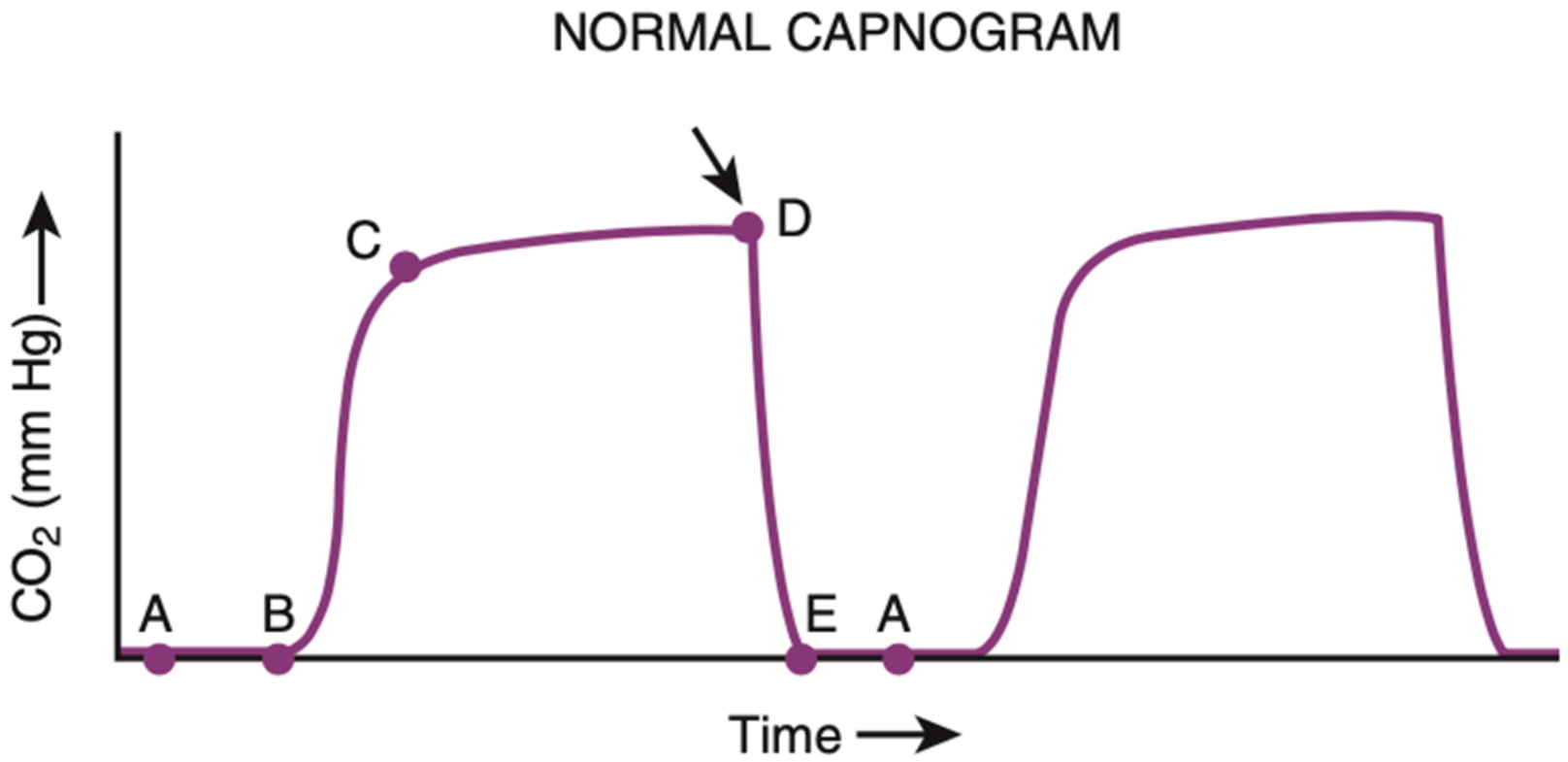
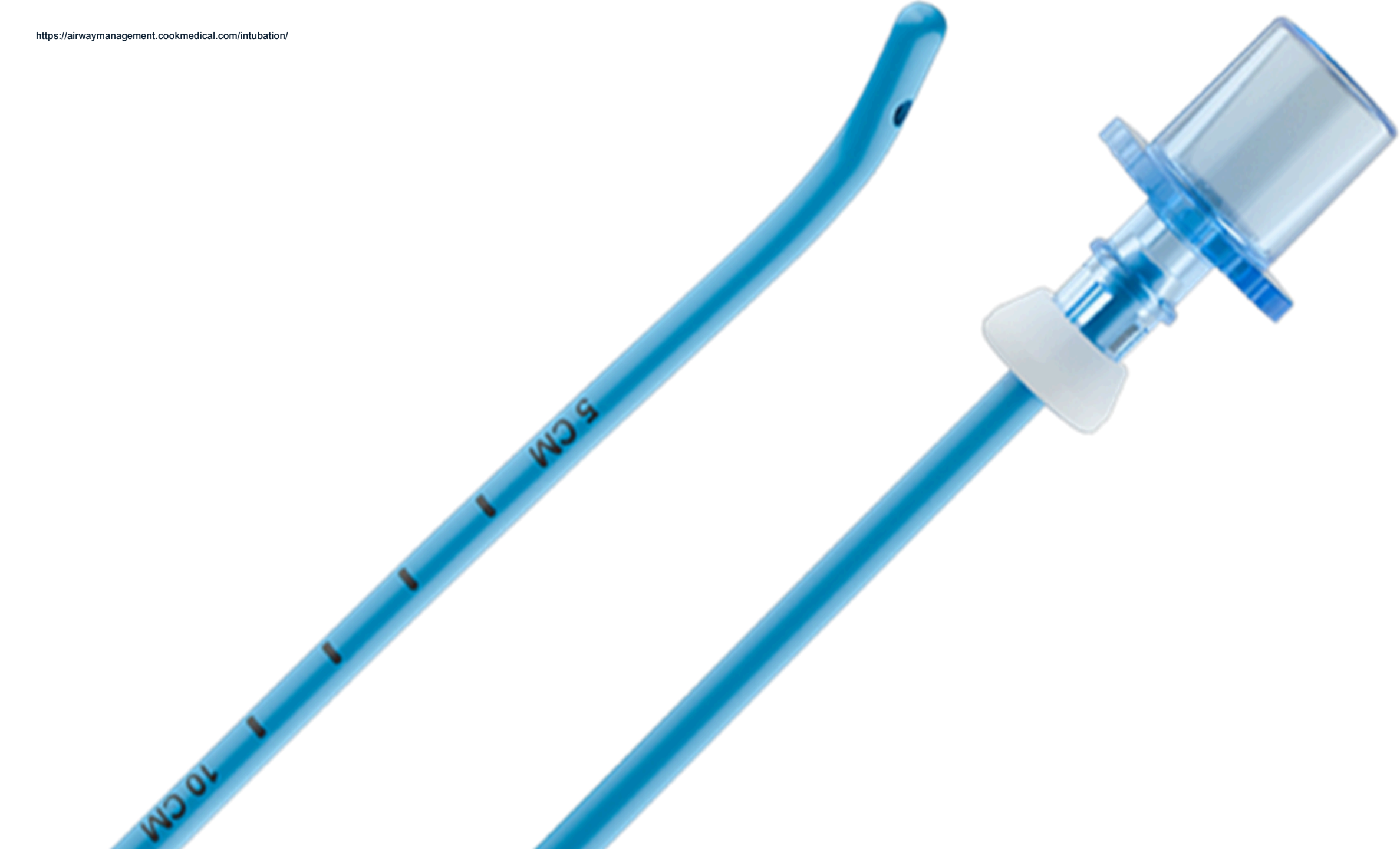


Figure 32-7 The CO_2 waveform. *A,* Expiratory pause begins. *A–B,* Clearance of anatomic dead space. *B–C,* Dead space air mixed with alveolar air. *C–D,* Alveolar plateau. *D,* End-tidal partial pressure of CO_2 registered by capnograph *(arrow)* and beginning of inspiratory phase. *D–E,* Clearance of dead space air. *E–A,* Inspiratory gas devoid of CO_2. (Modified from May WS, Heavner JE, McWorther D, Racz G: *Capnography in the operating room: An introductory directory,* New York, 1985, Raven Press, p 1.)

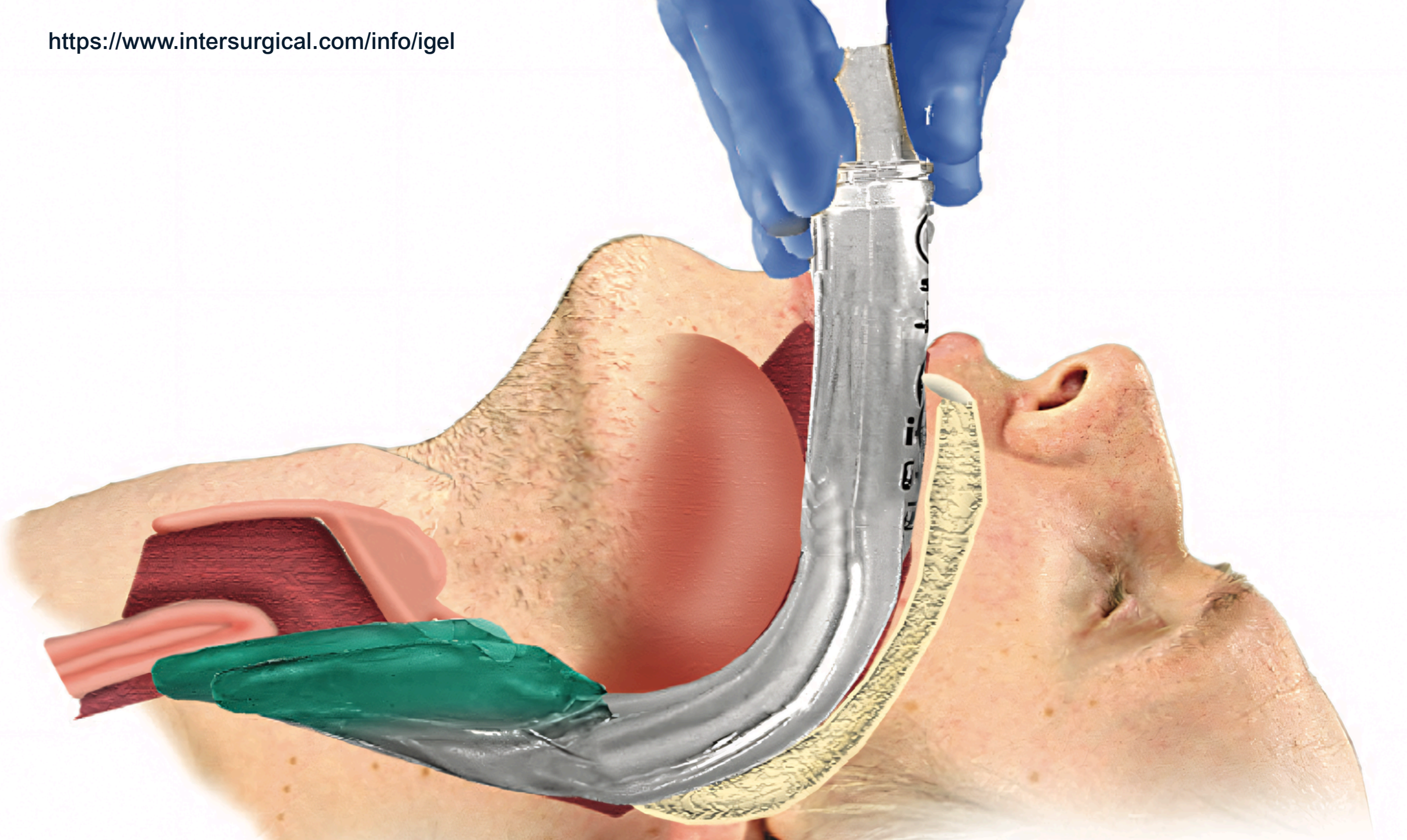
Hagberg CA, Benumof JL, eds. Benumof and Hagberg's Airway Management. 3rd ed. Elsevier Saunders; 2013.

https://airwaymanagement.cookmedical.com/intubation/




The operator manipulates the coude tip into the larynx.
YouTube

https://www.intersurgical.com/info/igel



Preparations for use

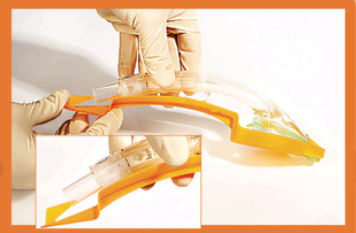
Adult patient

1



Open the **i-gel** package, and on a flat surface take out the protective cradle containing the device.

2



Remove the **i-gel** and transfer it to the palm of the same hand that is holding the protective cradle, supporting the device between the thumb and index finger.

3



Place a small bolus of a water-based lubricant, such as K-Y Jelly, onto the middle of the smooth surface of the protective cradle in preparation for lubrication.

4



Grasp the **i-gel** with the opposite (free) hand along the integral bite block and lubricate the back, sides and front of the cuff with a thin layer of lubricant.

5



Place the **i-gel** back into the protective cradle in preparation for insertion.

Step 6

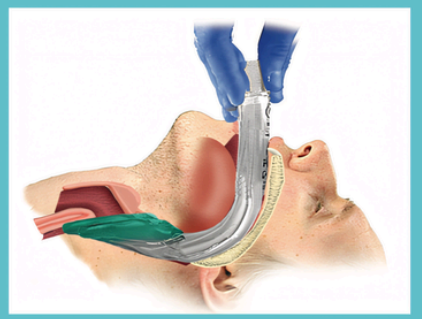
Insertion technique

6



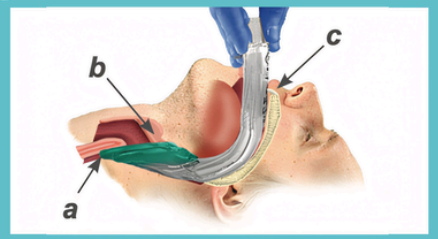
Remove the i-gel from the protective cradle. Grasp the lubricated i-gel firmly along the integral bite block. Position the device so that the **i-gel** cuff outlet is facing towards the chin of the patient. The patient should be in the 'sniffing the morning air' position with head extended and neck flexed. The chin should be gently pressed down before proceeding. Introduce the leading soft tip into the mouth of the patient in a direction towards the hard palate.

7



Glide the device downwards and backwards along the hard palate with a continuous but gentle push until a **definitive resistance** is felt .

8



The tip of the airway should be located into the upper oesophageal opening (a) and the cuff should be located against the laryngeal framework (b). The incisors should be resting on the integral bite-block (c).

9

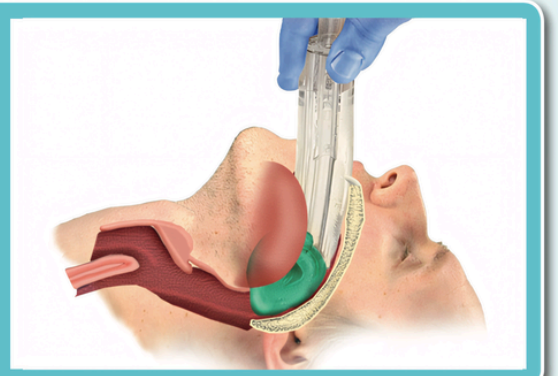


i-gel should be taped down from 'maxilla to maxilla'.

10



If there is early resistance during insertion a 'jaw thrust' (above) or 'Insertion with Deep Rotation' (right) is recommended.

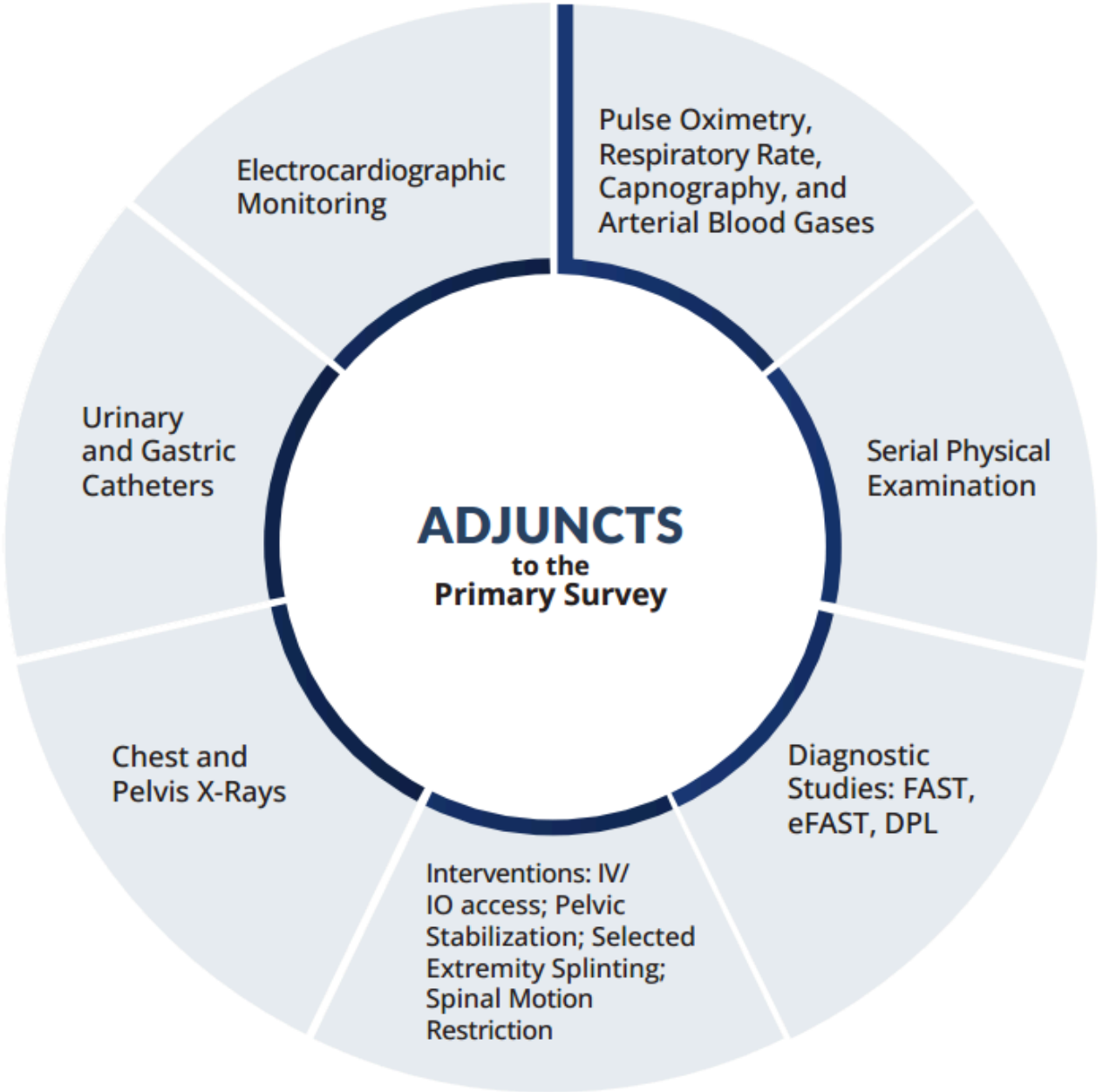




The "DOPE" Mnemonic

D	**D**isplacement: Endotracheal tube displacement (right mainstem) or dislodgement
O	**O**bstruction: Mucous plug, kink in tubing
P	**P**neumothorax: Simple or tension, due to positive-pressure ventilation
E	**E**quipment failure: Disconnect from ventilator and perform manual ventilation

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



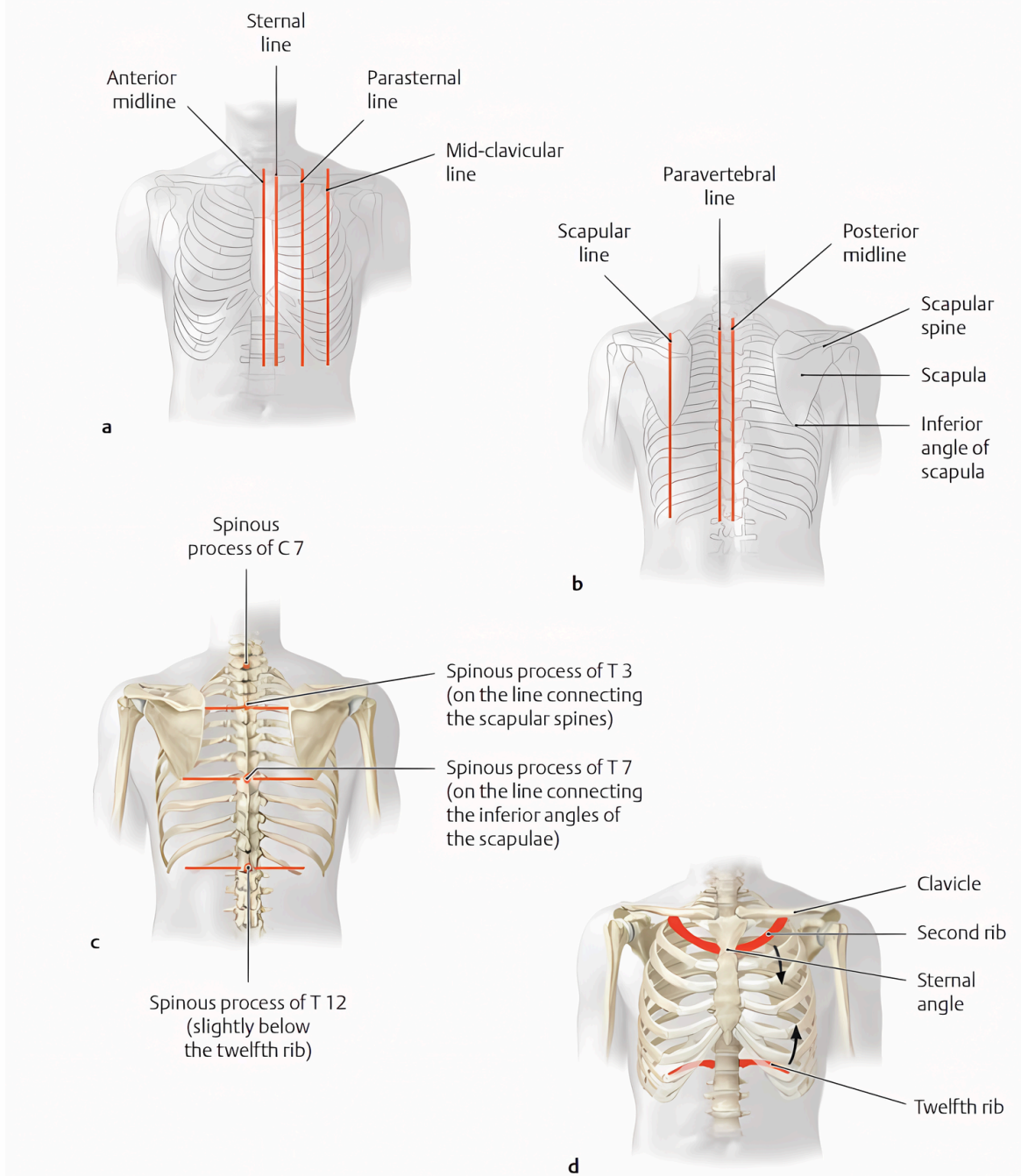
American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

Chest trauma

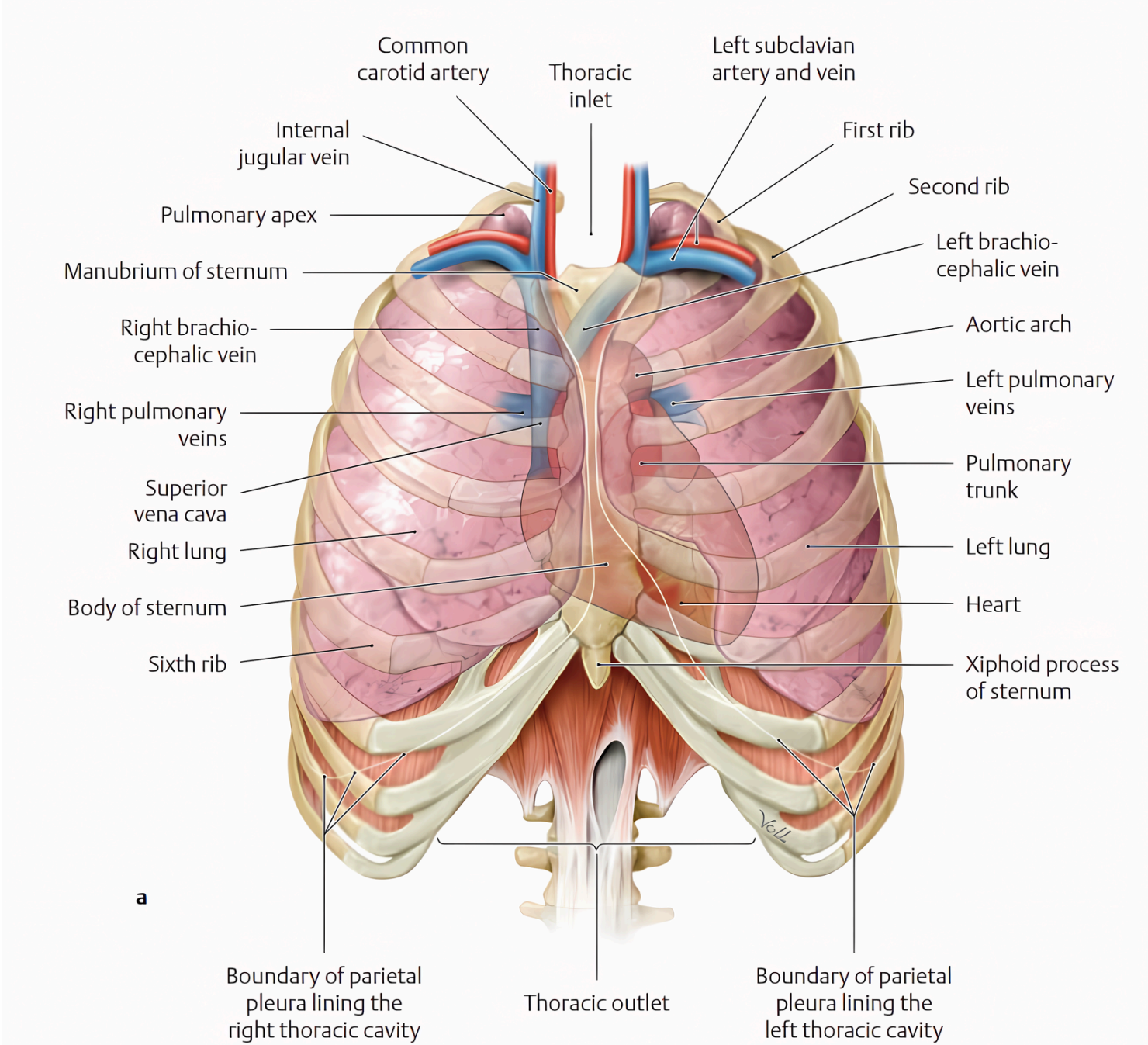
Thoracic Trauma: Injury Assessment and Management

- **Chest wall and thoracic cavity injuries**
  Common sequelae of trauma, involving bony structure, viscera, and great vessels
- **Impaired ventilation and oxygen exchange**
  Leading to respiratory and metabolic acidosis, impaired cardiac function
- **Urgent procedures**
  Tube thoracostomy to temporize and treat injuries
- **Severe rib fractures**
  Significant mortality and morbidity, need for multimodal pain relief
- **Alternative diagnostic strategies**
  For resource-limited settings
- **Special considerations**
  Pregnant, morbidly obese, and pediatric patients

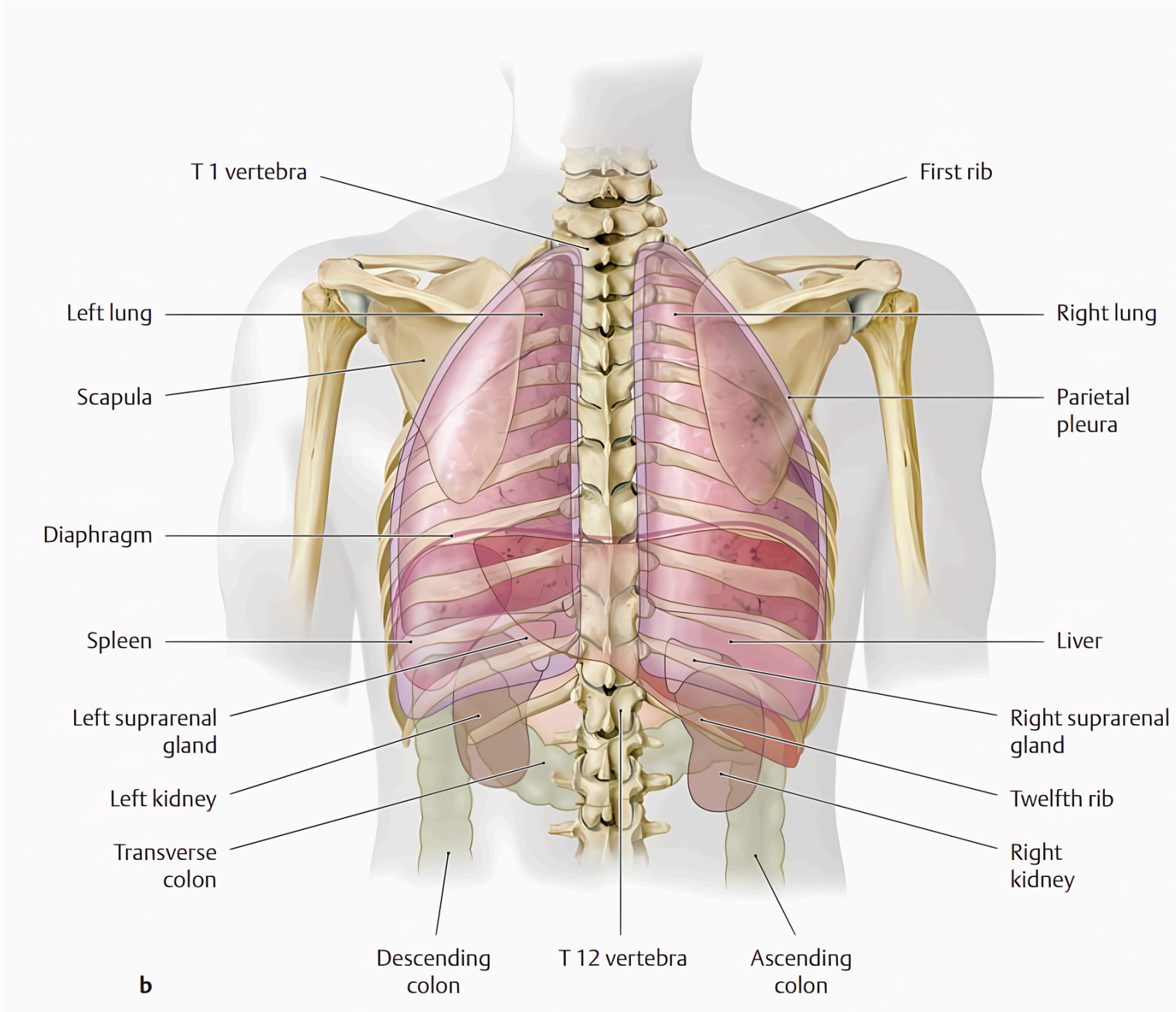
salvatore.sardo@unica.it



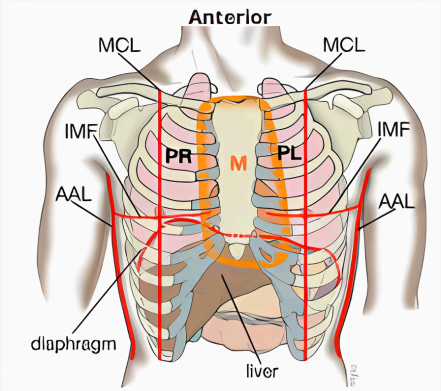
Gilroy AM, MacPherson BR, Wikenheiser JC, Schünke M, Schulte E, Schumacher U. *Atlas of Anatomy*. 4. Auflage. Thieme Medical Publishers; 2020.

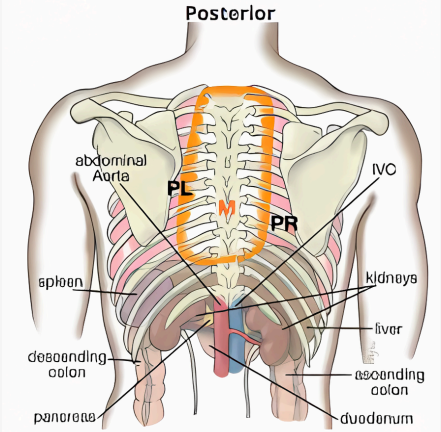


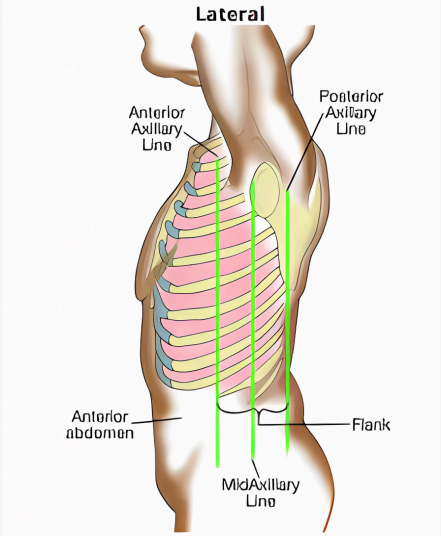
Gilroy AM, MacPherson BR, Wikenheiser JC, Schünke M, Schulte E, Schumacher U. *Atlas of Anatomy*. 4. Auflage. Thieme Medical Publishers; 2020.



Gilroy AM, MacPherson BR, Wikenheiser JC, Schünke M, Schulte E, Schumacher U. *Atlas of Anatomy*. 4. Auflage. Thieme Medical Publishers; 2020.







Based on surface landmarks, the chest extends from the sternal notch to the costal margin anteriorly.

Posteriorly it extends from the base of the neck to the costal margin.
Diaphragm movement varies with respiratory depth.

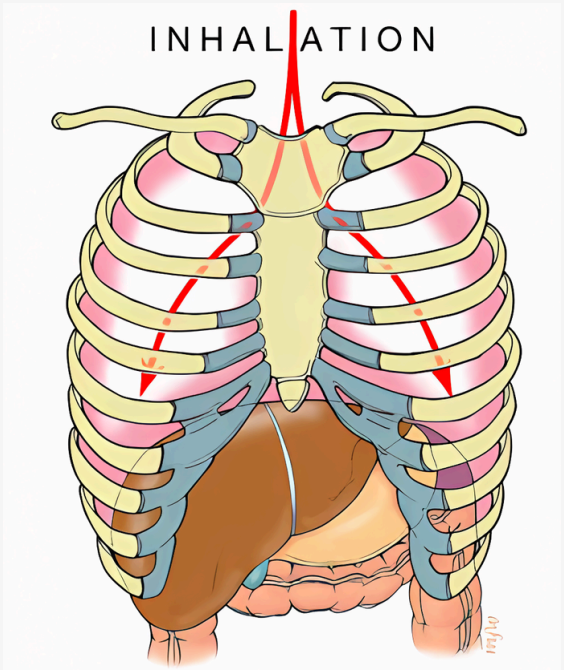
With deep exhalation, abdominal structures can reach the nipple line or inframammary fold and the tip of the scapula.

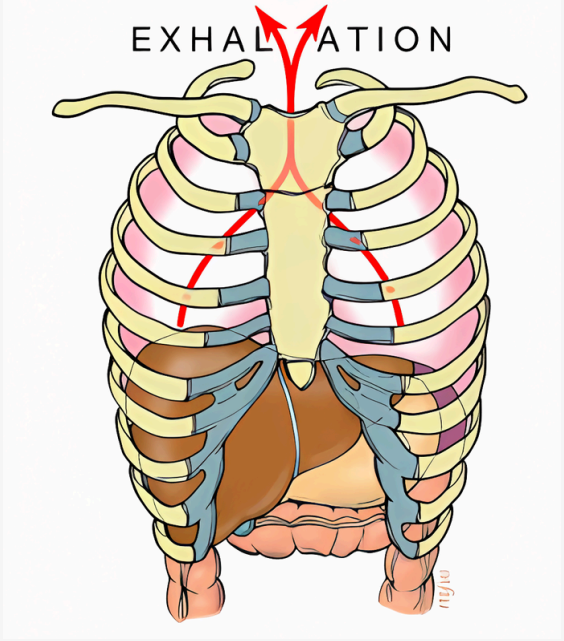
The nipple line intersects the midclavicular line at the fourth intercostal space, although its position varies with age and gender.

The inframammary fold usually lies along the fifth to sixth rib.
The inframammary fold is more reliable than the nipple line in patients with breast tissue.

The inframammary fold denotes the region of the fourth to fifth intercostal spaces.
These spaces allow safe needle decompression or tube thoracostomy.
Using this landmark reduces the risk of unintentionally entering the peritoneal cavity.

American College of Surgeons Committee on Trauma. Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count. 11th ed. American College of Surgeons; 2025.

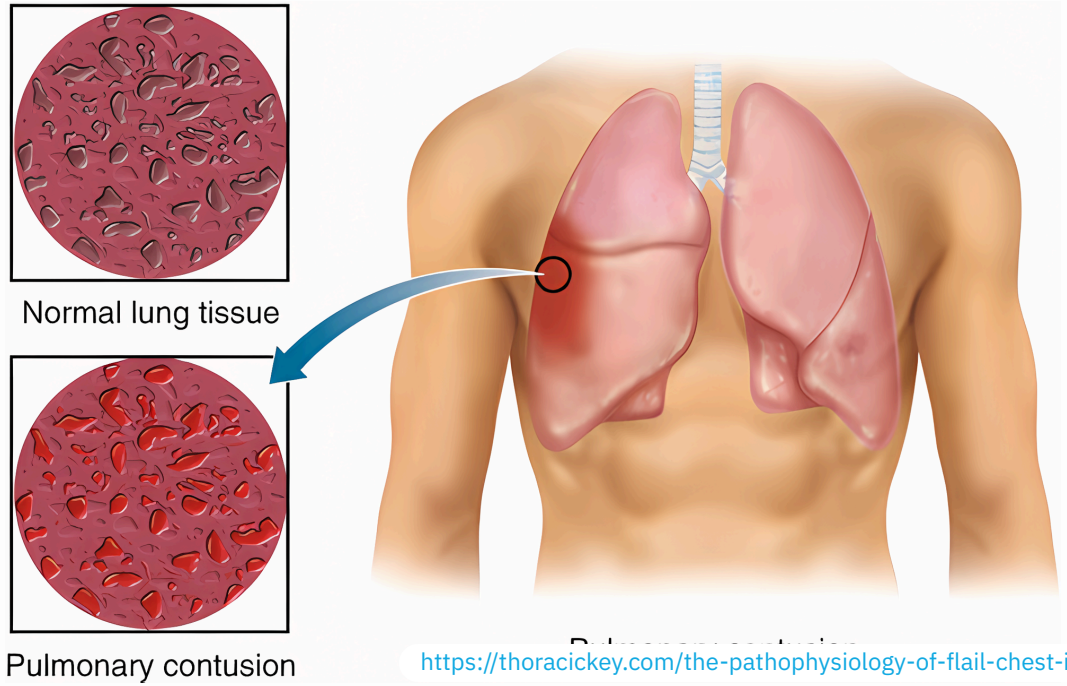




Pulmonary Contusion

- **Pulmonary contusion involves injury to the alveolar capillaries without a tear in lung tissue.**
  - It is the most common intrathoracic injury and may occur with or without rib fractures. Children often show contusions without fractures because of flexible chest walls.
  - Most young patients with isolated contusion recover, while patients with multiple injuries face higher risk of respiratory failure and death.
  - Blunt trauma produces contusion through force transmission, while penetrating or explosive trauma causes injury through direct damage or cavitation.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



https://thoracickey.com/the-pathophysiology-of-flail-chest-injury/

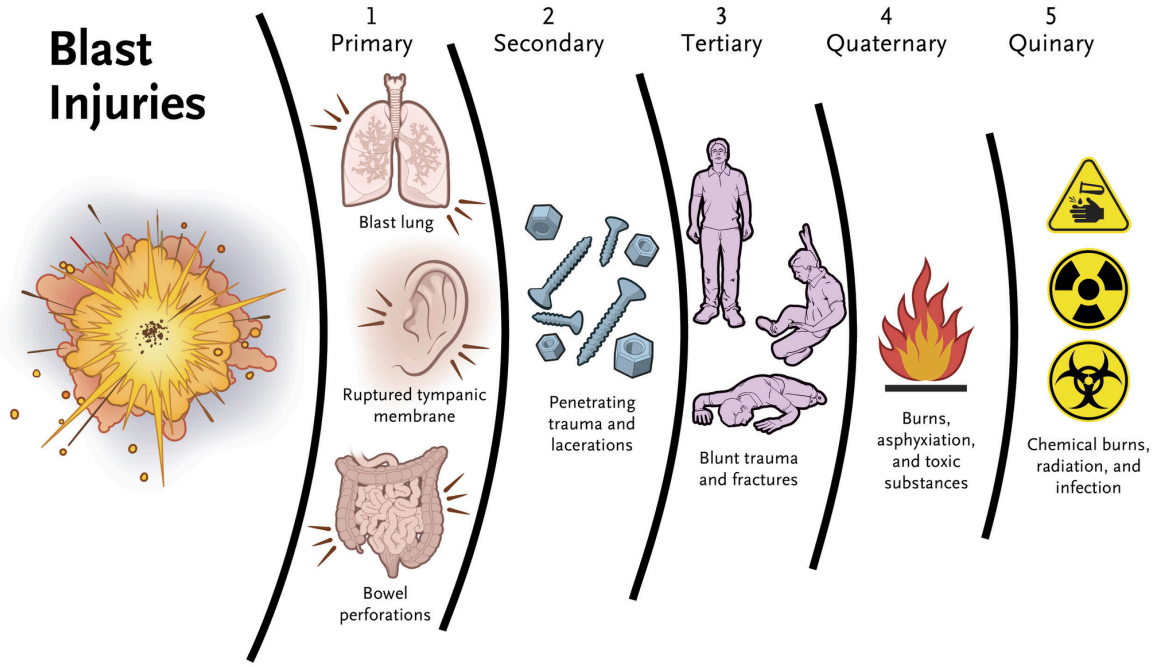
The injury leads to alveolar disruption with blood and fluid accumulation, ventilation–perfusion mismatch, intrapulmonary shunt, reduced compliance, hypoxemia, and hypercapnia.

Size and clinical severity often increase over the first 24 to 72 hours.

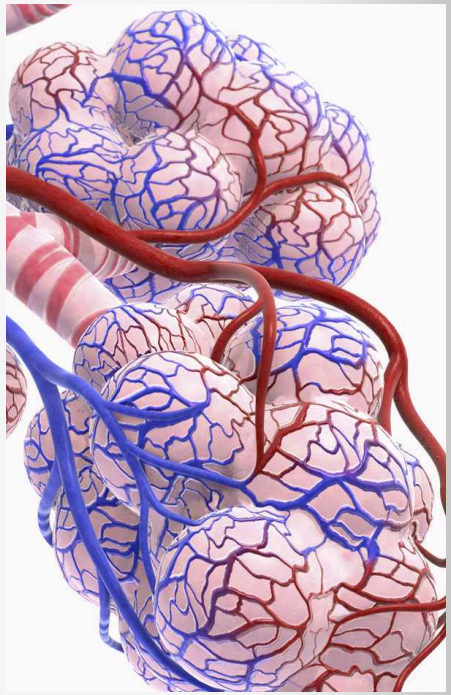
Clemedson identified three main mechanisms responsible for pulmonary contusion after blast exposure.

- The spalling effect causes alveolar disruption at gas–liquid interfaces when a shock wave meets lung tissue.
- The inertial effect results from different acceleration rates of light alveolar tissue and heavier hilar structures, leading to tissue separation.
- The implosion effect arises from rebound or overexpansion of gas bubbles as pressure waves pass. Excessive distension can further tear the parenchyma.

Cohn SM, DuBose JJ. Pulmonary contusion: an update on recent advances in clinical management. *World J Surg*. 2010;34(8):1959-1970. doi:10.1007/s00268-010-0599-9

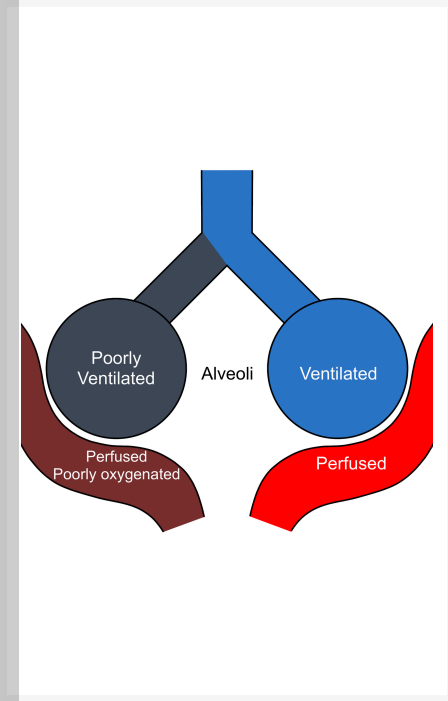


Ortega R, Vietor R, Arbelaez C, Feliu D, Nozari A, Ortoleva J. Blast injuries. *N Engl J Med*. 2024;391(23):e58. doi:10.1056/NEJMvcm2408353



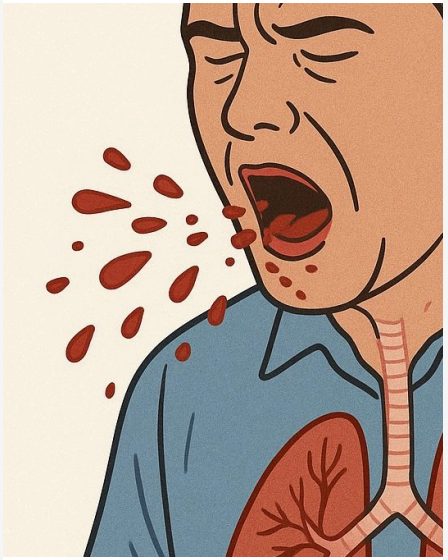
Parenchymal lung injury

Leads to bleeding, bronchospasm, impaired alveolar function, excess mucus, reduced mucus clearance, and reduced surfactant production



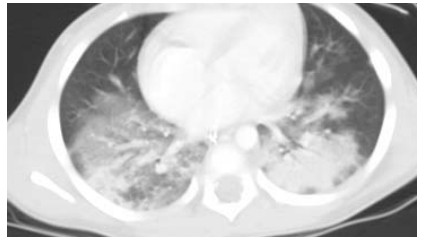
Ventilation–perfusion mismatch

Increased intrapulmonary shunt, lung fluid accumulation, and reduced compliance



Clinical features

Hypoxemia, hypercarbia, increased work of breathing, tachypnoea, wheeze or rhonchi, and sometimes haemoptysis



Radiographic changes

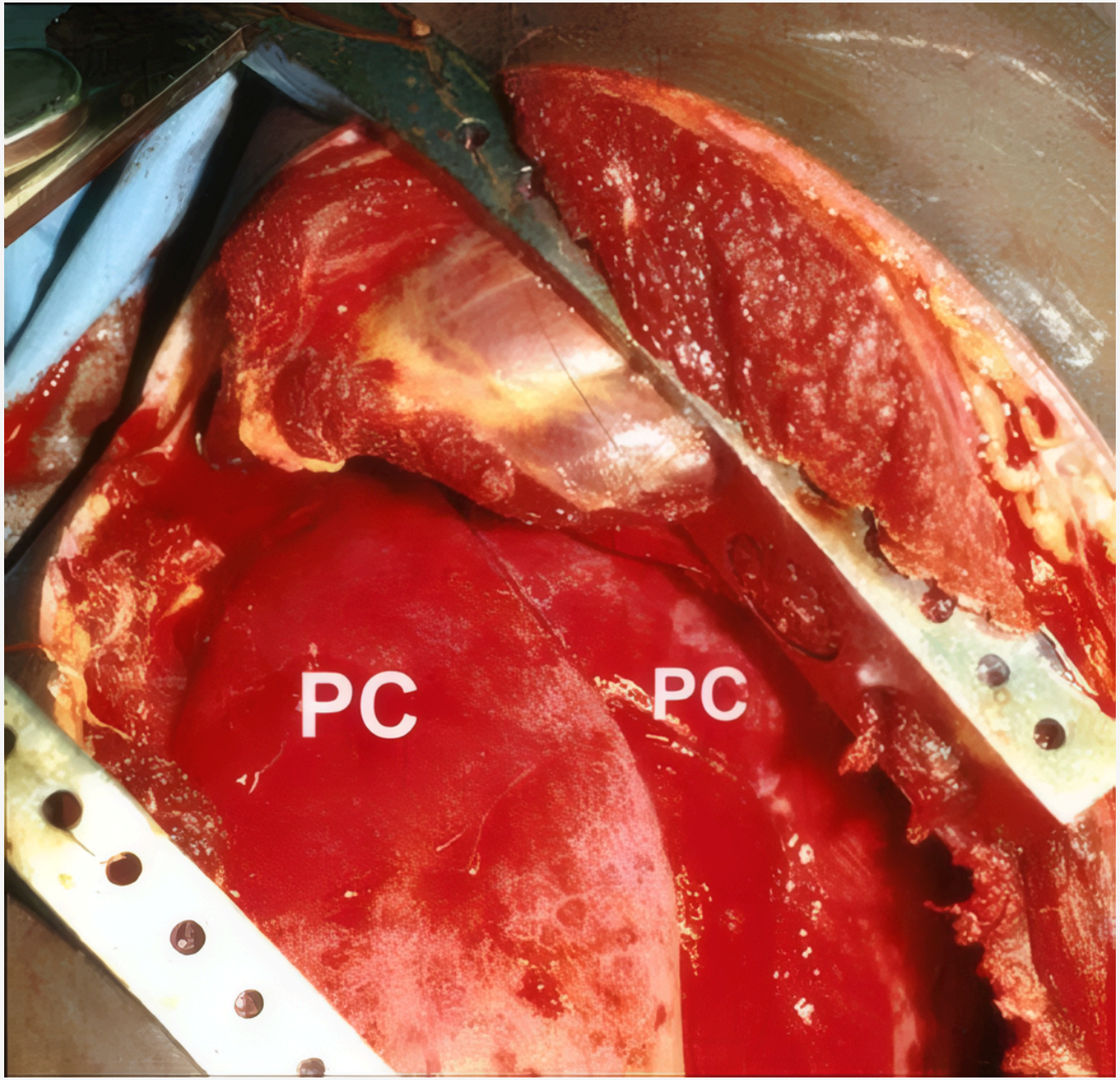
May appear only after 4 to 6 hours



Disease course

Most patients improve within 3 to 5 days, but delayed deterioration and later pulmonary dysfunction can occur

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Cohn SM, DuBose JJ. Pulmonary contusion: an update on recent advances in clinical management. *World J Surg*. 2010;34(8):1959-1970. doi:10.1007/s00268-010-0599-9

Flail chest

Rib fractures range from simple nondisplaced breaks to displaced or complex injuries. Fractures may be segmental, and when adjacent ribs are broken in multiple places they can form a radiographic flail segment. This is defined as two or more ribs fractured in two or more locations on imaging.

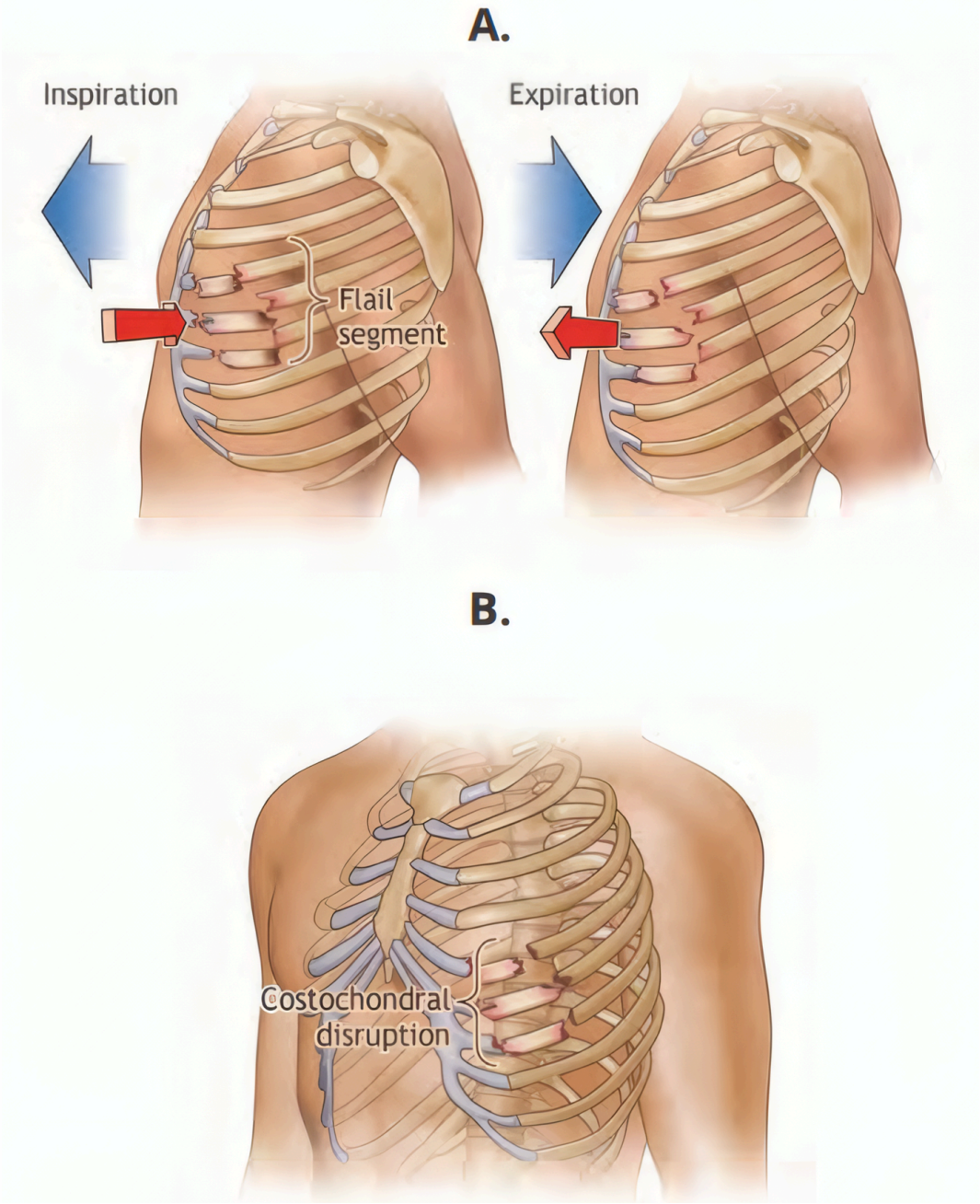
Clinical flail chest refers to paradoxical chest wall movement during respiration. Radiographic flail can occur without clinical flail, but clinical flail chest does not occur without radiographic flail.

Paradoxical movement develops because the flail portion is no longer mechanically linked to the rest of the chest wall. The injured segment moves inward during spontaneous inspiration and outward during exhalation.

During positive pressure ventilation this movement is less evident.

Rib fractures may lead to hypercapnic respiratory failure because pain limits adequate inspiration.

Severe hypoxaemia usually reflects associated pulmonary contusion or pneumothorax.



American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



Diaphragm Injury

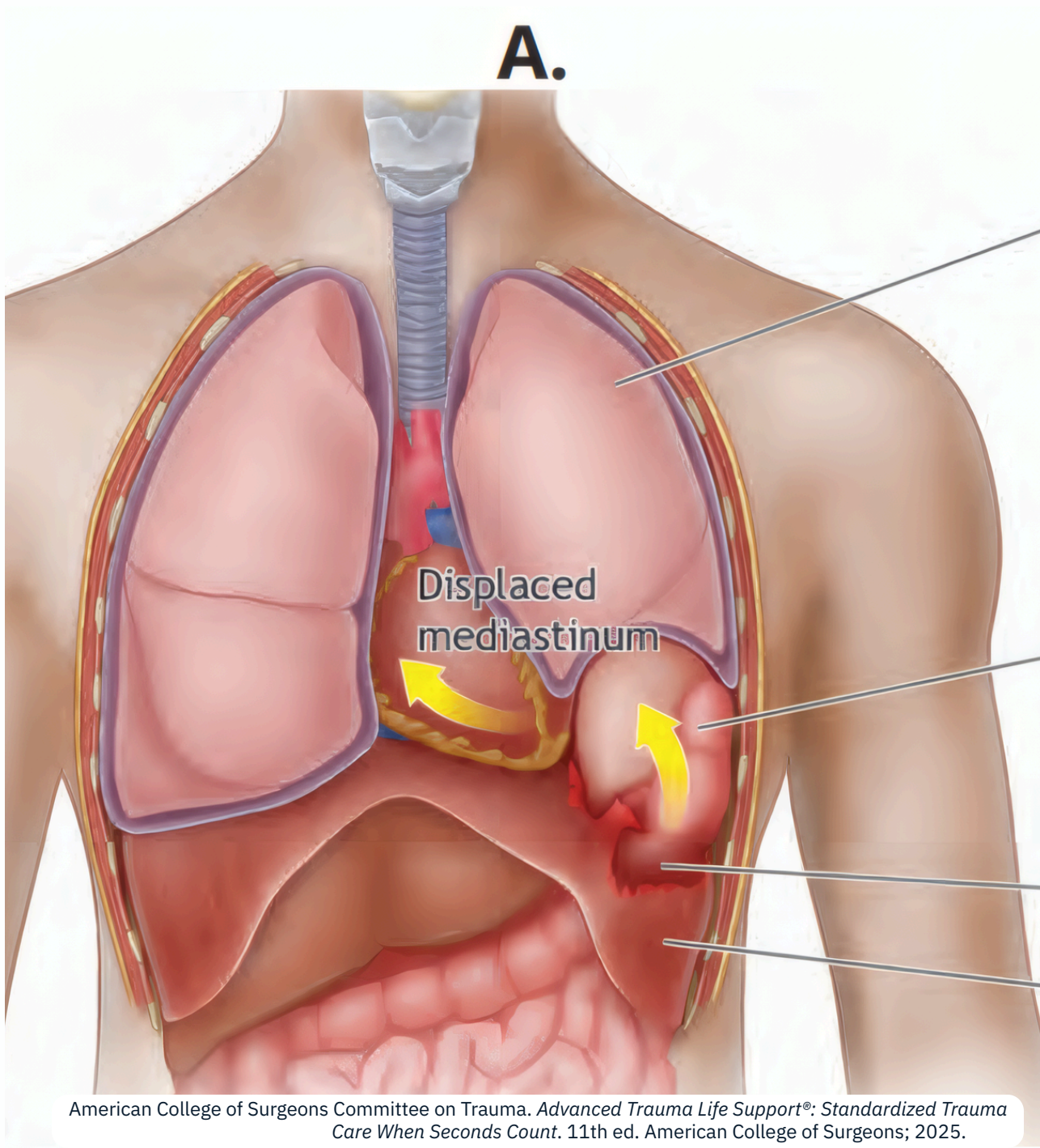
Occurs in about 0.5 percent of trauma patients, with two thirds due to penetrating mechanisms.

Blunt trauma usually causes large radial tears, while penetrating trauma produces smaller perforations, often less than 2 cm and harder to detect.

Left sided ruptures are more common because the liver protects the right hemidiaphragm.

Both mechanisms can lead to acute or delayed herniation of abdominal organs.

Respiratory compromise may occur due to lung compression by herniated viscera.



American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



Niknejad M, Diaphragmatic rupture. Case study, Radiopaedia.org (Accessed on 08 Dec 2025) https://doi.org/10.53347/rID-21690

Tracheobronchial injury

Injury to the trachea or a major bronchus is uncommon but can be fatal.

Most injuries occur within 1 inch of the carina.

Survivors who reach hospital often have high mortality due to associated trauma, loss of tidal volume, inadequate airway, or tension pneumothorax.

Mechanisms include crushing forces, shear forces from rapid deceleration, and penetrating trauma.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

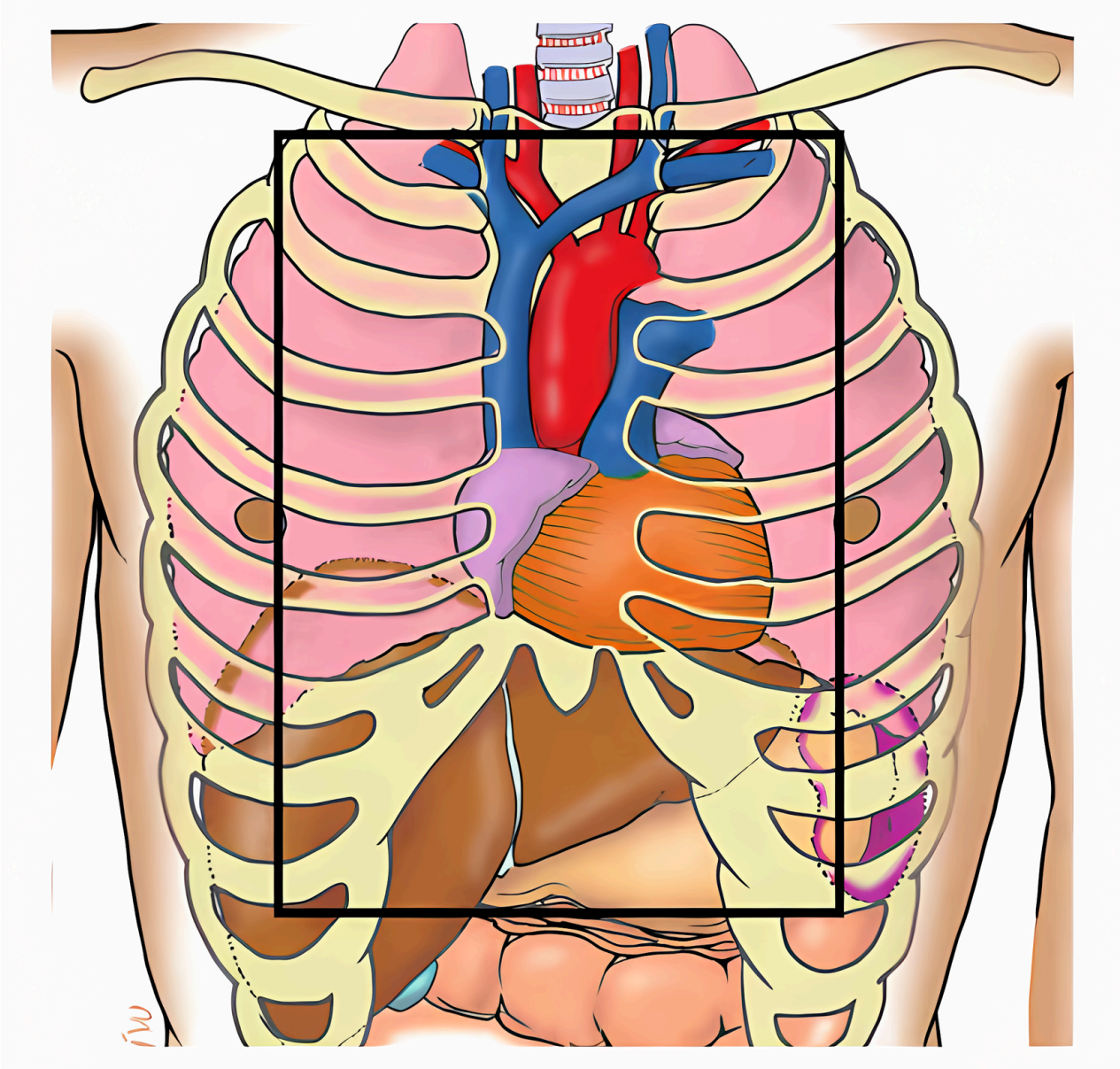


Aliev D, Metelmann I, Keller M, et al. Tracheobronchial injury after blunt thoracic trauma—lessons to learn in diagnosis, treatment, and postoperative care: a case series. *J Thorac Dis*. 2024;16(9). doi:10.21037/jtd-24-579

Condition	PHYSICAL SIGN				Comment
	Breath Sounds	Tracheal Position	Neck Veins	Blood Pressure	
Pulmonary Contusion	Normal to Decreased	Midline	Unaffected	Unaffected	Signs vary based on severity
Simple Hemo/ Pneumothorax	Normal to Decreased	Midline	Unaffected	Unaffected	
Tension Hemo/ Pneumothorax	Decreased	Deviated AWAY from injury	Distended*	Decreased	Critical clinical diagnosis
Open Pneumothorax	Abnormal and decreased	Midline	Unaffected	Unaffected	Chest wound
Massive Hemothorax	Decreased	Midline**	Flat	Decreased	Hemorrhagic shock
Flail Chest	Decreased	Midline	Unaffected	Unaffected	Severity of associated pulmonary contusion determines degree of hypoxia
Diaphragm Injury	Decreased	Midline**	Unaffected***	Unaffected***	
Tracheobronchial	Decreased and abnormal	Midline**	Unaffected****	Unaffected	Continued air leak after chest tube insertion. Potential for massive subcutaneous empysema.

* Distended neck veins are difficult to assess and may be absent if concurrent hemorrhagic shock is present.
** May shift away from injury side if tension physiology.
*** Massive herniation of abdominal contents into left chest may produce tension physiology. Neck veins may be distended and blood pressure decreased if tension physiology. Blood pressure may be decreased if hemorrhagic shock is present due to abdominal injury.
**** May be distended if concurrent tension pneumothorax.

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The Cardiac “Box”

The cardiac “box” is defined by the area medial to the nipple lines anteriorly, medial to the tips of the scapulae posteriorly, bounded superiorly by the sternal notch and inferiorly by the costal margins.

Penetrating injuries to this region have the potential to injure several vital structures.

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Circulatory disorders and shock

Shock

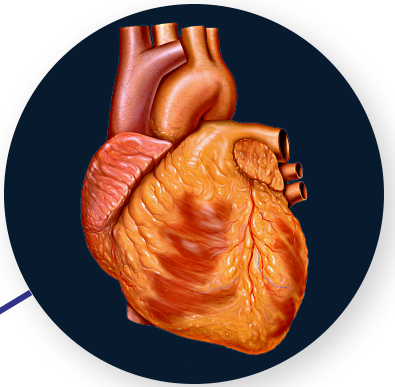
"Shock is best defined as a life-threatening, generalized form of acute circulatory failure associated with inadequate oxygen utilization by the cells"



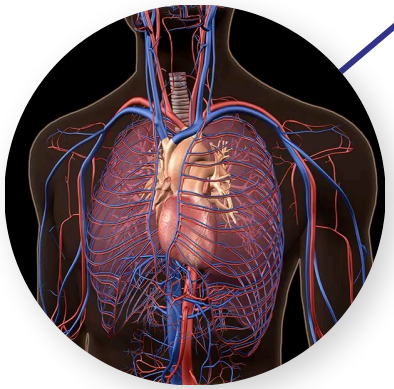
Cecconi M, De Backer D, Antonelli M, et al. Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. Intensive Care Med. 2014;40(12):1795-1815. doi:10.1007/s00134-014-3525-z

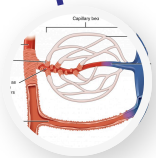
salvatore.sardo@unica.it

What's inside MAP...



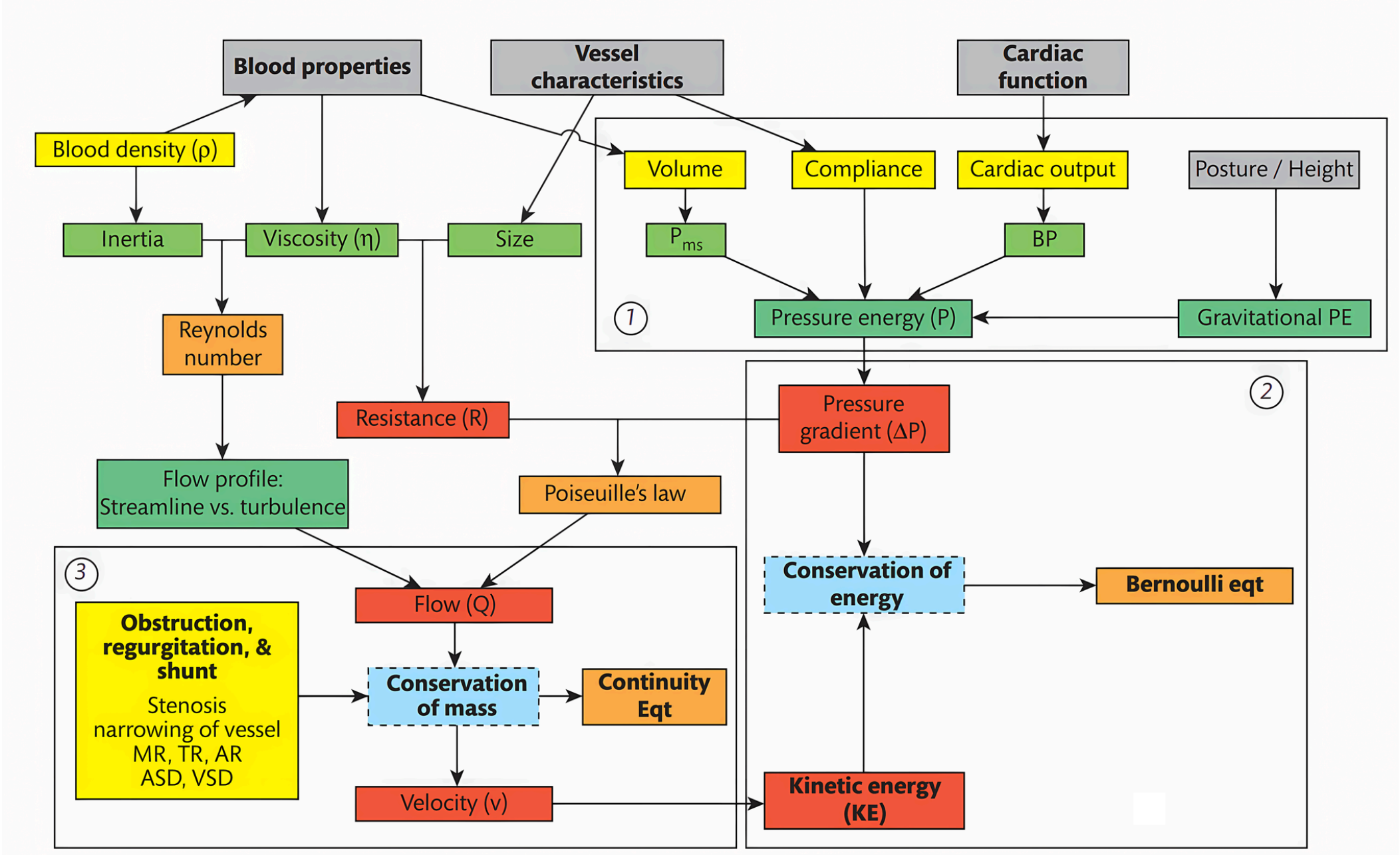
$$\text{Arterial Pressure} = \text{CO} \times \text{SVR} = (\text{SV} \times \text{HR}) \times \text{SVR}$$



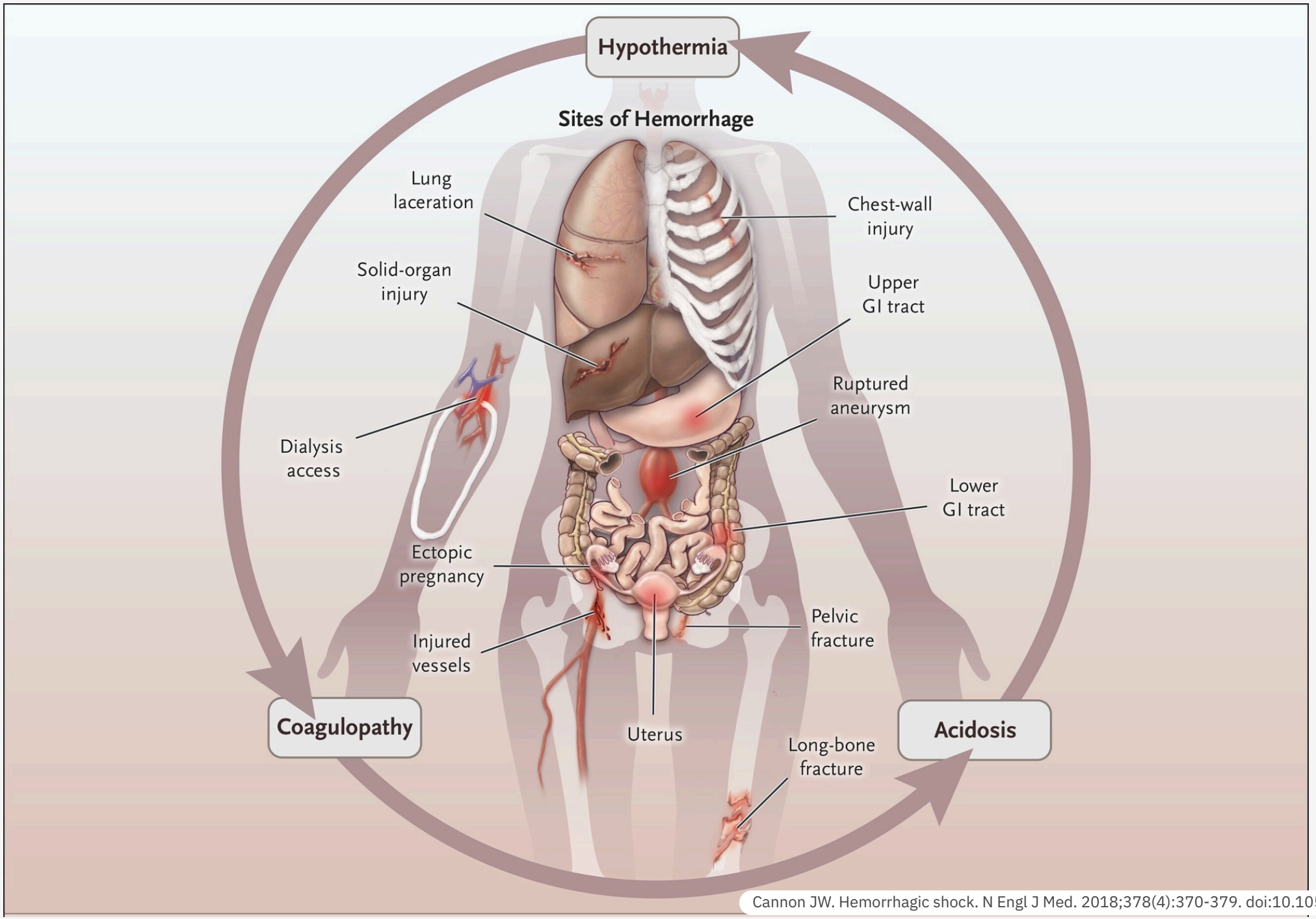


Arteriolar resistance, vasomotor tone, blood viscosity, and relative flow distribution across organs

salvatore.sardo@unica.it



McLean A, Huang S, Hilton A, eds. *Oxford Textbook of Advanced Critical Care Echocardiography*. 1st ed. Oxford University Press; 2020. doi:10.1093/med/9780198749288.001.0001

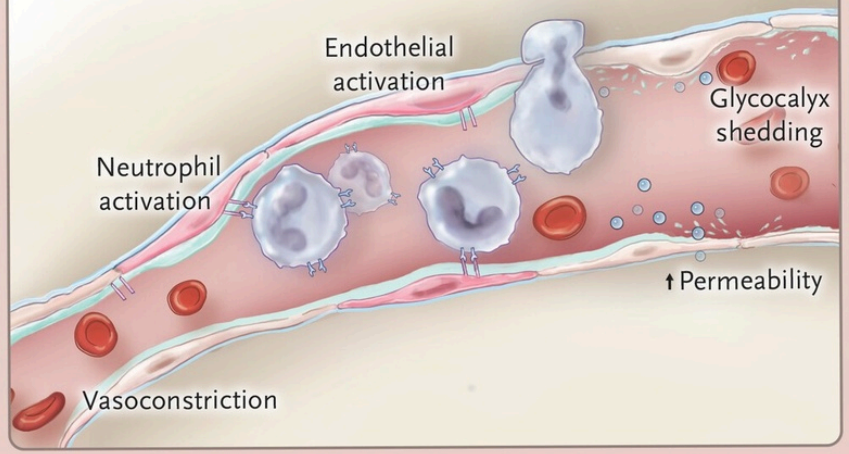


Cannon JW. Hemorrhagic shock. N Engl J Med. 2018;378(4):370-379. doi:10.1056/NEJMra1705649

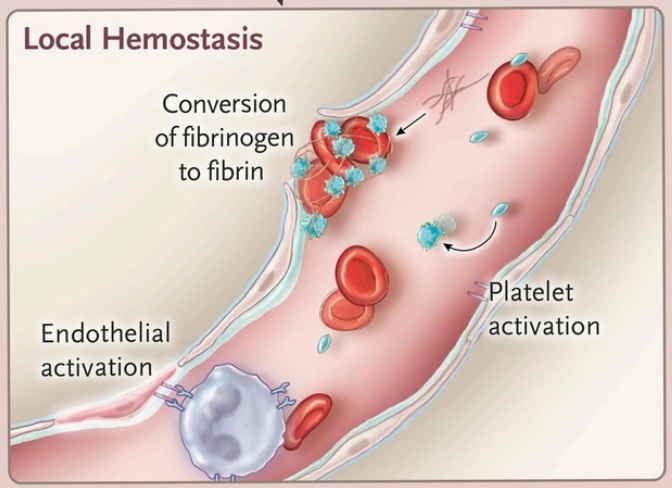
Iatrogenic Factors
Cold crystalloid infusion
Hemodilution
Hypothermia
Non–anion gap acidosis
Environmental exposure

Blood Loss
Depletion of red cells, clotting factors, and platelets
Decreased platelet margination (anemia)
Decreased clotting-factor activity (heat loss and acidosis)

Sympathoadrenal Response



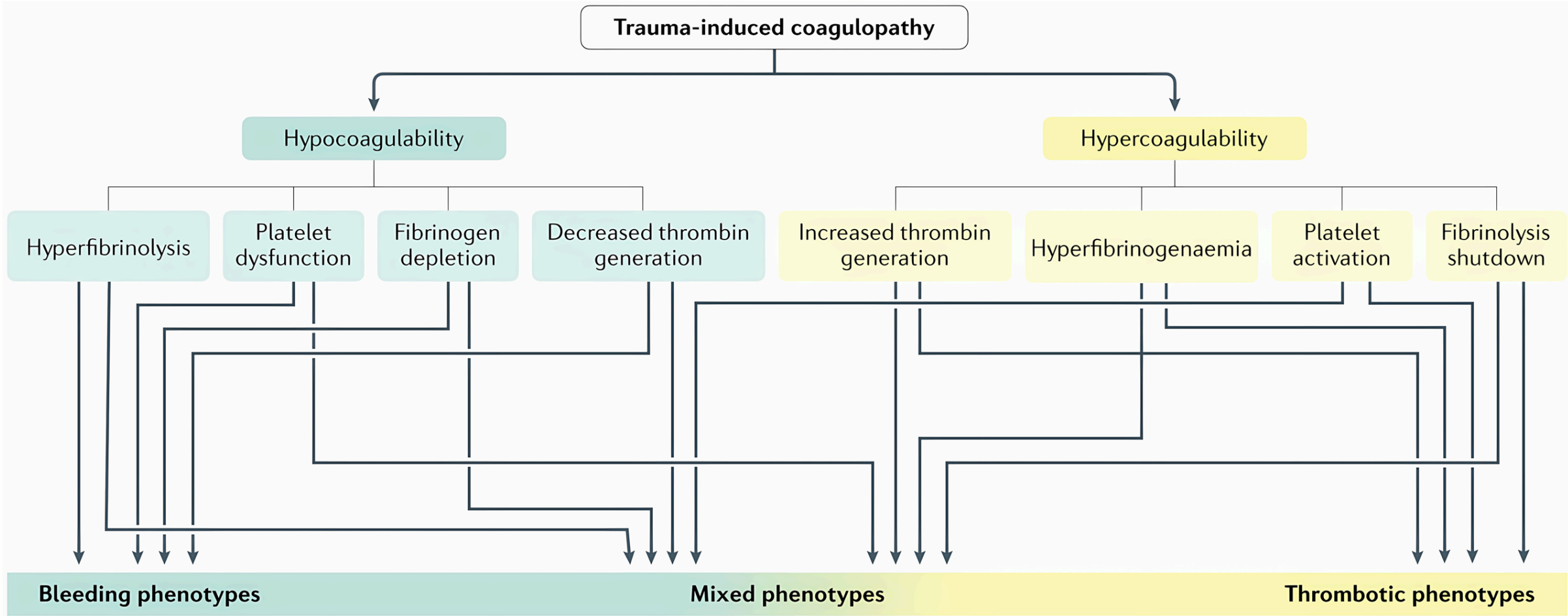
Local Hemostasis



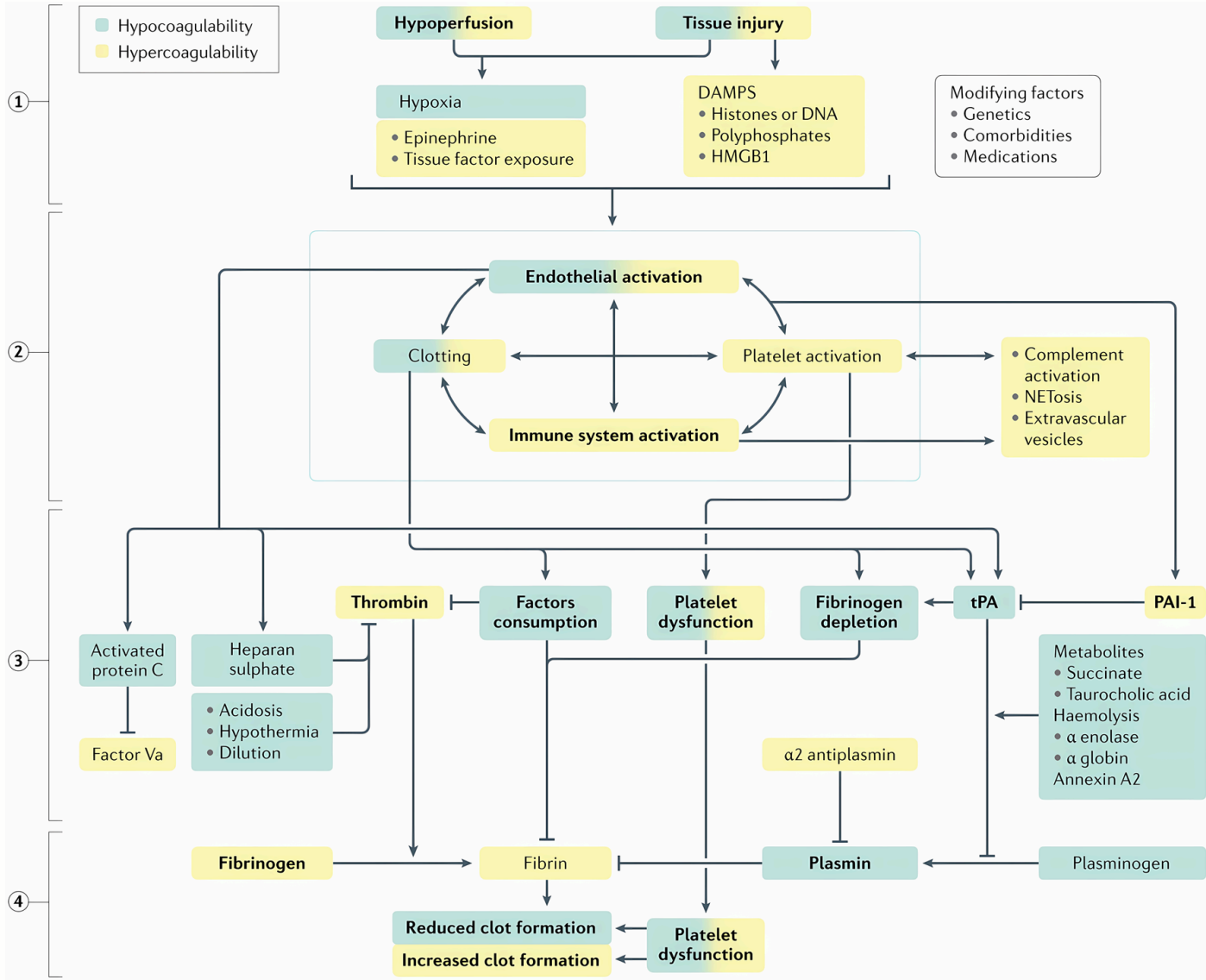
Genetic Response
Up-regulated innate immunity genes
Down-regulated adaptive immunity genes

Tissue Injury and Hypoperfusion
Release of formyl peptides and mitochondrial DNA (DAMPS or alarmins)
Activated protein C
Inactivated factors V and VIII
Increased plasmin
Release of tPA
Increased soluble thrombomodulin

Cannon JW. Hemorrhagic shock. N Engl J Med. 2018;378(4):370-379. doi:10.1056/NEJMra1705649



Moore EE, Moore HB, Kornblith LZ, et al. Trauma-induced coagulopathy. Nat rev, Dis primers. 2021;7(1):30. doi:10.1038/s41572-021-00264-3



Moore EE, Moore HB, Kornblith LZ, et al. Trauma-induced coagulopathy. Nat rev, Dis primers. 2021;7(1):30. doi:10.1038/s41572-021-00264-3

	HEMORRHAGE SEVERITY →			
PARAMETER	**MINOR**		**MODERATE**	**MAJOR**
Heart Rate (beats/min)	Unchanged	Unchanged to Increased	Increased (>100)	Markedly Increased (>120)
Systolic Pressure (mm Hg)	Unchanged	Unchanged	Unchanged to Decreased	Markedly Decreased (<90)
Pulse Pressure	Unchanged	Decreased	Decreased	Decreased
Respiratory Rate	Unchanged	Unchanged to Increased	Increased	Increased
Level of Consciousness	Unchanged	Unchanged	Decreased	Markedly Decreased
Base Deficit (meq/L)	0 to -2	-2 to -6	-6 to -10	-10 and less
Blood Products	Unlikely	Possibly	Likely	Massive Transfusion Protocol
Response to Resuscitation	Rapid and Sustained	Fairly Rapid and Sustained	Transient	Minimal to None
Operative Hemorrhage Control	Unlikely	Possibly	Likely	Extremely Likely

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Clinical Abnormalities in Shock and Signs of Improved Perfusion

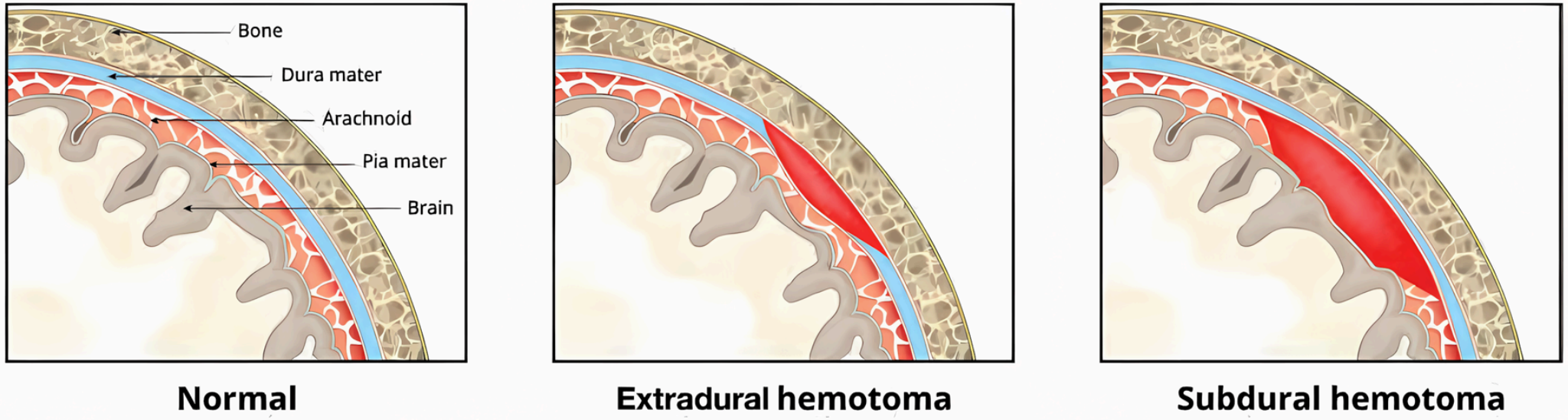
System	Shock	Improved perfusion	Limitations
CNS	Agitation in early shock Lethargy in late shock	Improvement in sensorium	May be confounded by concomitant TBI, intoxication, and medications Can't be assessed in intubated patient Interobserver variability
Skin	Cold, clammy Prolonged capillary refill time (<3 secs)	Improved temperature Improved capillary refill time	Subjective Confounded by external temperature
Renal	Pre-renal failure Oliguria anuria	Urine output adequate*	Evaluation takes hours No immediate index Chronic kidney disease Diuretics hyperglycemia
Hemodynamic	Tachycardia* Low-volume pulse by palpation Hypotension*	Trend towards normalization Palpable pulse volume improvement Normalization of BP	Noninvasive device not accurate Values may be falsely assuring Normalization of macrohemodynamics doesn't signify normalization of perfusion at cellular level Medication effects, preinjury diseases

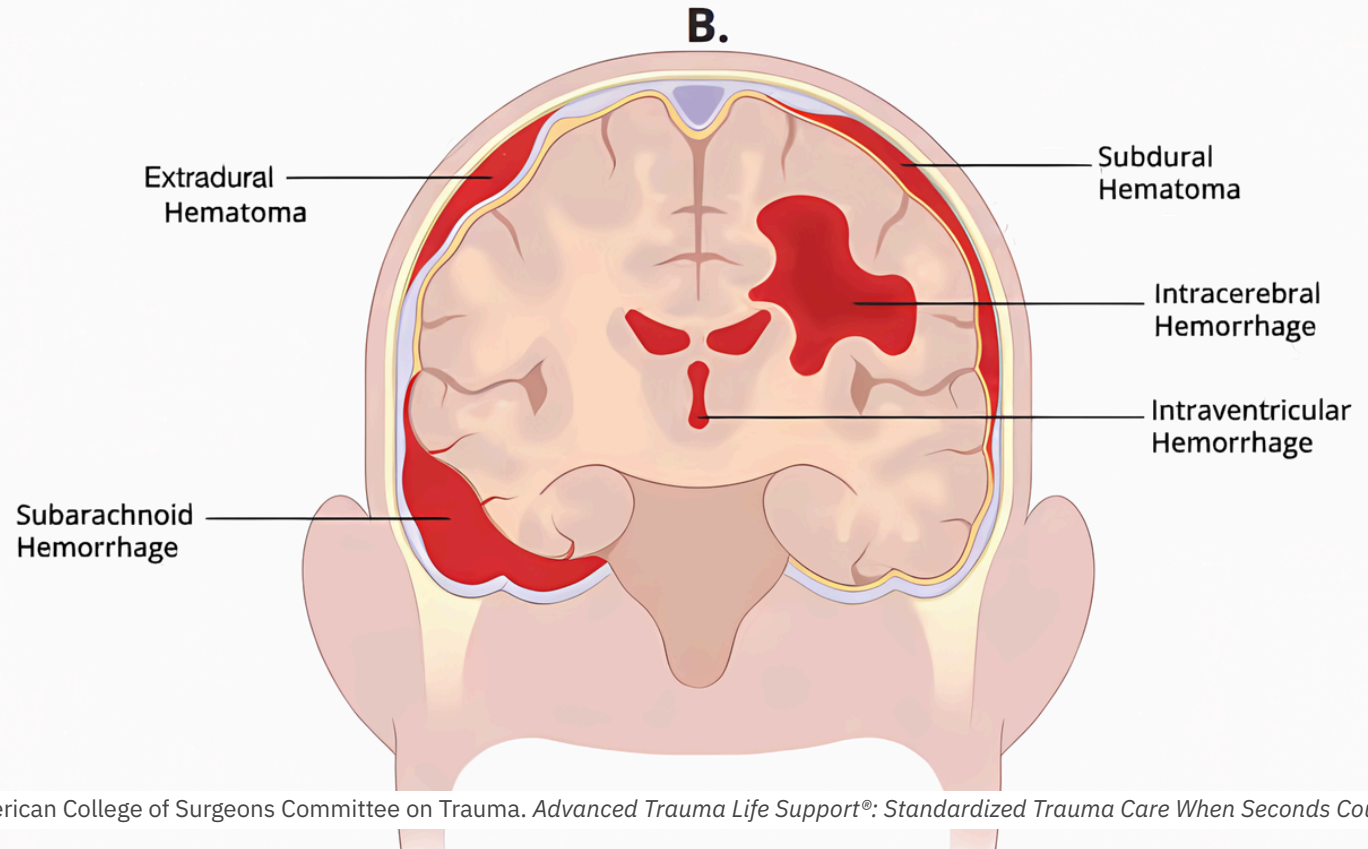
***Clinical values are evaluated as adjusted for age. BP, Blood pressure; TBI, Traumatic brain injury**

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Disability - TBI


YouTube





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Extradural haematoma is a collection of blood between the inner table of the skull and the parietal layer of the dura mater.

The bleeding is confined by cranial sutures, which gives the lesion a characteristic biconvex shape on imaging. More than 75 %of cases result from rupture of the middle meningeal artery and are commonly associated with skull fractures.

A vertex extradural haematoma usually arises from venous bleeding of the superior sagittal sinus and may cross the midline.

Primary brain injury is typically minimal in isolated extradural haematoma.

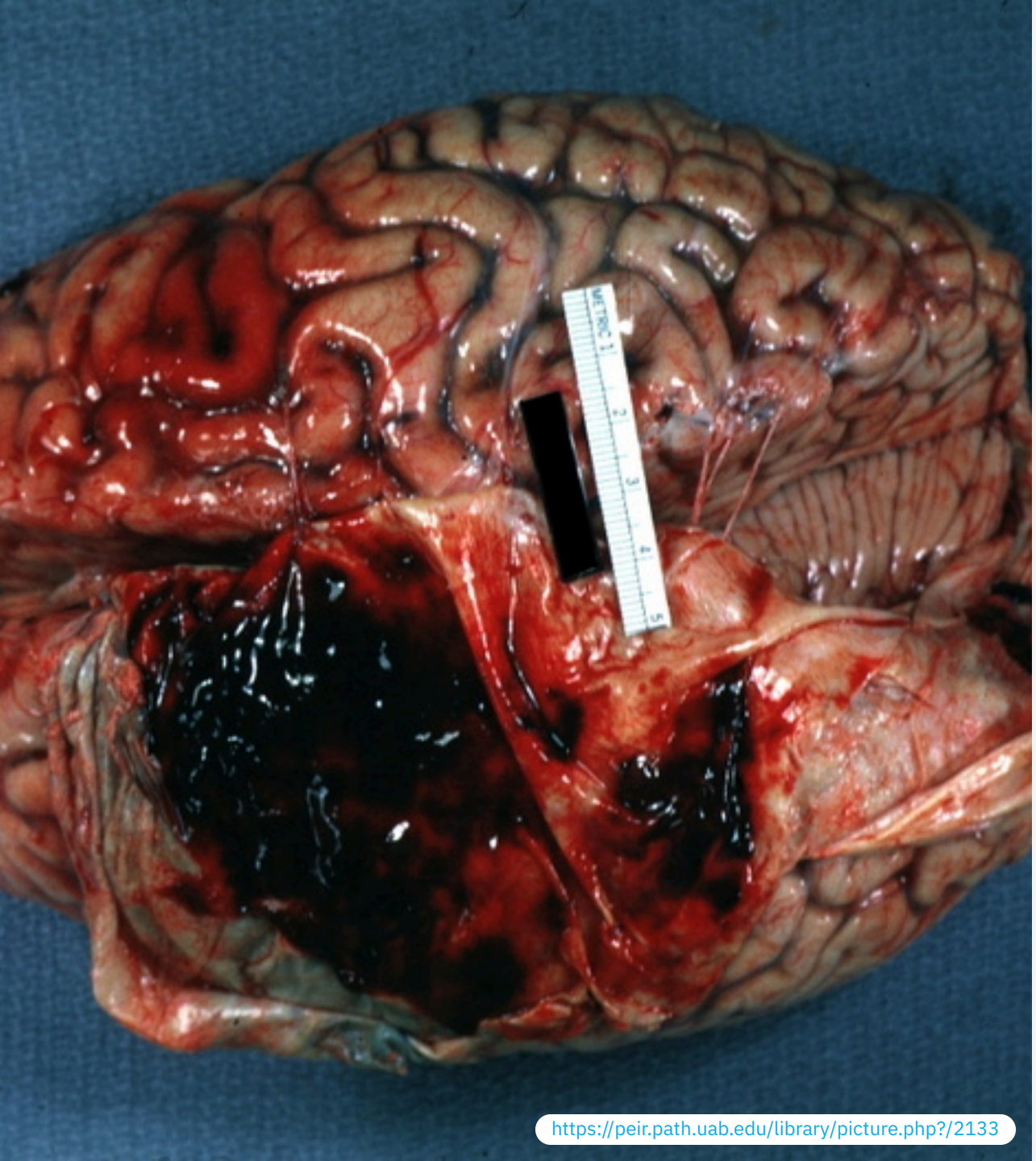
Neurological deterioration occurs mainly due to mass effect and midline shift.

With prompt surgical evacuation, most patients recover with little or no permanent neurological deficit.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



Hosn S, Epidural haematoma (gross pathology). Case study, Radiopaedia.org (Accessed on 13 Dec 2025) https://doi.org/10.53347/rID-20372



Subdural haematoma is a collection of blood within the subdural space, between the dura mater and the arachnoid.

It usually follows rapid deceleration of the head, during which the brain continues to move within the skull, causing stretching and rupture of bridging cortical veins.

Associated injury to the arachnoid allows cerebrospinal fluid to mix with blood in the subdural space. On CT imaging, subdural haematomas have a characteristic crescent shape.

They are not limited by cranial sutures but are constrained by dural reflections such as the falx cerebri, tentorium, and falx cerebelli.

Acute subdural haematomas, particularly in younger patients, are frequently associated with underlying brain contusions.

In adults, most subdural haematomas are unilateral. In infants, they are more often bilateral. Isolated interhemispheric or parafalcine subdural haematomas are commonly seen in non accidental injury and should raise concern for physical abuse.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

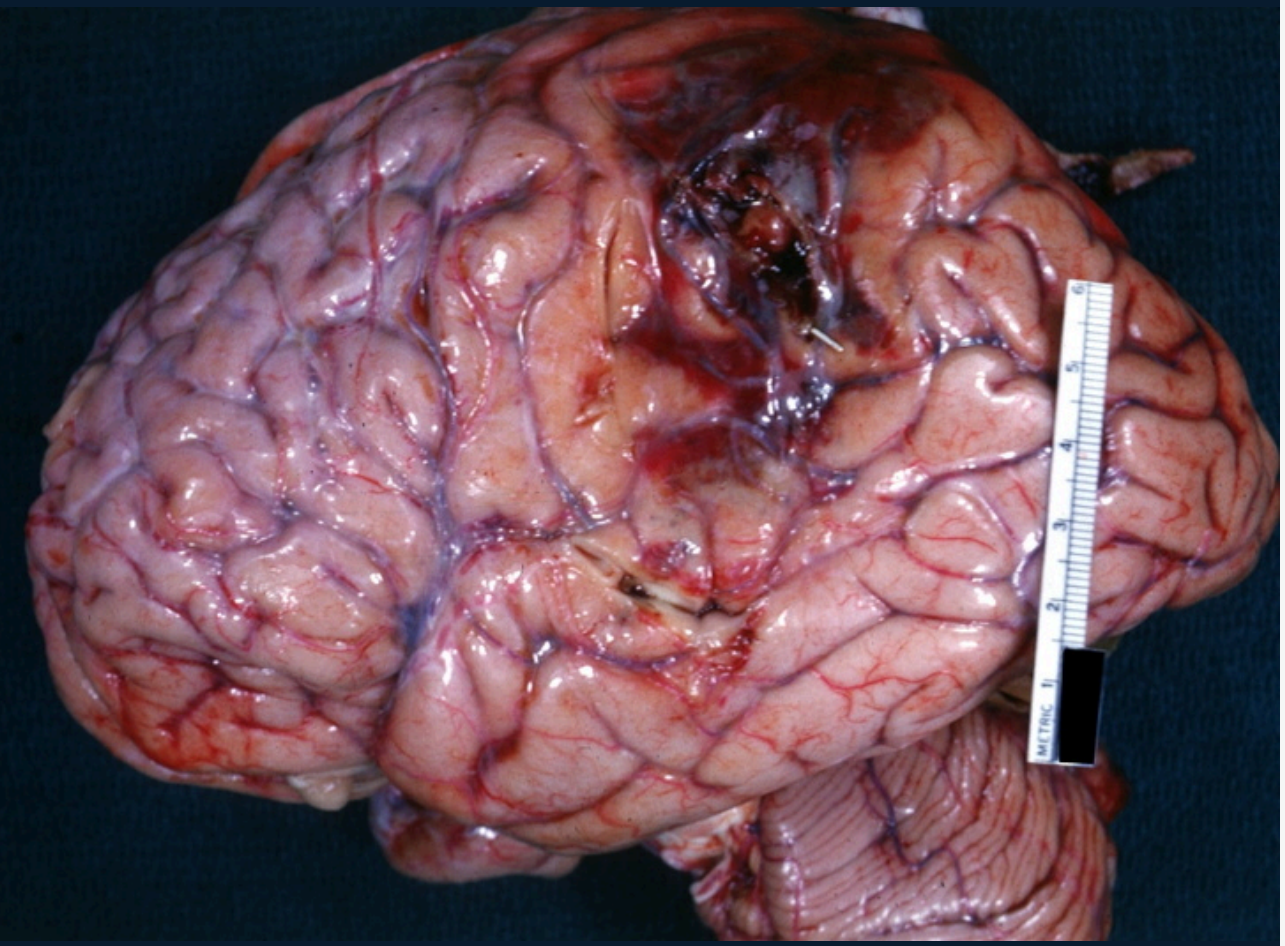
Traumatic subarachnoid haemorrhage is defined by the presence of blood within the subarachnoid spaces, including cerebral sulci, fissures, and basal cisterns.

The underlying mechanism is not fully established. Proposed mechanisms include shearing injury to small cortical veins or arteries, traumatic arterial dissection, and direct bleeding from adjacent cerebral contusions.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



https://www.rcemlearning.co.uk/modules/secondary-headache/lessons/intracranial-vascular-disorders/topic/subarachnoid-haemorrhage-sah/



https://peir.path.uab.edu/library/picture.php?/4052/tags/787-cerebral_cortical_contusion_skull_fracture

A cerebral contusion is a focal injury of the brain parenchyma.

Damage occurs either from direct external impact or from rapid deceleration that causes the brain to strike the inner surface of the skull.

Brain movement within the cranial vault can result in coup injuries at the site of impact or contrecoup injuries on the opposite side.

Contusions most commonly affect the frontal and temporal lobes, where the brain contacts the irregular surface of the anterior cranial fossa.

Cerebral contusions may enlarge during the first 24 hours due to ongoing bleeding and evolving pathophysiological changes. Reported rates of radiological or clinical progression range from 16 to 75%.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.

Traumatic axonal injury results from rotational acceleration or deceleration forces that produce shearing stress on neurons.

Grey and white matter have different densities, which causes relative movement between these tissues when the brain rapidly decelerates.

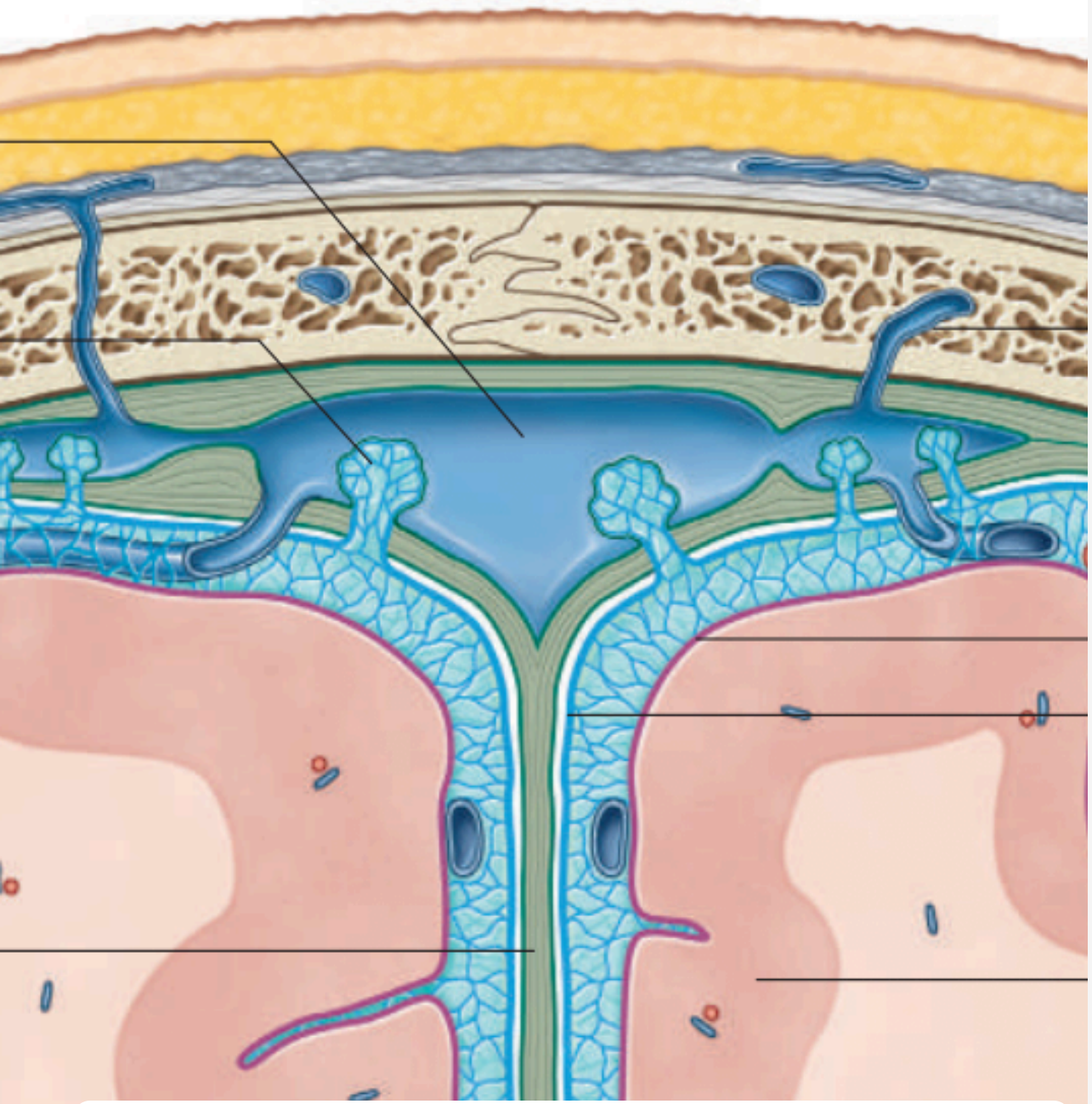
This differential movement leads to axonal stretching and shearing within the white matter, followed by axonal swelling and disconnection.

Diffuse axonal injury is characterised by multifocal involvement rather than a single focal lesion.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



https://www.pathologyoutlines.com/topic/forensicsneuropathologytai.html



Standring S, Ananad N, Gray H, Gray H, eds. *Gray's Anatomy: The Anatomical Basis of Clinical Practice*. 41. Elsevier; 2016.

Properties of the skull

- The skull comprises eight bones with a diploic structure of two cortical layers surrounding trabecular bone, allowing absorption and redistribution of impact energy. Individual anatomical variability results in differences in biomechanical response to head trauma.
- Impact energy is mainly absorbed by trabecular bone, while compact bone is less deformable.
- Oblique impacts are absorbed more effectively than perpendicular forces, and skull shape influences regional load transfer, particularly between temporal and parietal areas.
- Skull thickness and density vary by region and affect force transmission. The densest bone is in the posteroinferior temporal region, while the least dense and thinnest areas are near the frontal bone and pterion.
- Thicker bone deforms less but spreads force over a wider area, reducing local stress on brain tissue. Certain regions, such as the temporoparietal skull and lateral anterior cranial fossa, transmit energy more readily and fracture at lower forces.
- The skull demonstrates two injury thresholds: deformation, which may cause dural injury and haemorrhage, and penetration, which releases residual energy as kinetic debris and leads to severe brain injury.

Orr TJ, Lesha E, Kramer AH, et al. Traumatic brain injury: a comprehensive review of biomechanics and molecular pathophysiology. *World Neurosurg*. 2024;185:74-88. doi:10.1016/j.wneu.2024.01.084



Gosling JA, Harris PF, Humpherson JR, Whitmore I, Willan PLT. *Human Anatomy, Color Atlas and Textbook*. 6th ed. Elsevier Health Sciences UK; 2016.

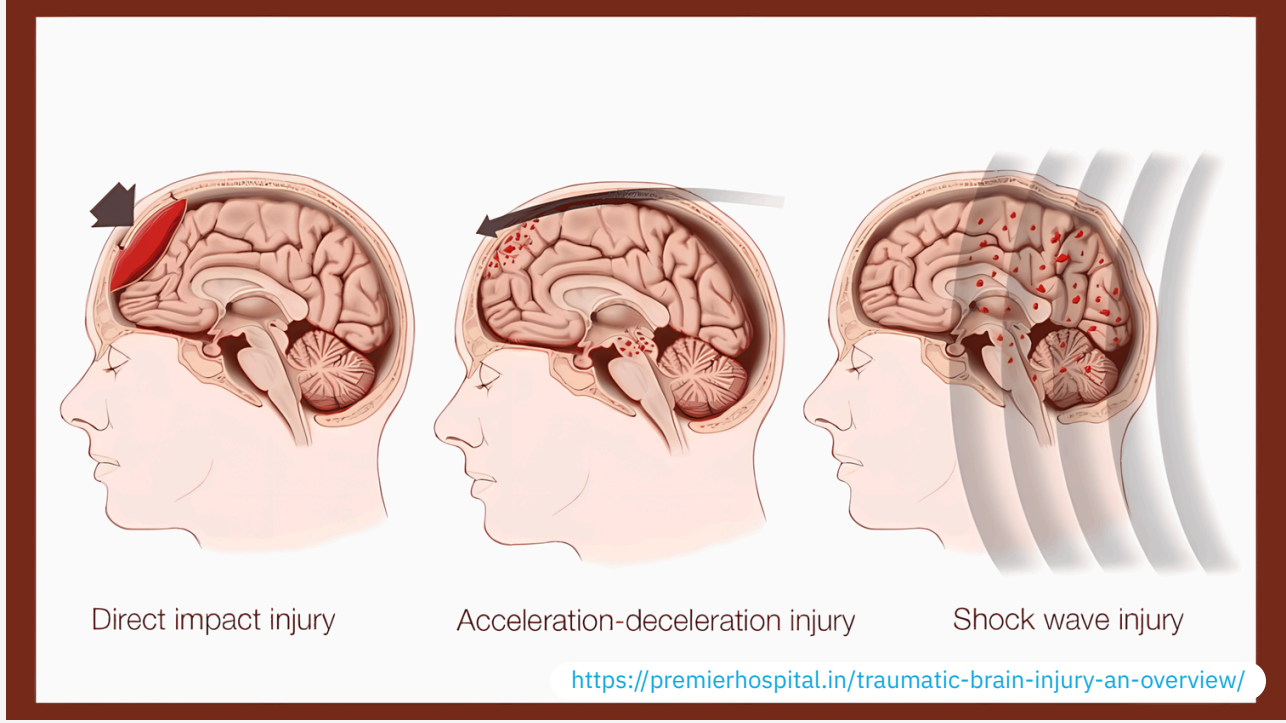
Properties of brain tissue

- Brain tissue is a soft, multicomponent solid with nonlinear, fluid-saturated, and largely incompressible poroviscoelastic properties. It is among the softest solid tissues in the body and demonstrates strain-dependent softening with repeated loading.
- Its mechanical behaviour reflects interaction between a solid matrix and a fluid phase composed of cerebrospinal fluid, interstitial fluid, and cerebral blood, all sensitive to osmotic and electrochemical changes.
- Mechanical properties vary between brain regions. The frontal, temporal, and occipital lobes are stiffer than the parietal lobe and cerebellum.
- White matter is significantly stiffer than grey matter, which is more compliant and less resistant to shear forces. These differences promote differential tissue movement during trauma.
- Regional and microstructural variability creates areas of mechanical mismatch, particularly at the grey–white matter junction. This predisposes these interfaces to injury and helps explain their frequent involvement in moderate to severe traumatic brain injury.

Orr TJ, Lesha E, Kramer AH, et al. Traumatic brain injury: a comprehensive review of biomechanics and molecular pathophysiology. *World Neurosurg*. 2024;185:74-88. doi:10.1016/j.wneu.2024.01.084

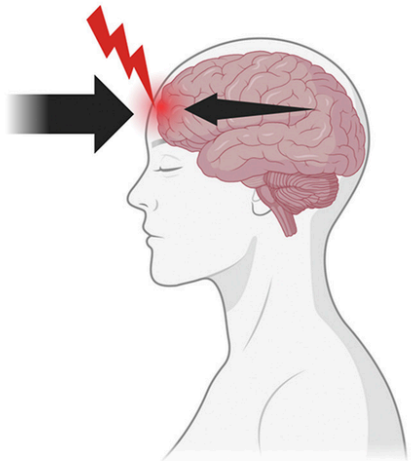
Biomechanics of Primary Injury

- Traumatic brain injury involves several categories of mechanical loading. These include impact loading, in which the head strikes or is struck by an object; blast overpressure, characterised by rapid pressure waves transmitted through brain tissue; and impulsive loading due to acceleration or deceleration of the head or body. In practice, these forces often occur together and are difficult to isolate.
- In closed-head injury, the dominant mechanisms are contact forces and inertial forces. Contact forces arise directly from impact and may cause focal skull damage at the site of contact or at distant regions, depending on energy magnitude and direction. At higher velocities, contact loading can result in skull fractures and associated injuries such as epidural haemorrhage, and is typically linked to focal brain injury in moderate to severe TBI.

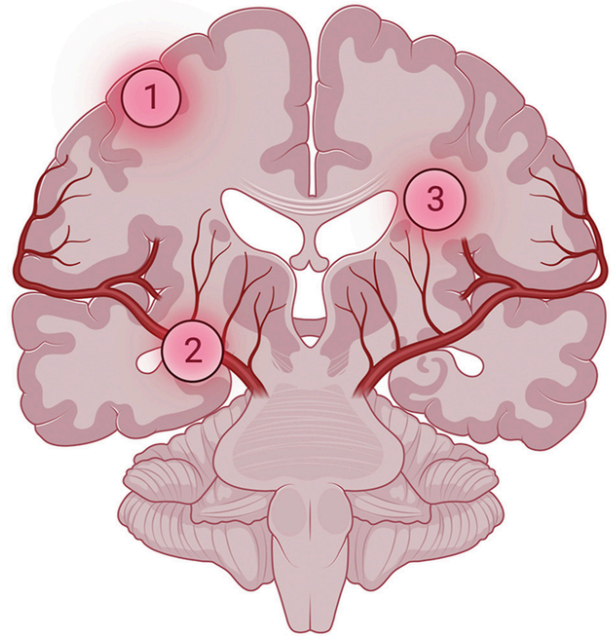


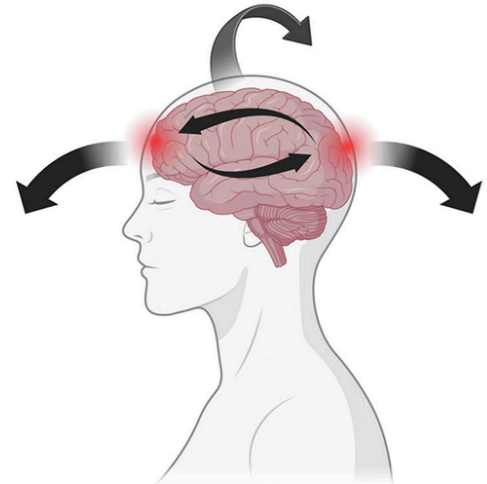
Inertial forces result from rapid acceleration or deceleration and act independently of direct contact. These forces produce widespread tissue deformation and are commonly associated with diffuse injuries, including subdural haematoma and diffuse axonal injury.

Orr TJ, Lesha E, Kramer AH, et al. Traumatic brain injury: a comprehensive review of biomechanics and molecular pathophysiology. *World Neurosurg*. 2024;185:74-88. doi:10.1016/j.wneu.2024.01.084

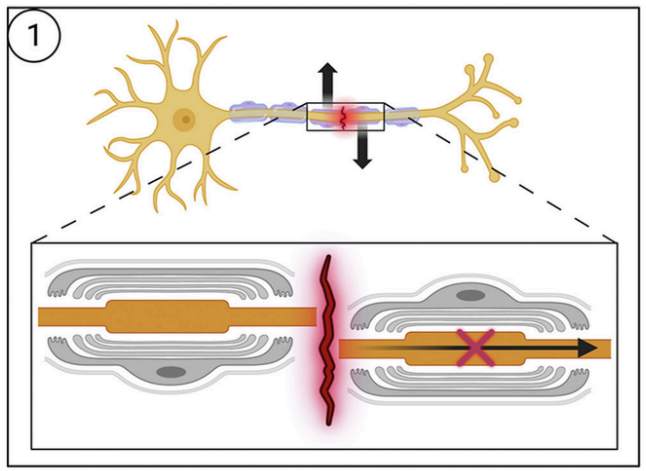


Direct Impact Loading

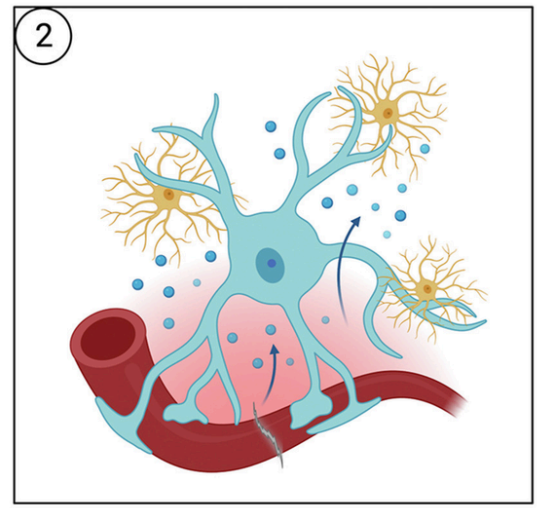




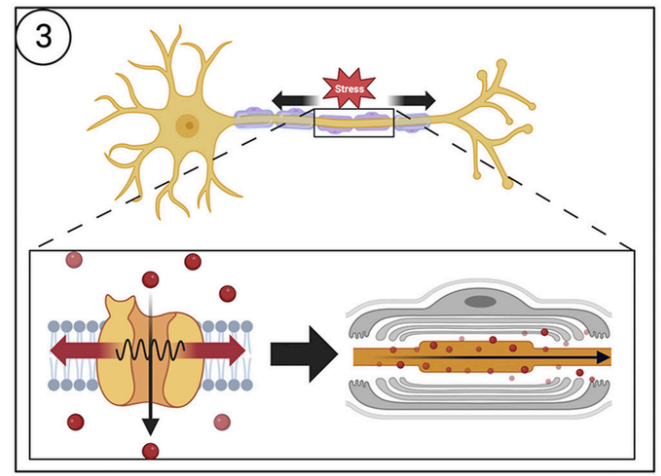
Rotational/Angular Inertia



Gray-White Matter Junction
Shearing Forces - Differences in stiffness leading to diffuse axonal injury

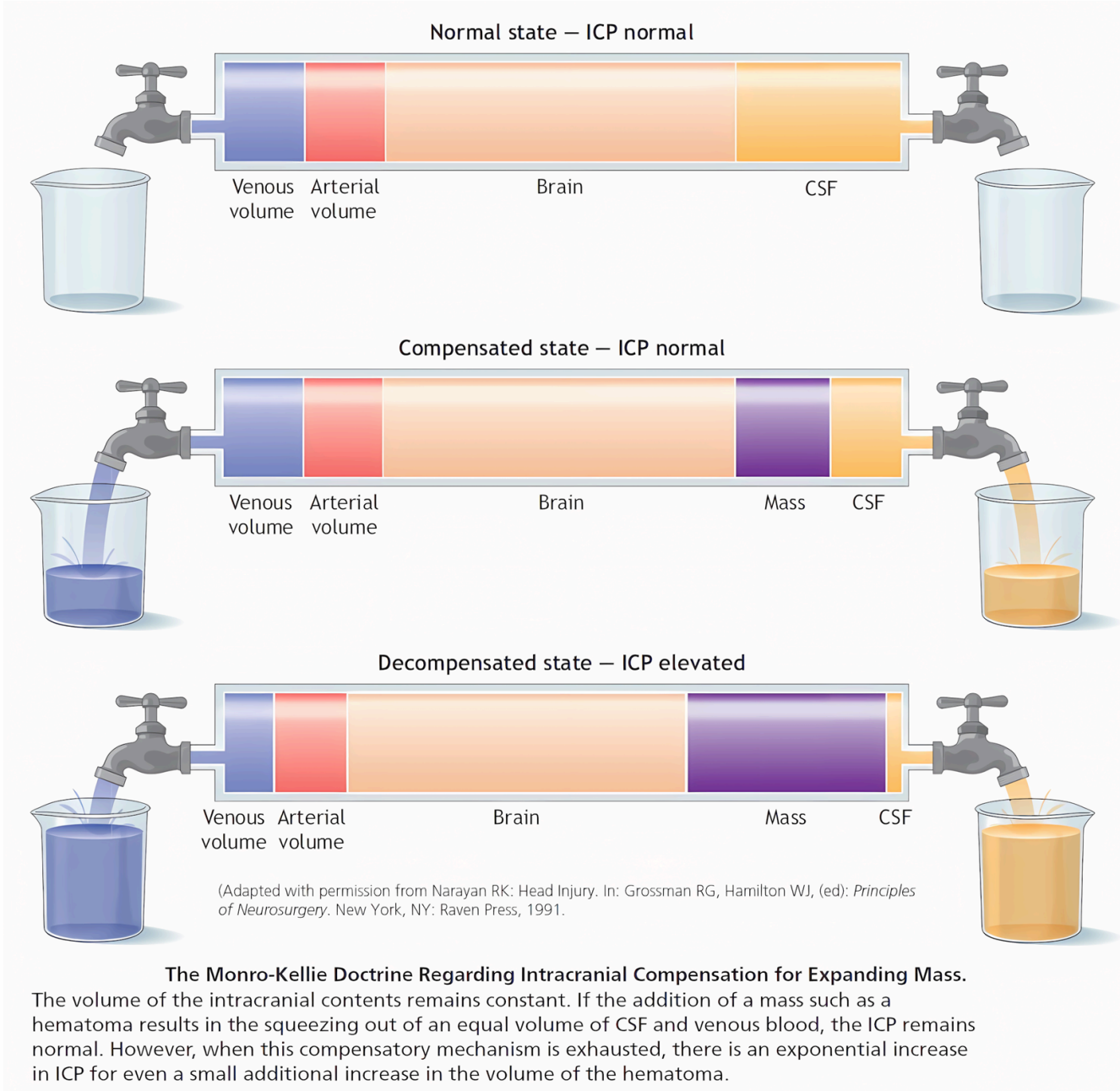


Cerebrovasculature
Shearing & Tensile Forces - Leads to BBB damage & neuroinflammation



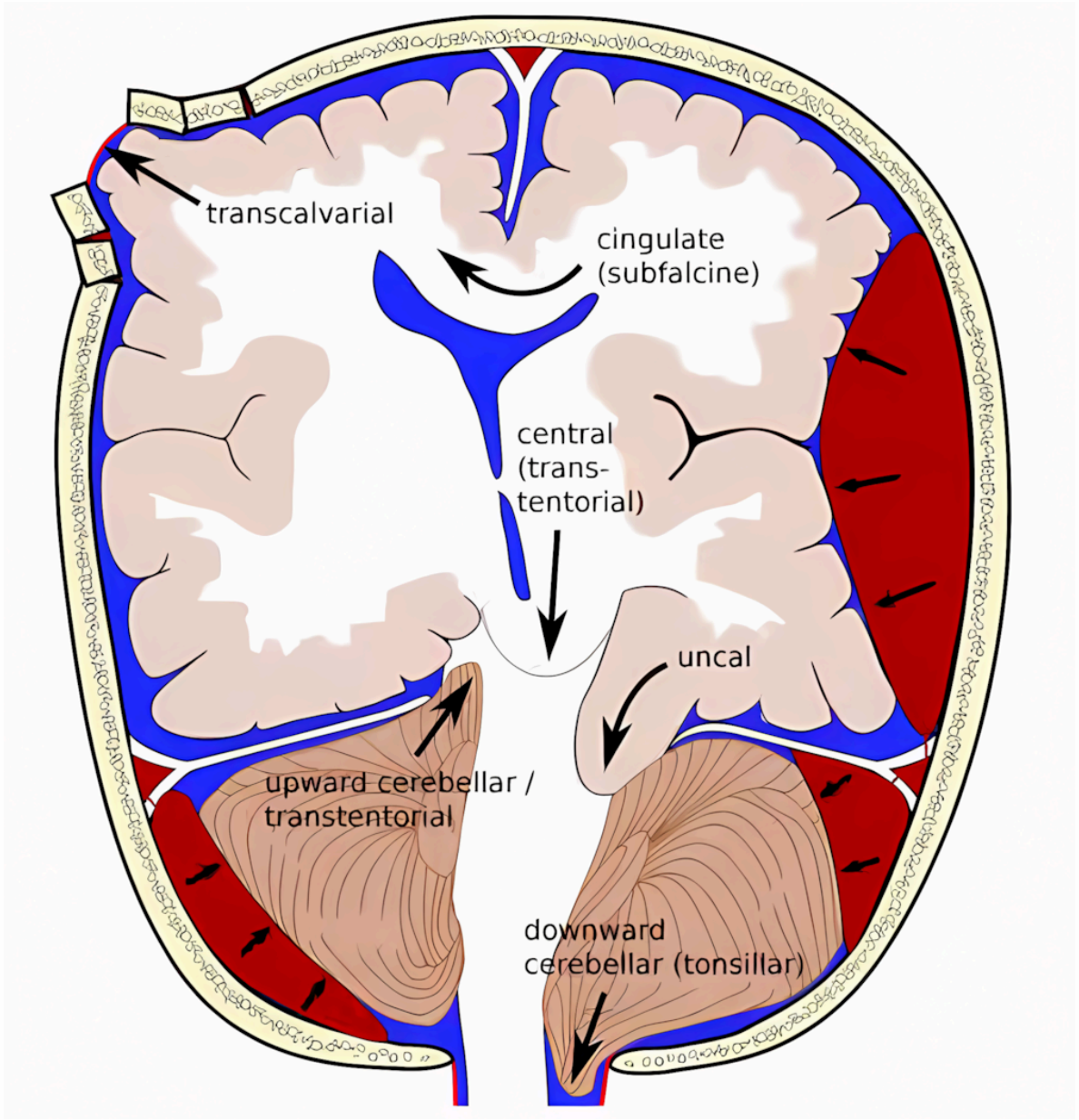
White Matter Axonal Tracts
Tensile Forces - Mechanotransduction leading to aberrant signaling & excitotoxicity

Orr TJ, Lesha E, Kramer AH, et al. Traumatic brain injury: a comprehensive review of biomechanics and molecular pathophysiology. *World Neurosurg*. 2024;185:74-88. doi:10.1016/j.wneu.2024.01.084

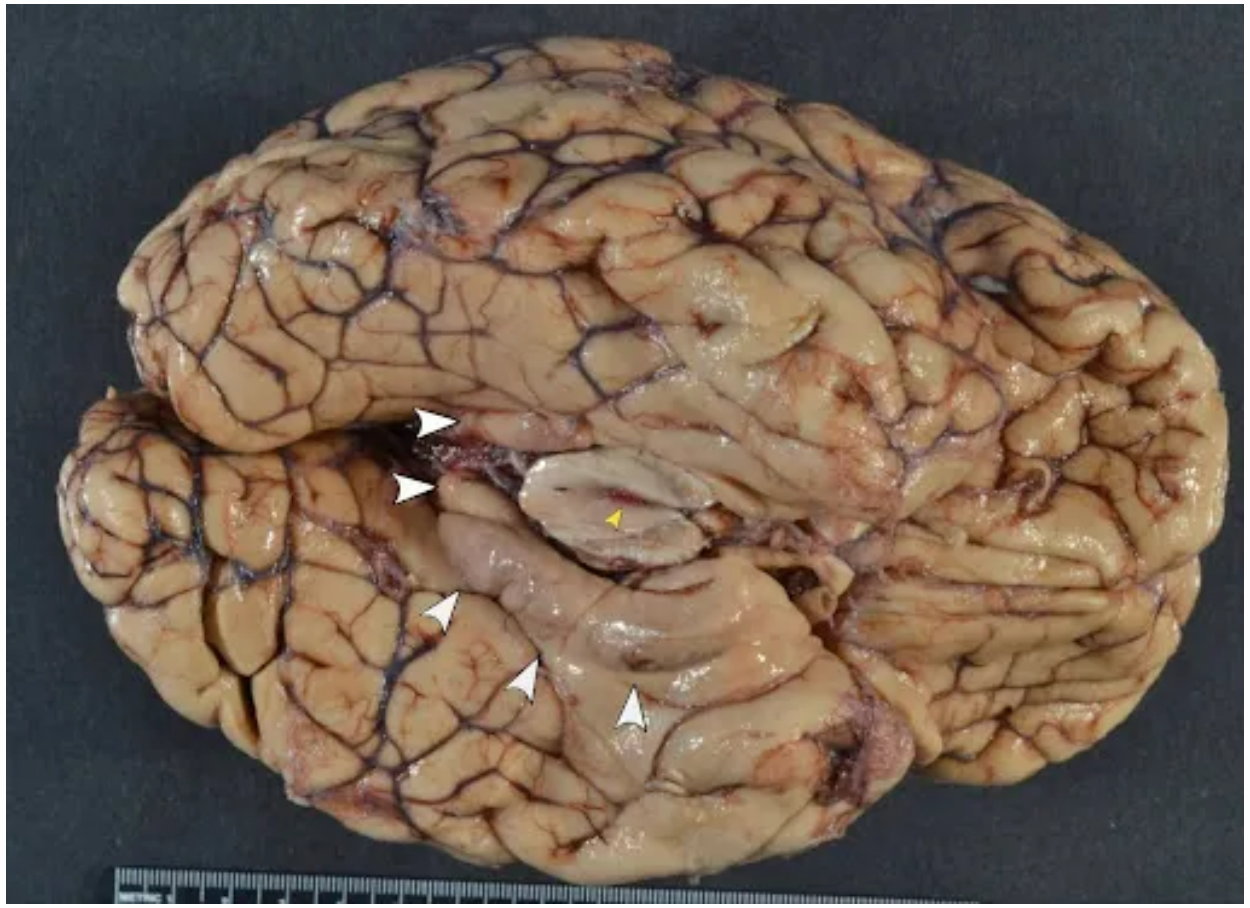


The Monro-Kellie Doctrine Regarding Intracranial Compensation for Expanding Mass.
The volume of the intracranial contents remains constant. If the addition of a mass such as a hematoma results in the squeezing out of an equal volume of CSF and venous blood, the ICP remains normal. However, when this compensatory mechanism is exhausted, there is an exponential increase in ICP for even a small additional increase in the volume of the hematoma.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



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Uncal herniation occurs when a lateral expanding mass in one cerebral hemisphere forces the medial temporal lobe to shift medially and downward through the tentorial notch.

- The cardinal clinical sign is an ipsilateral fixed and dilated pupil caused by compression of the oculomotor nerve. Impaired eye movements are usually present, although these may be difficult to assess in a reduced level of consciousness.
- Oculocephalic testing often reveals third nerve dysfunction even when voluntary eye movements cannot be assessed.
- A reduced level of consciousness is a key accompanying feature and reflects disruption of ascending arousal pathways in the midbrain or diencephalon, or ischaemia due to vascular distortion.
- The association between pupillary dilation and impaired consciousness is strong. A unilateral dilated pupil with preserved consciousness should prompt consideration of alternative causes, as this presentation is rare in uncal herniation.
- Hemiparesis may develop due to compression of the cerebral peduncle. Weakness may be contralateral or ipsilateral to the lesion because of midbrain displacement and Kernohan's notch phenomenon. For this reason, the side of motor deficit does not reliably localise the lesion.
- In contrast, the side of the dilated pupil identifies the side of herniation in more than 90 percent of cases.

Posner JB, Saper CB, Schiff ND, Claassen J. *Plum and Posner's Diagnosis and Treatment of Stupor and Coma*. 5th ed. Oxford University Press; 2019. doi:10.1093/med/9780190208875.001.0001



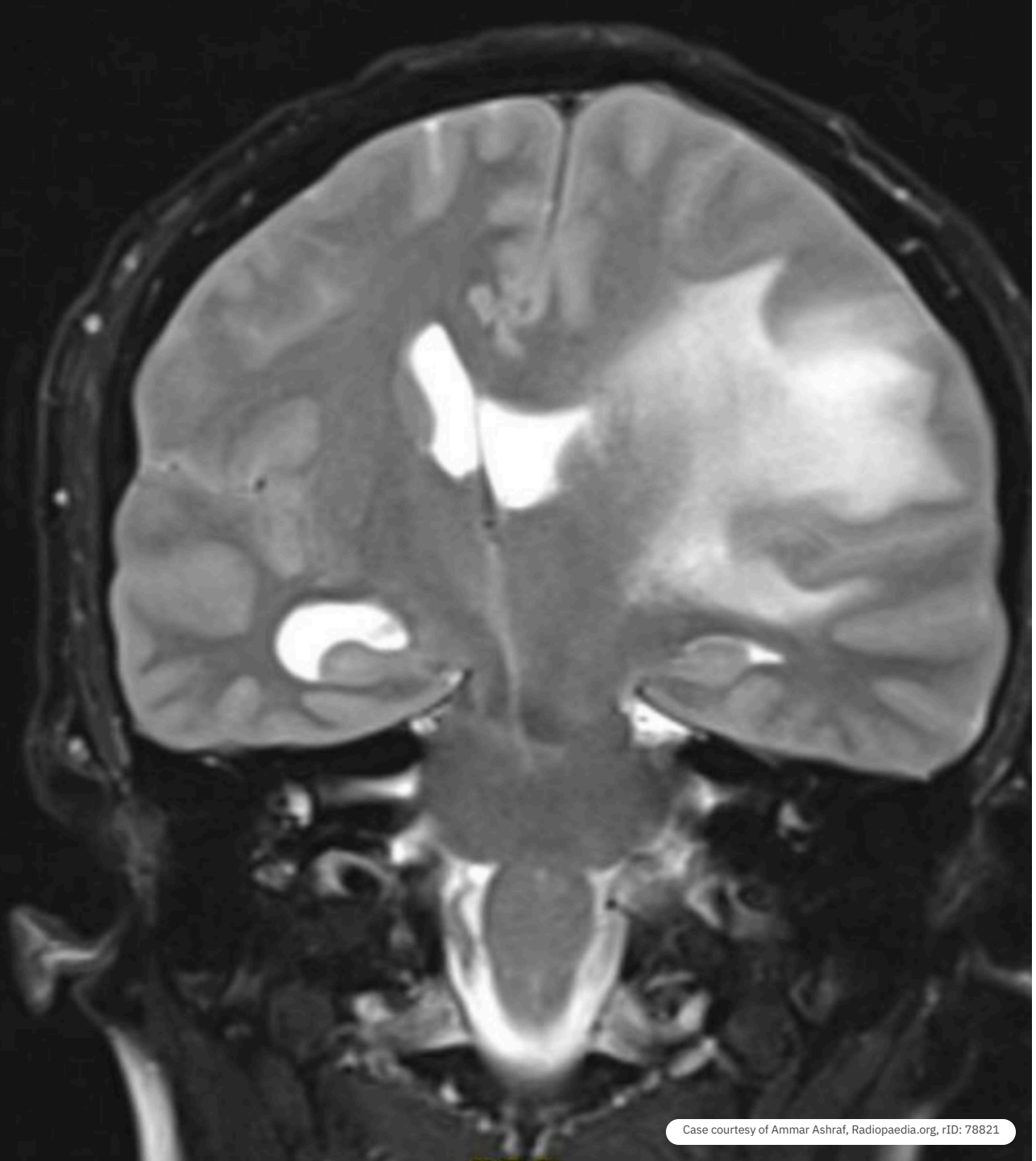
Videos from the "Adult NeuroLogic Exam" and "Pediatric NeuroLogic Exam" websites are used by permission of Paul D. Larsen, M.D., University of Nebraska Medical Center and Suzanne S. Stensaas, Ph.D., University of Utah School of Medicine. Additional materials were drawn from resources provided by Alejandro Stern, Stern Foundation, Buenos Aires, Argentina; Kathleen Digre, M.D., University of Utah; and Daniel Jacobson, M.D., Marshfield Clinic, Wisconsin. The movies are licensed under a Creative Commons Attribution-NonCommerical-ShareAlike License.





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Case courtesy of Ammar Ashraf, Radiopaedia.org, rID: 78821

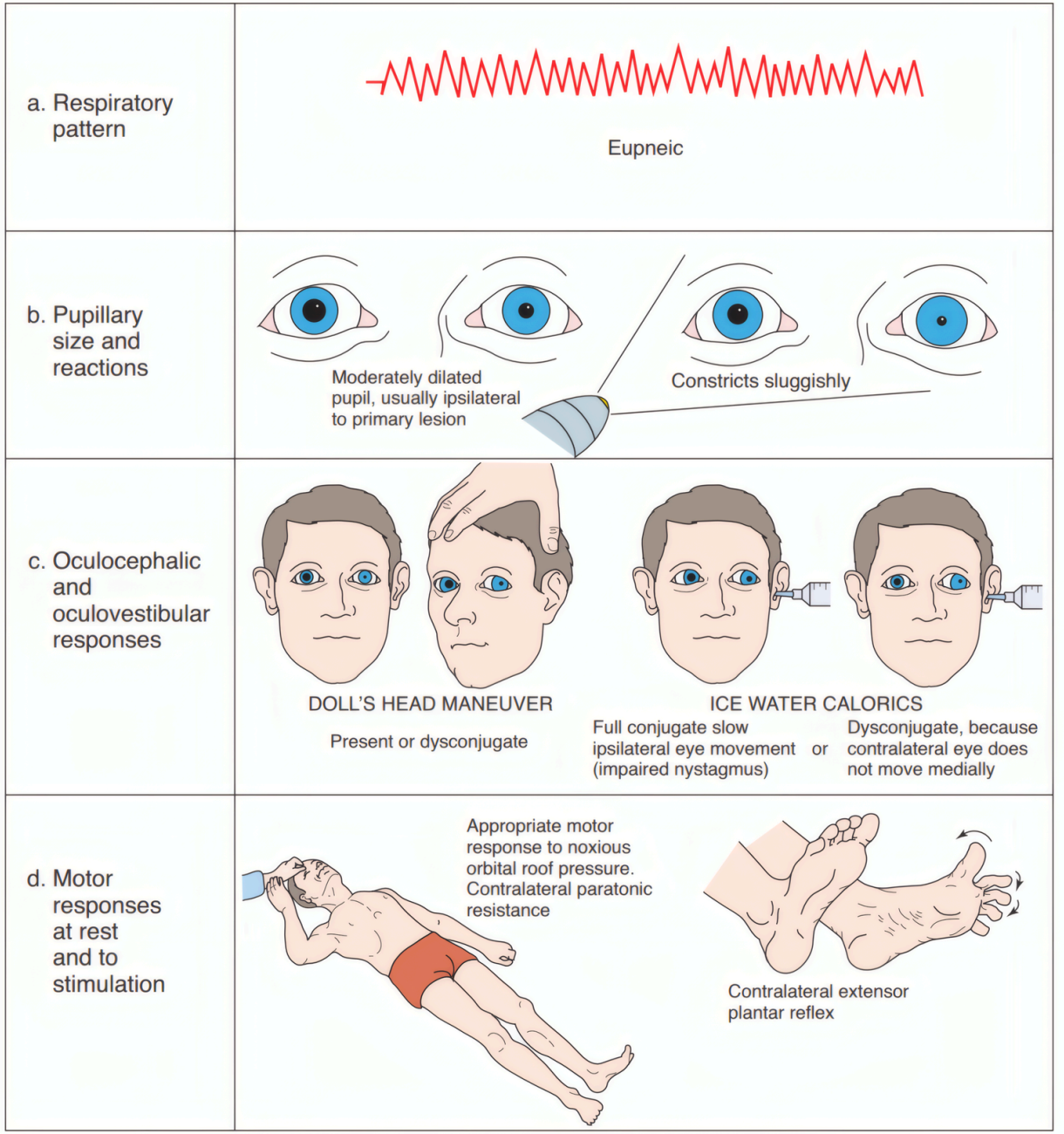


Figure 3–9. Signs of uncal herniation, early third nerve stage.

Levesque RJR. *Plum and Posner's Diagnosis of Stupor and Coma.* 4th ed. Oxford University Press USA - OSO; 2007.

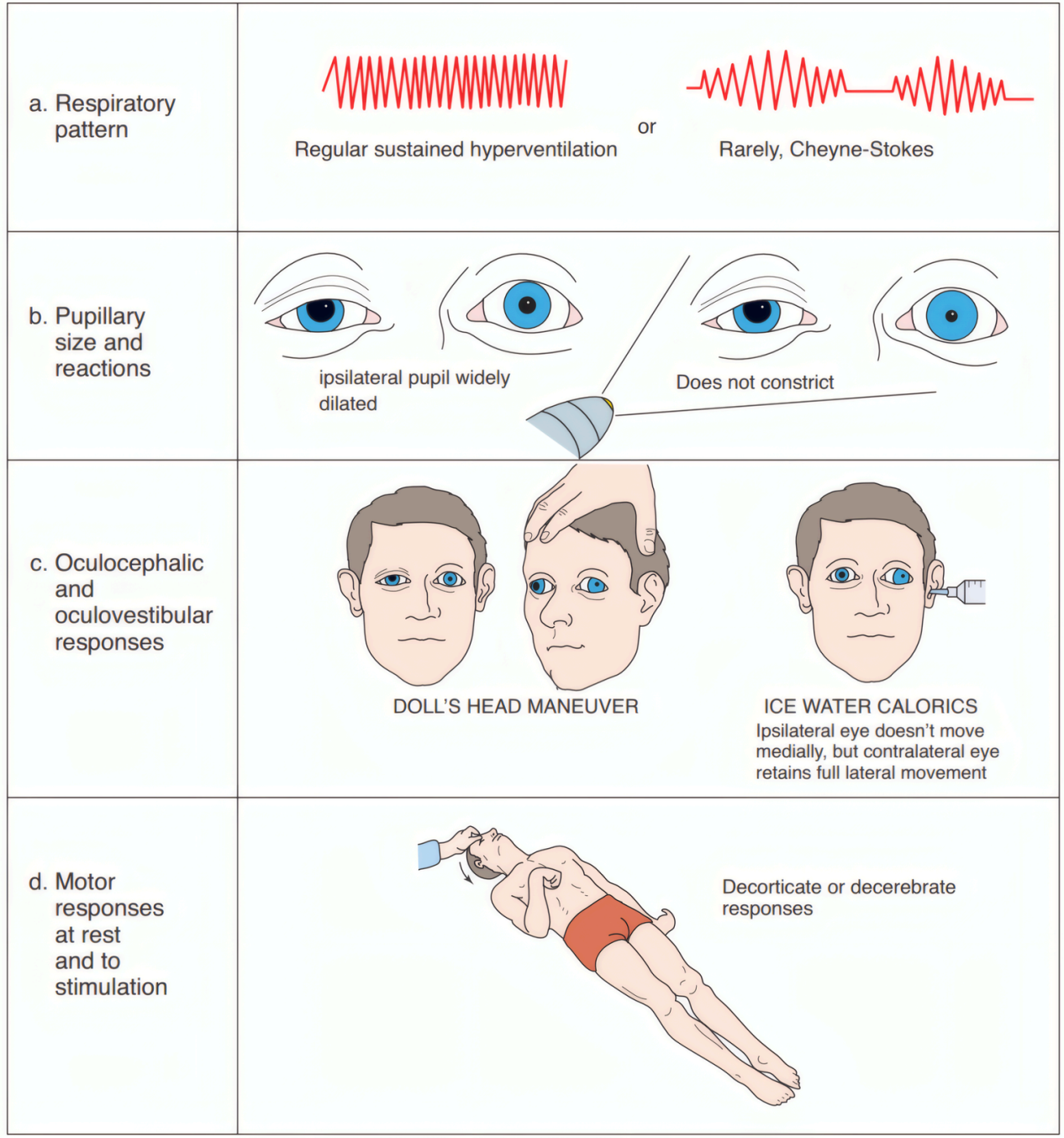
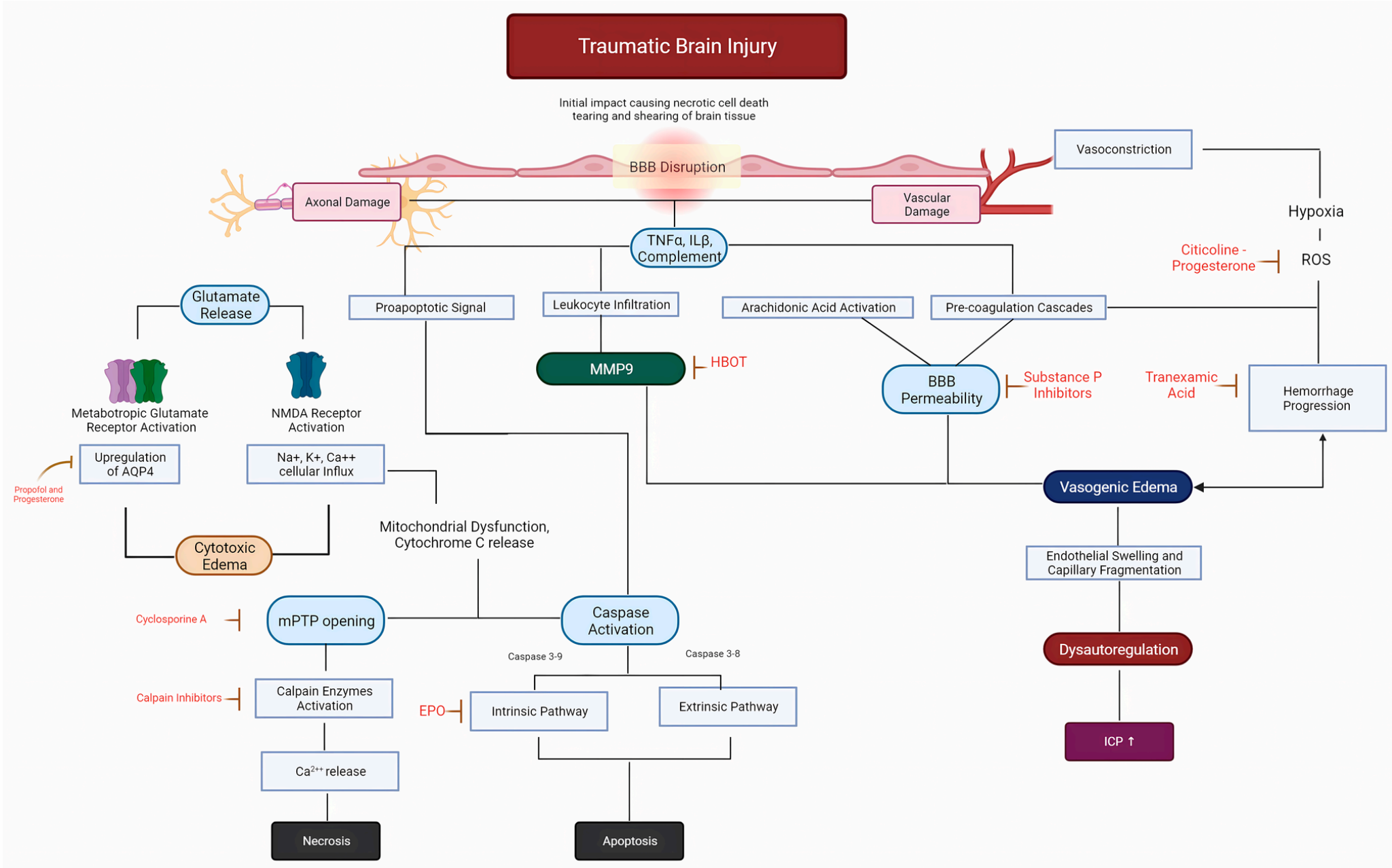
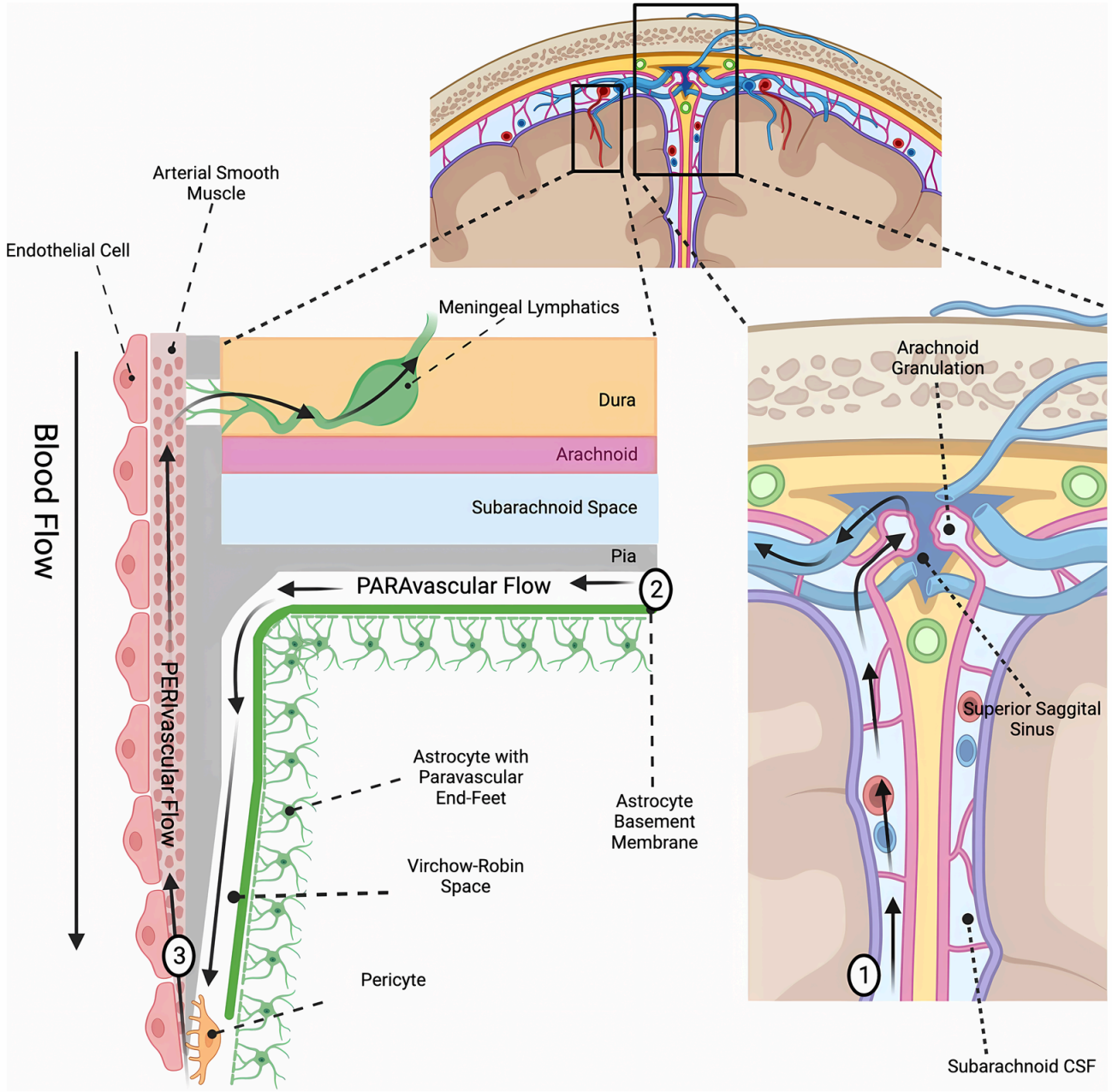


Figure 3–10. Signs of uncal herniation, late third nerve stage.

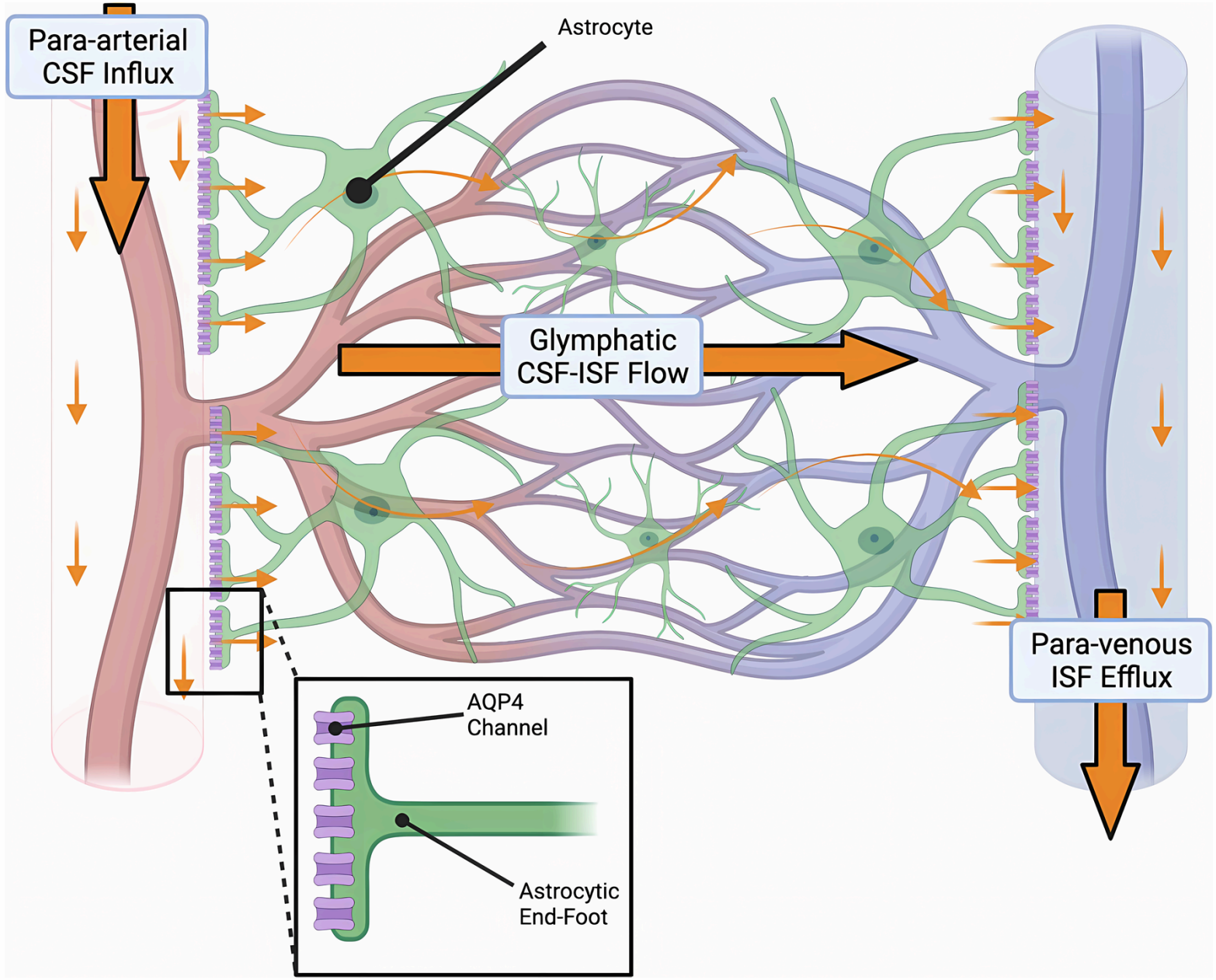
Levesque RJR. *Plum and Posner's Diagnosis of Stupor and Coma*. 4th ed. Oxford University Press USA - OSO; 2007.



de Macedo Filho L, Figueredo LF, Villegas-Gomez GA, et al. Pathophysiology-based management of secondary injuries and insults in TBI. *Biomedicines*. 2024;12(3):520. doi:10.3390/biomedicines12030520



Peters ME, Lyketsos CG. The glymphatic system's role in traumatic brain injury-related neurodegeneration. *Mol Psychiatry*. 2023;28(7):2707-2715. doi:10.1038/s41380-023-02070-7



Peters ME, Lyketsos CG. The glymphatic system's role in traumatic brain injury-related neurodegeneration. *Mol Psychiatry*. 2023;28(7):2707-2715. doi:10.1038/s41380-023-02070-7

Traumatic brain injury causes acute and persistent damage to the glymphatic system, with effects lasting beyond one month and contributing to long term neurodegeneration.

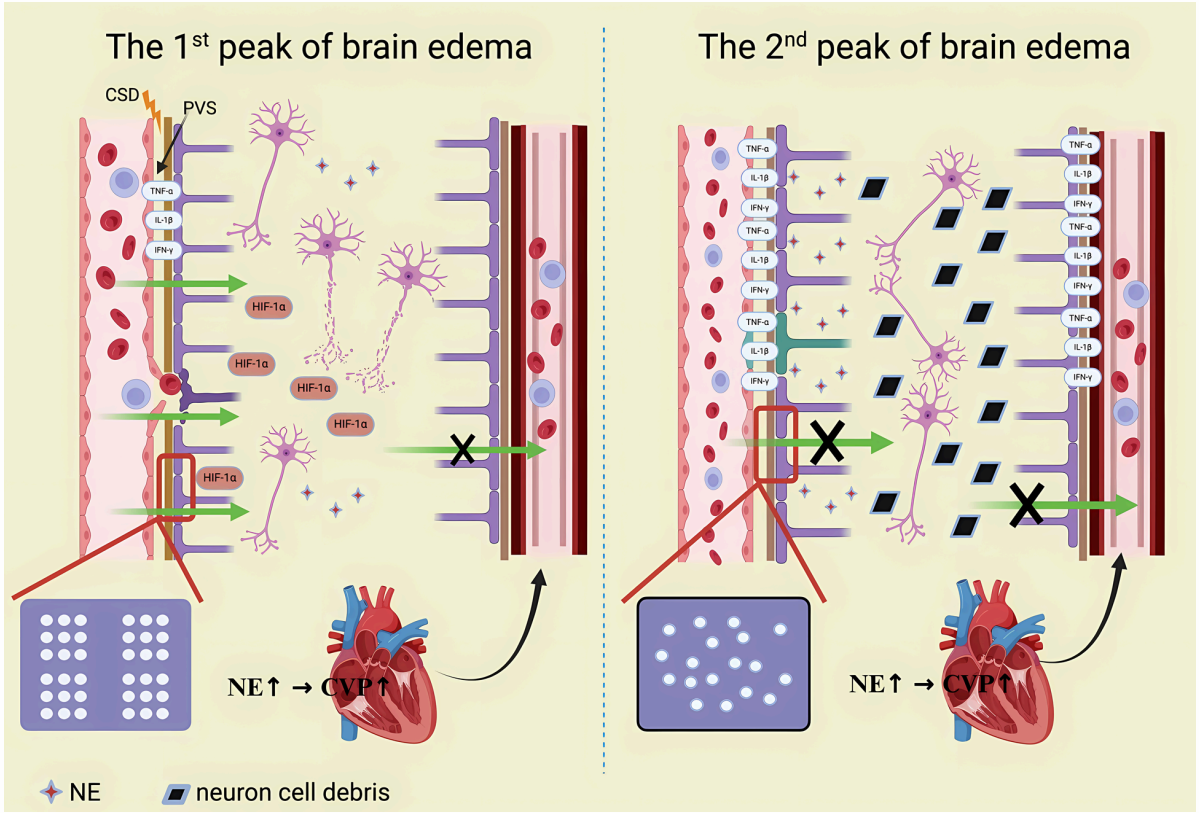
Mechanical forces injure the choroid plexus and ventricles, reduce cerebrospinal fluid production and distribution, and promote edema, inflammation, and astrogliosis.

Perivascular spaces, the main CSF inflow pathway, show reduced flow, structural disruption, and enlargement, which appear early, persist for years, and associate with impaired waste clearance, cognitive deficits, and sleep disturbance.

Neuroinflammation within the perivascular space further increases resistance to fluid movement. Astrocytes undergo reactive changes and scarring, altering water transport.

Aquaporin 4 expression increases after injury with time dependent effects.

Early AQP4 polarization contributes to cytotoxic edema, while sustained depolarization impairs glymphatic flow, maintains vasogenic edema, and reduces protein clearance. These processes follow a biphasic edema pattern and support time specific therapeutic targeting of glymphatic dysfunction.



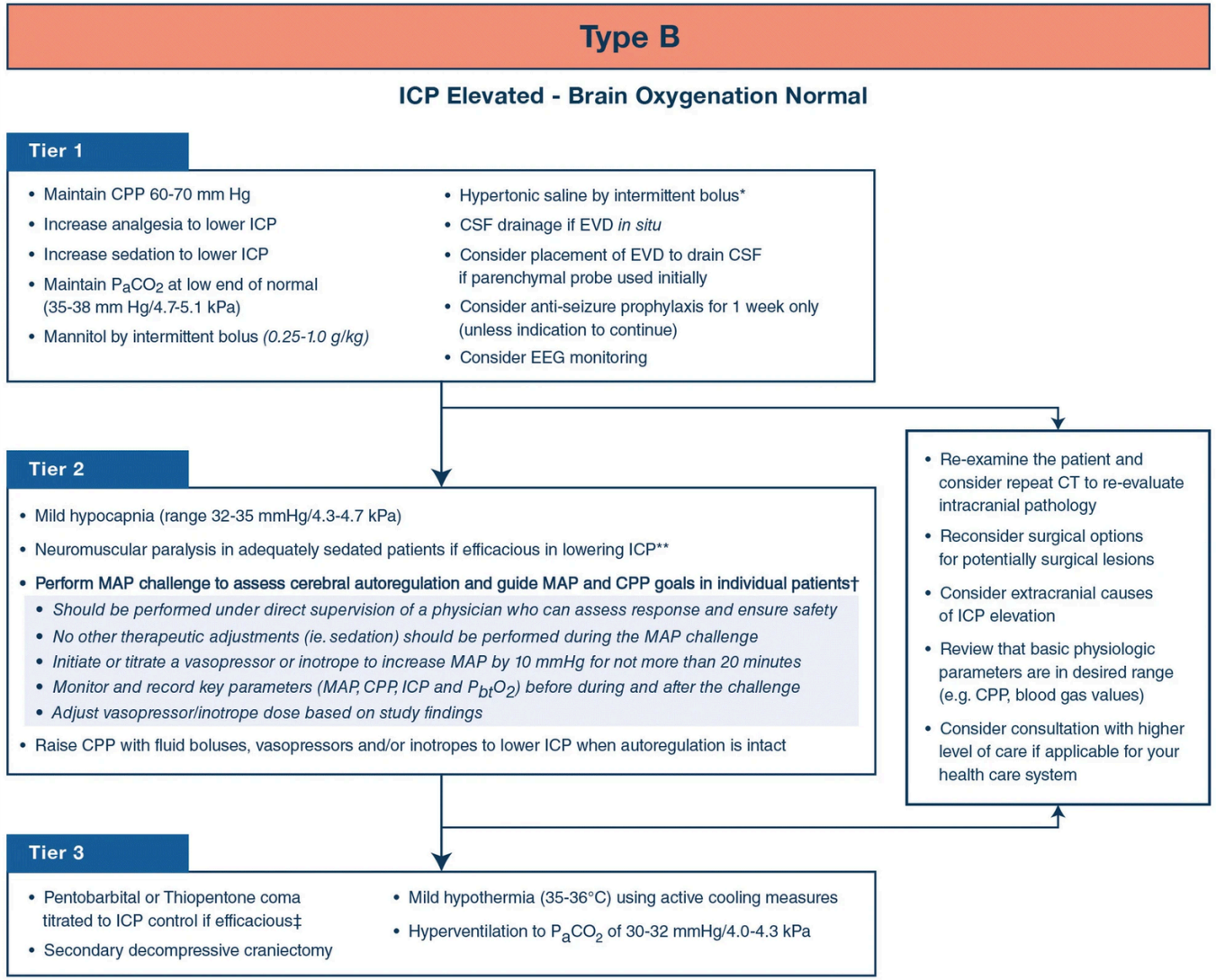
Yang Z, Li P, Wei J. Perceiving traumatic brain injury from glymphatic system. Mol Psychiatry. 2025;30(11):5546-5562. doi:10.1038/s41380-025-03126-6

Optimal Values in TBI Management

Category	Parameter	Optimal Value
Clinical Parameters	Systolic blood pressure	≥100 mm Hg
	Mean arterial pressure	>80 mm Hg
	Temperature	36–38°C
	Pulse oximetry	≥94%
Laboratory Parameters	Glucose	100–180 mg/dL
	Hemoglobin	> 7 g/dl
	International normalized ratio (INR)	≤1.4
	Serum sodium	135–145 meq/dL
	Serum osmolality	≤320 mOsm
	PaO_2	80–100 mm Hg
	$PaCO_2$	35–45 mm Hg
	pH	7.35–7.45
	Platelets	≥75 X 103 mm^3
Neurologic Monitoring Parameters	Cerebral perfusion pressure	60–70 mm Hg*
	Intracranial pressure	<22 mm Hg*
	$PbtO_2$	≥15 mm Hg

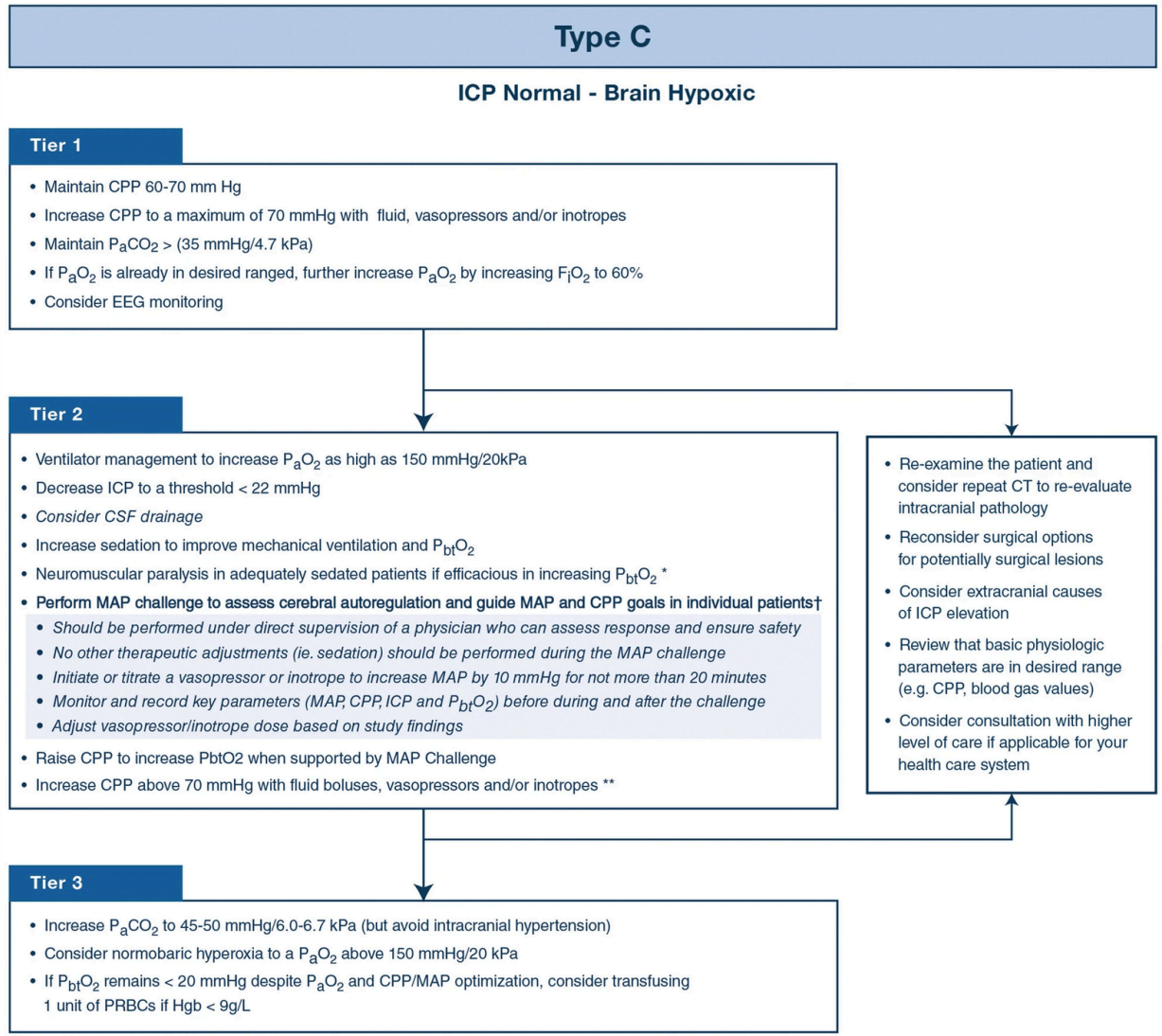
TBI, Traumatic brain injury; PbtO2, Partial brain tissue oxygenation.

American College of Surgeons Committee on Trauma. *Advanced Trauma Life Support®: Standardized Trauma Care When Seconds Count*. 11th ed. American College of Surgeons; 2025.



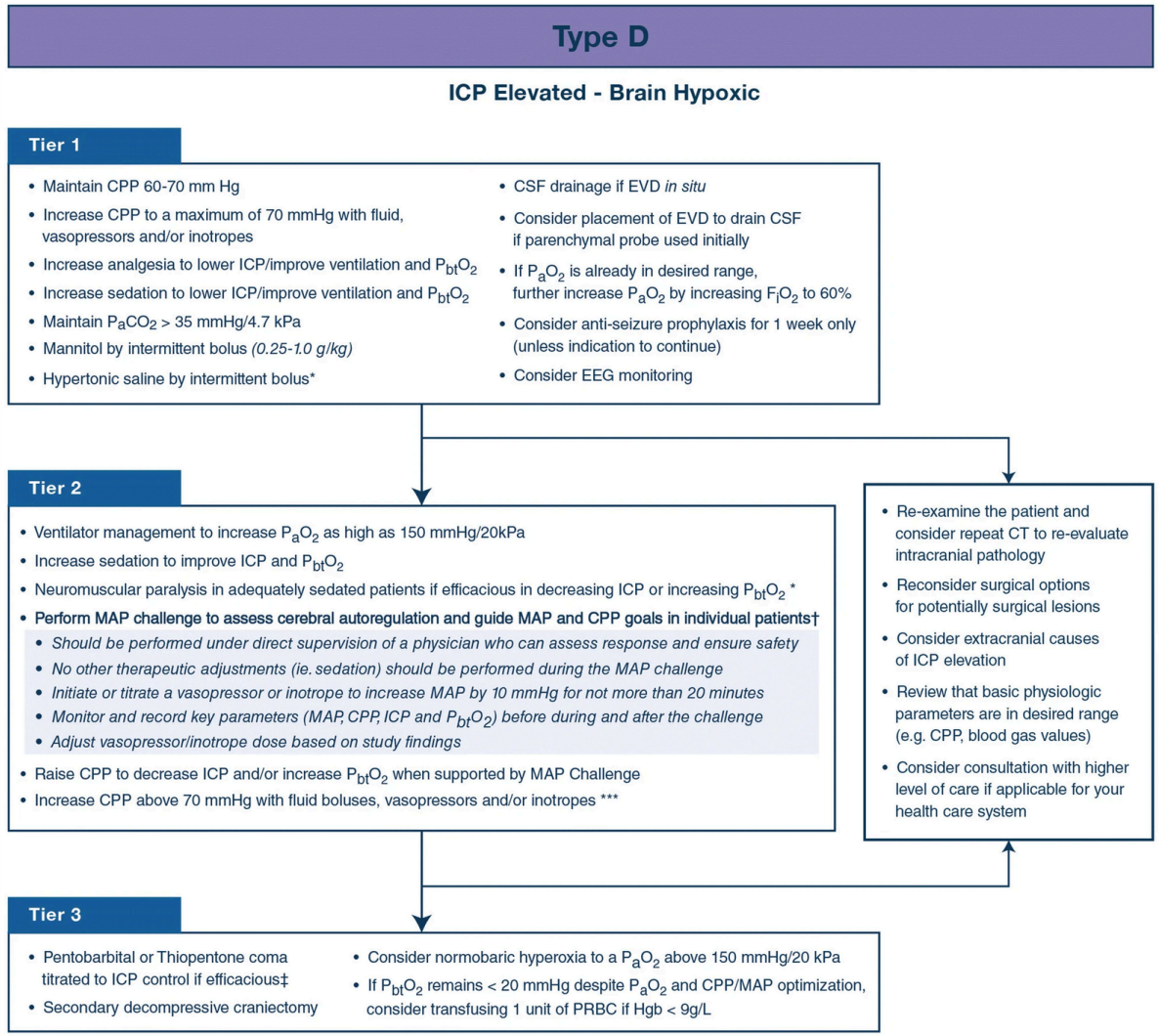
* We recommend using sodium and osmolality limits of 155 mEq/L and of 320 mEq/L respectively as administration limits for both mannitol and hypertonic saline.
** We recommend a trial dose of neuromuscular paralysis and only proceeding to a continuous infusion when efficacy is demonstrated.
† Rosenthal G. et al 2011
‡ Barbiturate administration should only be continued when a beneficial effect on ICP is demonstrated

Chesnut R, Aguilera S, Buki A, et al. A management algorithm for adult patients with both brain oxygen and intracranial pressure monitoring: the seattle international severe traumatic brain injury consensus conference (SIBICC). *Intensive Care Med*. 2020;46(5):919-929. doi:10.1007/s00134-019-05900-x



* We recommend a trial dose of neuromuscular paralysis and only proceeding to a continuous infusion when efficacy is demonstrated.
** Careful monitoring for respiratory complications is required when CPP is raised above 70 mmHg (Robertson et al, 1999)
† Rosenthal G. et al 2011

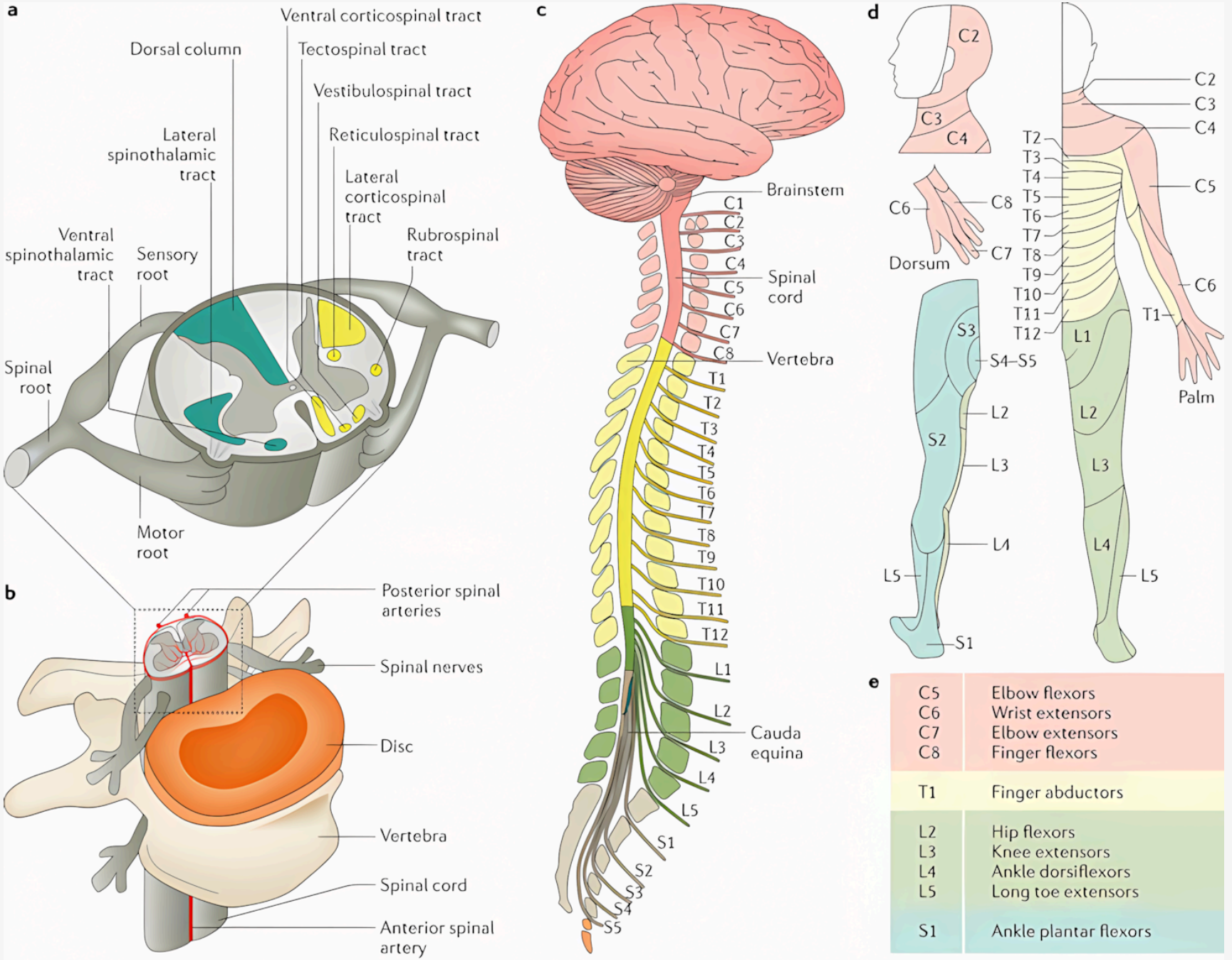
Chesnut R, Aguilera S, Buki A, et al. A management algorithm for adult patients with both brain oxygen and intracranial pressure monitoring: the seattle international severe traumatic brain injury consensus conference (SIBICC). *Intensive Care Med*. 2020;46(5):919-929. doi:10.1007/s00134-019-05900-x



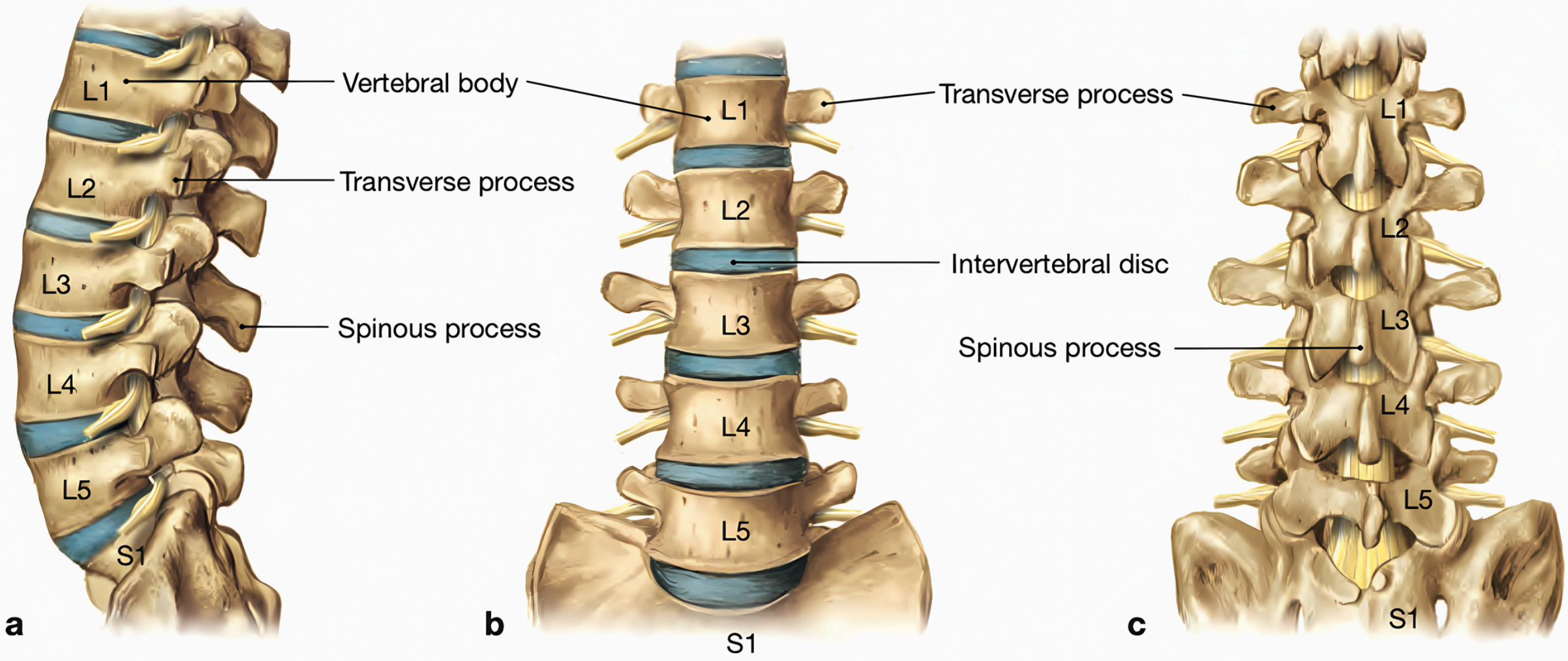
* We recommend using sodium and osmolality limits of 155 mEq/L and of 320 mEq/L respectively as administration limits for both mannitol and hypertonic saline.
** We recommend a trial dose of neuromuscular paralysis and only proceeding to a continuous infusion when efficacy is demonstrated.
† Rosenthal G. et al 2011
‡ Barbiturate administration should only be continued when a beneficial effect on ICP is demonstrated.
Titrate barbiturate to achieve ICP control but do not exceed the dose which achieves burst suppression.
Hypotension must be avoided when barbiturates are administered.

Chesnut R, Aguilera S, Buki A, et al. A management algorithm for adult patients with both brain oxygen and intracranial pressure monitoring: the seattle international severe traumatic brain injury consensus conference (SIBICC). *Intensive Care Med*. 2020;46(5):919-929. doi:10.1007/s00134-019-05900-x

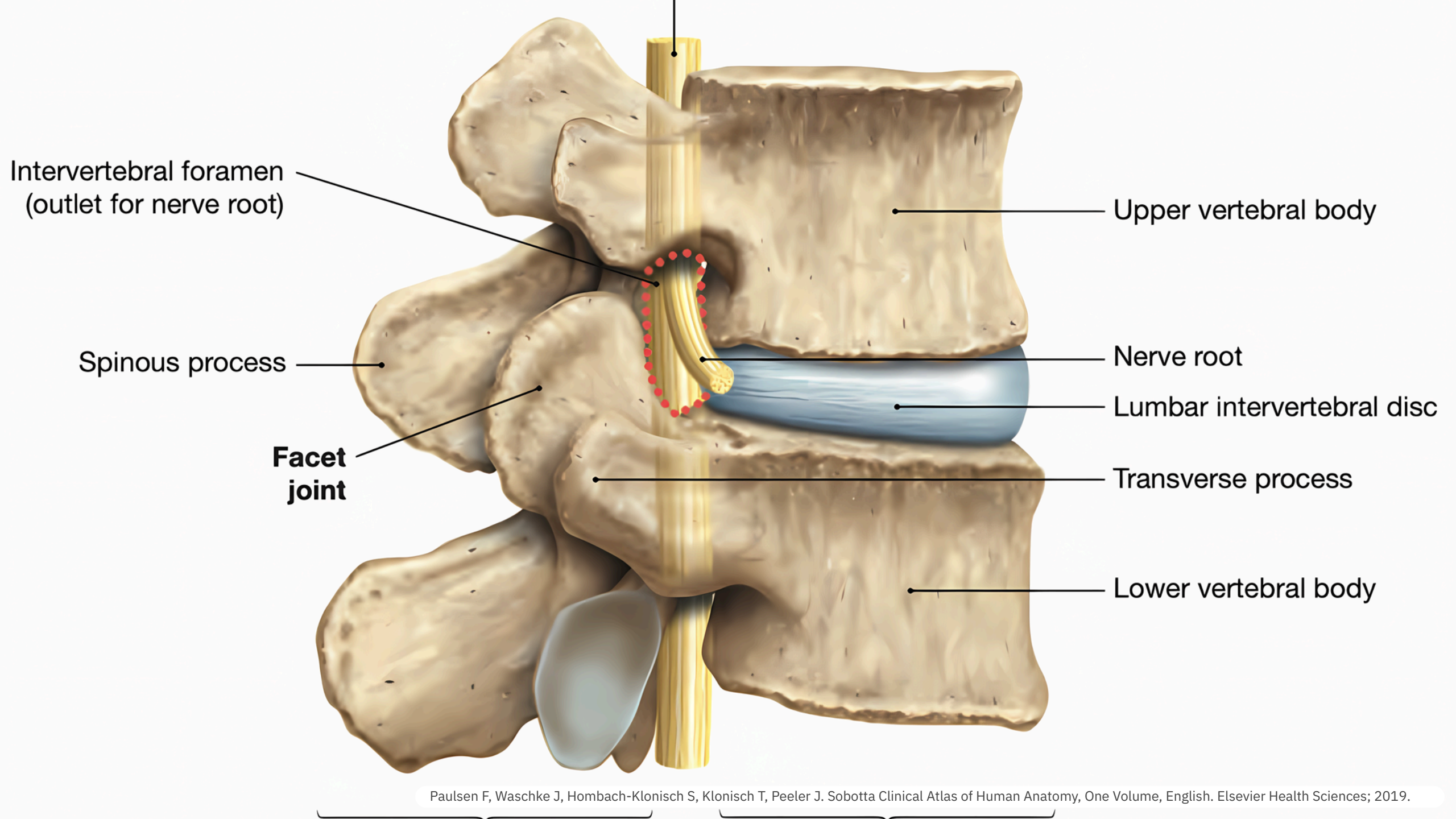
Spinal injury



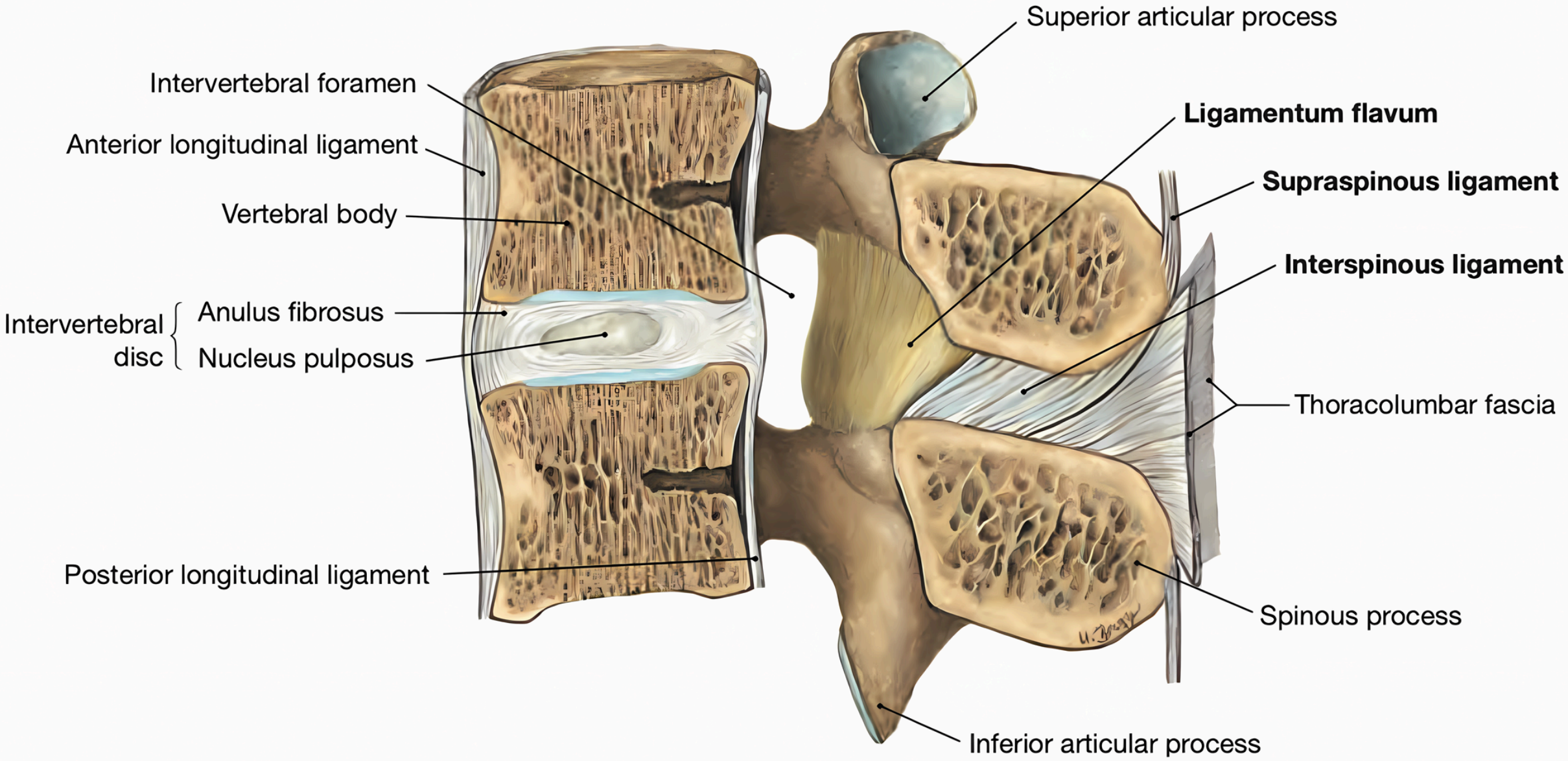
Ahuja CS, Wilson JR, Nori S, et al. Traumatic spinal cord injury. *Nat Rev Dis Primers*. 2017;3(1):17018. doi:10.1038/nrdp.2017.18



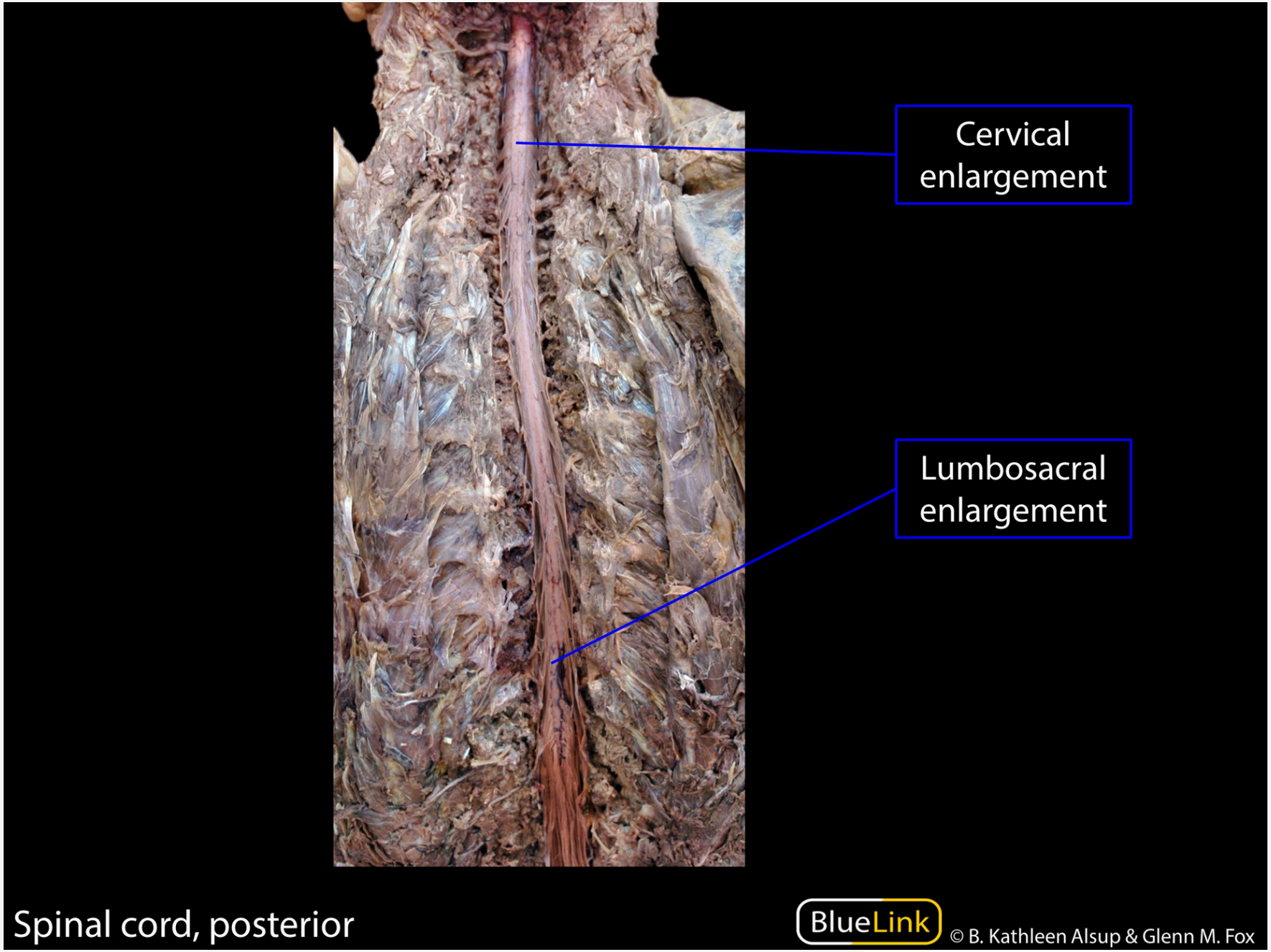
Paulsen F, Waschke J, Hombach-Klonisch S, Klonisch T, Peeler J. Sobotta Clinical Atlas of Human Anatomy, One Volume, English. Elsevier Health Sciences; 2019.



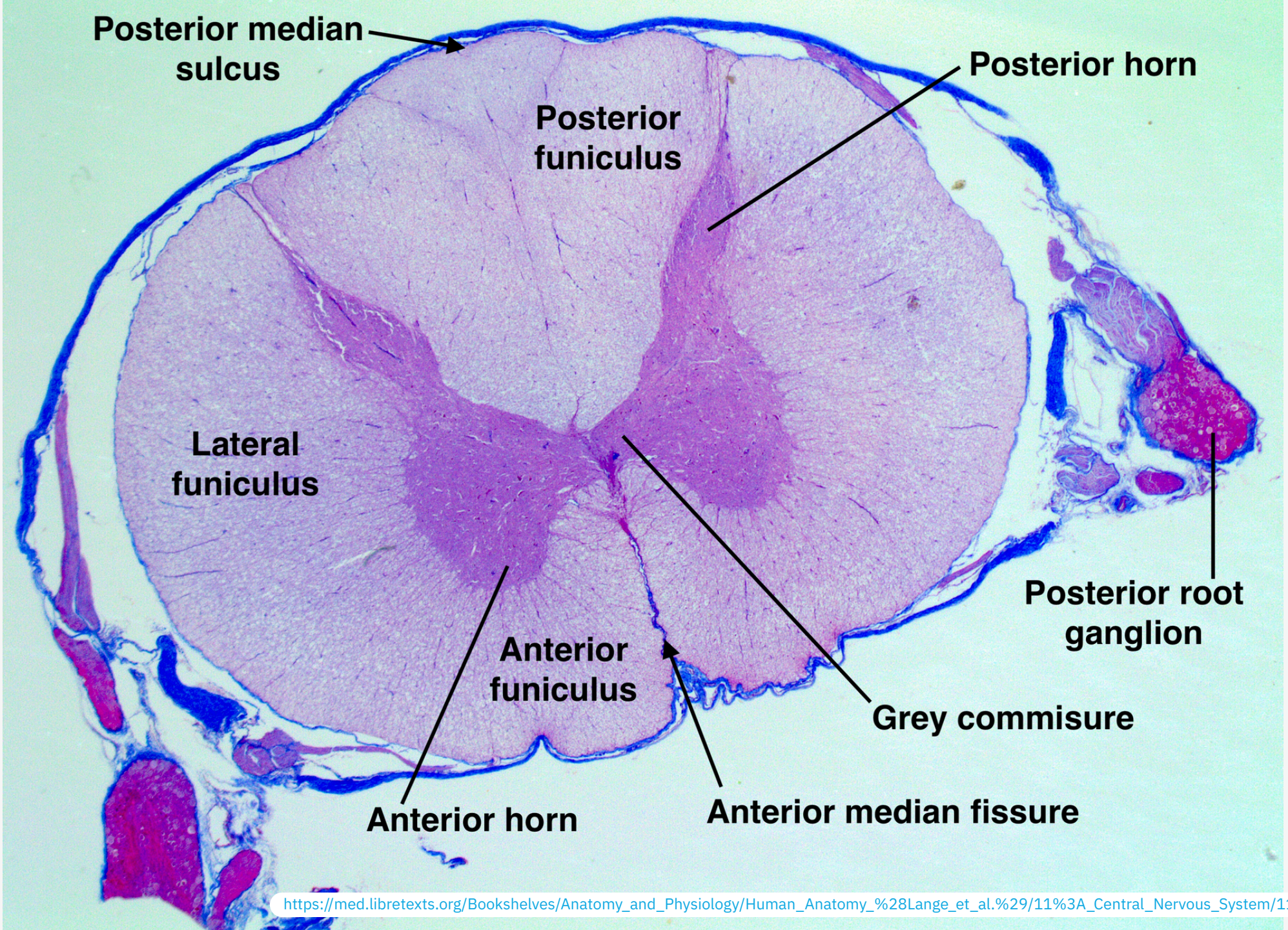
Paulsen F, Waschke J, Hombach-Klonisch S, Klonisch T, Peeler J. Sobotta Clinical Atlas of Human Anatomy, One Volume, English. Elsevier Health Sciences; 2019.



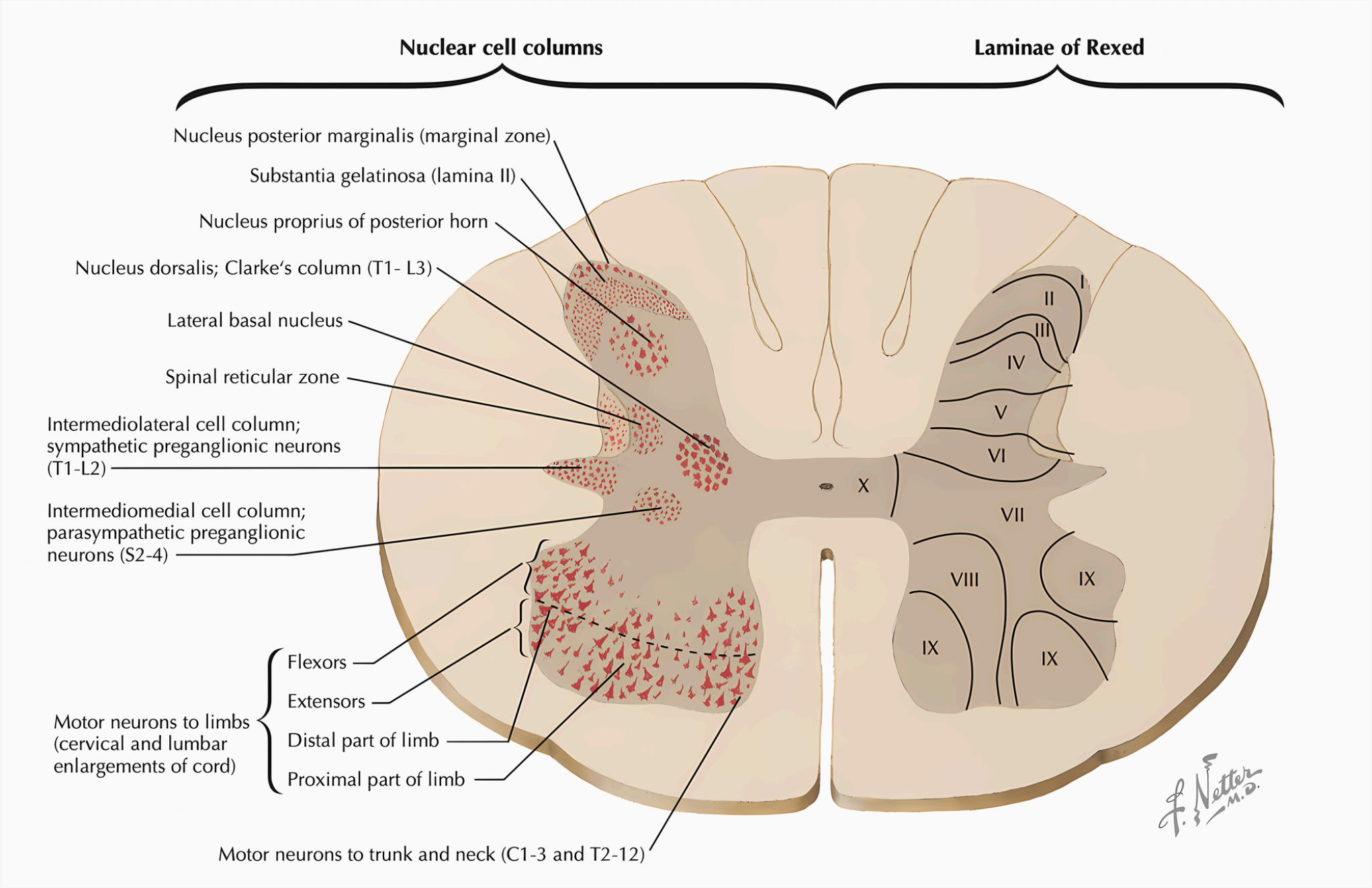
Paulsen F, Waschke J, Hombach-Klonisch S, Klonisch T, Peeler J. Sobotta Clinical Atlas of Human Anatomy, One Volume, English. Elsevier Health Sciences; 2019.



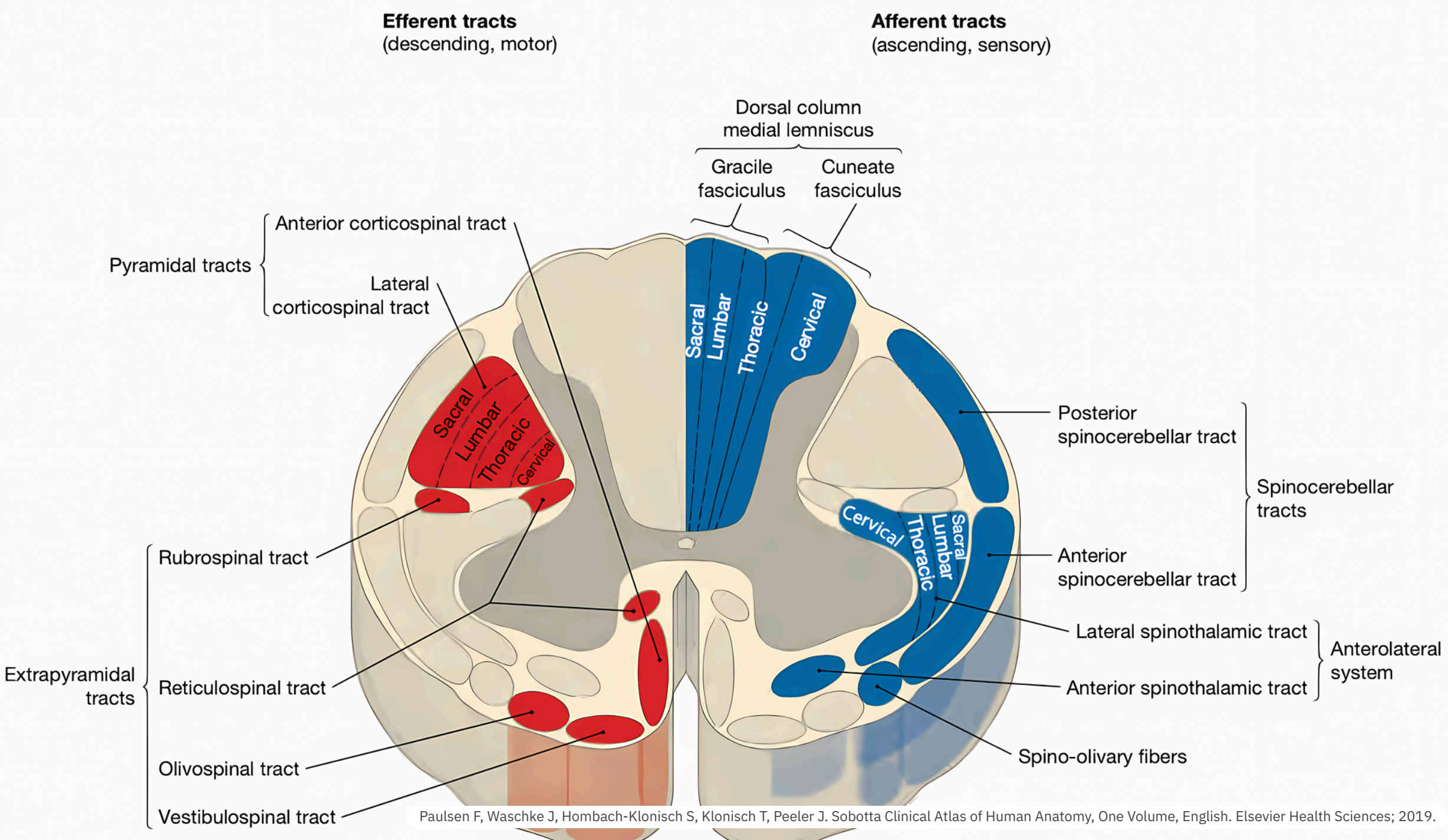
https://wisc.pb.unizin.org/virtualhumananatomylabmanual/chapter/__unknown__-2/



https://med.libretexts.org/Bookshelves/Anatomy_and_Physiology/Human_Anatomy_%28Lange_et_al.%29/11%3A_Central_Nervous_System/11.05%3A_Spinal_Cord

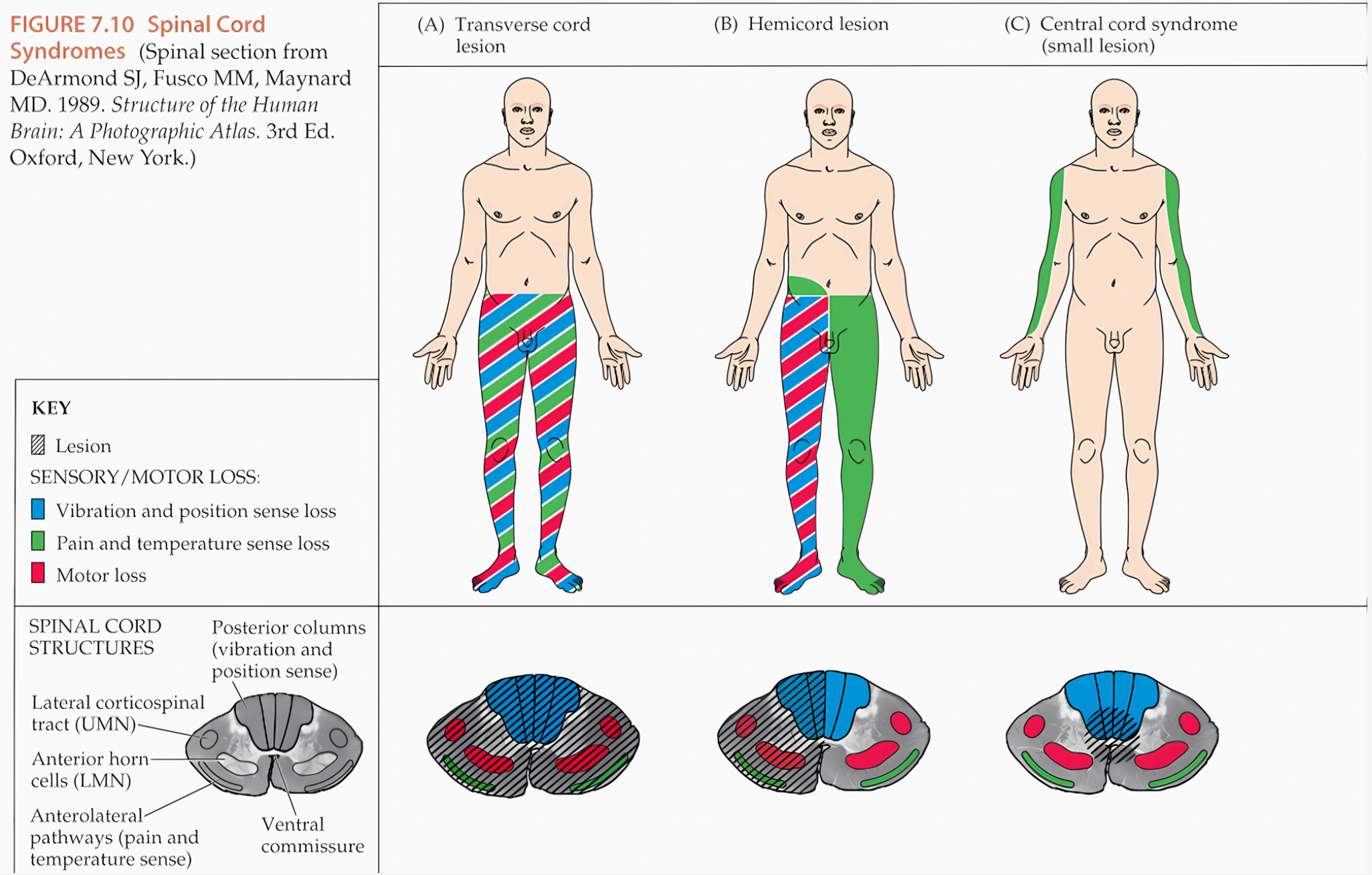


Felten DL, O'Banion MK, Maida ME. Netter's Atlas of Neuroscience. Elsevier Health Sciences; 2015.

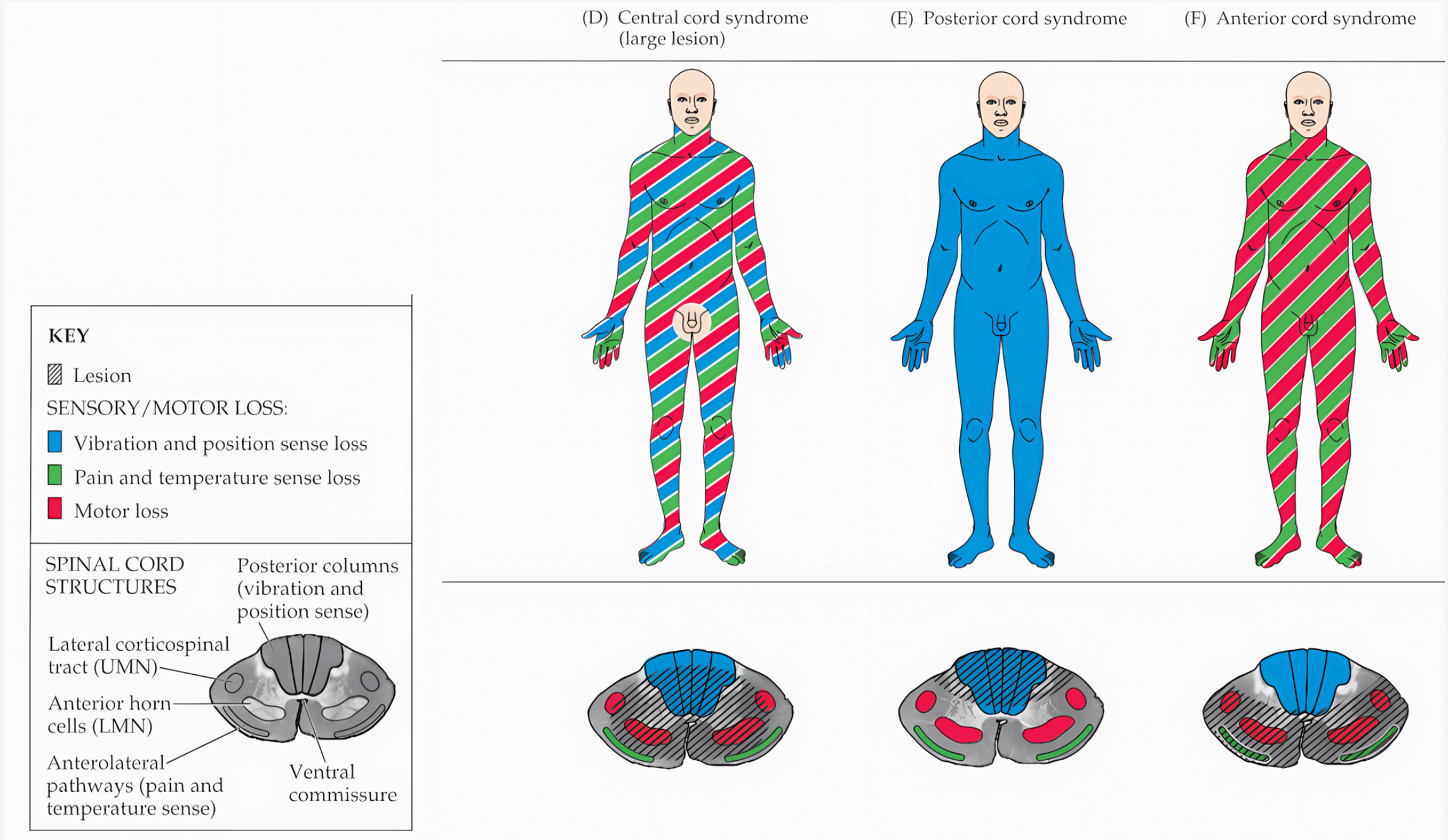


Paulsen F, Waschke J, Hombach-Klonisch S, Klonisch T, Peeler J. Sobotta Clinical Atlas of Human Anatomy, One Volume, English. Elsevier Health Sciences; 2019.

FIGURE 7.10 Spinal Cord Syndromes (Spinal section from DeArmond SJ, Fusco MM, Maynard MD. 1989. *Structure of the Human Brain: A Photographic Atlas.* 3rd Ed. Oxford, New York.)

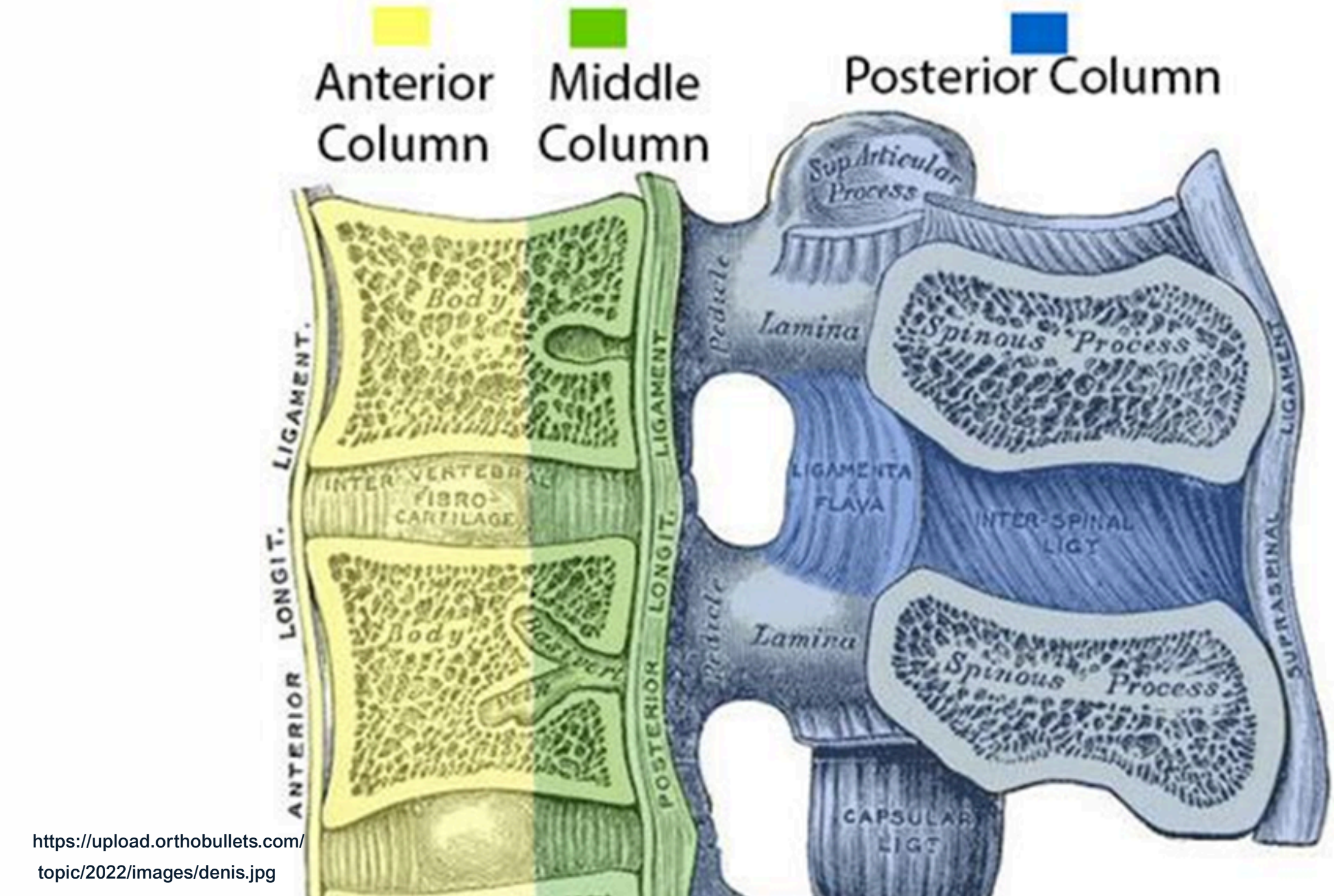


Blumenfeld H. Neuroanatomy through Clinical Cases with Ebook. Sinauer; 2011.



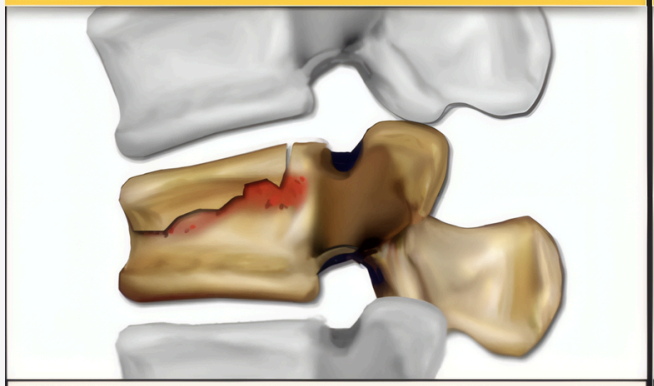
Blumenfeld H. Neuroanatomy through Clinical Cases with Ebook. Sinauer; 2011.


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ANNUAL CONFERENCE
YouTube



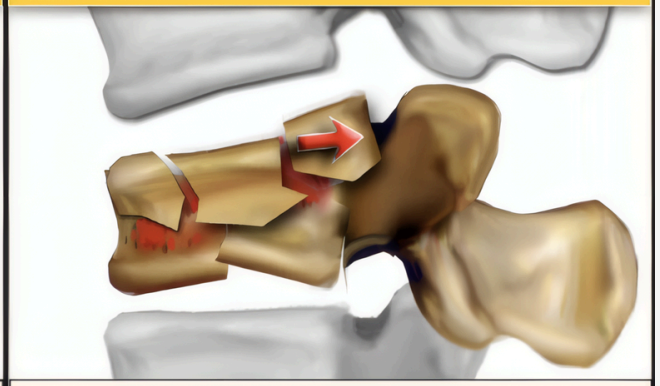
https://upload.orthobullets.com/topic/2022/images/denis.jpg

Compression 1 pnt



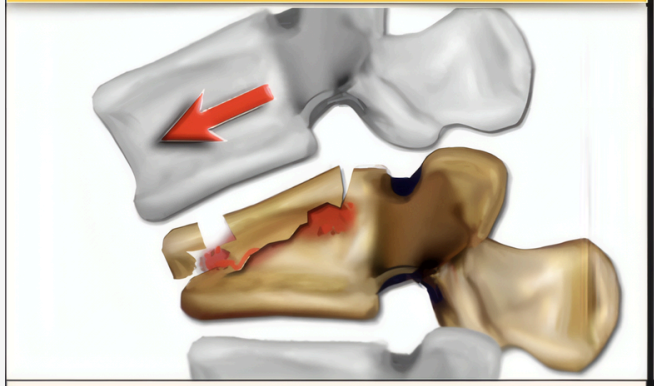
- Simple compression
- Wedge deformity

Burst 2 pnts



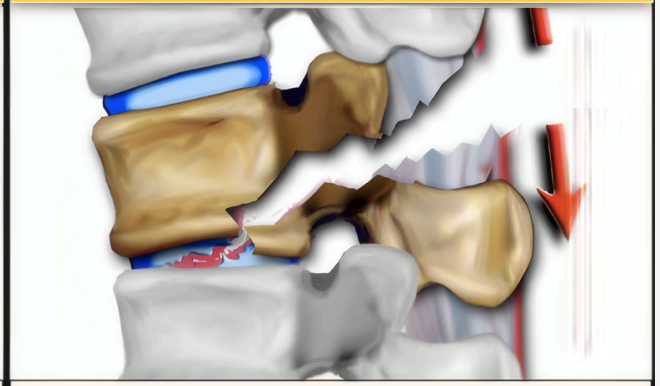
- Compression with retro-pulsion of superoposterior body fragment

Translation/rotation 3 pnts



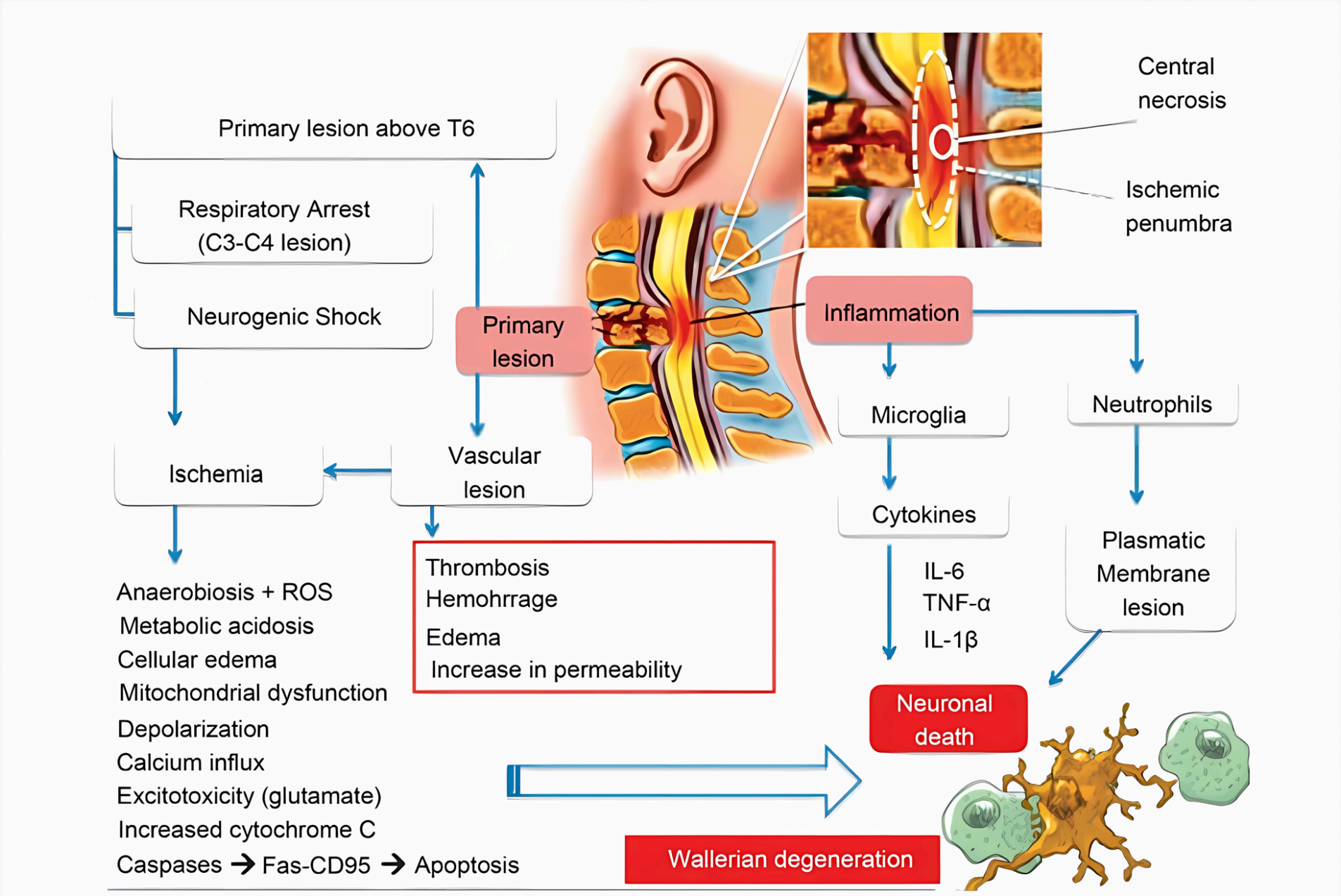
- Rotatory / shearing
- Anterior or lat displacement
- Facet joint displacement

Distraction 4 pnts

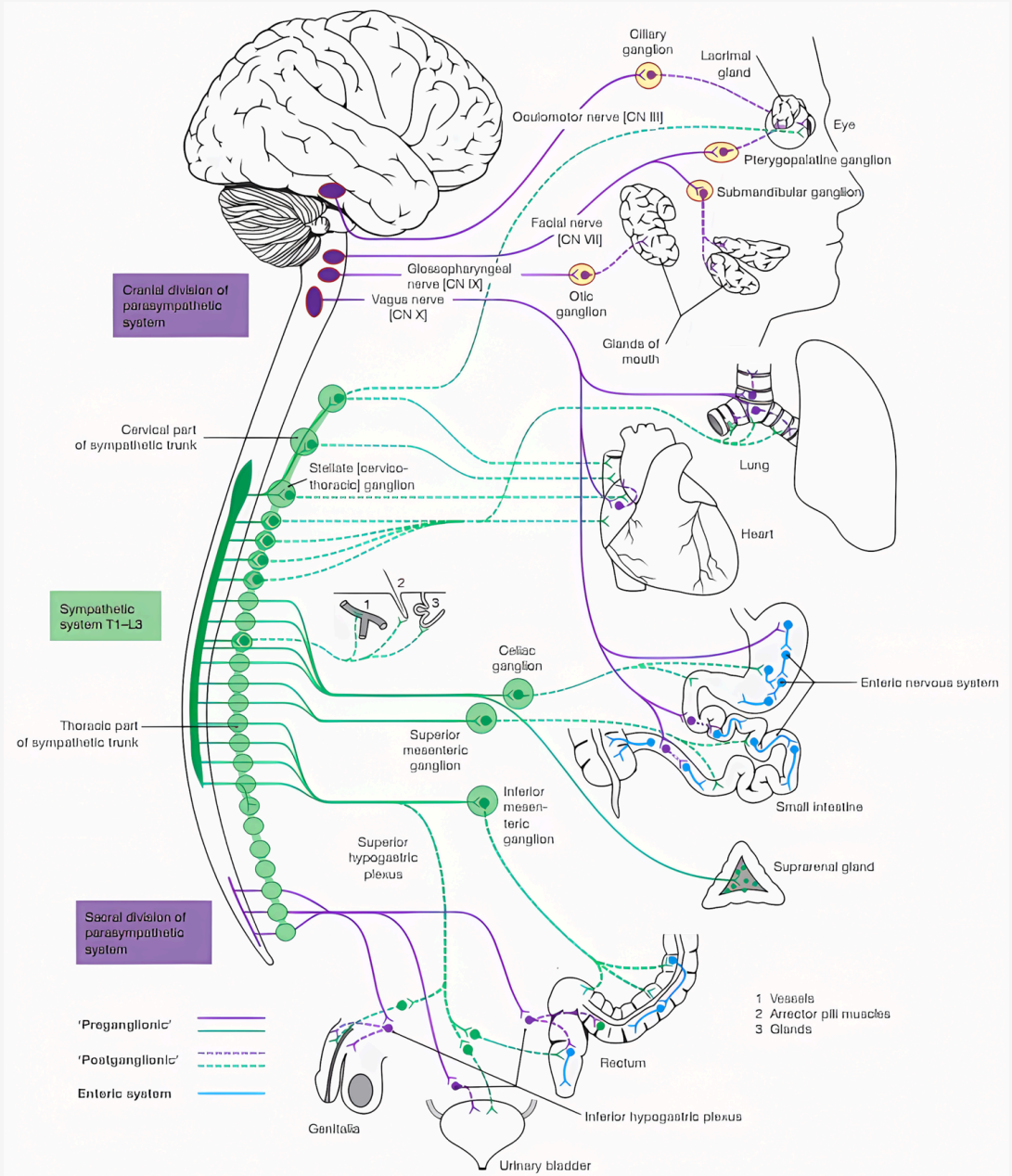


- Horizontal fracture of posterior elements
- Separation of posterior elements

https://radiologyassistant.nl/neuroradiology/spine/tlics-classification



Damiani D. Neurogenic shock: clinical management and particularities in an emergency room. *Arq Bras Neurocir: Braz Neurosurg.* 2018;37(3):196-205. doi:10.1055/s-0036-1584887

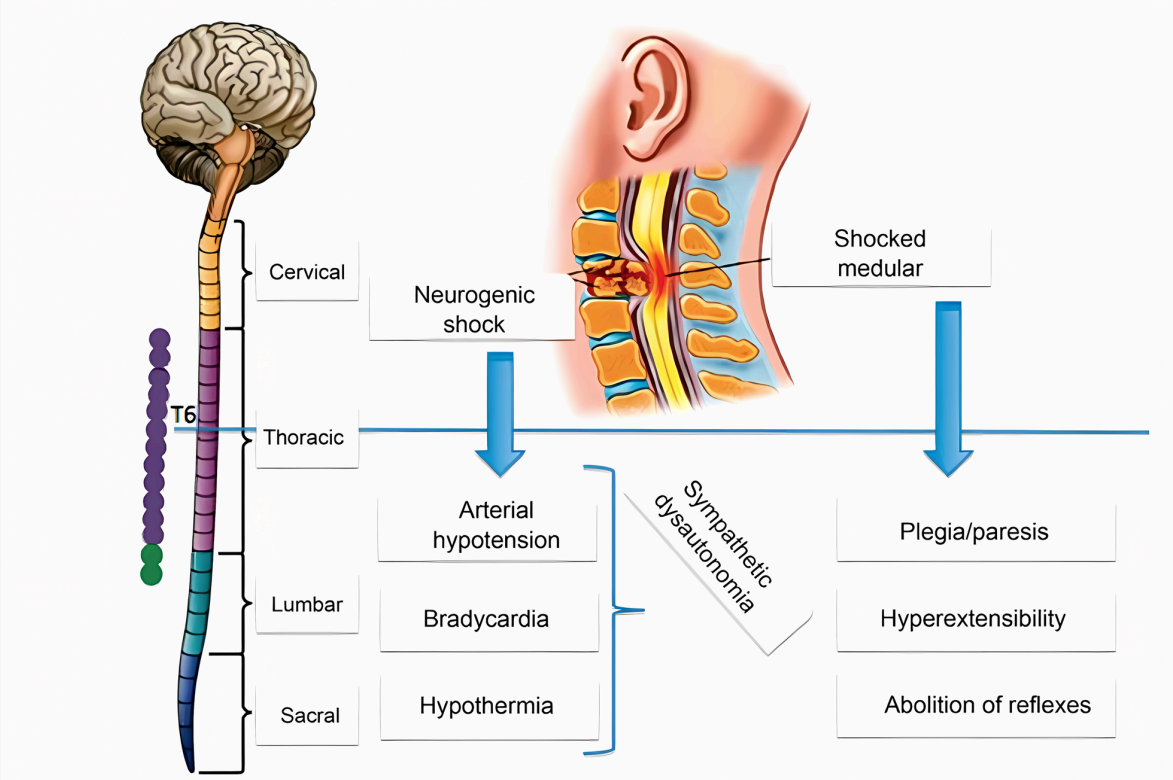


Hombach-Klonisch S, Klonisch TJ, Peeler J, eds. *Sobotta Clinical Atlas of Human Anatomy*. 1. Elsevier; 2019.

Neurogenic shock results from loss of sympathetic vascular tone, leading to systemic vasodilation and tissue hypoperfusion.

- It most commonly follows spinal cord injury at the cervical or upper thoracic level, particularly above T6.
- It must be distinguished from spinal shock, which is a transient loss of neurological function below the injury level.
- Typical haemodynamic features include hypotension with relative bradycardia.
- It is an uncommon but clinically significant complication of spinal cord injury.

Damiani D. Neurogenic shock: clinical management and particularities in an emergency room. *Arq Bras Neurocir: Braz Neurosurg*. 2018;37(3):196-205. doi:10.1055/s-0036-1584887



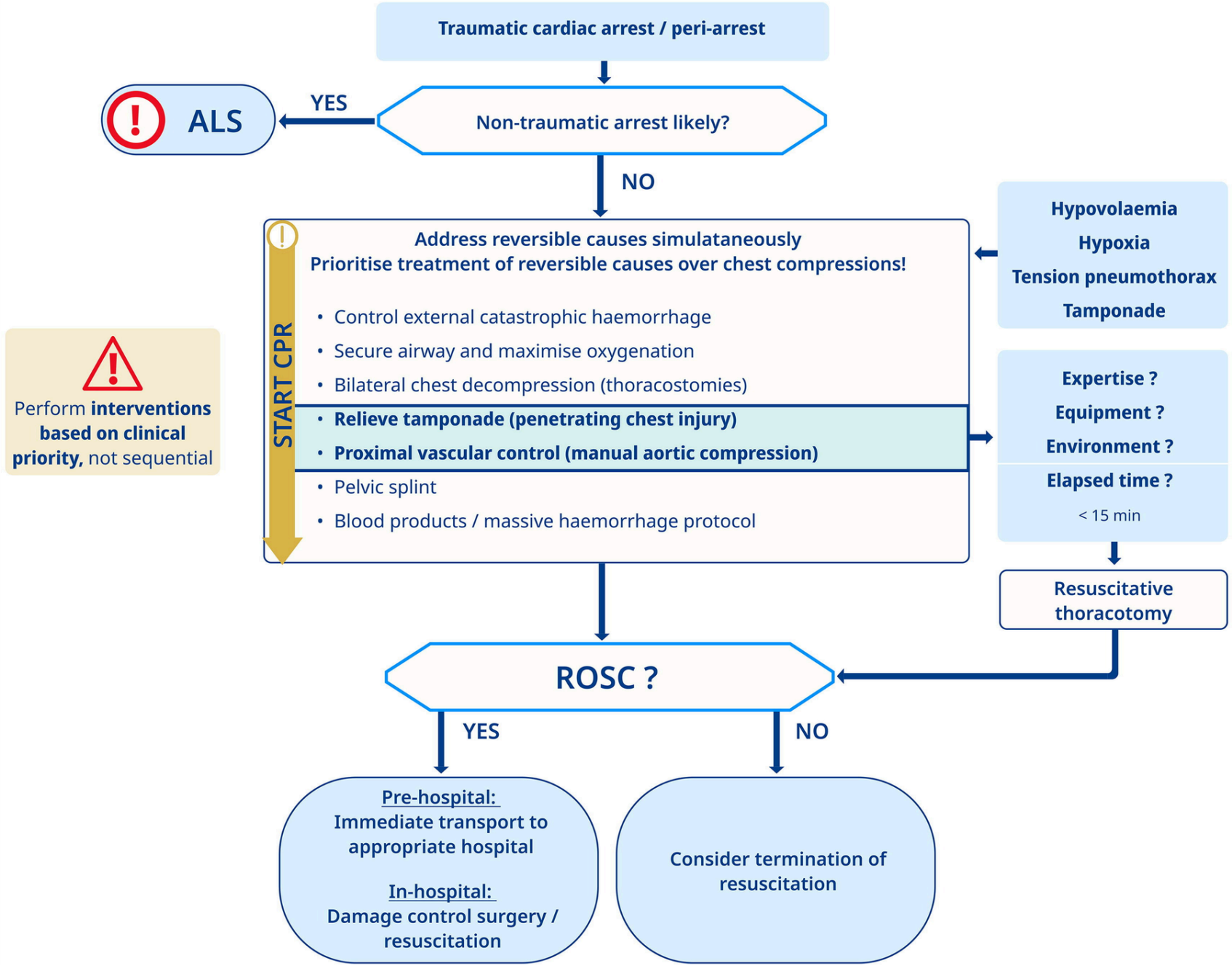
Neurogenic shock results from loss of sympathetic vascular tone, leading to systemic vasodilation and tissue hypoperfusion.

- Disruption of descending sympathetic pathways causes unopposed parasympathetic activity.
- Loss of sympathetic tone leads to venous pooling, reduced preload, and hypotension.
- Reduced cardiac sympathetic input results in bradycardia due to dominant vagal tone.
- Peripheral vasodilation produces warm, pink skin, unlike hypovolaemic shock.
- Hypotension may worsen secondary spinal cord injury through reduced spinal perfusion.

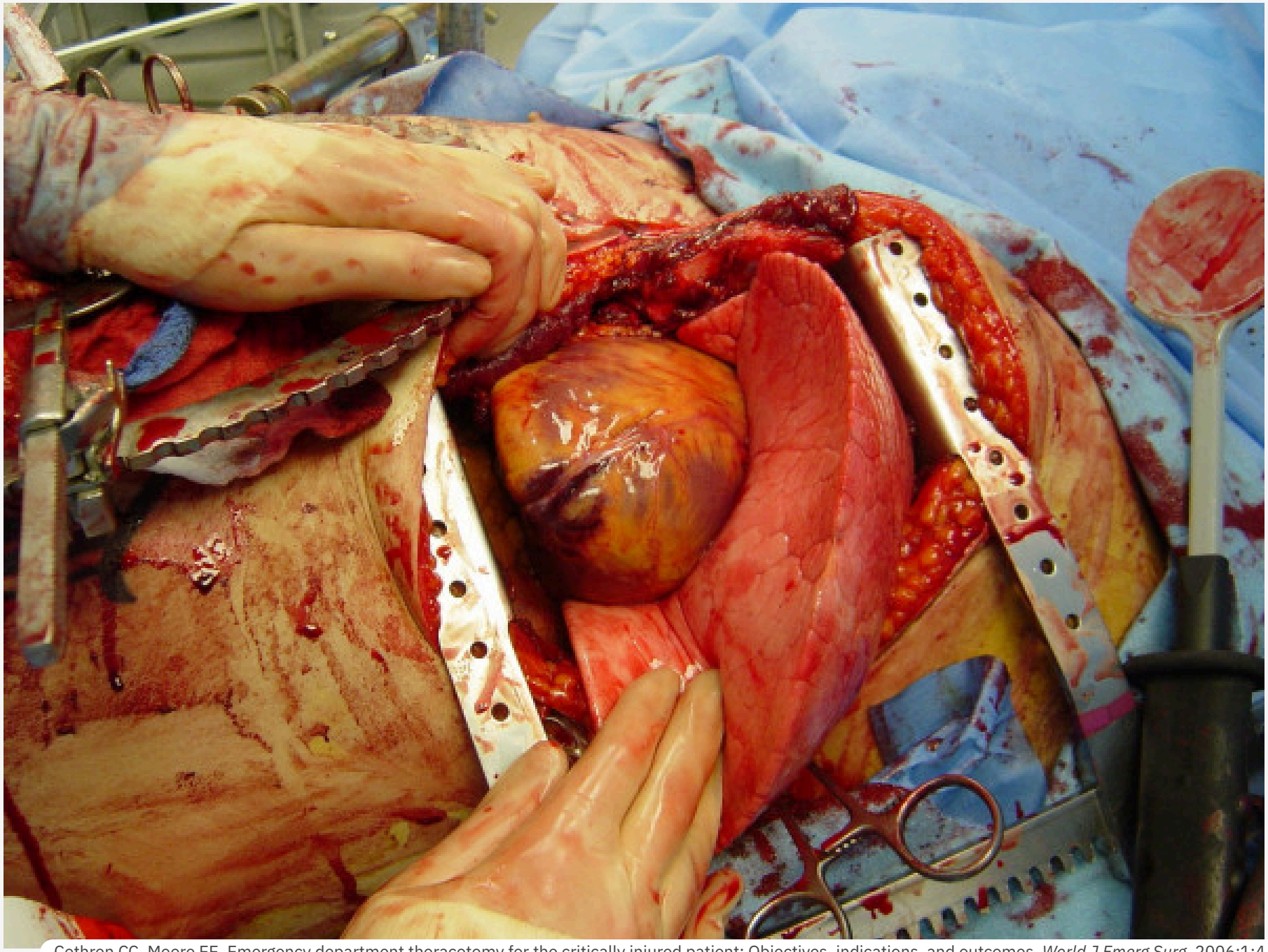
Damiani D. Neurogenic shock: clinical management and particularities in an emergency room. *Arq Bras Neurocir: Braz Neurosurg*. 2018;37(3):196-205. doi:10.1055/s-0036-1584887

Damiani D. Neurogenic shock: clinical management and particularities in an emergency room. *Arq Bras Neurocir: Braz Neurosurg*. 2018;37(3):196-205. doi:10.1055/s-0036-1584887

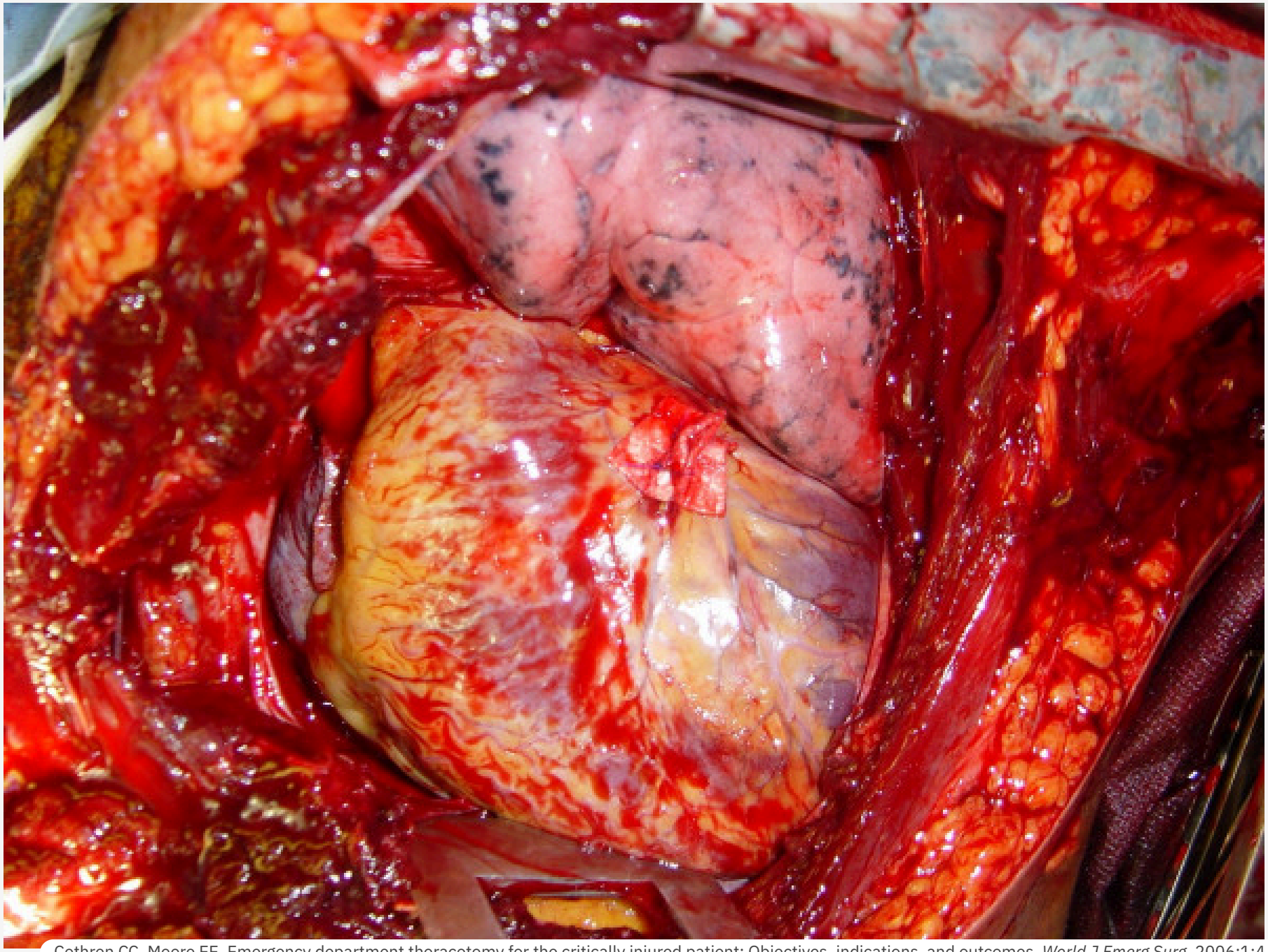
Traumatic cardiac arrest (TCA)



Lott C, Karageorgos V, Abelairas-Gomez C, et al. European resuscitation council guidelines 2025 special circumstances in resuscitation. *Resuscitation*. 2025;215. doi:10.1016/j.resuscitation.2025.110753



Cothren CC, Moore EE. Emergency department thoracotomy for the critically injured patient: Objectives, indications, and outcomes. *World J Emerg Surg*. 2006;1:4. doi:10.1186/1749-7922-1-4



Cothren CC, Moore EE. Emergency department thoracotomy for the critically injured patient: Objectives, indications, and outcomes. *World J Emerg Surg*. 2006;1:4. doi:10.1186/1749-7922-1-4

Open Thoracotomy

YouTube

Viewer Discretion Advised

Damage Control Principles Across Subspecialties

Principle	Application
Haemorrhage Control	Thoracoabdominal Trauma, Vascular Surgery, Emergency General Surgery
Contamination Control	Thoracoabdominal Trauma, Emergency General Surgery
Abdominal/Chest Packing and Temporary Closure	Thoracoabdominal Trauma, Emergency General Surgery
Definitive Reconstruction and Closure	Thoracoabdominal Trauma, Orthopedic Surgery, Emergency General Surgery
Correction of Acidosis, Hypothermia, and Coagulopathy	Resuscitation

Chung CY, Scalea TM. Damage control surgery: old concepts and new indications. *Curr Opin Crit Care*. 2023;29(6):666. doi:10.1097/MCC.0000000000001097



salvatore.sardo@unica.it