



CALS Comprehensive
Advanced Life Support
RURAL EMERGENCY MEDICAL EDUCATION

COMPREHENSIVE ADVANCED LIFE SUPPORT

**CALS PROVIDER MANUAL
THE FIRST 30 MINUTES
EDITION 15 2019**

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www.calsprogram.org

NOTE TO USERS

The CALS course was developed for rural and other healthcare providers who work in an environment with limited resources, but who are also responsible for the emergency care in their often geographically isolated communities. Based on needs assessment and ongoing provider participant feedback, the CALS course endorses practical evaluation and treatment recommendations that reflect broad consensus and time-tested approaches.

The organization of the CALS Provider Manual reflects the CALS Universal Approach. **The FIRST THIRTY MINUTES (previously known as Volume 1)** of patient care. **ACUTE CARE ALGORITHMS/ TREATMENT PLANS/AND ACRONYMS** provide critical care tools and are designed for quick access. The **STEPS** describe a system to diagnose and treat emergent patients. The **FOCUSED CLINICAL PATHWAYS** provide a brief review of most conditions encountered in the emergency setting.

Supplement to the First Thirty Minutes (previously known as Volume 2) is composed of **RESUSCITATION PROCEDURES** divided into appropriate areas of clinical expertise, which illustrate hands-on techniques. Reference material for the First thirty Minutes (previously known as Volume 3) is composed of **DIAGNOSIS, TREATMENT, AND TRANSITION TO DEFINITIVE CARE PORTALS**, is also divided into appropriate areas of clinical expertise. In conjunction with the **FOCUSED CLINICAL PATHWAYS**, these detail further specialized guidelines on many conditions.

Recommendations in the CALS course manual are aligned with those from organizations such as the American Heart Association, American College of Surgeons, American Academy of Family Physicians, American College of Emergency Physicians, American Academy of Neurology, American College of Obstetricians and Gynecologists, American Academy of Pediatrics, National Institutes of Health, and Centers for Disease Control. The CALS Program has selected and modified some recommendations to meet the specific needs and constraints of our rural and remote participants.

CALS teaches basic sound management principles and intentionally refrains from making definitive statements advocating or condemning rational alternatives for achieving a desired clinical result. The CALS position is to present reasonable options unless the evidence base clearly supports one choice over another.

CALS PROVIDER MANUAL EDITION 15- 2019

The CALS Provider Manual is available in a variety of formats including online.

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DISCLAIMER

The information presented through the Comprehensive Advanced Life Support Program (including the CALS Manual, Provider Course and Benchmark Skills Lab – “CALS Program” or “Program”) is made available for educational purposes through a collaborative effort. The effort has involved a multidisciplinary group of individuals and organizations. The information is not intended to represent the *only* – nor necessarily in every circumstance the *best* – methods or procedures appropriate for the medical situations discussed. Rather, the information is intended to present an approach, view, statement, or opinion of the persons involved in the development of the Program, which may be helpful to others who face similar situations.

Best efforts have been made to assure the accuracy of the information presented through the Program. Nevertheless, medicine is constantly changing, and not all therapies are clearly established. In addition, human error is always possible. Therefore, physicians and other members of the health care team must confirm the accuracy of the material presented in the CALS Program. They may wish to check current information regarding specific details, such as drug doses, indications and contraindications, drug interactions, and other information in appropriate, current standard sources (such as package inserts or standard medical reference textbooks) prior to clinical application.

CALS, for itself and for the persons and other organizations participating in the development and distribution of the Program, disclaims any and all liability for injury, or any other damages, resulting to any individual participating in the Program and for all claims that may arise out of the use of the information written or presented or techniques demonstrated or discussed therein by such individuals, whether such claims may be asserted by an individual who participated in the Program or by any other person.

- Much of the information in the Obstetrics portals was obtained from the *Advanced Life Support in Obstetrics (ALSO) Syllabus*. The ALSO Program and its copyright are owned by the American Academy of Family Physicians.
- The American Heart Association strongly promotes knowledge and proficiency in BLS, ACLS, and PALS and has developed instructional materials for this purpose. Use of these materials in an educational course does not represent course sponsorship by the American Heart Association. Any fees charged for such a course, except for a portion of fees needed for AHA course material, do not represent income to the Association.

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CONTRIBUTORS

The CALS Program gratefully acknowledges the generosity of many volunteer authors, reviewers, and instructors who have selflessly donated hundreds of hours of time. These individuals have shared in the creation of this CALS Provider Manual.

Great appreciation is also extended to the many organizations* that have made it possible for CALS to grow from a fledgling, grassroots volunteer group into a flourishing, self-supporting organization.

The Minnesota Academy of Family Physicians (MAFP), Minnesota American College of Emergency Physicians (MNACEP), and Department of Emergency Medicine at the University of Minnesota Medical School provided the support, framework, and organizational structure for the development of CALS. These three organizations continue to promote CALS within their own organizations and beyond, which has led to broad acceptance of CALS in the fields of family medicine, emergency medicine, and trauma.

MNACEP provided invaluable assistance, which led to the financial sustainability of the CALS Program. The Emergency Medical Services Regulatory Board and the Office of Rural Health and Primary Care, Minnesota Department of Health have provided broad institutional support.

The following organizations have also collaborated with and offered support in a variety of ways:

- Emergency Medical Services for Children
- Hennepin County Medical Center
- Hennepin County Medical Center Trauma Services
- Hennepin County Medical Center Traumatic Brain Injury Center
- Methodist Hospital
- Minneapolis Medical Research Foundation
- Minnesota Poison Control System
- National Highway Traffic and Safety Administration
- Regions Medical Center
- U Care Foundation
- University of Minnesota Medical School

We acknowledge the CALS instructors and staff whose hard work and dedication make the CALS program viable.

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* All organizations are in Minneapolis-St. Paul (Minnesota).

PREFACE:
CALS CONCEPTS, GOALS, AND OBJECTIVES

The Comprehensive Advanced Life Support (CALs) Program was conceived and developed from the understanding that success in improving rural emergency and critical care depends on providing existing teams of rural physicians and other team members with the tools to better care for critically ill/injured patients. The CALs curriculum is designed as an effective approach (1) to diagnose and treat the majority of emergency/critical care patients as well as (2) to help rural health care providers become skilled in treating undifferentiated emergencies.

Mission of CALS:

CALs improves patient care by providing advanced life support education to rural healthcare providers.

The focus of the CALs Program is to train medical personnel in a team approach to anticipate, recognize, and treat life or organ-threatening illness or injury. The CALs Program is designed for physicians, physician assistants, nurse practitioners, RNs, LPNs, paramedics and other allied health care professionals (eg, nurse anesthetists and respiratory therapists) who work in rural or remote settings with limited medical resources. In these settings, a broad range of medical emergencies must be addressed, but there is a lack of or limited access to subspecialty health care providers, organized and knowledgeable emergency teams, and/or technologically advanced diagnostic and treatment equipment. CALs has been specifically designed to help minimize the adverse effects of these limitations, which are common to many rural and remote settings.

The CALs curriculum, conducted in a collaborative environment, consists of four components: Home Review of the CALs Provider Manual, the CALs Provider Course classroom sessions, CALs Benchmark Skills Lab, and CALs Trauma Module. Home Review involves studying the CALs Provider Manual in preparation for the interactive components. The two-day CALs Provider Course is comprised of interactive presentations and real-world, scenario-based skill stations in cardiac, trauma, pediatric, obstetric, neonatal, and medical advanced life support as well as difficult airway management. The one-day CALs Benchmark Skills Lab includes skills needed for resuscitation, stabilization, and management of critically ill or injured patients. The Benchmark Skills Lab targets both skills development and team building and teaches participants to

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work collaboratively in caring for patients. The 3-hour Trauma Module consists of expanded trauma skills. CALS curriculum encourages team interaction, a cornerstone of the CALS teaching, which recognizes the value of all emergency team members.

The CALS Curriculum Committee reviews the CALS Provider Manual and course components on a regular bases. The following principles guide course content and recommendations:

- Medical literature is ever changing, often inconclusive, and open to interpretation.
- Course content is based on the best available evidence whenever possible.
- More than one approach may be acceptable for a given problem, and practice may vary from one locale to another. Thus, the material taught, while not always the only approach to a problem is one acceptable method of treatment.

The CALS Program continues to be a work in progress as new evidence prompts changes in knowledge and skills.

Vision of CALS:

The quality of emergency critical care provided in rural communities by primary care teams can be enhanced by the use of the components of the CALS Resuscitation Diamond which consists of:

- Emergency skills and knowledge
- Effective provider teams
- Patient-focused care and systems
- Appropriate equipment



Figure 1 CALS Diamond

The greatest need during resuscitation is to support life and stabilize patients. The CALS Program teaches Rural Emergency Response Team (ERT) members emergency skills and knowledge, proper use of needed emergency equipment, and a systemic approach based on the needs of the patient. With the use of these components of the Resuscitation Diamond as a base, CALS teaches a practical approach to all critically ill/injured patients through the use of the CALS Universal Approach to Undifferentiated Emergencies, which includes the CALS Compass (see Figure 2).

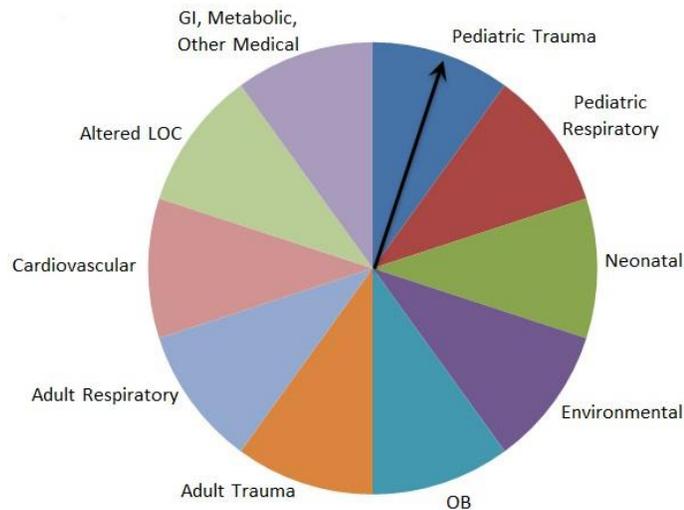


Figure 2 CALS Compass

This approach guides the team through a systematic resuscitation approach focused on the needs of the patient rather than on a perceived diagnosis. The CALS Approach teaches providers to respond first to life-threatening problems and includes safeguards to avoid following an inadequate or improper mode of treatment. The team leader begins with the assessment of airway, breathing, circulation, and disability with major threats to the patient’s well being rapidly addressed. CALS is intended to help providers function effectively as individuals and as team members at times of greatest patient vulnerability, in the most stressful and technically difficult situations.

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Key Concepts, Goals, Objectives, and Principles:

- CALS training emphasizes the value of the team approach to emergency patient management. To this end, CALS training is intended for the whole emergency team and is best taught with local team members working and learning together to gain confidence in the value of teamwork and comfort in its use in their institution.
- The CALS Program presents an educational experience in advanced life support that encompasses all areas of emergency care.
- CALS courses are designed to provide material in a variety of instructional formats to allow for self-directed learning and to provide a balance of cognitive, effective, and psychomotor skills. Through these varied teaching methods, CALS education is designed to maximize the “transfer of learning” as best demonstrated by, as appropriate, a positive change in the techniques and patterns of care provided by rural providers and their teams.
- CALS materials are designed to serve as an information resource, a rapid retrieval system with the use of algorithms and treatment plans, and a reference on more complex issues as contained in the CALS Provider Manual.
- CALS provides a means to update and maintain knowledge and skills.
- CALS teaches team members individually and collectively to problem solve in a variety of clinical situations and to develop safeguards to avoid missing treatable conditions by basing care on the needs of the patient rather than on some assumed diagnosis that may be incomplete or incorrect.
- CALS helps the provider to develop the ability to problem solve in a variety of clinical situations.
- CALS training is designed to complement other Advanced Life Support courses, by providing a bridge over a gap in knowledge and skill set that leads to improved quality of emergency care.
- CALS teaches the knowledge and skills necessary to effectively treat organ or life-threatening emergencies for patients (ranging from newborns to geriatric) before serious organ injury or cardiac arrest occurs.
- The CALS Program teaches a comprehensive, realistic, team-based approach to providing emergency medicine in rural hospitals.
- CALS promotes standardization of high-quality rural emergency care through the use of appropriate patient care systems, equipment, and provider skills and knowledge.

Impact of CALS Training on Rural Emergency and Critical Care:

- CALS training has enhanced the quality of airway management in rural communities. Specifically, significant improvement has been observed in the success rates for endotracheal intubation and other advanced airway techniques. Transport teams and referral centers who receive these rural referrals have reported improvement in airway management by rural hospitals that have participated in the CALS Program.
- The CALS Benchmark Skills Lab increases comfort levels of rural emergency personnel by exposing them to procedures rarely encountered on the job.
- Rapid Sequence Intubation (RSI) is now successfully performed by primary care providers in numerous rural hospitals throughout Minnesota and western Wisconsin, representing a significant change from a decade ago.
- CALS training forms an excellent foundation upon which to develop an ERT in a rural hospital. ERTs are valuable resources for rural hospitals as they respond to critical patients with the hospital's limited resources.
- CALS establishes a rural-based standard for assessing the medical equipment needs of small hospitals and clinics.
- CALS teaches participants to anticipate and prepare for a patient's needs prior to arrival, improving the speed and efficiency of treatment and leading to better patient outcomes.
- CALS has improved the speed and efficiency of transferring critical patients to higher levels of care.
- CALS training improves the care of time-sensitive conditions, such as acute stroke, trauma, acute coronary syndrome, shock, and critical airway management.
- CALS training not only improves the emergency care provided by rural care facilities, but also helps to facilitate the decision-making process as to which patients need to be rapidly stabilized and transported to higher levels of care.

Certification and Completion of CALS:

Successful completion of the CALS Course components results in each participant receiving a certificate of completion of each individual segment of CALS training. This certificate is not a license to perform any skill learned. One's own licensing board grants licensure, and competency is awarded by the participant's employment institution. The CALS certificate signifies satisfactory completion of the CALS course consistent with the participant's role on the CALS team. This includes:

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- Review of the CALS manual or other preparatory materials prior to the CALS Provider Course and completion of a pretest
- Attendance at the CALS didactic/skills training sessions
- Active participation in the training and the ability to use the algorithms and treatment flow diagrams to problem-solve critical patient presentations
- Active participation in the CALS Labs (either Benchmark Skills or Trauma Module)

Conclusion:

More than twenty-five years ago, the all-volunteer, grassroots, rural and urban emergency care provider coalition that founded the CALS Program embarked on its mission to improve emergency patient care through advanced education of rural health care providers, especially those practicing in remote settings or in areas of limited health care resources. Now an independent 501(c) (3) organization, the CALS Program continues to expand and develop in the hopes of achieving its mission to improve emergency patient care and thus patient outcomes in the Twenty-First century.

Given an ever increasing number of patients presenting to both rural and urban Emergency Departments and the growing complexity of state-of-the-art emergency care; adequate preparation, knowledge, and skills training is essential for all emergency health care providers. The CALS Program has been shown to be successful in improving rural emergency care and thus is one approach to helping rural hospitals and providers improve emergency care. We trust that the users of the CALS Provider Manual and participants in the CALS training programs will find the use of these tools valuable in their goals to deliver the best quality of emergency care possible in their work settings.

Further information about the CALS Program and the online version of the CALS Provider Manual as well as current updates may be obtained on the CALS website: www.calsprogram.org.

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COMPREHENSIVE ADVANCED LIFE SUPPORT CALS

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ACUTE CARE 1: UNIVERSAL APPROACH FOR CRITICAL PATIENT CARE

Steps	Patient Approach	Intervention	
Step 1	Notification of Patient Arrival	Alert team /Appropriate protocols /Consider resources	
Step 2	Immediate Control Immobilization	Restrain/ Immobilize Adequate lighting Appropriate work surface Receive EMS report	
Step 3 Team Leader	Initial (Primary) Survey '10 Seconds of Silence' Assess life threats Airway inadequate? Breathing inadequate? Circulation inadequate? Disability/LOC/Defib	Immediate transfer? Treat life threats Correct problems Control bleeding/Defib IV access/ Replace volume/ AVPU/GCS/DONT	A -Alert D-Dextrose V-Voice O-Oxygen P-Pain N-Narcan U-Unres T-Thiamine
↓ Simultaneous Actions		Sample history	
▲			
Team Members	Assist Team Leader	Apply oxygen Gain exposure Obtain vitals/SAO ₂ ECG monitor IV access /Medications Obtain labs and x-rays Urinary catheter Dextrose evaluation Other	S=Signs/symptoms A=Allergy M=Meds P=Past med history L=Last meal E=Events
Step 4	Preliminary Impression	Focused evaluation (secondary survey) Focused physical exam Diagnostic tests Diagnostic procedures	Focused Pathways as needed OB Neonatal Respiratory Cardiovascular Trauma Gastrointestinal/ Abdominal Altered LOC/ unknown
Step 5	Working Diagnosis/Ongoing Care/Disposition <div style="border: 1px solid gray; padding: 5px; margin-top: 10px;">No patient response or patient deteriorates – return to initial (primary) survey</div>	Refer to diagnostic treatment portals Continue to reassess Consultation Stabilization Disposition	Admit Transfer Discharge
Step 6	Team Process Review	Team input/assess need for debriefing	

ACUTE CARE 2: PATIENT TRANSPORT ALGORITHM

Follow this list to prepare a patient for transfer to another facility.

Airway Needs

1. Reassess airway and consider need for airway protection during transport.
2. Intubate for the following indications: GCS < 11, confused or combative, respiratory distress, or any condition with potential loss of airway.
3. Secure the ET tube or Supraglottic airway and check position with capnography and chest x-ray.
4. Insert orogastric tube if intubated.
5. Suction mouth and upper airway frequently.
6. Ensure adequate paralysis, sedation, and analgesia for transfer.
7. Secure patient's hands to prevent accidental extubation by patient.
8. To decrease aspiration risk, slightly elevate HOB pre-/post- intubation; consider spinal issues.

Breathing Needs

9. Insert chest tube for pneumothorax or hemothorax with repeat chest x-ray.
10. Use continuous exhaled CO₂ monitoring of intubated patients during transfer and suction if needed.

Circulation Needs

11. Secure IV lines or I/O (minimum of two). Convert to large bore if patient is hypovolemic.
12. Number IV bags to keep track of amount of fluids being given.
13. Use O-negative or O-positive blood (males or non-child-bearing females) for patients in shock.
14. Consider giving TXA if giving blood and at least one SBP <90 (adult).
15. Keep the patient warm. Use warm fluids. Monitor and document the patient's temperature.

Disability Needs

16. Examine patient's back for any injuries. Maintain spinal immobilization if indicated/suspicion for trauma. Remove from spine board if possible or use padding or air cushion and check for Neuro changes and CMS before and after removal.
17. Insert Foley catheter or suprapubic catheter as required. Empty and document output from Foley before transfer.
18. Splint fractures. Use sheet or commercial pelvic binder as appropriate to splint pelvic fractures. Confirm distal pulses before transport.

Infection Control

20. Tetanus prophylaxis and consider antibiotics.

Copies of all Patient Records and Communication

21. Include copies of all lab tests, x-ray and CT studies, and flow sheets. Include the SAMPLE history.
22. Communicate directly doctor-to-doctor and nurse-to-nurse from sending to receiving facilities. Send copy of patient demographic sheet.
23. Include estimated patient weight and initial GCS.
24. Include list of known meds, including OTC.
25. Include family/emergency contact if known.

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Acute Care 3

RAPID SEQUENCE INTUBATION (RSI) ALGORITHM

(Also known as Medication Assisted Intubation)

1. Prepare: Equipment, meds, team
Patient (basic airway management, positioning)
Discuss plan including a backup plan
 2. Preoxygenate: 100% O₂, 3 to 5 minutes and add nasal cannula @ 4L/min
 3. Premedicate: Treat Hypotension and Hypoxia
-
- TIME OUT
-
4. Push the sedative: Use only ONE:
 - Ketamine 1 to 2 mg/kg IV (bronchodilator/useful in hypotension) or
 - Etomidate 0.3 mg/kg IVIncrease O₂ by nasal cannula to ADULTS 15 L/min,
PEDS 10 L/min (children), 5 L/min (infants)
 5. Paralyze: Use only ONE:
 - Rocuronium 1 to 1.5 mg/kg IV (PEDS: preferable) or
 - Succinylcholine 2 mg/kg IV (Avoid in hyperkalemia, neuromuscular disease, or ocular trauma)Wait for relaxation (45-60 sec). Do not bag unless hypoxic
 6. Position airway: Head/neck position; BURP or tracheal manipulation (if needed)
 7. Pass & assess tube: Maintain in-line cervical immobilization in head/neck trauma
Inflate cuff (Careful not to overinflate)
Use esophageal intubation detector, check breath sounds, continuous or colorimetric EtCO₂
Record depth of tube. Secure the tube. CXR for depth confirmation
 8. Pass next tube: Pass OG or NG. Empty stomach & then intermittent suction
 9. Post-intubation plan: Drugs and dosages depend on medications used during intubation
Titrate all meds and watch for hypotension and remember sedation/analgesia have shorter half-lives than paralytics

Sedation: Midazolam 0.1 mg/kg IV
Paralysis (if needed): Vecuronium 0.1 mg/kg IV or repeat
Rocuronium 1mg/kg IV
Analgesia: Fentanyl 1 to 2 MICROgrams/kg IV or
Morphine 0.05 to 0.15 mg/kg IV
- Helpful Reminders: If capabilities exist, can consider sedation/analgesia drips for transport
RSI medications can be given IV/IO
Consider need for seizure prevention
Elevate Head of Bed if able

ACUTE CARE 4: RAPID SEQUENCE INTUBATION MEDICATIONS

Sedation for Rapid Sequence Intubation

Agent/Class	Dose	Onset	Duration	Key Notes
Ketamine/ dissociative	1 to 2 mg/kg	1 minute	10 to 30 minutes	Bronchodilation, amnesia, analgesia
Etomidate/ imidazole derivative	0.3 mg/kg given over 30 to 60 seconds	< 1 minute	5 to 14 minutes	Best all- around profile, Suppresses cortisol
Fentanyl/ opiate	1 to 2 <u>micrograms</u> /kg IV	< 1 minute	30 to 60 minutes	Analgesia, Reversible; Give slow IV push
Propofol	1 to 2 mg/kg	30 to 60 seconds	3 to 5 minutes	May cause hypotension

Paralytics for Rapid Sequence Intubation

Agent Class	Dose	Intubation	Duration	Key Notes
Succinylcholine/ depolarizing	2 mg/kg	45 to 60 seconds	6 to 12 minutes	Avoid in hyperkalemia, neuromuscular disease, or ocular trauma.
Rocuronium/ non depolarizing	1 mg/kg	45 to 75 seconds	25 to 60 minutes	Alternative to succinylcholine for initial paralytic agent
Vecuronium/ non depolarizing	0.1 mg/kg	90 to 240 seconds	25 to 120 minutes	Most useful after intubation for longer-term paralysis; PEDS: <u>may be preferable</u>

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ACUTE CARE 5: ENDOTRACHEAL INTUBATION FLOW SHEET

Name:		Date:		Actual Weight:	
				Ideal Weight:	
Reason:					
Preparation: Equipment including rescue airways, meds, team, & patient.				Time:	
Initial VS:	BP:	Pulse:	RR:	O ₂ Sats:	EtCO ₂ :
					Temp: GCS:
Preoxygenate via nasal cannula:		ADULTS 15 L/min		PEDS 10 L/min	
				Infants 5 L/min	
Position airway: <i>head/neck, laryngeal manipulation, and consider if the patient needs ramping position</i>					
100%O₂ via:		NRB	BVM	BIPAP	CPAP
		DSI (if needed)			
Premedicate: <i>Treat Hypoxia and Hypotension. Dose calculation based on Actual Weight.</i>					
RSI TIME OUT					
Push sedative: (Provider calculate dose)				Time:	
<input type="checkbox"/>	Ketamine (Ketalar)	1 to 2 mg/kg	IV Push	_____ mg	_____ mL
<input type="checkbox"/>	Etomidate (Amidate)	0.3 mg/kg	IV Push	_____ mg	_____ mL
Paralyze: (Provider calculate dose)				Time:	
<input type="checkbox"/>	Rocuronium (Zemuron)	1 to 1.2 mg/kg	IV Push	_____ mg	_____ mL
<input type="checkbox"/>	Succinylcholine:	2 mg/kg	IV Push	_____ mg	_____ mL
Do not ventilate unless acutely desaturating					
Pass the tube:		ETT size	Lips/teeth	cm	Time:
By:					
Pass the OG:		Size	Time:		
Post Intubation Airway Assessment:					
Direct Visualization	EDD	BBS	CO ₂ (EtCO ₂ /colorimetric)	SpO ₂	CXR
POST INTUBATION PLAN					
Analgesia:				Time:	
<input type="checkbox"/>	Morphine	2 to 4 mg		_____ mg	_____ mL
<input type="checkbox"/>	Fentanyl	1 to 2 mcg/kg		_____ mcg	_____ mL
				Drip: 1 to 2mcg/kg/hr	
<input type="checkbox"/>	Ketamine	0.5 – 1 mg/kg/hr		_____ mg/min	
				Drip: (500mg in 500ml) Titrate to 4mg/kg/hr	
Sedation:				Time:	
<input type="checkbox"/>	Midazolam (Versed)	0.05 to 0.3 mg/kg		_____ mg	_____ mL
<input type="checkbox"/>	Propofol (Diprivan)	0.5 – 1mg/kg		_____ mg/kg	
				Drip: Titrate 10 to 100 mcg/kg/min	
Paralytic: (if needed)				Time:	
<input type="checkbox"/>	Vecuronium	0.1 mg/kg		_____ mg	_____ mL
<input type="checkbox"/>	Rocuronium	1 to 1.2 mg/kg		_____ mg	_____ mL
POST INTUBATION ASSESSMENT					Time:
Vital Signs:		BP:	Pulse:	RR:	O ₂ Sats:
					EtCO ₂ : Temp:
Used Bougie:		Yes	No	Goal RASS of -2 to 0	
				RASS:	
Rescue Airway:		Yes	No	Type:	
Vent Settings & Mode:		FiO ₂	TV	Rate	PEEP
					Mode
Issues: <i>None Aspiration - pre- or post-induction or during intubation</i>					
<i>Esophageal intubation Bleeding Dental trauma</i>					
		<i>Tube not at proper depth</i>		<i>Repositioned at _____ cm _____</i>	
		<i>Other:</i>			
Additional Notes:					
Signature:					

ACUTE CARE 6: RAPID SEQUENCE INTUBATION DRUG CALCULATOR

Patient Weight: (kgs) _____

Date: _____

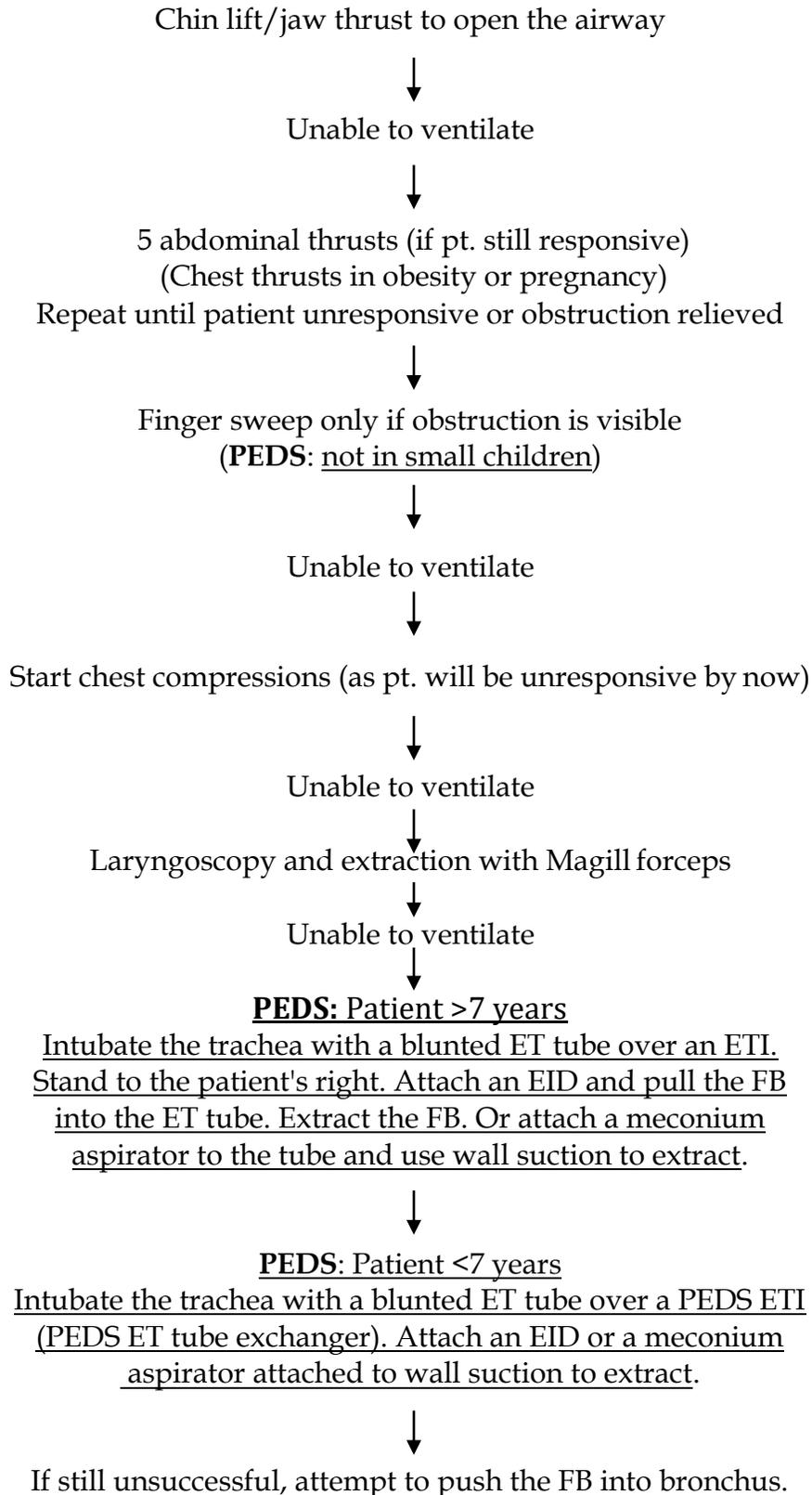
	Medication	Dose (mg/kg)	X	Pt. Wt. (kgs)	=	Dose (mg)	÷	mg/ mL	=	Volume (mL)
1	Etomidate	0.3 mg/kg	X		=		÷	2 mg/mL	=	
2	Ketamine	1 to 2 mg/kg	X		=		÷	50 mg/mL	=	
3	Succinylch.	2 mg/kg	X		=		÷	20 mg/mL	=	
4	Rocuronium	1 mg/kg	X		=		÷	10 mg/mL	=	
5	Vecuronium (continued paralysis)	0.1 mg/kg	X		=		÷	1 mg/mL	=	
6	Fentanyl	1 to 2 µg/kg	X		=		÷	50 µg/mL	=	
7	Morphine	0.05 to 0.15 mg/kg	X		=		÷	Various concentrations	=	
8	Versed	0.05 to 0.3 mg/kg	X		=		÷	Various concentrations	=	
9										

Put patient's weight in **kilograms** on the top line and then in all the rows in the column under it. Use calculator to arrive at the dosages in both **milligrams** and **milliliters** for the nurses to draw up.

REMEMBER: Someone should be in charge to make sure that the medications ordered are **always** in the same concentrations or else the formulae will change.

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ACUTE CARE 7: OBSTRUCTED AIRWAY ALGORITHM – ADULT AND PEDIATRIC



ACUTE CARE 8: CPR STEPS FOR ADULTS, CHILDREN, AND INFANTS (For Healthcare Providers)^{1,2}

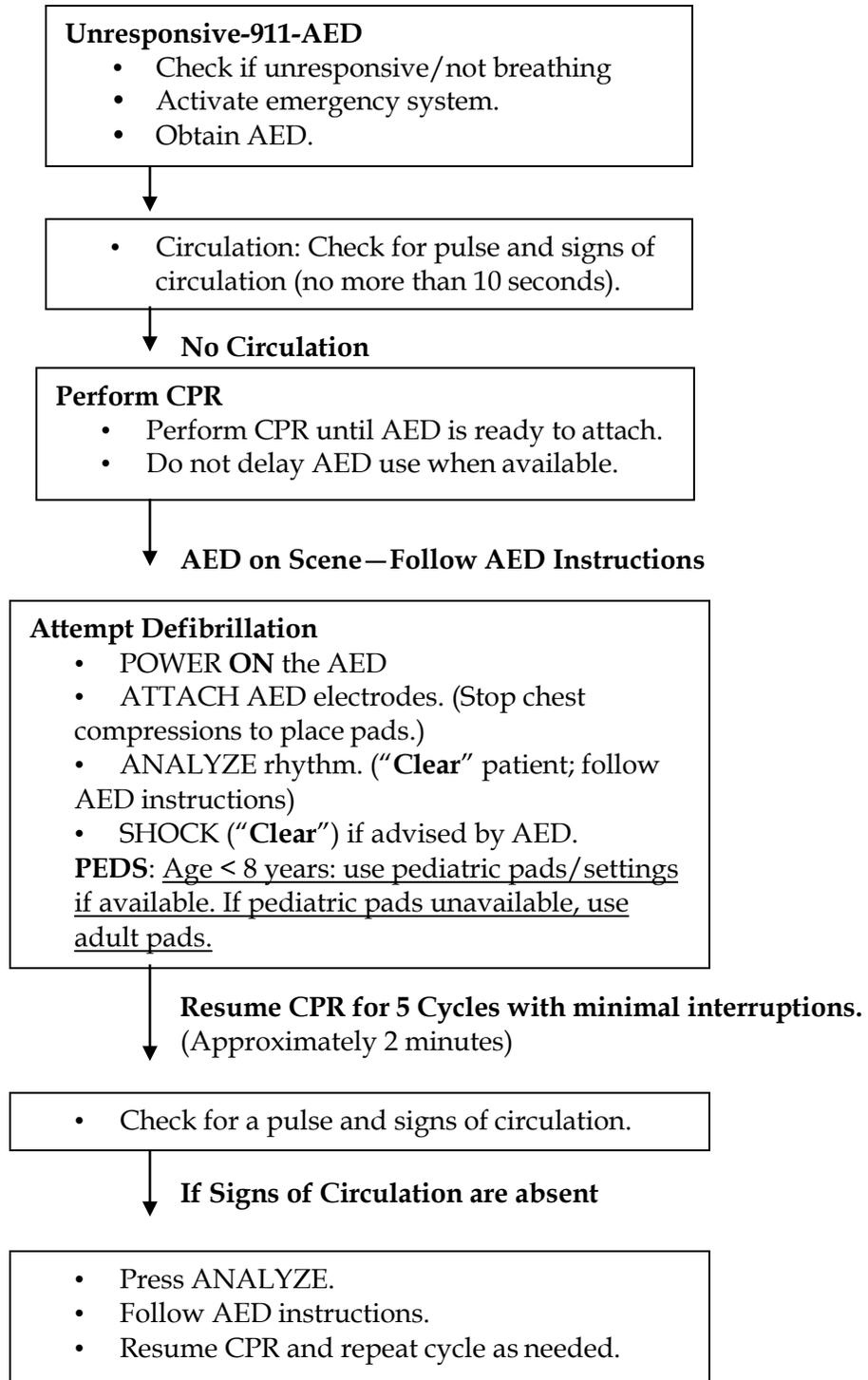
Assessments/ Interventions (all age groups)	Age-Specific Recommendations		
	Adult/ Older Child (puberty and older)	Child (1-year-old to puberty)	Infant (less than 1-year-old)
<p>Check for response; check for breathing</p> <p>If unresponsive and/or not breathing and/or agonal respirations</p>	<p>Leave victim to activate emergency response system and retrieve AED</p> <p style="text-align: center;">or</p> <p>Get or send for AED or monitor/defibrillator and begin CPR</p>	<p>Lone rescuer (witnessed sudden collapse) or more than one rescuer: activate emergency response system</p> <p>Get or send for AED or monitor/defibrillator</p> <p>Lone rescuer (no sudden collapse witnessed): Check pulse and do 2 minutes of CPR, then activate emergency response system/AED</p>	
<p>Check pulse</p> <p>At least 5 seconds and no more than 10 seconds</p> <p>If no pulse, start compressions at rate of</p> <p>If pulse present go to Open Airway and recheck pulse every 2 minutes</p>	<p>Check carotid pulse</p> <p>If no pulse, start CPR</p> <p>100 - 120 per minute</p>	<p>Check carotid pulse</p> <p>If no pulse or pulse <60 bpm with signs of poor perfusion, start CPR</p> <p>100 – 120 per minute</p>	<p>Check brachial pulse</p> <p>If no pulse or pulse <60 bpm with signs of poor perfusion, start CPR</p> <p>100 – 120 per minute</p>
<ul style="list-style-type: none"> • Compression location 	Center of breastbone between nipples		Just below nipple line, on breastbone
<ul style="list-style-type: none"> • Compression method 	Use heel of 1 hand, with other hand on top (For smaller victims, use 1 hand)		<p>One-person CPR: Use 2 fingers.</p> <p>Two-person CPR: Encircle chest with hands; use both thumbs.</p>
<ul style="list-style-type: none"> • Compression depth 	At least 2 inches (5 cm)	At least 1/3 depth of chest (5 cm)	At least 1/3 depth of chest (4 cm)

• Compression-ventilation ratio	30:2 (1- or 2-rescuer CPR)	30:2 for 1-rescuer CPR (15:2 for 2-rescuer CPR)	3:1 for newborns (up to 1-month-old)
	If advanced airway inserted, give 8-10 breaths per minute, not synchronized, with compressions		
Open the Airway	Use head tilt/chin lift; if suspected spine trauma use jaw thrust.		
Check breathing If the victim is not breathing, give 2 breaths that make the chest rise.	Take at least 5 seconds and no more than 10 seconds. Do not interrupt compressions for longer than ten seconds.		
If pulseless	Resume CPR; attach and use AED when available (See AED Algorithm, p. 17.)		
If pulse present Continue rescue breathing; Recheck pulse every 2 minutes	Adults: 8-10 breaths/minute	Infants and children: 12-20 breaths/minute	

REFERENCES

1. Neumar RW, Shuster M, Callaway CW, et al. Part 1: executive summary: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(18)(suppl 2). In press.
2. Hazinski MF, Nolan JP, Aicken R, et al. Part 1: executive summary: 2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation*. 2015;132(16)(suppl 1). In press.

ACUTE CARE 9: AUTOMATED EXTERNAL DEFIBRILLATOR ALGORITHM



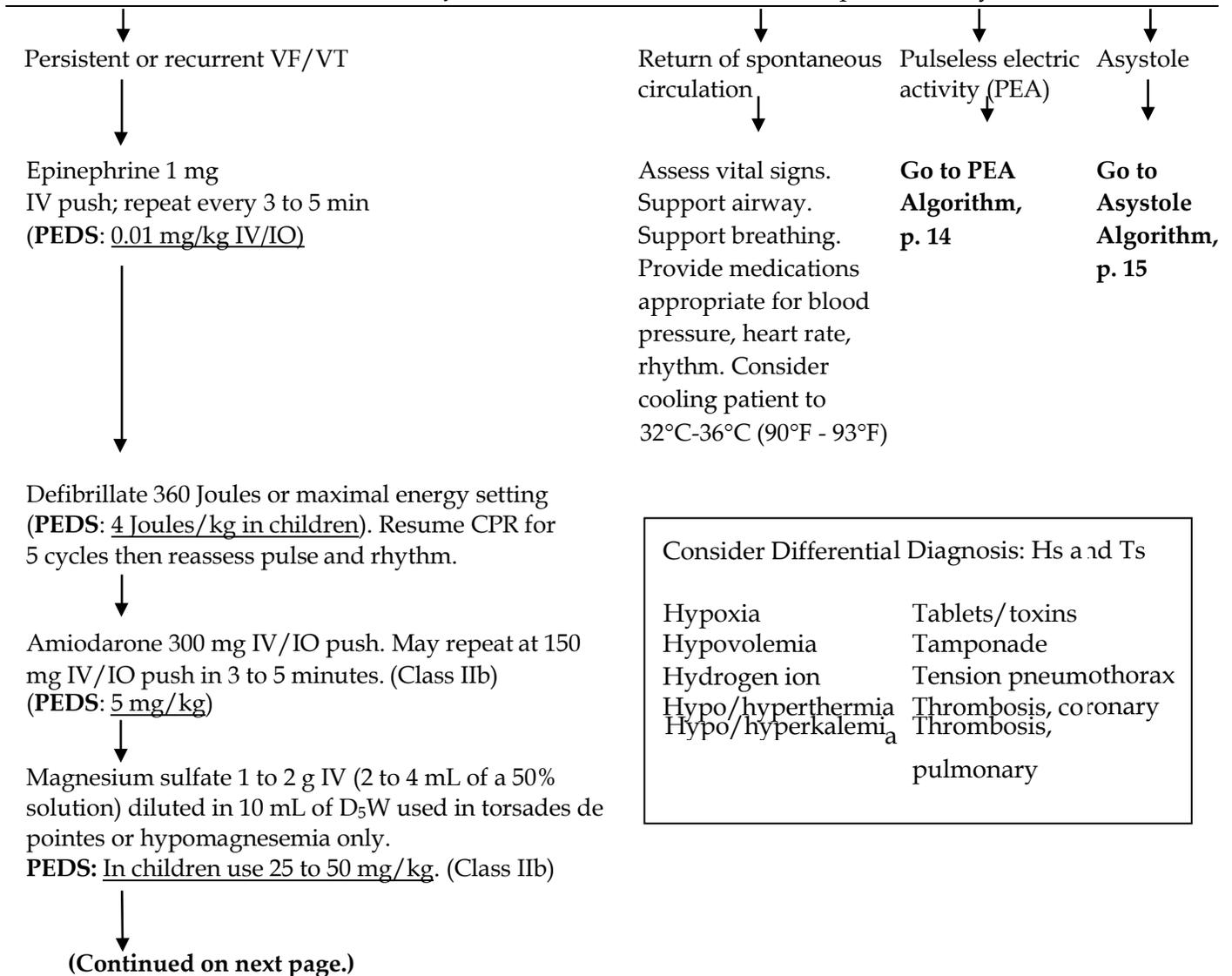
ACUTE CARE 10: VENTRICULAR FIBRILLATION/ PULSELESS VENTRICULAR TACHYCARDIA ALGORITHM

Assess responsiveness/breathing.
Activate emergency response system
Get AED or Monitor/defibrillator



Check for pulse (no more than 10 seconds)
If no pulse, begin CPR with minimal interruptions.

As soon as defibrillator is available: Defibrillate 200 Joules (or default) for first shock.
Use maximal energy setting for subsequent shocks. **PEDS: In children, use 2 Joules/kg for first shock, 4 Joules/kg for subsequent shocks.**
Resume CPR for 5 cycles or attach mechanical device if available.
Secure airway. Obtain IV/IO access; then reassess pulse and rhythm.



Repeat defibrillation at maximal energy levels. Do 5 cycles of CPR and reassess. **PEDS: ≥ 4 Joules/kg up to a 10 Joules/kg or adult dose.**

If patient remains in VF/VT, sequence follows: drug intervention, defibrillation, 5 cycles of CPR, reassess

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ACUTE CARE 11: PULSELESS ELECTRICAL ACTIVITY ALGORITHM— ADULT AND PEDIATRIC

Assess the patient for responsiveness/breathing.
 Activate the emergency response system.
 Get AED or Monitor/Defibrillator.



Check pulse (no more than 10 seconds)



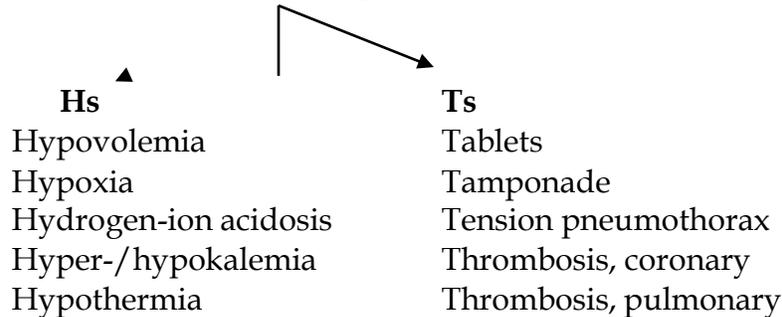
If no pulse, begin CPR with minimal interruptions. Apply mechanical device if available.



PEA – Rhythm on the monitor but the patient has no pulse.
 Complete the primary and secondary ABCD survey.



Place advanced airway; confirm and secure.
 Establish effective oxygenation and ventilation.
 Identify the rhythm on the monitor.
 Administer appropriate drugs for rhythm and condition.
 Search for and treat reversible causes.
 If available, bedside ultrasound may help to determine
 contractility and filling status of the heart.



Give epinephrine 1 mg IV push. **PEDS:** Epinephrine 0.01 mg/kg IV/IO. Repeat every 3 to 5 minutes.

Atropine is not routinely recommended in PEA. However, in bradycardic PEA, it may be pharmacologically reasonable to administer atropine 1 mg IV/IO. Repeat as needed at 3- to 5-minute intervals to a total dose of 0.04 mg/kg (approximately 3 mg for adults). **PEDS:** Dose is 0.02 mg/kg IV/IO. Repeat at 3- to 5-minute intervals to a total dose of 0.04 mg/kg (~3 mg for adults).

ACUTE CARE 12: ASYSTOLE ALGORITHM – ADULT AND PEDIATRIC

Assess the patient for responsiveness/breathing.
 Activate the emergency response.
 Get AED or monitor/defibrillator.



Check for pulse (no more than 10 seconds).
 If patient has no pulse, begin CPR with minimal interruptions. Apply mechanical device if available.



Identify asystole in more than one lead.
 Look for evidence not to start resuscitation, such as obvious signs of death or DNR order.



Administer epinephrine 1 mg (**PEDS: 0.01 mg/kg**) IV/IO push.
 Repeat every 3 to 5 minutes.



Place advanced airway; confirm and secure.
 Establish effective oxygenation and ventilation.
 Administer appropriate drugs for rhythm and condition.
 Search for and treat reversible causes. (See below.)



If asystole persists:
 Stop resuscitation?
 Is support for family present?

Potentially Reversible Causes of Cardiac Arrest

Hs	Ts
Hypoxia	Tablets/toxins
Hypovolemia	Tamponade
Hydrogen ion	Tension pneumothorax
Hypo/hyperthermia	Thrombosis, coronary
Hypo/hyperkalemia	Thrombosis, pulmonary

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ACUTE CARE 13: BRADYCARDIA ALGORITHM¹

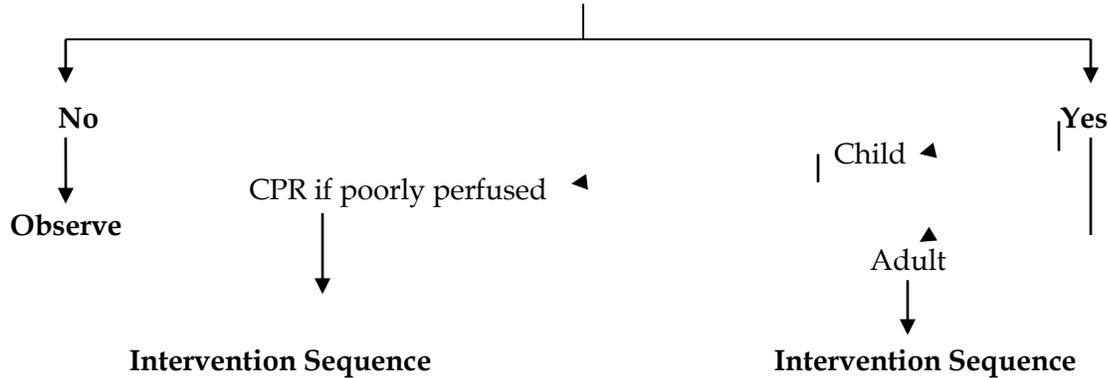
The Team Should:

Assess airway, Start IV, Assess vital signs,
Determine O₂ saturation, give supplemental O₂, if required.
Order 12-lead ECG, Obtain history/physical

Bradycardia, either absolute rate (< 50 bpm)
or relative (slower than expected for underlying condition)

Serious signs or symptoms?

(Chest pain, shortness of breath, decreased LOC, poor perfusion, shock,
pulmonary edema, heart failure, acute MI)



Intervention Sequence	Intervention Sequence
<ul style="list-style-type: none"> • Epinephrine 0.01 mg/kg (1:10,000 0.1 mL/kg) IV or IO. Repeat every 3 to 5 min • Atropine 0.02 mg/kg with minimum dose of 0.1 mg and a maximum single dose of 0.5 mg. Repeat once if needed. • Transcutaneous pacing for Type II second- or third-degree block or any bradycardia unresponsive to atropine. 	<ul style="list-style-type: none"> • Atropine 0.5 mg IV/IO; repeat if needed at 3 to 5 min intervals to total dose of 0.04 mg/kg (3 mg for adults) • Transcutaneous pacing for Type II second- or third-degree block or any bradycardia unresponsive to atropine • If pacing is unavailable or ineffective: <ul style="list-style-type: none"> - Dopamine 2 to 10 µg/kg/min - Epinephrine 2 to 10 µg/min • Prepare for transvenous pacing.

In pediatric and adult patients, consider reversible causes of bradycardia.

Hs	Ts
Hypoxia	Tablets/toxins
Hypovolemia	Tamponade
Hydrogen ion	Tension pneumothorax
Hypo/hyperthermia	Thrombosis, coronary
Hypo/hyperkalemia	Thrombosis, pulmonary

1. Hazinski MF, Samson R, Schexnayder S, editors. *2010 Handbook of Emergency Cardiovascular Care*. Dallas, TX: American Heart Association, 2010 pp 9, 74.

ACUTE CARE 14: TACHYCARDIA ALGORITHM

Is this a sinus tachycardia?

Yes →

No ↓

Treat the underlying medical problem:
sepsis, hypovolemia, hypoxia, pain, etc

Is the patient unstable with signs or symptoms caused by the tachycardia (chest pain, pulmonary congestion/pulmonary edema, decreased level of consciousness, hypotension /shock, acute myocardial infarction)?

No ↓

Yes ↓

Prepare for immediate electrical cardioversion. Consider sedation and airway management.

Initial energy levels:

- Narrow complex or atrial flutter: 50-100 Joules
 - Monomorphic V tach: 100 Joules
 - Atrial Fib: 200 Joules (monophasic) 120-200 Joules (biphasic)
 - Polymorphic VT: unsynchronized defibrillation (same as VF)
- PEDS: 0.5 to 1.0 Joules/kg**

Is the rhythm atrial fibrillation with wide complexes (≥ 0.12 sec) and rate >150 ?

No ↓

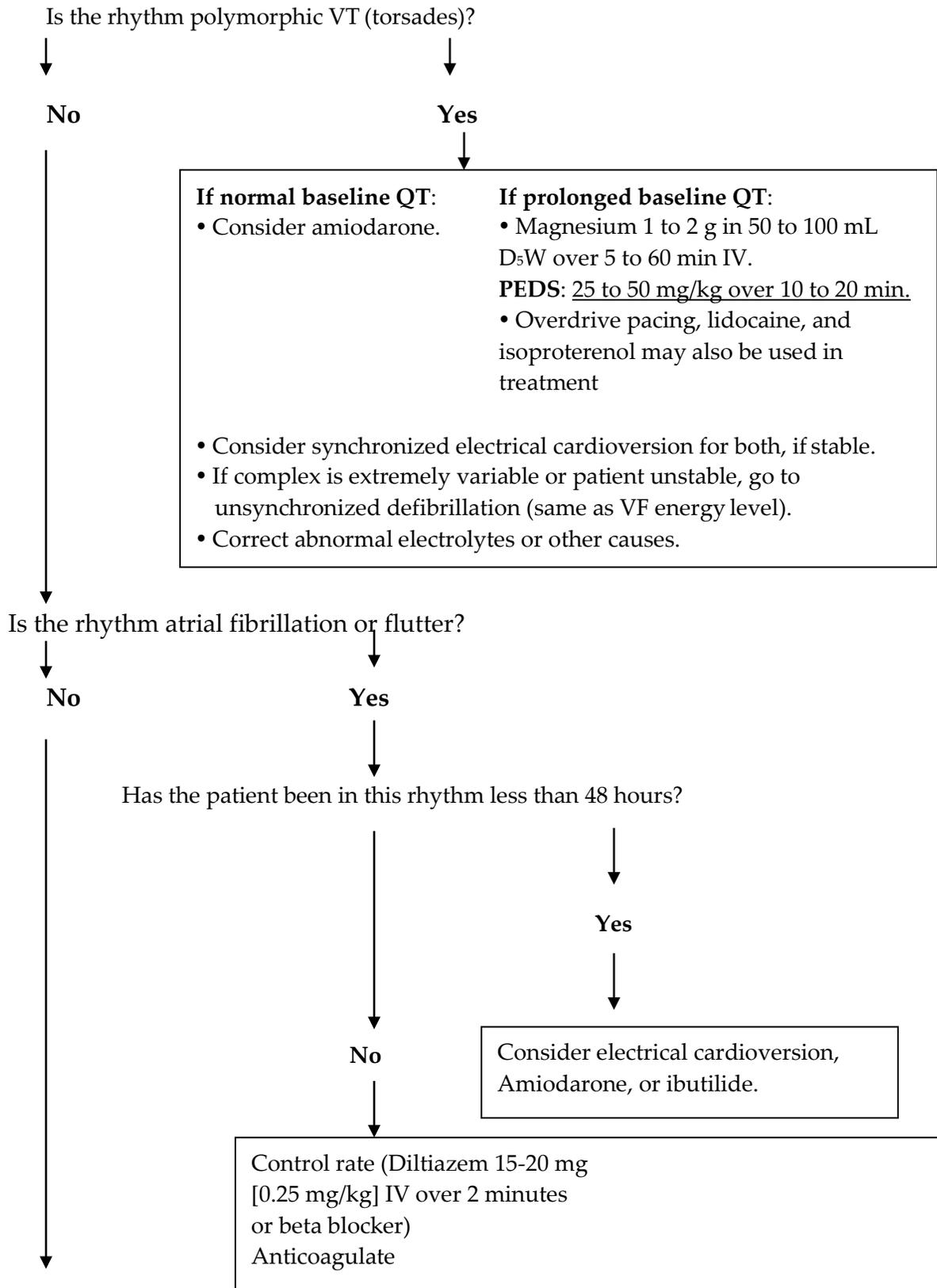
Yes ↓

Consider possible WPW.
Avoid calcium channel blockers, Beta blockers, and digoxin.

- Amiodarone 150 mg IV over 10 to 20 min; repeat if needed.
PEDS: 5 mg/kg over 10 to 20 min; may repeat to max 15 mg/kg.
- Procainamide 20 mg/min IV to 17 mg/kg
PEDS: 15 mg/kg IV over 30 to 60 min

If AF present > 48 hours, avoid cardioversion.

Tachycardia Algorithm (continued)



Tachycardia Algorithm (continued)

Is the tachycardia narrow or wide complex?

Regular narrow complex tachycardias	Regular wide complex tachycardias
<p>First try Vagal (Valsalva) maneuvers If ineffective in converting rhythm: Adenosine 6 mg IV rapid push and flush. PEDS: 0.1 mg/kg. If ineffective, may double dose and repeat x 2</p> <p>Calcium channel blockers and Beta blockers may also be considered.</p> <p>Consider synchronized electrical cardioversion.</p>	<p>Amiodarone 150 mg IV over 10 to 20 min; repeat if needed. PEDS: 5 mg/kg over 20 to 60 min; may repeat to 15 mg/kg max/24 hours</p> <p>Procainamide 20 mg/min to 17 mg/kg IV PEDS: 15 mg/kg IV over 30 to 60 min</p> <p>Lidocaine 1 to 1.5 mg/kg IV push; total loading dose 3 mg/kg PEDS: same</p> <p>Consider synchronized electrical cardioversion.</p>

Defibrillator Energy Delivery for Cardioversion ¹		
Synchronized		
Rhythm	Monophasic Defibrillator	Biphasic Defibrillator
Atrial fibrillation	200 Joules	120 - 200 Joules
Atrial flutter	50 - 100 Joules	NA*
PSVT (narrow complex, regular)	50 - 100 Joules	NA*
VT-monomorphic (wide complex, regular)	100 - 360 Joules	NA*
Unsynchronized		
VT-polymorphic (wide complex irregular)	200 Joules (unsynchronized); deliver subsequent shocks at maximal energy setting	Device specific (or 200 Joules) (unsynchronized); deliver subsequent shocks at maximal energy setting
NA*= insufficient data to recommend energy levels; use the same or less than monophasic.		

REFERENCE

Neumar RW, Otto CW, Link MS, Kronick SL, Shuster M, Callaway CW, et al. Part 8. Adult Advanced Cardiovascular Life Support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010; 122(suppl 3):S729-S768.

Tachycardia Algorithm (continued)

Paroxysmal Supraventricular Tachycardia (PSVT) Algorithm

Vagal Maneuvers:

Valsalva Maneuver

Modified Valsalva Maneuver

From a semi-recumbent position, have patient blow into a 12 mL syringe for 15 seconds (enough to move plunger). Immediately place patient in supine position and perform passive leg raise to 45° angle.

Adenosine: 6 mg rapid IV push over 1 to 3 seconds followed by 20 mL fluid flush

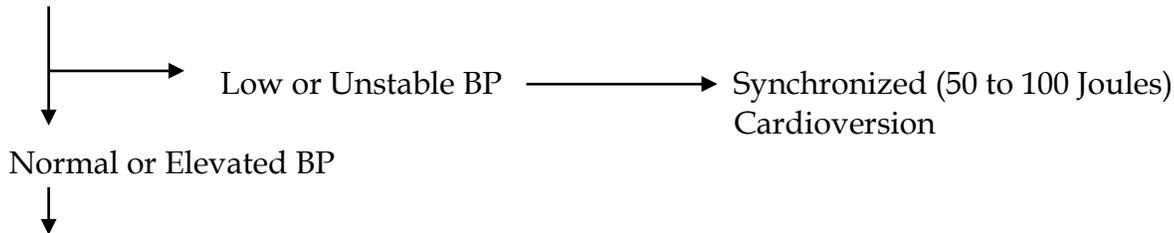
↓ **PEDS:** 0.1 mg/kg

If fails to convert in 1 to 2 minutes, give:

↓ **Adenosine:** 12 mg rapid IV push. May repeat the 12 mg dose once after 1 to 2 minutes.
PEDS: 0.2 mg/kg

If rhythm fails to convert or recurs, consider other treatments.

↓
Assess Blood Pressure



Calcium Channel Blockers and Beta Blockers:

↓ **Verapamil:** 2.5 to 5.0 mg IV over 2 minutes. If rhythm does not convert in 15 to 30 minutes, a second dose of 5 to 10 mg IV over 2 minutes may be given. (Do not give to patients with impaired hearts.)
Diltiazem: 15 to 20 mg (0.25 mg/kg) IV over 2 minutes. If rhythm does not convert in 15 minutes, give a 2nd dose of 20 to 25 mg (0.33 mg/kg) IV over 2 minutes.
If hypotension occurs with these agents, place patient in Trendelenburg position or slowly infuse calcium chloride 0.5 to 1.0 g IV.

Digoxin (Lanoxin) or **Beta Blockers** (Propranolol or Esmolol) may be tried. (Use extreme caution with use of beta blockers after use of calcium channel blockers.)

↓
Synchronized Cardioversion

January 2019

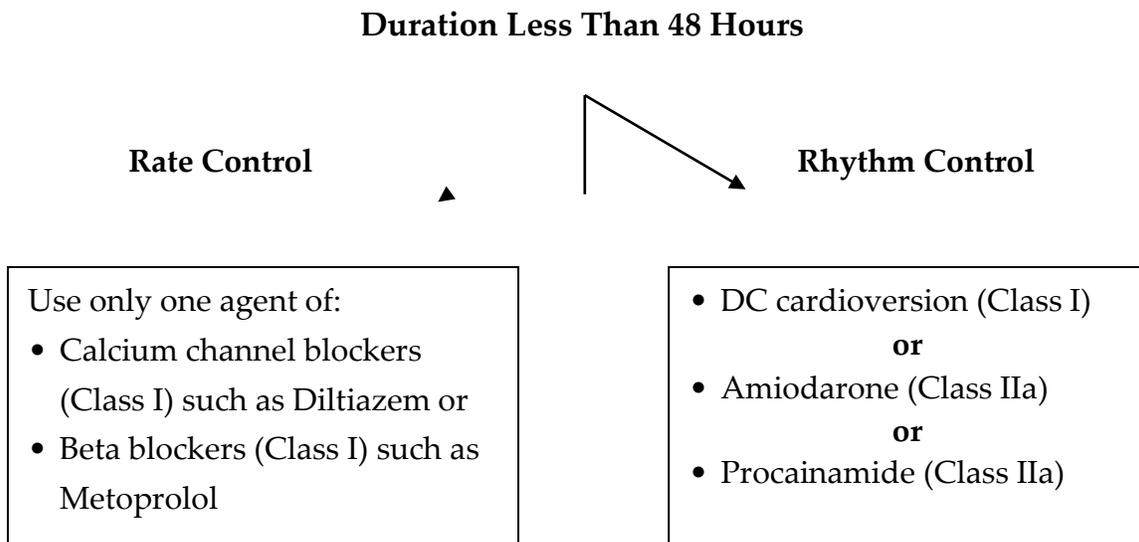
ACUTE CARE 15: ATRIAL FIBRILLATION/ATRIAL FLUTTER ALGORITHM

Perform immediate synchronized cardioversion if patient is unstable (shock, decreased LOC) and has poor perfusion that seems to be due to atrial fibrillation/atrial flutter with rapid ventricular response.

If patient is stable, determine the duration of atrial fibrillation/atrial flutter.
If wide complex, consider Wolff-Parkinson-White syndrome. (See page 30.)

Attempt to convert to NSR using medication or electrical cardioversion, when the patient is stable, even if you are certain patient has been in AF 48 hours.

Factors to consider include level of comfort with sedation, potential airway management, need to avoid anticoagulation, and patient's preference. Consultation is helpful.



Synchronized DC Cardioversion Energy Levels:

Atrial Fibrillation: 120 J – 200 J Biphasic; 200 J Monophasic

Atrial Flutter: 50 J – 100 J

Atrial Fibrillation/Atrial Flutter (continued)

Duration More Than 48 Hours or Unknown

Rate Control

Use only one agent of:

- Calcium channel blockers
Diltiazem (Class I)
- Beta blockers –
Metoprolol (Class I)

Methods of Converting Rhythm When Duration More Than 48 Hours

Caution Note: Avoid non-emergent cardioversion unless anticoagulation or clot precautions are taken.

Urgent Cardioversion (Unstable Patients):

- Begin Heparin IV immediately.

Delayed Cardioversion:

- Anticoagulation x 3 to 4 weeks at therapeutic levels
- Cardioversion
- Anticoagulation x 4 additional weeks

Atrial Fibrillation/Atrial Flutter (continued)

Wolff-Parkinson-White (in Known WPW or Wide Complex Very Rapid Atrial Fibrillation and Atrial Flutter)

Note: In patients in atrial fibrillation/atrial flutter with an accessory pathway or WPW, the following drugs are Class III (can be harmful) and should not be used as they may cause a paradoxical increase in the ventricular rate.

- Adenosine
- Beta blockers
- Calcium channel blockers
- Digoxin

To Convert if Duration Less Than 48 Hours:

If the duration of the atrial fibrillation/atrial flutter with WPW is < 48 hours in duration:

- DC cardioversion is Class I or recommended treatment.

If drug therapy is used and ventricular function is preserved, consider the following Class II drug recommendations:

- Amiodarone
- Procainamide
- Flecainide
- Propafenone
- Sotalol

If duration is 48 hours or less and the ventricular function is impaired:

- Use amiodarone only.

To Convert if Duration More Than 48 Hours:

Urgent Cardioversion:

1. Begin Heparin IV immediately.
2. Perform transesophageal echocardiogram to exclude atrial clot.
3. Cardioversion within 24 hours followed by:
Anticoagulation x 4 additional weeks

Delayed Cardioversion:

1. Anticoagulation x 3 to 4 weeks at therapeutic levels
2. Then cardioversion followed by:
Anticoagulation x 4 additional weeks

Atrial Fibrillation/Atrial Flutter (continued)

Pharmacological Agents-Atrial Fibrillation-Atrial Flutter

Rate Control

Vagal Maneuvers^a (Ice water or Valsalva maneuver) or **Adenosine** (6 to 12 mg IV) temporarily slows the ventricular rate to confirm presence of atrial flutter. **In this setting, vagal maneuvers and adenosine are used for diagnostic purposes only.**

Calcium Channel Blockers^a

Diltiazem: 0.25 mg/kg IV over 2 minutes. If ventricular rate does not slow in 15 minutes, give 2nd dose of 0.33 mg/kg IV over 2 minutes.

Verapamil: 5 mg IV over 2 minutes. If rate does not slow in 15 to 30 minutes, give 2nd dose of 5 to 10 mg over 2 minutes.

If hypotension occurs with these agents, place patient in Trendelenburg position or slowly infuse calcium chloride 0.5 to 1.0 g IV.

Beta Blockers^a

Propranolol (Inderal): 0.5 to 1.0 mg/min IV to maximum total dose of 0.1 mg/kg.

Esmolol (Brevibloc): Load with 0.5 mg/kg IV over 1 minute, then infuse 0.05 to 0.2 mg/kg/min IV as needed to slow the ventricular rate.

Metoprolol (Lopressor): 5 mg IV over 2 to 5 minutes. Repeat at 5-minute intervals to total dose of 15 mg.

Atenolol (Tenormin): 5 mg IV over 5 min. May repeat in 10 min to total dose of 10 mg.
(Use extreme caution with use of beta blockers after use of calcium channel blockers.)

Digoxin^a (Lanoxin): Give 0.25 to 0.5 mg IV followed by 0.125 to 0.25 mg every 2 hours as needed to a maximum total dose of 0.75 to 1.5 mg (10 to 15 µg/kg) in the first 24 hours.

Chemical Cardioversion

Ibutilide (Corvert): (Class III anti-arrhythmic agent) Infuse 1 mg IV over 10 minutes. (For patients < 60 kg, use 0.01 mg/kg.) May repeat the 1 mg infusion 10 minutes after completing the first infusion.

Propafenone (Rythmol): (Class Ic anti-arrhythmic agent) Initial dose of 150 mg PO every 8 hours; may increase every 3 to 4 days up to 300 mg every 8 hours.

Amiodarone (Cordarone): (Class III anti-arrhythmic agent) Load with 800 to 1000 mg per day PO for 1 to 3 weeks, then 400 to 800 mg per day for 2 to 4 weeks, then 100 to 400 mg per day as maintenance.

Sotalol (Betapace): (Class II and III anti-arrhythmic agent) Initial dose of 80 mg bid PO; increase to a maximum total daily dose of 320 mg/day.

Flecainide (Class Ic anti-arrhythmic agent) Load with 2 mg/kg IV slow infusion at rate up to 10 mg/min. **Must be infused slowly. (IV form not approved in the US)**

Procainamide (Class Ia anti-arrhythmic agent) Loading dose of 20 to 30 mg/min IV to max of 17 mg/kg.

Anticoagulants: Heparin, Enoxaparin (Lovenox), Warfarin (Coumadin), and Dabigatran (Pradexa).

Caution: If any of these agents are used for treatment of patients with atrial fibrillation or atrial flutter, monitor the patient while observing for prolongation of the QT interval. This may indicate that the patient is at risk for developing a serious arrhythmia. If this happens, consider discontinuing the offending drug.

^a Contraindicated in WPW.

ACUTE CARE 16: ELECTRICAL CARIOVERSION ALGORITHM— ADULT AND PEDIATRIC

Synchronized cardioversion is used to treat supraventricular tachycardia (SVT) or ventricular tachycardia (VT) patients who have a pulse and are symptomatic with poor perfusion, hypotension, or heart failure.

If patient is unstable and rhythm is not sinus tachycardia, junctional tachycardia, or multifocal atrial tachycardia, prepare for immediate cardioversion. May give brief trial of medications based on specific arrhythmias. Immediate cardioversion generally not needed for rates < 150 bpm.



Team Actions to Prepare for Planned Electrical Cardioversion

1. Prepare for airway management
2. Monitor, O ₂ , IV line
3. BP and O ₂ saturation monitor
4. Trial of antiarrhythmic medications as appropriate
5. Prepare the defibrillator



Pre-medicate whenever possible.
Use sedatives with or without analgesic agents.
Sedation must not delay cardioversion in emergency situations.

Sedative Choices	Analgesic Choices
Etomidate — Adult and Pediatric: 0.1 mg/kg IV	Fentanyl — Adult and Pediatric: 1 to 5 µg/kg IV 2.0 µg/kg IN
Midazolam (Versed) — Adult: 0.02 to 0.1 mg/kg IV; 0.3 mg/kg IN—max 10 mg (Use 5 mg/mL concentration) Pediatric: 0.05 to 0.15 mg/kg IV; 0.3 mg/kg IN—max 10 mg (Use 5 mg/mL concentration)	Morphine — Adult and Pediatric: 0.05 to 0.15 mg/kg IV
Propofol — Adult: 1 to 2 mg/kg IV Pediatric: 1 to 2.5 mg/kg IV	
Ketamine — Adult and Pediatric: 1.5 to 2 mg/kg IV; 4 mg/kg IM	

Electrical Cardioversion (continued)

Defibrillator Energy Delivery for Adult Cardioversion¹		
Synchronized		
Rhythm	Monophasic Defibrillator	Biphasic Defibrillator
Atrial fibrillation (narrow complex, irregular)	200 J	120 J to 200 J
Atrial flutter	50 J to 100 J	NA*
PSVT (narrow complex, regular)	50 J to 100 J	NA*
VT-monomorphic (wide complex, regular)	100 J to 360 J	NA*
Unsynchronized		
VT-polymorphic (wide complex, irregular)	360 J (unsynchronized)	Device specific (or 200 J) (unsynchronized)
*NA= insufficient data to recommend energy levels; use the same as or less than monophasic		

Synchronized Cardioversion for Pediatric Patients

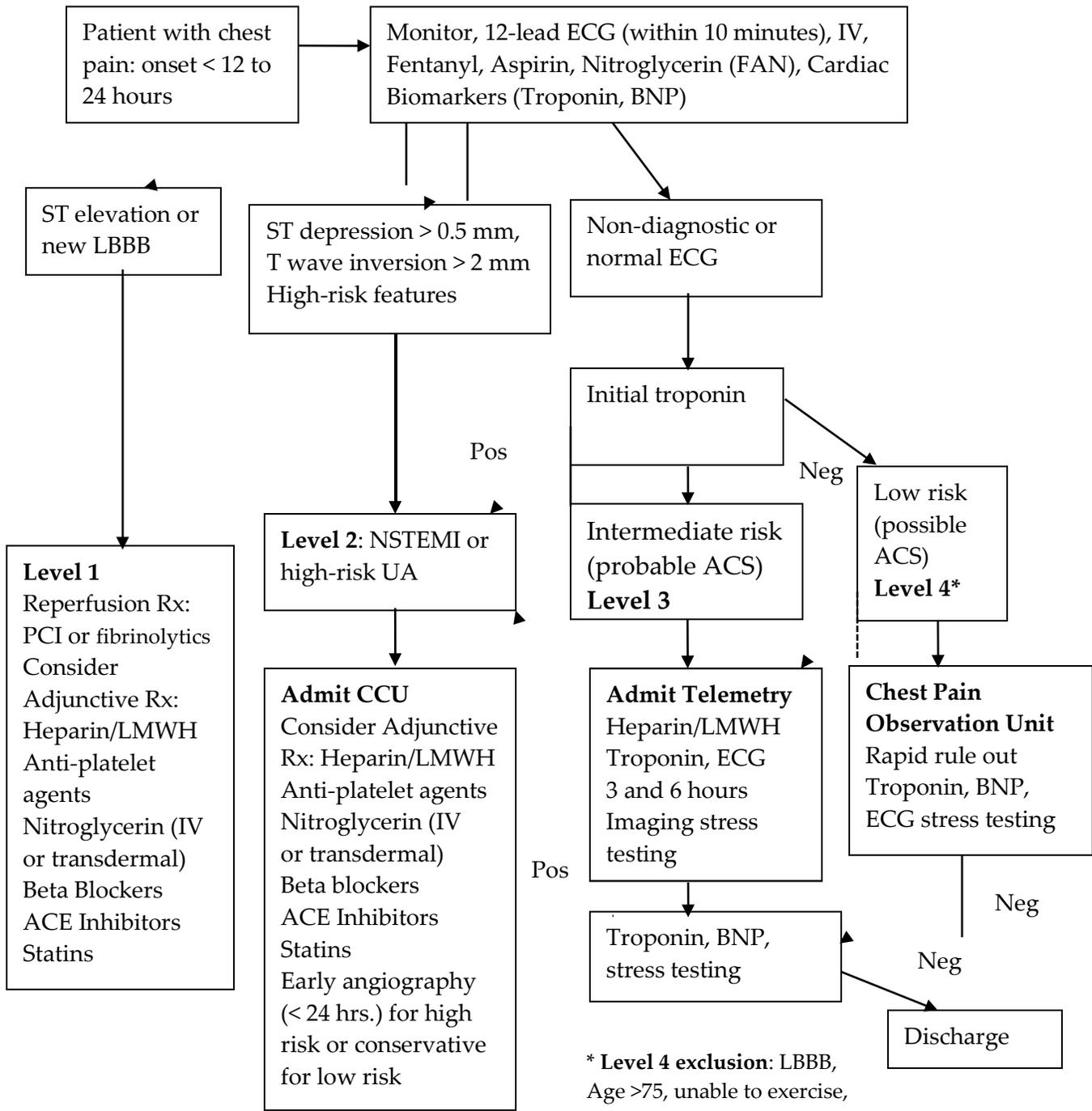
- Attempt initial cardioversion with 0.5 to 1 J/kg, mono- or bi-phasic
- May increase to 2 J/kg if initial dose is ineffective
- If the second shock is unsuccessful or tachycardia rapidly recurs, evaluate for correctable causes of the tachycardia such as hypoxemia, acidosis, hypoglycemia, or hypothermia. If a correctable cause is not found, consider antiarrhythmic therapy before delivering the third shock.

REFERENCE

Link MS, Atkins DL, Passman RS, Halperin HR, Samson RA, White RD, Cudnik MT, Berg MD, Kudenchuk PJ, Kerber RE. Part 6: electrical therapies: automated external defibrillators, defibrillation, cardioversion, and pacing: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010; 122(suppl 3):S706 –S719.

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ACUTE CARE 17: CHEST PAIN EVALUATION ALGORITHM



ACUTE CARE 18: TARGETED TEMPERATURE MANAGEMENT AFTER CARDIAC ARREST

Targeted Temperature Protocol

1. Patient Inclusion Criteria:

- An adult patient successfully resuscitated from a witnessed out-of-hospital cardiac arrest of presumed cardiac cause (a patient with in-hospital cardiac arrest may also benefit) *and*
- Is comatose at 10 minutes after return of spontaneous circulation (ROSC) *and*
- Had an initial rhythm of ventricular fibrillation or non-perfusing ventricular tachycardia (Patients presenting with asystole or PEA may also benefit.) *and*
- Is hemodynamically stable *and*
- Is 18 years of age or older (May consider using in younger pediatric patients if treating physician or consultant determine it advantageous.) *and*
- Has an initial temperature of $> 35^{\circ}\text{C}$ ($> 95^{\circ}\text{F}$)

2. Patient Exclusion Criteria:

- Initial temperature $< 30^{\circ}\text{C}$
- Comatose before the cardiac arrest
- Pregnancy
- Terminally ill or DNAR patients
- Coagulopathy
- MAP < 60 mm Hg for > 30 minutes
- O₂Sat $< 85\%$ for > 15 minutes
- Active bleeding
- Multisystem organ failure
- Sepsis
- ARDS
- QTc > 550 ms
- Recent major surgery with risk of bleeding

3. Targeted Temperature Management:

- Initiate Targeted Temperature Management as soon as possible after the cardiac arrest in eligible patients who are hemodynamically stable and within six hours of the arrest.
- Protect patient's airway with ETT or other advanced airway.
- Use hemodynamic support to maintain systolic BP > 90 mm Hg (90 mm Hg to 110 mm Hg) or MAP \geq 65.
- Use adequate sedation, analgesics, and paralysis to maintain patient comfort and to prevent shivering.
- Monitor core temperature with an esophageal, rectal, or bladder probe.
- Surface cool with ice packs or chemical cooling products applied to the head, neck, and torso (especially axilla and groin) as able.
- Cooling temperature goal is 32°C to 36°C (89.6°F to 96.8°F).
- Maintain the patient's temperature in the 32°C to 36°C (89.6°F to 96.8°F) range for 12 to 24 hours.
- Consider using treatments to prevent DVT, ventilator-associated pneumonia, and stress ulcers.
- Elevate the head of the bed (HOB) 30 degrees (decreases ventilator-associated pneumonia).
- Insert a Foley catheter (diuresis often occurs).
- If unable to guarantee that patient will stay cold in transport, delay cooling until arrival at referral center.
- Contact and consider referral to a Therapeutic Cooling Center.
- After 12 to 24 hours of hypothermia, rewarm slowly at a rate of 0.3°C to 0.5°C/ hour.

4. Patient monitoring:

- Vital signs every 5 minutes
- Temperature every 15 minutes
- ABG/VBG every 4 hours
- Serum potassium
- Serum magnesium
- Serum phosphorus
- Blood sugar

- WBC
- Platelet count
- ECG rhythm monitoring
- PT, PTT
- Central venous pressure

5. Watch for These Potential Adverse Effects of Targeted Temperature:

- Hypokalemia
- Hypomagnesemia
- Hypophosphatemia
- Hyperglycemia
- Leucopenia
- Thrombocytopenia
- Coagulation disorder
- Non-convulsive seizures
- Bradycardia (HR<40)
- Dysrhythmia (such as AF)

REFERENCE

Callaway CW, Donnino MW, Fink EL, et al. Part 8: Post-Cardiac Arrest Care: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015; 132(18 suppl 2): S465-482.

January 2019

ACUTE CARE 19: SHOCK ACRONYM – SHRIMPCAN

One way of defining the cause of shock and the appropriate treatment is:

1. Cardiac rate problem
 - heart contracts too slowly to meet the metabolic demands
 - heart beats too rapidly to allow effective heart filling during diastole
 - results in a significant decrease in cardiac output.
2. Pump problem
 - primary heart abnormalities resulting in inadequate cardiac output
 - due to a decrease in preload
 - vascular obstruction resulting in inadequate venous return to the heart
 - decrease in cardiac output
3. Volume problem
 - vascular volume loss either from hemorrhage or systemic fluid loss
 - results in vasodilation or third space fluid loss such that hypotension occurs

Consider the mnemonic **SHRIMPCAN** to help develop a broad differential if the cause is still unknown to you.

- S** Sepsis – gram-negative infection or other overwhelming infection
- H** Hypovolemia resulting from hemorrhage, dehydration, vomiting, peritonitis, pancreatitis, leaking aneurysm, ectopic pregnancy
- R** Respiratory compromise resulting in hypoxia, tension pneumothorax, massive pulmonary embolus
- I** Ingestion of a toxic substance, drug overdose
- M** Metabolic causes including diabetic ketoacidosis, hyperosmolar coma, myxedema, adrenal insufficiency, electrolyte abnormalities
- P** Psychiatric causes such as water intoxication
- C** Cardiogenic shock, acute myocardial infarction, cardiomyopathy, cardiac tamponade, dysrhythmias, severe cardiac failure, valvular heart disease, atrial myxomas
- A** Anaphylactic shock
- N** Neurogenic causes such as spinal shock, herniation syndromes, and intracranial bleed

ACUTE CARE 20: SYMPTOM RECOGNITION / THERAPY RECOMMENDATIONS*—CATEGORY A BIOAGENTS

Obtain up-to-date recommendations in consultation with CDC and/or state/local health departments and/or infectious disease experts. Anthrax and smallpox are of particular concern because they are hardy organisms easily grown in large quantities.

Agent	Symptom Complex	Incubation	Therapy	Immunization	Prophylaxis
Smallpox	Prodrome of fever, headache, nausea/vomiting, malaise; 2-3 days later: macular>deep pustular synchronous face/upper extremity rash. High to moderate lethality.	7-17 days	Cidofovir undergoing trial	Limited amounts of live virus vaccine at CDC. Smallpox vaccine, 1 dose by scarification	Live vaccine (or vaccinia immune globulin) if within 3 days of exposure; > 3 days exposure warrants both.
Inhalational anthrax (See other sources for skin and GI forms.)	Influenza-like illness but with no sore throat or rhinorhea plus shortness of breath. Very high lethality.	Usually 1-6 days but can be up to 8 weeks	STAT IV multidrugs: Cipro or doxycycline plus one of: rifampin, vancomycin, penicillin, ampicillin, imipenem, clindamycin, clarithromycin, chloramphenicol	Bioport military vaccine: multiple doses and annual boosters	Confirmed exposure: 60 days of either Cipro 500 mg every 12 hours or doxycycline 100 mg every 12 hours
Plague	Fever, dyspnea, hemoptysis, followed by fulminant pneumonia and respiratory failure. High lethality without treatment	2-3 days	IV drugs: streptomycin or gentamycin or doxycycline, or chloramphenicol	Greer inactivated vaccine: multiple doses and boosters	Doxycycline 100 mg twice daily or Cipro 500 mg twice daily or tetracycline 500 mg four times daily: 7 days or duration of exposure
Botulism	Bulbar palsies followed by descending symmetrical flaccid paralysis. High lethality without respiratory support (long term)	1-5 days	DOD heptavalent antitoxin. CDC trivalent antitoxin. Antibiotics not effective.	DOD (IND) pentavalent toxoid	NA
Tularemia	Flu-like illness followed by pulmonary infection and/or sepsis. Mod lethality without treatment.	1-21 days	10-14 day course of IM streptomycin or IV/IM gentamycin or IV Cipro	IND-live attenuated vaccine> incomplete protection	Doxycycline 100 mg twice daily or Cipro 500 mg twice daily x 2 weeks
Viral hemorrhagic fevers	High fever, HA, pains, followed by GI/mucous membrane bleeding. High lethality	4-21 days	Ribavirin IV may help some arenaviruses; passive antibody for AHF, BHF, Lassa, and CCHF	Several IND vaccines	Post exposure oral ribavirin may be effective.
AHF—Argentine hemorrhagic fever; BHF—Bolivian hemorrhagic fever; CCHF—Crimean Congo hemorrhagic fever; DOD—Department of Defense; GI—gastrointestinal; HA—headache; IND—Investigational New Drug. *Sources: CDC (2002)/National Center for Infectious Diseases (NCID).					

ACUTE CARE 21: BLAST INJURIES

Key Concepts

- Bombs and explosions can cause unique patterns of injury seldom seen outside combat
- Expect half of all initial casualties to seek medical care over a one-hour period
- Most severely injured arrive after the less injured, who bypass EMS triage and go directly to the closest hospitals
- Predominant injuries involve multiple penetrating injuries and blunt trauma
- Explosions in confined spaces (buildings, large vehicles, mines) and/or structural collapse are associated with greater morbidity and mortality
- Primary blast injuries in survivors are predominantly seen in confined space explosions
- Repeatedly examine and assess patients exposed to a blast
- All bomb events have the potential for chemical and/or radiological contamination
- Triage and life saving procedures should never be delayed because of the possibility of radioactive contamination of the victim; the risk of exposure to caregivers is small
- Universal precautions effectively protect against radiological secondary contamination of first responders and first receivers
- For those with injuries resulting in non-intact skin or mucous membrane exposure, give hepatitis B immunization (within 7 days) and age-appropriate tetanus toxoid vaccine (if not current). Consider HIV prophylaxis discussion.

Blast Injuries

- Primary: Injury from over-pressurization force (blast wave) impacting the body surface
 - TM rupture, pulmonary damage and air embolization, hollow viscus injury
- Secondary: Injury from projectiles (bomb fragments, flying debris)
 - Penetrating trauma, fragmentation injuries, blunt trauma
- Tertiary: Injuries from displacement of victim by the blast wind
 - Blunt/penetrating trauma, fractures and traumatic amputations
- Quaternary: All other injuries from the blast
 - Crush injuries, burns, asphyxia, toxic exposures, exacerbations of chronic illness

Primary Blast Injury

- **Lung Injury**
 - Signs usually present at time of initial evaluation, but may be delayed up to 48 hrs
 - Reported to be more common in patients with skull fractures, >10% BSA burns, and penetrating injury to the head or torso
 - Varies from scattered petechiae to confluent hemorrhages
 - Suspect in anyone with dyspnea, cough, hemoptysis, or chest pain following blast
 - CXR: "butterfly" pattern
 - High flow O₂ sufficient to prevent hypoxemia via NRB mask, CPAP, or ET tube
 - Fluid management similar to pulmonary contusion; ensure tissue perfusion but avoid volume overload

Blast Injuries (continued)

- Endotracheal intubation for massive hemoptysis, impending airway compromise or respiratory failure
 - Consider selective bronchial intubation for significant air leaks or massive hemoptysis
 - Positive pressure may risk alveolar rupture or air embolism
- Prompt decompression for clinical evidence of pneumothorax or hemothorax
- Consider prophylactic chest tube before general anesthesia or air transport
- Air embolism can present as stroke, MI, acute abdomen, blindness, deafness, spinal cord injury, claudication
 - High flow O₂; prone, semi-left lateral, or left lateral position
 - Consider transfer for hyperbaric O₂ therapy
- **Abdominal Injury**
 - Gas-filled structures most vulnerable (esp. colon)
 - Bowel perforation, hemorrhage (small petechiae to large hematomas), mesenteric shear injuries, solid organ lacerations, and testicular rupture
 - Suspect in anyone with abdominal pain, nausea, vomiting, hematemesis, rectal pain, tenesmus, testicular pain, unexplained hypovolemia
 - Clinical signs can be initially subtle until acute abdomen or sepsis is advanced
- **Ear Injury**
 - Tympanic membrane most common primary blast injury
 - Signs of ear injury usually evident on presentation (hearing loss, tinnitus, otalgia, vertigo, bleeding from external canal, otorrhea)

Other Injury

- Traumatic amputation of any limb is a marker for multi-system injuries
- Concussions are common and easily overlooked
- Consider delayed primary closure for grossly contaminated wounds, and assess tetanus immunization status
- Compartment syndrome, rhabdomyolysis, and acute renal failure are associated with structural collapse, prolonged extrication, severe burns, and some poisonings
- Consider possible exposure to inhaled toxins (CO, CN, MetHgb) in industrial and terrorist explosions
- Significant percentage of survivors will have serious eye injuries

Disposition

- No definitive guidelines for observation, admission, or discharge
- Discharge decisions will also depend upon associated injuries
- Admit 2nd and 3rd trimester pregnancies for monitoring
- Close follow-up of wounds, head injury, eye, ear, and stress-related complaints
- Patients with ear injury may have tinnitus or deafness; communications and instructions may need to be written

ACUTE CARE 22: CAUSES OF ANION AND NON-ANION GAP ACIDOSIS

Presence of an increased anion gap signifies a base deficit and a metabolic acidosis. Normal anion gap is $12 \pm 2-4$ mEq/L. Anion gap acidosis means the value is above the normal

Anion gap = $NA^+ - [Cl^- + HCO_3^-]$ = unmeasured anions - unmeasured cations

Normal sodium value = 140 mEq/L ± 4 mEq/L

Normal chloride = 100 if the sodium is 140 (roughly Na^+ divided by 1.4).

An abnormal chloride level is a red flag for some sort of acid/base abnormality, even if HCO_3^- is normal.

Normal HCO_3^- = $22 - 26$ mEq/L

To recall causes of anion gap acidosis, use the mnemonic MUDPILES:

M – Methanol

U – Uremia

D – Diabetic ketoacidosis

P – Paraldehyde

I – Iron or isoniazide, inhalants (CO, CN, H₂S)

L – Lactic acid

E – Ethanol (ketoacidosis), ethylene glycol

S – Salicylates, solvents (toluene), starvation (ketoacidosis)

To recall causes of non-anion gap acidosis, use the mnemonic USED CARP:

U – Ureteroenterostomy

S – Small bowel fistula

E – Extra chloride **D** –

Diarrhea

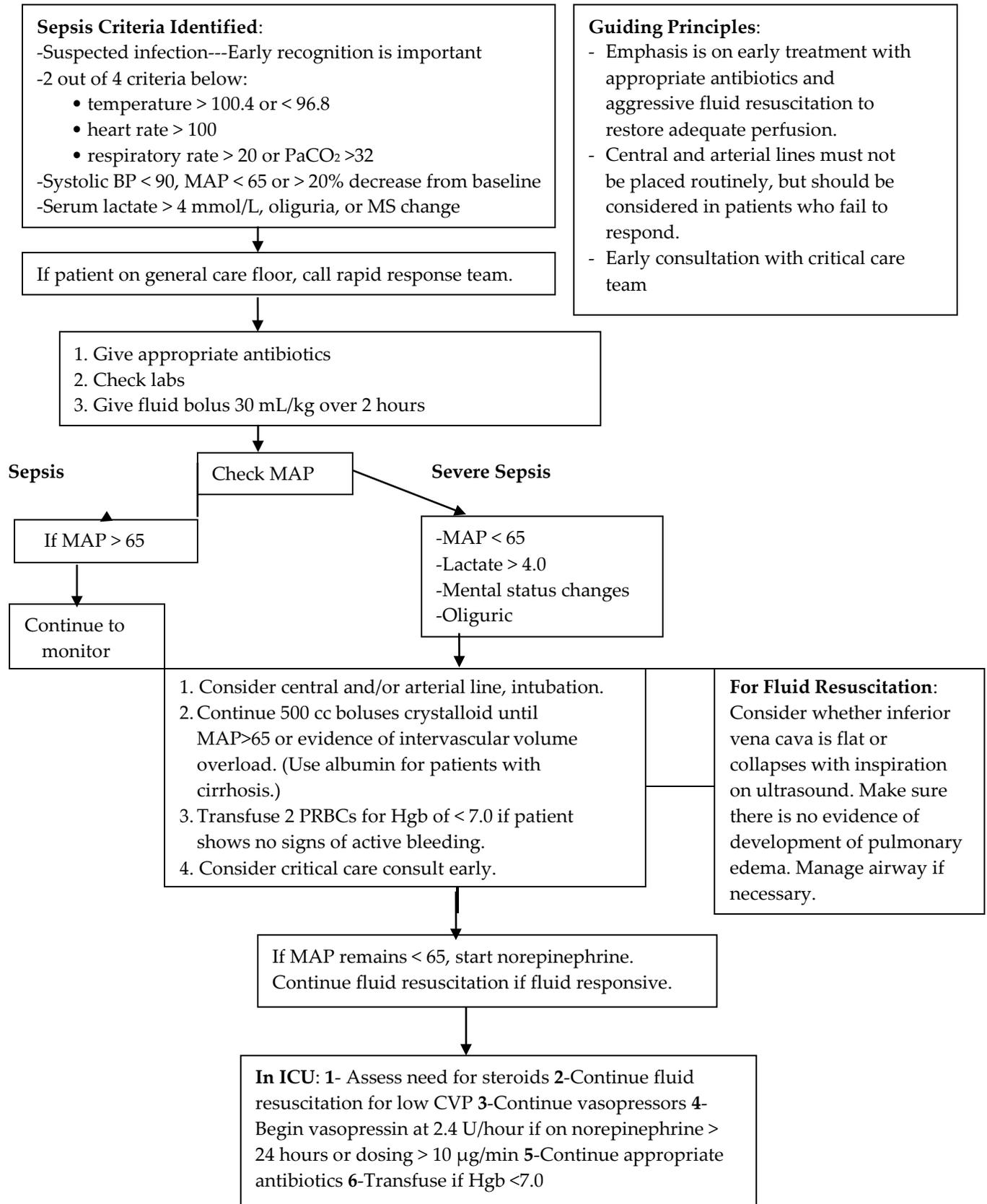
C – Carbonic anhydrase inhibitors

A – Adrenal insufficiency **R** –

Renal tubular acidosis **P** –

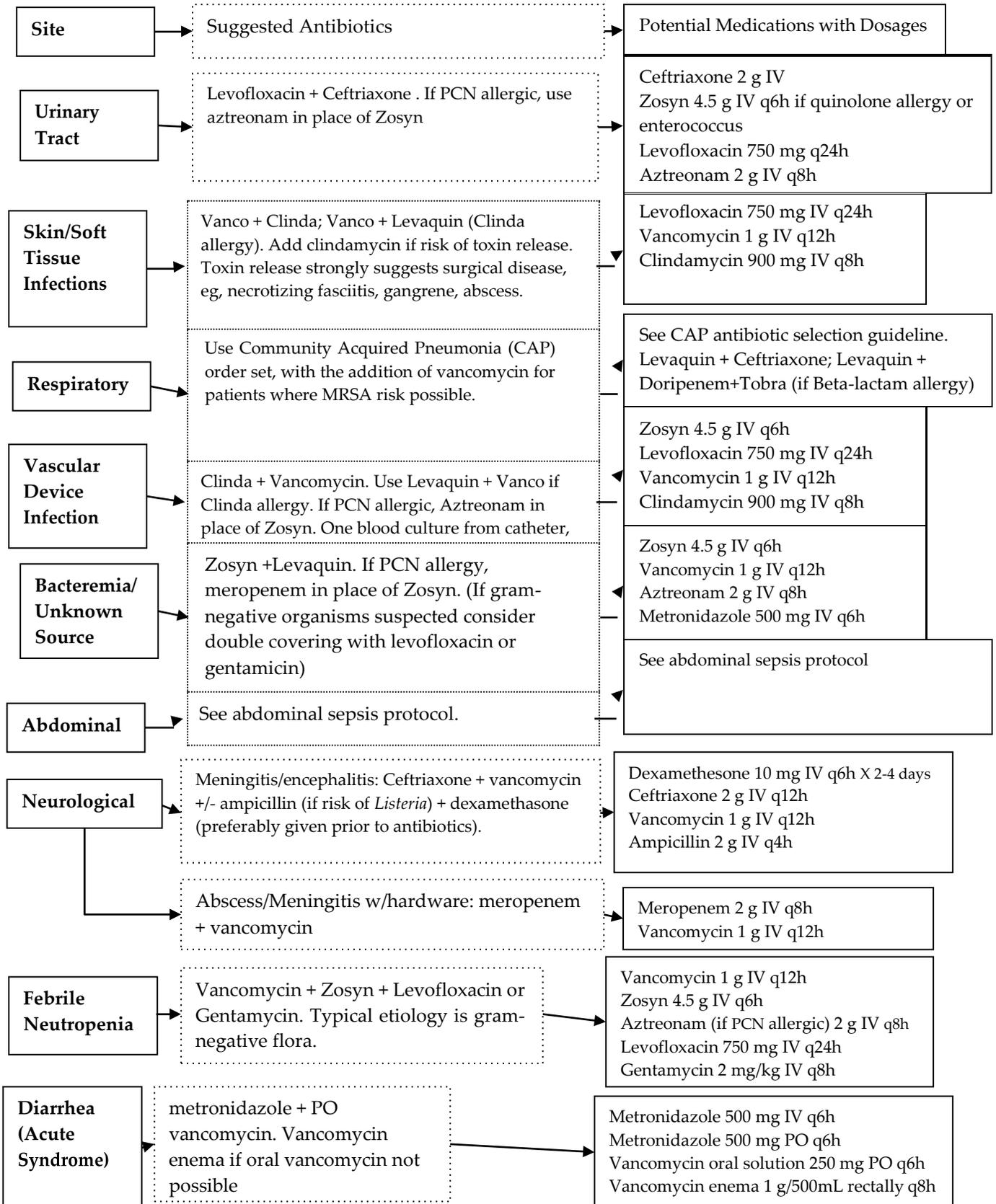
Pancreatic fistula

ACUTE CARE 23: SEPSIS GUIDELINES*



Antibiotic Selection for Sepsis Protocol

(Note: Antibiotic guidelines may vary by clinical setting and location.)



Community Acquired Pneumonia Guidelines

Physician Orders	Nursing Guidelines/Orders
Routine patient orders: oxygen, cardiac monitor, IV access Obtain pneumonia severity index (PSI) score http://pda.ahrq.gov/clinic/psi/psicalc.asp	<ul style="list-style-type: none"> ▪ Oxygen at 2 to 4 L/min per nasal cannula or more to obtain SpO₂ > 90% ▪ Place on cardiac monitor ▪ Continuously monitor SpO₂, RR, HR, and intermittent BP readings. Insert at least one IV
Labs: <ul style="list-style-type: none"> ▪ Chem-8 ▪ Hemogram, platelets with differential ▪ Lactate ▪ Blood cultures X 2 ▪ Urinalysis, conditional UC ▪ ABG if critical patient ▪ Sputum culture and smear ▪ Urine antigen if PSI > 90 	<ul style="list-style-type: none"> ▪ Draw rainbow of tubes, including a lactate on ice ▪ Obtain first blood culture with initial draw ▪ Obtain second blood culture 15 minutes after first blood culture obtained, or at minimum, less than 1 hour after antibiotics started ▪ Draw ABG and place on ice; send to lab. ▪ May call Respiratory Therapy to obtain sputum sample
Diagnostic Testing: ECG 12-lead; Chest X-ray PA and lateral (portable if critical patient)	If patient is monitored, the RN will accompany to x-ray.
Respiratory Medications: Albuterol nebulizer treatment Ipratropium nebulizer treatment	Obtain peak flow before and after administering nebulizer treatments.
Antipyretics: APAP or ibuprofen for temp > 101.5°F	Consider rectal APAP if unable to swallow.
Antibiotics: For patients to be admitted: azithromycin 500 mg IV x 1 and one of the following: <ol style="list-style-type: none"> 1. ceftriaxone 1 g IV x 1 2. levofloxacin 750 mg IV x 1 For patients to be discharged: azithromycin 500 mg oral x 1 in ED levofloxacin 500 mg oral x 1 in ED Discharge prescriptions: azithromycin (Z-pack) 250 mg oral cefuroxime 500 mg PO BID x 7days	Infuse azithromycin over 60 min via infusion pump. 1. Infuse ceftriaxone over 30 min via infusion pump. 2. Infuse 750 mg IV over 90 min via infusion pump. Antibiotics need to be started within 4 hours of arrival.
Admission: Admit to the hospitalist service in either medical or telemetry bed unless patient is in critical condition.	Complete admission note before transport.

Severe Sepsis Protocol Checklist*

Based on the Evaluation for Severe Sepsis Screening Tool

- Does patient history suggest a new infection? If yes,
- Does patient present with 2 or more new signs or symptoms of infection? If yes,
- Does the patient have evidence of organ dysfunction due to the infection?

If answers to ALL screening elements are YES, initiate Severe Sepsis Protocol.

- Determine time of presentation, which is equal to ED triage time or documentation (date/time) supporting diagnosis of severe sepsis in progress notes for non-ED admissions.

Quality Indicators to Measure

Sepsis Resuscitation Bundle—The goal is to perform all indicated tasks 100% of the time within the first 6 hours of identification of severe sepsis.

- Measure serum lactate.
- Obtain blood cultures prior to antibiotic administration.
- Administer broad-spectrum antibiotic **within 3 hours of ED admission and within 1 hour of non-ED admission.**

Admission

In the event of hypotension and/or a serum lactate > 4 mmol/L

- Deliver an initial minimum of 30 mL/kg crystalloid or an equivalent
- Apply vasopressors for hypotension not responding to initial fluid resuscitation to maintain mean arterial pressure (MAP) > 65 mm Hg

In the event of persistent hypotension despite fluid resuscitation (septic shock) and/or lactate > 4 mmol/L

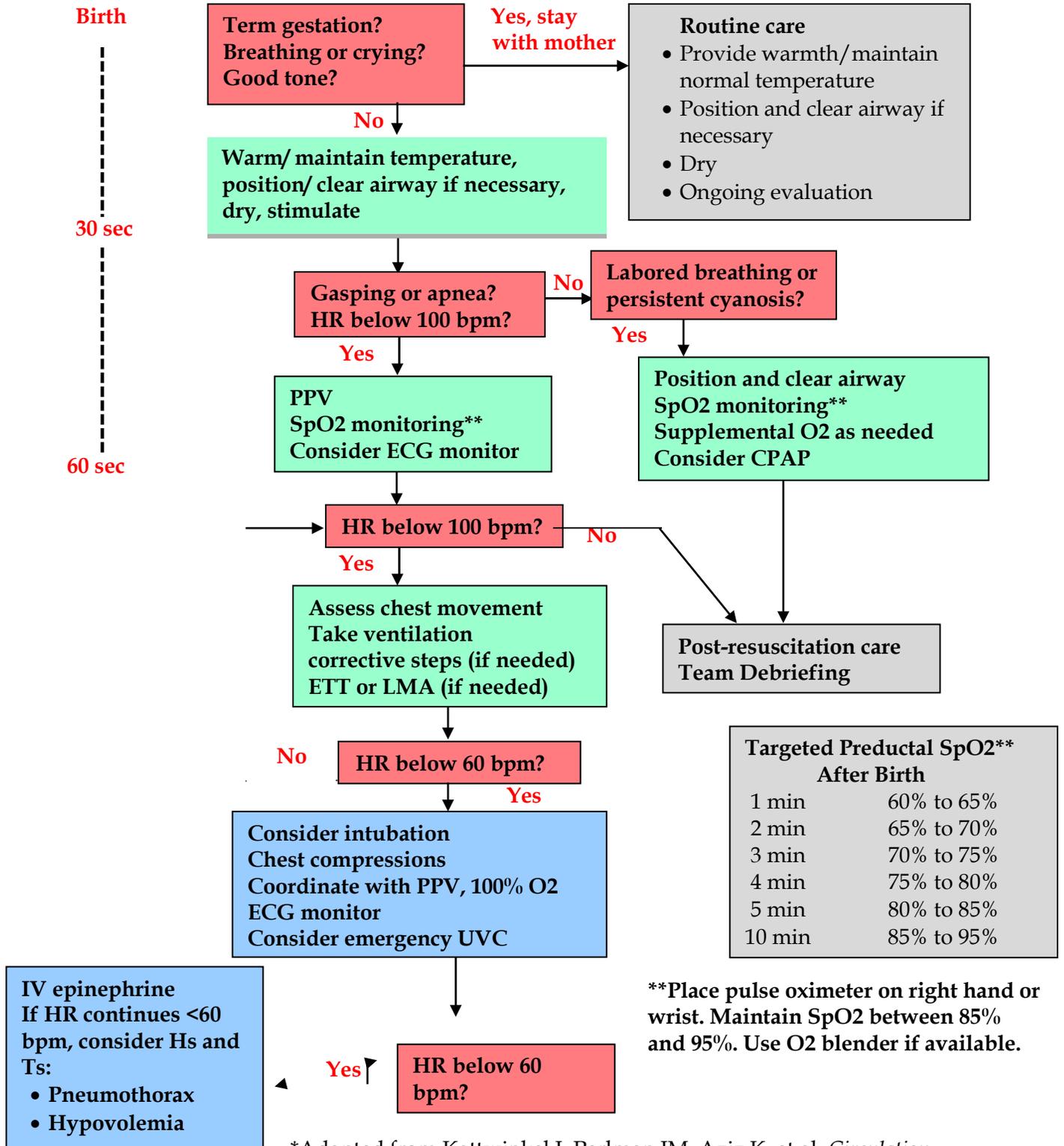
Sepsis Management Bundle

Begin efforts to accomplish these goals immediately, but these items may be completed within 24 hours of presentation for patients with severe sepsis or septic shock:

- Do not use corticosteroids in adult septic shock patients if adequate fluid resuscitation and vasopressor therapy are able to restore hemodynamic stability.
- Maintain glucose control ≥ 70 , but < 180 mg/dL
- Maintain a median inspiratory plateau pressure (IPP) < 30 cm H₂O for mechanically ventilated patients.

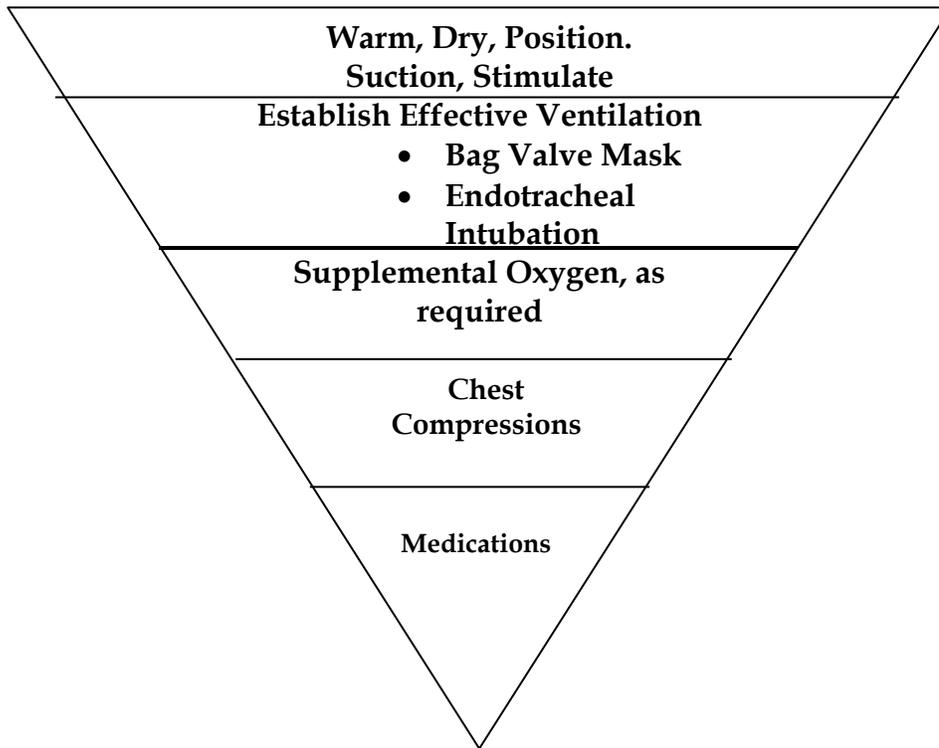
* Adapted from Regions Hospital Emergency Department, St. Paul, Minnesota, 2013.

ACUTE CARE 24: NEWBORN RESUSCITATION ALGORITHM*



*Adapted from Kattwinkel J, Perlman JM, Aziz K, et al. *Circulation* 2010;122:S909-S919 and Myra H, Wyckoff et al. *Circulation*. 2015; 132:S543-S560.

ACUTE CARE 25: INVERTED TRIANGLE/APGAR SCORE



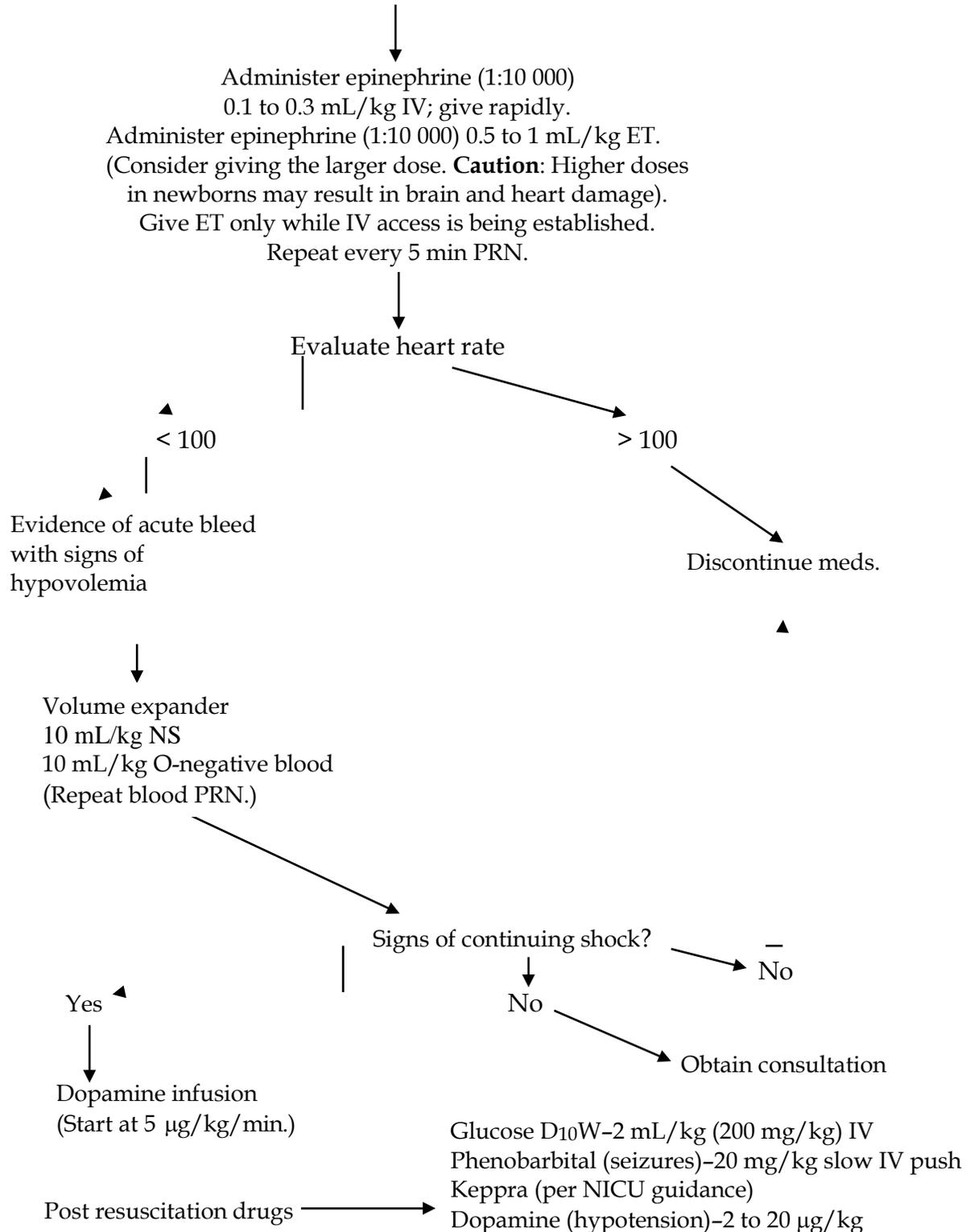
APGAR Score: Measure at 0 and 5 minutes.

Sign	0	1	2
Heart rate/ minute	Absent	Slow (< 100)	> 100
Respirations	Absent	Weak cry, hypoventilation	Good, crying
Muscle Tone	Limp		Active motion
Reflex irritability (Catheter in nostrils)	No response	Grimace	Cry or active withdrawal
Color	Blue or pale	Pink body blue extremities	Completely pink

Reviewed January 2019

ACUTE CARE 26: DRUGS IN NEONATAL RESUSCITATION ALGORITHM

Indications: Heart rate < 60 after 30 sec CPR and PPV with 100% O₂



ACUTE CARE 27: ALTERED LEVEL OF CONSCIOUSNESS

Causes (Tips from the Vowels):

T – Trauma, Temperature, Tumor I – Infection P – Poisoning, Psychogenic S – Stroke, Shunts, Shocks	A – Alcohol E – Epilepsy, Encephalopathy I – Insulin O – Oxygen, Opiates, and Drug Abuse U – Uremia and Other Metabolic Abnormalities
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Assessment Tools

DO the DON'T

D – Dextrose IV for possible hypoglycemia, unless a bedside glucose has been determined to be normal. For adults and children weighing >26 kg, give 50 mL IV bolus of 50% glucose.

PEDS: For children weighing 5 kg to 25 kg, give 2 mL/kg of 25% glucose. For infants weighing < 5 kg, give 2 to 4 mL/kg of 10% dextrose.

Do not give dextrose to a brain-injured patient without checking blood glucose level first.

O – Oxygen

N – Naloxone for possible opiate intoxication (after the patient is secure), give 0.4 to 2 mg IV every 2 to 3 minutes in adults; or give 0.1 mg/kg IN – max 2 mg (2 mg/2 mL concentration).

PEDS: For infants, give 0.1 mg/kg IV or IN – max 2 mg (same as adult concentration).

(Beware of patients on chronic opioids. In such patients, titrate Narcan for respiratory support. Avoid acute withdrawal.)

T – Thiamine 100 mg IV push in adults; give before glucose.

Mini Neuro Exam

1. Level of Consciousness – AVPU Method

	LOC	GCS equivalent
A	Alert	14 to 15
V	Responds to voice	12 to 13
P	Responds to pain	8
U	Unresponsive	3 to 4

2. **Pupils and Vision** – Conjugate or disconjugate gaze?
 Pupil size, equality, and reactivity
 Vision check with finger counting
3. **Tympanic Membranes** – Hemotympanum?
4. **Neck Tenderness** Midline tenderness?
5. **Extremities** – Movement and strength on command or pain
 Withdraws, purposeful or none
 Ankle, patellar and brachial reflexes and clonus
 Babinski signs
 Sensation and position sense
6. **Trunk** – Priapism, saddle sensation, anal sphincter tone, sensation level

If there is a depressed LOC with lateralization:

Check for dilated pupil and limb weakness on the opposite side. If present, uncal herniation may be occurring. For adults, give mannitol 1 g/kg IV. **PEDS:** In children, give 0.25 to 0.5 g/kg IV. Orotracheally intubate, maintain ventilation rate at 10-12 breaths per minute, and complete the initial survey. Do not hyperventilate aggressively. $p\text{CO}_2 < 30$ may be harmful.

If there is seizure activity:**Initial management:**

Lorazepam (Ativan): Adult: 0.1 mg/kg IV at a max rate of 2 mg/ min to total dose of 8 mg; **PEDS:** 0.1 mg/kg IV at a max rate of 2 mg/ min to total dose of 4 mg. Alternatively, in adults, give 4 mg IV; repeat in 3 to 5 minutes if patient is still seizing. Administer oxygen by mask. If patient has a depressed gag reflex, oro-tracheally intubate. Prepare for vomiting, aspiration, and cardiac arrhythmias. Complete the initial survey.

Diazepam (Valium): Adult: 0.2 mg/kg at 5 mg/min IV. **PEDS:** 0.3 mg/kg at 5 mg/min IV. For both, may repeat in 3 to 5 minutes if patient is still seizing.

Diazepam may also be given PR at double the IV dose to a max of 10 mg.

Midazolam (Versed): Adult: 0.02 mg/kg IM, 10 mg max; **PEDS:** 0.1 mg/kg IV slowly over minimally 30 sec; 0.05 mg/kg to 0.15 mg/kg IM, 10 mg max; 0.2 mg/kg IN (via atomizer), 10 mg max.

If the patient is posturing:

Check for decorticate (arm flexion) or decerebrate (arm and leg extension) posturing. If present, for adults, give 1 g/kg mannitol IV. **PEDS:** In children, give 0.25 to 0.5 g/kg IV. (Other choice is Hypertonic Saline, either 3 or 5%). Orotracheally intubate, maintain ventilation rate at 10-12 breaths per minute, and complete the initial survey. Do not hyperventilate aggressively. $p\text{CO}_2 < 30$ may be harmful.

If there is evidence of a spinal cord lesion or injury:

Look for paralysis, sensory deficit, and priapism. A more thorough mini neuro exam may be done later. But if RSI must be done quickly, it should be preceded by a mini neuro exam first.

Stabilize the patient's neck before intubation.

August 2017

ACUTE CARE 28: GLASGOW COMA SCALE– GCS-P

ADULT, PEDIATRIC, INFANT

Eye Opening

Infant (<1 year)

Pediatric (>1 year)

Adult

Spontaneous	Spontaneous	Spontaneous	4
Voice	Voice	Speech	3
Pain	Pain	Pressure	2
None	None	None	1

Verbal Response

Infant (<2 years)

Pediatric (>2 years)

Adult

Coos, babbles	Appropriate word/ phrase	Oriented	5
Irritable but consolable	Disoriented/ converses	Confused	4
Persistent cries/ screams	Inappropriate word	Words	3
Moans/grunts to pain; restless	Incomprehensible sounds	Sounds	2
None	None	None	1

Motor Response

Infant (<1 year)

Pediatric (>1 year)

Adult

Spontaneous	Obeys	Obeys	6
Localizes pain	Localizes pain	Localizes pain	5
Flexion- withdrawal	Flexion- withdrawal	Withdraws	4
Flexion/ decorticate	Flexion/ decorticate	Abnormal flexion (decorticate)	3
Extension/ decerebrate	Extension/ decerebrate	Abnormal extension (decerebrate)	2
None	None	None	1
			3 to 15

Note; Changes made in 2014 such that previously when eye opening or verbal response could not be tested (swelling or intubation), the value was scored as a 1. This has now been changed, so that those components, that cannot be tested are marked "NT" and the total score is not calculated.

ACUTE CARE 29: TIPS FROM THE VOWELS – AEIOU

Tips from the Vowels

T – Trauma, Temperature, Tumor

- Traumatic head injury
- Hypothermia
- Hyperthermia
- Tumor of the brain

I – Infection

- Meningitis/Encephalitis
- Sepsis
- Pneumonia
- Urosepsis

P – Poisoning, Psychogenic Poisoning (Drug Overdose, Toxic Effects)

- Acetaminophen overdose
- Aspirin overdose
- Tricyclic overdose
- Anticholinergic drug overdose
- Benzodiazepine overdose
- Hypnotic drug overdose, in line with poisoning-toxic substance ingestion
- Carbon monoxide
- Cyanide
- Organophosphate (insecticide)
- Heavy metal poisoning (iron, lead, mercury)
- Hydrocarbon ingestions
- Psychogenic coma (catatonic state)

S – Stroke, Shunts, Shock (CNS Insults)

- Thrombotic or embolic strokes
- Intracerebral hemorrhage
- Subarachnoid hemorrhage
- Subdural hematoma
- Epidural hematoma
- Increased intracranial pressure
- Shock (from any source)
- Shunt malfunction

A – Alcohol (Intoxication, Withdrawal, Toxic Effects)

- Ethyl alcohol intoxication
- Ethyl alcohol withdrawal
- Methanol intoxication
- Ethylene glycol intoxication
- Isopropanol intoxication

E – Epilepsy, Encephalopathy

- Status epilepticus/postictal
- Hypertensive encephalopathy
- Hepatic encephalopathy

I – Insulin and Other Hormones

- Diabetic ketoacidosis
- Non-ketotic hyperosmolar coma
- Hypoglycemia
- Ketotic hypoglycemia
- Addisonian crisis
- Thyroid storm (hyperthyroidism)
- Myxedema coma

O – Oxygen, Opiates, and Other Drugs of Abuse

- Hypoxia
- High altitude illness
- Opiate/heroin/narcotic overdose
- Cocaine ingestion
- Amphetamine intoxication
- Phencyclidine intoxication

U – Uremia and Other Metabolic Abnormalities

- Acute renal failure
- Electrolyte abnormalities
- Acid base abnormalities

ACUTE CARE 30: NIH STROKE SCALE (ABBREVIATED)

The NIH Stroke Scale is a relatively reproducible and standardized evaluation tool used to help estimate the severity of a patient's stroke and to monitor the patient's progression of improvement or deterioration over time.

Item	Scale Definition	Scale	Score
1a. Level of Consciousness (LOC) <i>(Alert, drowsy, etc.)</i>	Alert	0	
	Drowsy	1	
	Stuporous	2	
	Coma	3	
1b. LOC Questions <i>(Month, age)</i>	Answers both correctly	0	
	Answers one	1	
	Answers neither	2	
1c. LOC Commands <i>(Open, close eyes; make fist, let go)</i>	Does both correctly	0	
	Does one	1	
	Does neither	2	
2. Best Gaze <i>(Both eyes open – patient follows examiner's finger)</i>	Normal	0	
	Partial gaze palsy	1	
	Forced deviation	2	
3. Visual <i>(Introduce visual stimulus/threat to patient's visual field quadrants)</i>	No visual loss	0	
	Partial hemianopia	1	
	Complete hemianopia	2	
	Bilateral hemianopia	3	
4. Facial Palsy <i>(Show teeth, raise eyebrows, and squeeze eyes shut)</i>	Normal	0	
	Minor	1	
	Partial	2	
	Complete	3	
5. Motor Arm <i>(Elevate extremity to 45° for 10 seconds and score drift/movement)</i>	No drift	0	Left
	Drift	1	
	Some effort vs gravity	2	
	No effort vs gravity	3	Right
	No movement	4	
	Injured (explain)	0	
6. Motor Leg <i>(Elevate extremity to 30° for 5 seconds and score drift/movement)</i>	No drift	0	Left
	Drift	1	
	Some effort vs gravity	2	
	No effort vs gravity	3	
	No movement	4	Right
	Injured (explain)	0	

7. Limb Ataxia (<i>Finger-nose, heel down shin</i>)	Absent	0	
	Present in 1 limb	1	
	Present in 2 limbs	2	
8. Sensory (<i>Pin prick to face, arm, trunk, and leg – compare sides</i>)	Normal	0	
	Partial loss	1	
	Severe loss	2	
9. Best Language (<i>Name items, describe a picture, and read sentences</i>)	No aphasia	0	
	Mild to moderate aphasia	1	
	Severe aphasia	2	
	Mute	3	
10. Dysarthria (<i>Evaluate speech clarity by patient repeating listed words</i>)	Normal articulation	0	
	Mild to moderate dysarthria	1	
	Near to unintelligible or worse	2	
	Unable to evaluate	0	
11. Extinction and Inattention (<i>Use information from prior testing to identify neglect or double simultaneous stimuli testing</i>)	No neglect	0	
	Partial neglect	1	
	Complete neglect	2	

Level of Stroke Severity: 0=no stroke; 1-4=minor stroke; 5-15= moderate stroke; 15-20=moderate/severe stroke; 21-42=severe stroke.

Strokes with scores greater than 4 may be considered for treatment with tPA.

REFERENCE

1. NIH Stroke Scale. National Institutes of Health. National Institute of Neurological Disorders. https://www.stroke.nih.gov/documents/NIH_Stroke_Scale_508C.pdf
2. Accessed December 20, 2018.

January 2019

ACUTE CARE 31: STATUS EPILEPTICUS TREATMENT PLAN

Status Epilepticus – Initial Management Algorithm – Age 12 to Adult¹⁻³

A – Open and Control **Airway**- (Administer O₂, suction, and place airway as needed)
B – Breathing – (Assess air exchange, ventilate with BVM, and intubate as needed)
C – **Circulation** – (Vitals – O₂ sat, BP, HR, RR, Temp) (ECG Monitor) (IV access)
D – Assess **Disability** (AVPU), **Do the DONT**

Benzodiazepines

- Lorazepam (Ativan) – 0.1 mg/kg IV or IO at 2 mg/min, up to 4 mg/dose. May repeat dose once in 10 to 15 minutes.
or
- Diazepam (Valium) – 0.2 mg/kg at 5 mg/min IV or IO up to 10 mg/dose. May repeat in 15 to 30 minutes.
or
- If IV/IO access not available – consider:
* Versed 10 mg IM
or
- Buccal or IM or IN midazolam 0.2 mg/kg to max of 10 mg
or
- * Keppra 20 mg IV loading dose to max of 2.5 to 3 gms. (Same for adults as pediatrics.)

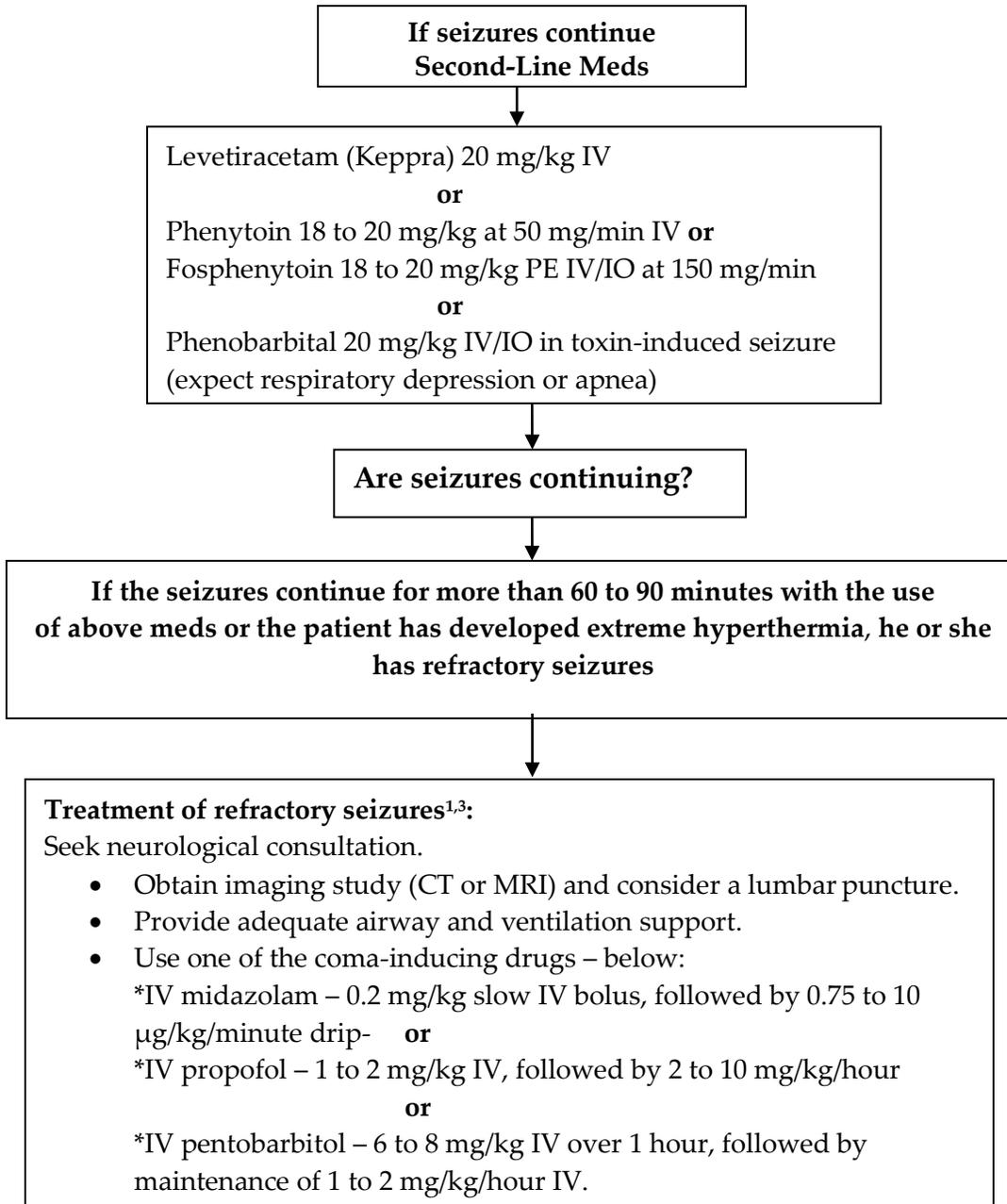
Focused History and Physical Exam

- History of seizures, infection, trauma, substance abuse
- Medications
- Focal neurological signs
- Signs of trauma
- Signs of medical illness (infection, renal, electrolyte, hepatic)

Laboratory Tests

- CBC, electrolytes, Ca, Mg, BUN, creatinine, glucose, liver function, antiepileptic drug levels, and toxicology screen
- Consider ABGs/VBGs
- Consider need for LP, head CT, and other tests to define cause

Antiepileptic Drug Therapy Algorithm–Age 12 to Adult
(Use algorithm if patient is actively seizing or has recurrent seizures)



REFERENCES

1. Leppik IE. Status Epilepticus Treatment in 2001- New Approaches, New Medicines. *MINCEP Epilepsy Reports*. 2001; Volume X: Number 2.
2. Bone RC. Treatment of Convulsive Status Epilepticus. *JAMA*. 1993;270:854-859.
3. Lowenstein, DH. Status Epilepticus. *N Engl J Med*. 1998;338:970-976.

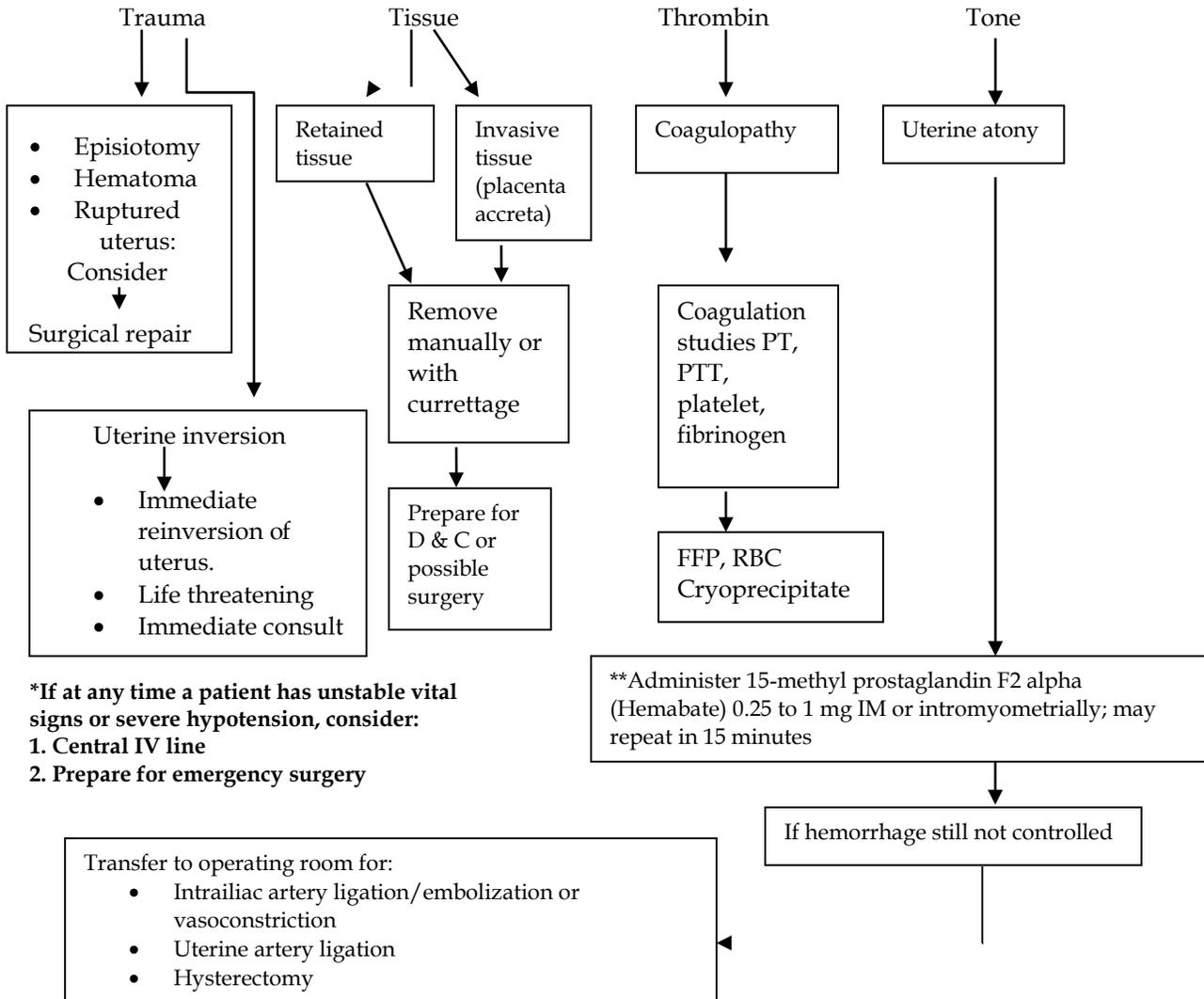
ACUTE CARE 32: POSTPARTUM HEMORRHAGE ALGORITHM

Perform uterine massage*
Administer oxygen
Insert large bore IV with crystalloid (NS or LR)
Start Oxytocin 10 U IM or 10 to 40 U/L IV @ < 250 mL/h Administer
Methergine, 0.2 mg IM (do not use for PPH in women with HTN)

Is hemorrhage controlled? → Yes → 1. Maintain IV access
2. Maintain Oxytocin for 24 h
3. Monitor vital signs and bleeding

No

Proceed to Hemabate below if hemorrhage continues**
Perform manual uterine exploration (consider procedural sedation)
Consider the causes of postpartum hemorrhage



***If at any time a patient has unstable vital signs or severe hypotension, consider:**
1. Central IV line
2. Prepare for emergency surgery

ACUTE CARE 33: SHOULDER DYSTOCIA ACRONYM—HELPERR

The mnemonic **HELPERR** is a useful guide for what to consider in in shoulder dystocia.

H—HELP

E—Consider **Episiotomy**

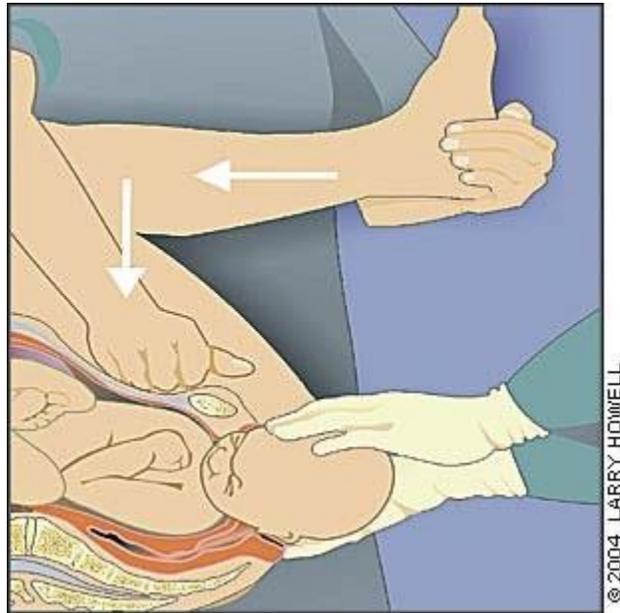
L- **Legs**—McRobert's maneuver

P - **Pressure**—Suprapubic **P**ressure externally

E- **Enter** the vagina using internal pressure to reduce impacted shoulder, finally using a Wood's screw maneuver to bring the shoulders into oblique diameter and 180 degrees rotation, if necessary.

R- **Remove** the posterior arm. Finally, if all other maneuvers fail, cephalic replacement may be used in certain circumstances.

R-**Rotate** the patient to her hands and knees



Reference; ALSO Mnemonic Reference Cards

ACUTE CARE 34: ASSISTED DELIVERY ACRONYM— ABCDEFGHIJ

Procedure: Delivery using the vacuum extractor or forceps should only be attempted by those trained in their use. The same acronym applies to both:

A—Anesthesia may be local or by pudendal block.

B—Bladder empty? Catheterize if needed.

C—Cervix completely dilated?

D—Determine position and think shoulder **Dystocia**.

E—Equipment and **Extractor** ready?

F—Insert cup and position on the **flexion** point. (See figure below.) Apply negative pressure with contraction.

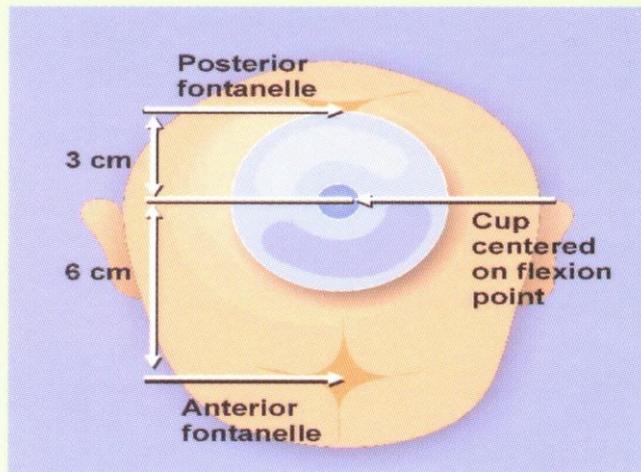
G—Gentle traction in the direction of the pelvic axis (timed with contraction).

H—Halt traction when the contraction is over.

I—Make Incision for the episiotomy when head is being delivered if perineum distends.

J—Remove vacuum cup/forceps when **Jaw** is delivered.

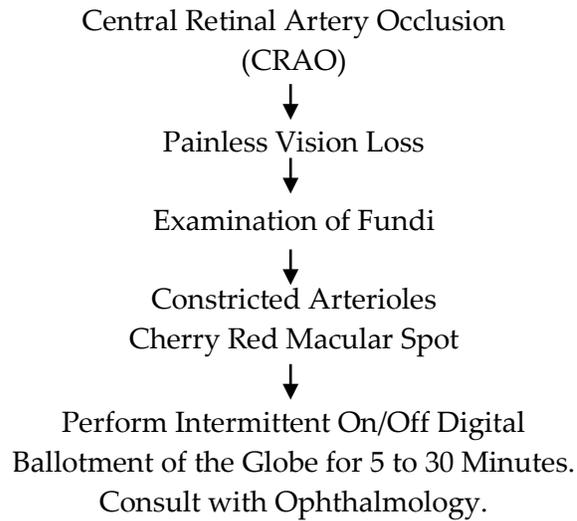
Cup Placement for Vacuum



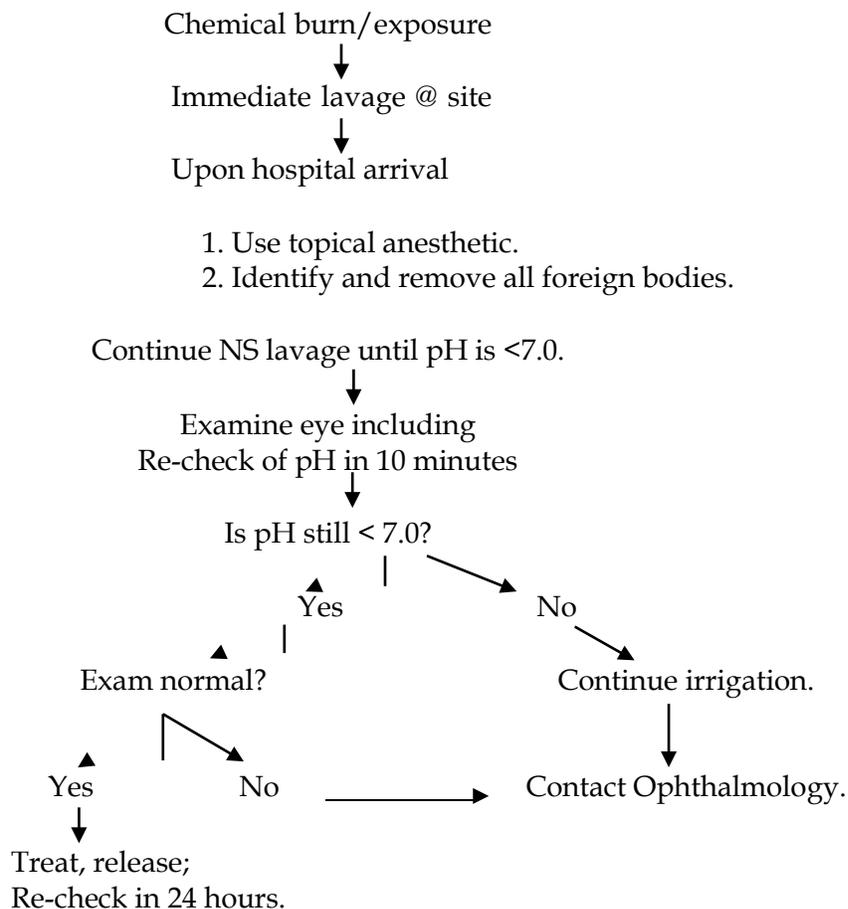
Proper cup placement on the flexion point is essential for safety and efficiency. Traction centered on the flexion point helps keep the neck flexed.

Reference; ALSO Mnemonic Reference Cards

ACUTE CARE 35: CENTRAL RETINAL ARTERY OCCLUSION



ACUTE CARE 36: CHEMICAL BURN EXPOSURE TO EYE ALGORITHM



ACUTE CARE 37: PEDIATRIC EQUIPMENT SIZES*

Pediatric ET Tube Sizes and Other Tubes

Age	ET Tube (mm ID)	Blade Size (French)	Tracheal Suction	Chest Tube (French)	Gastric (French)	Foley (French)
Premie	2.5-3.0	0	6.5	10-14	5	5
Neonate	3.0-4.0 cuffed	1	6.5	12-18	8	6
6 months	3.5-4.0 cuffed	1	8	14-22	10	8
1 year	4.5 cuffed	2	8	16-26	12	10
2 years	4.5 cuffed	2	8	16-26	12	10
3 years	5.0 cuffed	2	10	18-30	14	12
4 years	5.0 cuffed	2	10	18-30	14	12
5 years	5.5 cuffed	2	10	18-30	14	12
6 years	5.5 cuffed	2	10	18-30	14	12
7 years	6.0 cuffed	2	12	18-30	14	12
8 years	6.0 cuffed	2	12	20-34	16	14
9 years	6.5 cuffed	3	12	20-34	16	14
10 years	6.5 cuffed	3	12	20-34	16	14
11 to 18 years	7.0-8.0 cuffed	3	14	20-38	16-18	14

Tips to Remember:

Infants ≤ 1 year: Uncuffed ET tube size = 3.5; Children from 1 to 2 years = 4.0. Children ≥ 2 years: Uncuffed ET tube size = 4 + (age/4); Cuffed ET tube size = 3.5 + (age/4). Use cuffed tubes in children > 1 year.

When using cuffed tubes, inflate **slowly** until air leak is lost.

Always have one size smaller and one larger available to fit correct size.

ET tube length (in cm) at the teeth = ET tube size x 3.

Pediatric chest tube size = ET tube size x 4.

Pediatric NG tube or Foley size = ET tube size x 2.

Reviewed January 2019

ACUTE CARE 38: MODIFIED LUND BROWDER CHART¹

Burned Area	Age, years					
	1	1 to 4	5 to 9	10 to 14	15	Adult
Head	19%	17%	13%	11%	9%	7%
Neck	2	2	2	2	2	2
Anterior trunk	13	13	13	13	13	13
Posterior trunk	13	13	13	13	13	13
Right buttock	2.5	2.5	2.5	2.5	2.5	2.5
Left buttock	2.5	2.5	2.5	2.5	2.5	2.5
Genitalia	1	1	1	1	1	1
R.U. arm	4	4	4	4	4	4
L.U. arm	4	4	4	4	4	4
R.L. arm	3	3	3	3	3	3
L.L arm	3	3	3	3	3	3
Right hand	2.5	2.5	2.5	2.5	2.5	2.5
Left hand	2.5	2.5	2.5	2.5	2.5	2.5
Right thigh	5.5	6.5	8	8.5	9	9.5
Left thigh	5.5	6.5	8	8.5	9	9.5
Right leg	5	5	5.5	6	6.5	7
Left leg	5	5	5.5	6	6.5	7
Right foot	3.5	3.5	3.5	3.5	3.5	3.5
Left foot	3.5	3.5	3.5	3.5	3.5	3.5

1. *Trauma Nursing Core Course Provider Manual*. 6th ed. Chicago, IL: Emergency Nurses Association; 2007.

ACUTE CARE 39: BURN MANAGEMENT TREATMENT PLAN

Initial Treatment of Burns

Remove all clothing, jewelry, and contact lenses.

Stop the burning process.

Immediately cool burn with water or saline if < 10% of BSA.

Cleanse wounds with saline.

Dress wounds with loose gauze dressing.

Elevate extremities.

Cover with a clean dry sheet if > 10% BSA.

Keep the patient warm.

Medications

Pain control – narcotic

Tetanus prophylaxis, 0.5 mL IM

Antibiotic IV

Consider peptic ulcer prophylaxis with an H2 blocker or proton pump inhibitor IV

Burn Transfers

If comprehensive burn treatment is not available at your facility, consider the following transfer guidelines:

Partial-thickness and full-thickness burns are > 10% of TBSA in patients (PEDS) ≤ 10 years and > 50 years of age.

Full-thickness burns over 5% TBSA in any age group

Partial-thickness and full-thickness burns > 20% TBSA in other age groups

Patients with burns and multiple injuries

Medical histories that might be complicated by a burn

Significant electrical injury including lightning

Partial-thickness and full-thickness burns of hands, feet, face, eyes, ears, or perineum

Carbon monoxide > 10%

Suspicion of abuse (child or adult) requiring special social service or long-term rehabilitation support

Evidence of pulmonary or respiratory distress

Transfer of any patient must be coordinated with the burn center physician.

Document all pertinent information regarding tests, temperature, pulse, fluids administered, urinary output, and treatments. Send these with the patient.

ACUTE CARE 40: TRANEXAMIC ACID

Clinical Indications: Anti-fibrinolytic hemostatic agent for trauma patients
When hemorrhagic shock is suspected, give early; outcomes are improved the earlier a patient receives TXA.

Dosing

- **Adult Dosing (IV)** Loading dose: 1 g over 10 minutes via infusion pump;
Infusion: 1 g over 8 hours via infusion pump
PEDS: Pediatric Dosing: 15mg/kg to max of 1 g over 10 minutes via infusion pump. Infusion: same dose over 8 hours via infusion pump

IV Preparation and Administration

- Adult doses (1000 mg/10 mL vials) --Must be initiated within 3 hours of trauma;
Loading dose: Add 1 g (10 mL) to a 50 mL bag of NS
-Flush line if infusion not available
-Maximum infusion rate: 100 mg/min. **CAUTION:** Too rapid infusion may cause hypotension
- **Infusion:** 1 g (10 mL)/250 mL bag of NS. Infused over 8 hours (32 mL/hour) via infusion pump.

Contraindications:

- Subarachnoid hemorrhage (Stop infusion if diagnosed.)
- Acute or current treatment of thrombosis
- Seizure in the course of current illness (Stop infusion if seizure occurs.)
- Factor concentrate administration
- Hypersensitivity to tranexamic acid

Caution:

- If serum creatine > 3 mg/dL, give only loading dose. **Do not give infusion.**
- Rate-related hypotension may occur.

Side Effects: Nausea, vomiting, diarrhea, giddiness, thromboembolic events

Nursing Considerations

- Maximum infusion rate: 100 mg/minute—too rapid of an infusion may cause hypotension
- If rate related hypotension (new SBP<80) develops, extend infusion by 20 minutes
- Stop infusion if seizures develop
- Stop infusion if subarachnoid hemorrhage diagnosed after the start of the administration

Compatible: Administer via dedicated IV line

Incompatible: Blood products, penicillin derivatives

ACUTE CARE 41: INITIAL CARE OF MAJOR TRAUMA PATIENTS

Prior to Arrival

- Consider transfer potential, destination, and type of transport. Call for transport. Transport decisions and calls may be made while patient is in the field.

Upon Arrival

- Combative patients require restraint; this can be accomplished mechanically or chemically while conducting initial assessment and treatment of life threats.

Initial Survey

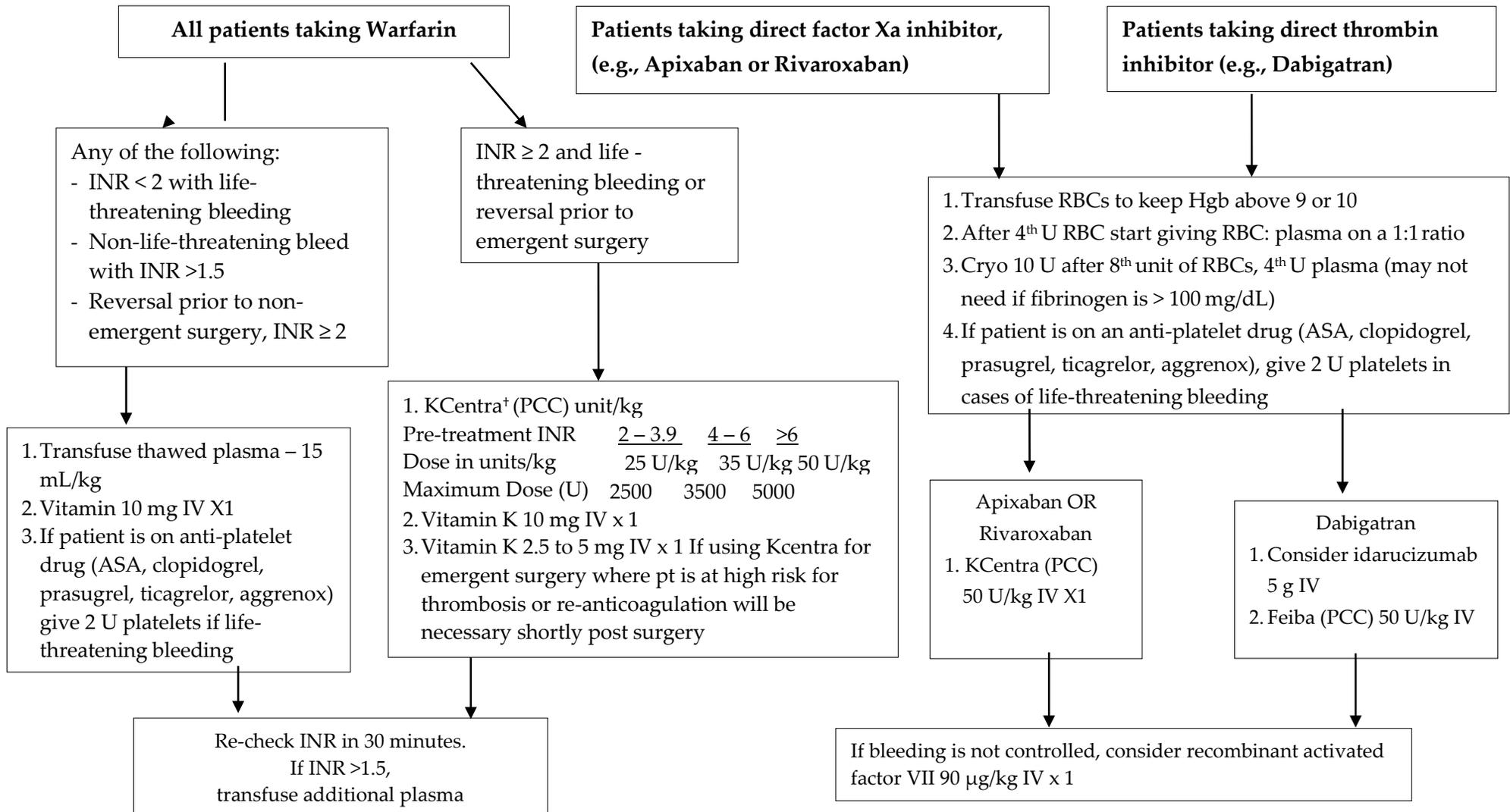
- Observe “10 seconds of silence” for the team leader to identify life threats needing immediate interventions (obstructed airway, tension pneumothorax, uncontrolled external hemorrhage, sucking chest wound, cardiac arrest). These conditions must be corrected before proceeding with routine measures.
- Team actions include exposure, IV access, monitors, blood draws (in order of importance, type and crossmatch, hemoglobin/CBC, chemistry panel, PTT/INR, urinalysis, pregnancy test, alcohol/tox screen).
- Consider advanced airway management techniques as needed. Indications for intubation include airway and breathing compromise, combative or uncooperative patients requiring significant sedation, and most critical patients requiring transport. If possible, perform mini-neuro exam prior to intubation.
- Initiate fluid resuscitation as needed. Consider blood administration. If giving blood consider TXA.

Focused Evaluation and Ongoing Care

- Repeat initial survey during focused trauma evaluation. Remember to examine the back and rectum of a trauma victim.
- Advanced airway management and ongoing fluid resuscitation may be needed.
- In pediatric patients, 20 mL/kg IV boluses of crystalloid (NS or LR) may be initiated for hypovolemic shock. Consider blood and TXA.
- Further tests, imaging, and interventions are based on results of the focused trauma evaluation. A chest x-ray is almost always indicated. In all cases of blunt trauma, obtain a pelvic x-ray.
- Treat and re-evaluate life threats. If the patient does not respond appropriately to the treatments provided or shows deterioration, return to the initial survey. If patient is to be transferred emergently, do not attempt to investigate and treat each and every injury.

ACUTE CARE 42: REVERSE ANTICOAGULATION THERAPY*

Warfarin and Novel Oral Anticoagulant (NOAC) Reversal for Life-Threatening Bleeding



† Kcentra contains heparin and cannot be used in patients with heparin allergy/HIT.

*Adapted with permission from Regions Hospital, St. Paul, Minnesota, 2017. August 2017

SECTION 2 **UNIVERSAL APPROACH**

UNIVERSAL APPROACH – CALS UNIVERSAL APPROACH

63

CALS UNIVERSAL APPROACH TO EMERGENCY ADVANCED LIFE SUPPORT

The CALS universal approach emphasizes team action. A team of emergency care providers can be much more efficient than a single provider. The team leader directly attends to the patient and sets treatment priorities. Team members anticipate needs and are skilled in performing their roles. They respect the team leader's overall control. The team leader appreciates and respects the preparedness of the team and considers their suggestions when something seems amiss.

The First 30 Minutes of Stabilization

Step 1: Activate the Team

As soon as it is known that a critically ill or injured patient is coming to the emergency facility, summon all needed personnel. Critical care transport may be activated. Size and composition of the team will vary. But even if a hospital's staff is made up of two people, those two individuals can form a team. For example, a nurse working an all-night shift may activate the team by calling others from areas within the hospital or calling in a physician or other health care personnel (e.g., lab or x-ray staff).

Step 2: Obtain Immediate Control and Immobilization

Position the patient so that he or she can be cared for safely. In blunt trauma, spine immobilization is needed. Agitated patients must be restrained. The patient is placed on a work surface stationed in a suitable space to give the team members access. After this is accomplished, the team leader is given "10 seconds of silence" by the team to mentally take in the scene while making direct contact with the patient.

Step 3: Perform the Initial (Primary) Survey for All Patients – Identify and Treat Life Threats and Obtain the SAMPLE History

While the team works, the team leader performs a rapid search for immediate life threats and corrects them if they are found. For example, if exsanguinating external bleeding is found, the team leader directs a team member to apply pressure to the site. The leader looks for airway, breathing, and circulatory problems (the ABCs). Life-threatening neurologic problems are addressed. As this step is finished, the leader obtains a SAMPLE history.

**CALS UNIVERSAL APPROACH
TO EMERGENCY ADVANCED LIFE SUPPORT**

Whenever possible, obtain a SAMPLE history before sedating the patient

S	Signs and symptoms	P	Past history, pregnancy
A	Allergies	L	Last meal
M	Medications	E	Events, environment

Step 3: The Team Acts Simultaneously with the Initial (Primary) Survey

Immediately after the 10 seconds of silence, the team members work rapidly to obtain exposure, measure vital signs, obtain venous access, and draw blood. They apply monitor leads. They begin fluid resuscitation as directed by the leader.

Step 4: Form a Preliminary Clinical Impression

The team leader develops a preliminary impression of the patient's condition by using the historical and clinical data obtained thus far. This impression causes the leader to select a pathway of action that will address the patient's problem. It may be as simple as recognizing the need for neonatal resuscitation or as complex as recognizing that the patient is obtunded with no clear cause. The team leader must remain flexible and remain willing to start over again. Perform a focused evaluation (secondary survey) in order to assist in formulating a working diagnosis.

Step 5: Form a Working Diagnosis and Arrange a Disposition

By this time, the team leader has enough information from clinical observations and initial laboratory results to form a working diagnosis. The leader continues to keep an open mind. Once the working diagnosis is clear, the portals in Volume III assist when additional information or treatment options are needed. Patient disposition is arranged. The team makes sure that transfer information is complete and checks to be sure that the patient's medical safety has been provided for as much as possible.

Step 6: Team Process Review and Debriefing

It is important for the team (including the team leader) to review the emergency experience, including what went right, what went wrong, whether needed supplies were easily available, and whether communications were optimal. After a difficult case, team members may need emotional support.

SECTION 3 STEPS 1-6

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STEP 1: ACTIVATE THE TEAM

As soon as it is known that a critically ill or injured patient is coming to the emergency facility, summon all of the needed personnel. Critical care transport may also be activated.

In most rural settings, a physician/ advanced provider is available 24 hours a day. If not present at the hospital, the physician/ advanced provider is usually available on call. In a remote (or wilderness) setting, however, the most experienced health care professional available must function as team leader. Under such circumstances, a physician assistant or nurse practitioner with emergency experience and training may fulfill this role, or a nurse till a provider arrives. Team membership may include prehospital personnel, laboratory and x-ray technologists, respiratory therapists, clerical personnel, physicians, and other allied health care givers (such as physician assistants, nurse practitioners, nurses, and nurse anesthetists).

Early notification of all team members is essential. Provide prehospital personnel with protocols regarding the early notification of ground or air inter-facility transport providers. In most rural areas, delivery of the patient to the rural ED for immediate stabilization maybe the most practical approach (as opposed to scene response), even when transfer to another facility will be necessary. Local geography and weather may dictate which approach to use. In a wilderness setting, an intercept point between the local prehospital personnel and an inter-facility critical care transport team may be arranged to good advantage.

Inter-facility transport by air or critical care transport should not delay the immediate resuscitation of a critically ill or injured patient. The rural ED team must initiate and continue the resuscitation until the patient is in as safe a condition as practical for safe transport.

For example the Minnesota State Trauma System has established model protocols for team activation at trauma stabilization facilities for the Minnesota Department of Health.¹ These are listed here with additional suggestions for non-trauma emergencies:

Indications for Team Activation

- A. Either after EMS notification or upon patient arrival, mobilize the hospital emergency care team for these conditions:
 - 1. Altered LOC secondary to trauma (GCS \leq 10 or $<$ "V" on the AVPU scale).
 - 2. Airway compromise that cannot be resolved by foreign body removal, airway adjuncts, or ventilatory support

<http://www.health.state.mn.us/traumasystem/hospresources/resourcemannual/> Accessed December 2018

ACTIVATE THE TEAM

STEP 1

3. Respiratory distress or respiratory rate outside of acceptable range.
Adult < 10 or > 30

Pediatric

Age	RR
1 to 12 months	<20 or >60
12 to 24 months	<10 or >50
2 to 5 years	<10 or >40
≥ 6 years	<10 or > 30

4. Evidence of shock or diminished perfusion
- a. Transient hypotensive episode
 - b. Vital signs outside of acceptable range
 - c. Adult SBP ≤90
 - d. **PEDS:** Child capillary refill > 2 seconds or age-specific hypotension

Age-Specific Hypotension (PEDS)

Age	SBP (mm Hg)
≤1 year	≤60
2-10 years	<70 + (2 x age in years)

- d. Persistent heart rate > 120 in adult or age-specific tachycardia in a child

Age-Specific Tachycardia (PEDS)

Age	HR
< 2 years	>180
2-5 years	>160

- e. Shock index ≥ 1 (HR÷BP)
 - f. SaO₂ ≤ 70% in adult
 - g. Provider impression of hypoperfusion
5. Uncontrolled hemorrhage; arterial tourniquet/wound packing indicated
6. Suspected cardiac or major vessel injury
7. Penetrating injury of the head, neck, chest, abdomen, or pelvis
8. Suspected severe orthopedic injuries:
- a. Pelvic fracture
 - b. Unstable facial fracture
 - c. More than one proximal long bone fracture
 - d. Femur fracture
 - e. Open long bone fracture
 - f. Knee dislocation
9. Amputation proximal to the wrist or ankle

10. Second- or third-degree burns of 20% or more of the total body surface area (TBSA) or in combination with other injuries
 - a. Facial burns
 - b. Suspected inhalation burn
 - c. Burns with concomitant trauma
11. Pregnancy >20 weeks with vaginal bleeding or contractions
12. Aeromedical launched by EMS
13. Traumatic paralysis or focal neurological signs/symptoms (ie, numbness, tingling)

In addition, scene personnel should strongly consider notification of inter-facility transfer vehicles (ground ALS, airplane, or helicopter).

- B. Be suspicious that critical injury may have occurred and that the emergency care team may need to be activated for the following:
 1. Motor vehicle crashes (MVC) where one of the following has occurred:
 - a. Ejection from the vehicle
 - b. Death in the same passenger compartment
 - c. Extrication time > 20 minutes (time spent accomplishing extrication)
 - d. Major deformity of the vehicle > 20 inches
 - e. Intrusion > 12 inches into the passenger compartment
 - f. Motorcycle, snowmobile, or ATV crash with separation of rider
 - g. Pedestrian/bicyclist struck (> 5 mph), thrown, run over; separation of rider
 2. Falls
 - a. > 15 feet
 - b. > 65 years old and from elevation or down stairs
 - c. (PEDS) < 10 years old: > 2 x patient's height
- C. Provider discretion for the following:
 - a. Severe single system injury
 - b. Multiple injuries (≥ 2 systems)
 - c. Co-morbid factors:
 - Anti-coagulant therapy
 - Age < 5 or > 55 years old
 - Multiple co-morbidities
- D. Strongly consider activating the emergency care team by prehospital or hospital personnel if one of the following non-trauma situations exists:
 1. Newborn infant:
 - a. With any signs of weak breathing or crying, limp or weak extremities, or cyanosis that does not improve with oxygen or 30 seconds of bagging.

ACTIVATE THE TEAM

STEP 1

- b. With a fetal heart rate (FHR) < 80 and any baby requiring chest compressions
 - c. Rhythmic activity that could be seizure activity
2. Obstetrical patients who exhibit:
 - a. Significant second or third trimester vaginal bleeding
 - b. FHR < 120 beats per minute (bpm)
 - c. Tetanic uterine contractions
 - d. Seizures
 - e. Hypotension or hypertension
 - f. Prolapsed umbilical cord or footling presentation
 - g. Shoulder dystocia manifested by the turtle sign
3. Pediatric patients who exhibit:
 - a. Unconscious or stuporous state
 - b. Marked respiratory distress or apnea from any cause including status asthmaticus, croup, foreign body aspiration, or unknown causes
 - c. Hypotension from any cause
 - d. Status epilepticus
 - e. Anaphylactic reaction and/or angioneurotic edema
4. Adult patients who exhibit:
 - a. Depressed LOC (unless cause is apparent and easily treated)
 - b. Marked respiratory distress or apnea, any cause, including tracheal FB, status asthmaticus, pulmonary edema, respiratory failure, or pulmonary embolus
 - c. Chest pain with any indication of probable myocardial infarction
 - d. Anaphylactic reaction and/or angioneurotic edema
 - e. Cardiac arrest
 - f. Unstable cardiac arrhythmias
 - g. Shock state or severe hypertension from any cause
 - h. Pulmonary edema
 - i. Severe hypothermia or hyperthermia
 - j. Evidence for cardiac tamponade
 - k. Evidence for tension pneumothorax
 - l. Evidence for leaking abdominal aortic aneurysm
 - m. Evidence for dissection of the thoracic aorta
 - n. Gastrointestinal bleed with unstable vital signs
 - o. Drowning patient

STEP 2: IMMEDIATE CONTROL AND IMMOBILIZATION

Position the patient so that he or she can be cared for safely. In blunt trauma, spine immobilization is needed. Agitated patients must be restrained and or medicated. The patient is placed on a work surface stationed in a suitable space to give the team members access. After this is accomplished, the team leader is given 10 seconds of "silence" by the team to mentally take in the scene while making direct contact with the patient.

Please Note: Comments, directions, and instructions specific to pediatric patients are underlined.

Prehospital personnel have often already immobilized patients who are being admitted to the ED. However, out-of-control and even dangerous but critically ill patients may arrive by private car. Emergency situations arising in a hospital setting may be as difficult to control as those that occur away from treatment facilities.

Immobilization and Control

Skeletal immobilization. In trauma situations, addressing spine immobilization may be needed. Long bone and pelvic stabilization may be delayed until the team begins the systematic pathway for trauma.

A. Cervical spine immobilization

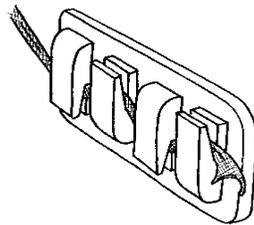
1. **Removing a helmet** is a two-person job. One person stabilizes the patient's head and neck in neutral position by placing one hand under the patient's occiput and, with the other hand, cupping the patient's chin. The second person releases the helmet chinstrap and spreads the sides of the helmet apart while slipping it over the patient's head. (A cast cutter may be needed to remove a facemask.)
2. **Apply a cervical collar** whenever a neck injury is possible. There are now rigid and "beanbag type" collars available to provide stabilization. They are available in a variety of sizes (from infant to adult), including no neck and long neck models. Apply without moving the neck. Magill forceps may be used to pull the collar behind the neck with no neck movement.
3. Complete immobilization of the cervical spine requires neck blocks, forehead straps, and sometimes a spine board. Be aware of the need to

pad the board. (The term backboards is being replaced with the term extrication board, as it is not necessary to keep patients on the boards for a prolonged time). **PEDS:** In small children, the backboard may result in neck flexion because of the relatively large posterior skulls of children. Place a pad or folded cloth under the shoulders to keep the neck in a neutral position.

B. Thoracic and lumbar spine immobilization

Logroll the patient to place the long board under the patient's back using only the degree of rolling necessary. The person stabilizing the head and neck directs the movement.

Physical restraints are sometimes needed in emergency situations. The urge to pull out ET or gastric tubes is great. When in doubt, apply restraints. They may be attached to the cart rails with brackets at the patient's calf level.



A. Use quick release restraint systems. Wrist restraints must not be tied to the cart with knots. If the patient vomits, he or she must be turned quickly. Boat-cleat type restraint holders (see illustration above) are strong and can be quickly released.

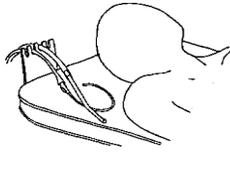
B. Hook and loop fastener straps attached to the resuscitation cart at the level of the ankles, knees, mid thigh, and abdomen are useful.

The out-of-control patient and chemical restraints: A head-injured or intoxicated patient may be so confused and so strong physically that immediate sedation is needed to gain control of the patient, to protect the team, and to protect the patient from excessive intracranial pressure (ICP) rise secondary to struggling. In such cases, it may not be possible to start a reliable IV. Administer Ketamine IM 4 mg/kg to allow placement of an IV for subsequent management. Ketamine takes effect in 1 to 2 minutes.

CONTROL AND IMMOBILIZATION STEP 2

Work Surfaces/Carts

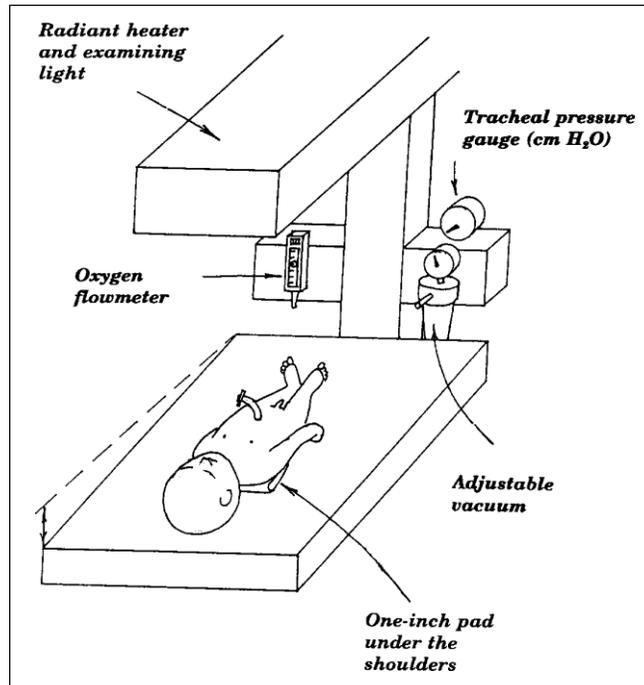
The **resuscitation cart** is highly important. Station the cart away from walls. If cables to and from a wall or monitor are unavoidable, arrange them so that they are in one area only. Access to the patient from the head of the cart is crucial. Large bore suction is a critical piece of equipment in airway management, yet it is frequently found on the floor when it is needed most. This can be avoided by placing a bracket at the head of the cart to hold the suction tubing in place.



Suction bracket holds suction tubing and other tubing

The brakes on the resuscitation cart must be reliable and should always be set prior to a patient's arrival. The resuscitation cart should have an adjustable backrest. A removable footboard keeps the patient from sliding off of the foot of the cart when the backrest is elevated. The cart should be easily adjustable to typical operating table height to facilitate patient care. A removable arm board is useful to facilitate chest tube and IV placement.

PEDS: Infants and neonates require a special resuscitation table. Equip the table with the following: overhead warming lights with 1 or 2 procedure lights, oxygen tank and oxygen flowmeter, and adjustable oxygen pressure-powered vacuum bottle mounted on the table for tracheal suction. Also mounted on the cart should be a low-pressure meter that may be connected to the infant BVM so that transtracheal pressure can be monitored to avoid producing a pneumothorax by use of excessive pressure.



Pediatric resuscitation cart

An airway cart containing all of the emergency airway equipment needed for both adult and pediatric patients must be situated within arm's reach. [See Vol II – AIR SKILLS 1, AIDS TO INTUBATION.](#)

10 Seconds of Silence

In most cases, it is wise for the team to leave hands off the patient and not talk to the patient during the next 10 seconds so that the team leader can soak in the scene and establish contact with the patient. During these few seconds, there is plenty for the team to do: Get the monitor leads ready and check lighting (warming lights, etc). In some cases, this brief delay is not possible. The patient may be flailing about, or there may be exsanguinating hemorrhage. The team leader identifies him- or herself and greets the patient.

STEP 3: INITIAL SURVEY

While the team works, the team leader performs a rapid search for immediate life threats and corrects them if found. For example, if exsanguinating, external bleeding is found, the team leader directs a team member to apply pressure to the site or apply a tourniquet or do wound packing. The leader looks for airway, breathing, and circulatory problems (the ABCs). Life-threatening neurologic problems are addressed. The leader obtains a SAMPLE history as this step is finished.

Perform the initial survey for all patients. Identify and treat life threats and obtain the SAMPLE history.

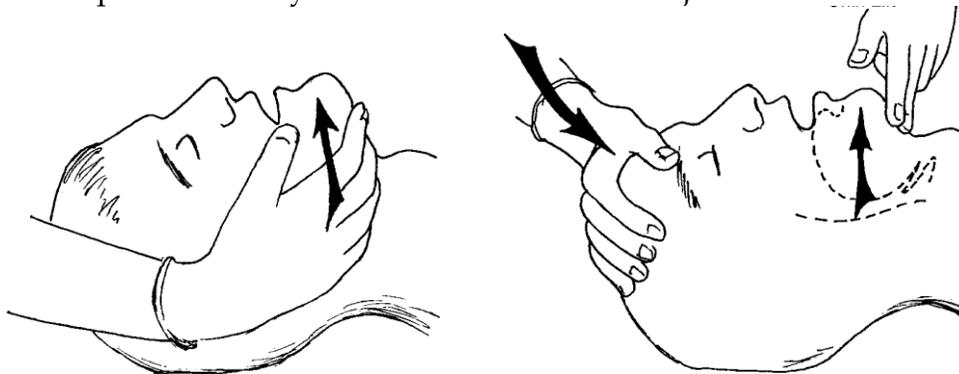
The pursuit of a correct diagnosis may interfere with the immediate needs of a patient during the critical, initial period of care. **Identify those problems that can be treated immediately.** The ABCDE mnemonic is the easiest way to commit the steps to memory. Another mnemonic is MARCH.

A stands for airway.

Patients who are awake and breathing are able to protect their airways. Look for retractions, increased work of breathing, abnormal rate of breathing, and pallor. **PEDS:** In small children signs of respiratory distress may be subtle. Nasal flaring or grunting may be the only indication.

If the patient is breathing but unconscious or semi-conscious:

1. Open the airway with either the chin lift or jaw thrust maneuver.

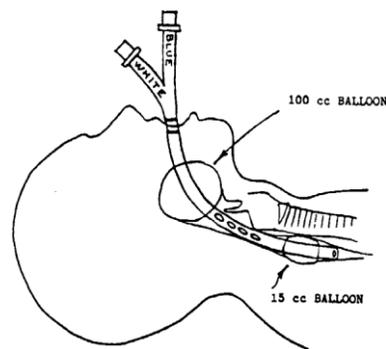


Trauma: Jaw thrust maneuver Non-trauma: Chin lift maneuver

PEDS: In infants, place a folded towel (about 1 inch thick) under the shoulders.

2. **PEDS:** In older children and adults, insert an oral airway unless it makes the patient gag. If so, insert a nasal trumpet.

3. Apply oxygen per facemask at 10 to 15 L/min.
4. **PEDS:** In infants, begin ventilation with a bag-valve-mask, taking care not to use too much pressure. In older children and adults, if a gag reflex is not present an orotracheal intubation maybe achieved with in-line immobilization and an endotracheal tube introducer. (Vol II – AIR SKILLS 2 BAG-VALVE-MASK, AIR SKILLS 3 OROTRACHEAL INTUBATION) If the patient can be ventilated with a bag-valve-mask, endotracheal intubation may be delayed. Or an oral or nasal airway can be used, or a supraglottic airway can be inserted. Such as an iGEL, King or some other supraglottic airway (Vol II – AIR SKILLS 6 KING AIRWAY)



5. Confirm correct ET tube placement with an esophageal intubation detector or colorimetric or continuous CO₂ detector. (Vol II – AIR SKILLS 1 AIDS TO INTUBATION) Follow this with listening for breath sounds.

If the patient is apneic or struggling to breathe

1. Open the airway with either the chin lift or jaw thrust maneuvers. **PEDS:** In infants, place a folded towel (about 1 inch thick) under the shoulders.
2. Attempt to ventilate with a bag-valve-mask with 100% oxygen; mouth-to-pocket facemask ventilation may also be used. Insert an oral airway if tolerated; otherwise, a nasal airway may be inserted.
3. If you observe good chest rise, oxygenate, ventilate, and complete the initial survey.
4. If ventilation is unsuccessful, foreign body presence is likely.

If ventilation is unsuccessful there is a possibility of a foreign body

1. Give 5 chest compressions, and then perform a finger sweep of the pharynx. In pregnant women and obese patients, use chest thrusts instead. **PEDS:** In infants and children ≤ 1 year, deliver 5 back blows and 5 chest thrusts. Do not use a finger sweep in children.
2. Remove the foreign body if it is visualized.
3. Re-attempt ventilation.

INITIAL SURVEY

STEP 3

4. If still unsuccessful, use a laryngoscope to visualize the glottis. Use a Magill forcep to grasp and remove the foreign body if it is visible.
5. If the foreign body cannot be seen, presume that a tracheal foreign body is present beyond the vocal cords. If the obstruction is incomplete, continue to attempt ventilation until you can arrange rigid or flexible bronchoscopic removal.
6. An 80% helium and 20% oxygen gas mixture (heliox) may be used, which will allow gas flow around the foreign body because of its decreased density and laminar flow properties. If the O₂ saturation is low, supplemental O₂ may be added with the use of a nasal cannula.

If the obstruction is life threatening or for complete tracheal obstruction, see [Vol II – AIR SKILLS 12 TRACHEAL FOREIGN BODY REMOVAL](#).

Traumatic neck hematoma or tracheal disruption

1. Try orotracheal intubation and attempt ventilation. ([Vol II – AIR SKILLS 3 OROTRACHEAL INTUBATION](#)) Be careful not to use too much force because a severed trachea can be pushed under the sternum.
2. If unsuccessful, perform tracheotomy. ([Vol II – AIR SKILLS 14 TRACHEOTOMY](#))
3. Oxygenate, hyperventilate, and complete the initial survey if you observe good chest rise.

Epiglottitis or angioneurotic edema

1. A bag-valve-mask may force some oxygen past the edema.
2. An 80% helium and 20% oxygen gas mixture (heliox) may help.
3. Try orotracheal intubation using an endotracheal tube introducer as a guide. ([Vol II – AIR SKILLS 1 AIDS TO INTUBATION](#))
4. If unsuccessful, use transtracheal needle ventilation. ([Vol II – AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION](#))
5. While ventilating, re-attempt orotracheal intubation.
6. Cricothyrotomy ([Vol II – AIR SKILLS 13 CRICOTHYROTOMY](#)) or **PEDS:** tracheotomy in children < 8 years, ([Vol II – AIR SKILLS 15 TRACHEOTOMY IN INFANTS](#)) may be necessary.
7. Oxygenate, hyperventilate, and complete the initial survey if you observe good chest rise.

If the patient is already intubated

1. Double check for correct placement by listening for breath sounds and by using an esophageal intubation detector or a colorimetric or continuous CO₂ detector. **PEDS:** In infants, use a CO₂ detector to confirm correct placement. ([Vol II – AIR SKILLS 1 AIDS TO INTUBATION](#))
2. Ultimately a chest x-ray is needed to confirm ETT depth.

3. If a supraglottic airway is already inserted and functioning well, there is no need to replace it. Continue the resuscitation.

Whenever possible, obtain a SAMPLE history before sedating the patient

S	Signs and symptoms	P	Past history, pregnancy
A	Allergies	L	Last meal
M	Medications	E	Events, environment

There are indications for Rapid Sequence Intubation (RSI) present:

1. Depressed level of consciousness (LOC) with probable increased intracranial pressure (ICP) as in head injury, cerebral edema, intracerebral hemorrhage, subarachnoid hemorrhage, hypertensive encephalopathy, hydrocephalus, and mass formation from any cause.
2. Agitated patient needing critical medical attention as in large tricyclic overdose or a head-injured patient with other possible severe injuries.
3. Muscle rigidity with jaw clenching in a patient who needs intubation.

There are relative contraindications for RSI present:

1. The patient's anatomy or the presence of a mass may result in a paralyzed patient you cannot intubate.
2. The patient has a large beard, another anatomic problem, or facial trauma that may result in a paralyzed patient that you cannot ventilate with a bag-valve-mask.
3. The patient may have elevated serum potassium, making the use of succinylcholine risky. Succinylcholine raises serum potassium. Chronic or acute renal failure, a crush injury or burn a few days previous, muscular dystrophy, muscle wasting, and extreme muscular exertion as in cocaine or amphetamine overdose are examples. In which case Rocuronium would be a good alternative.
4. A relative contraindication is the possibility of a globe injury of the eye. In such a case, succinylcholine can be replaced with Rocuronium.
5. If the patient is in cardiac arrest without reflexes or muscle tone, RSI offers no advantage and is not needed. Proceed with intubation.

RSI entails the use of sedation and paralysis to ease orotracheal intubation while preventing adverse responses to tracheal intubation. [See Vol II – AIR SKILLS 12 TRACHEAL FOREIGN BODY REMOVAL](#) for more discussion and optional rescue airways.

The RSI procedure should not be feared. RSI greatly facilitates orotracheal intubation because of the good relaxation it affords. RSI eliminates laryngospasm and gag reflexes. If the intubator is prepared to go to another airway if intubation fails, the risk is greatly reduced. A supraglottic airway can often be easily inserted.

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Remember the 9 Ps for RSI:

1. **Prepare:** Equipment, meds, team, patient (basic airway management, positioning). Discuss plan including back up plan
2. **Preoxygenate:** 100% O₂, 3 to 5 minutes; add nasal cannula @ 4 L/min
3. **Premedicate** **Treat Hypotension and Hypoxia**

RSI TIME OUT

4. **Push the sedative:** **Use ONE:**
Ketamine 1 to 2 mg/kg IV (bronchodilator/ useful in hypotension)
or Etomidate 0.3 mg/kg IV
Also increase O₂ by nasal cannula to 15 L/min (adult), (PEDS) 10 L/min (children), 5 L/min (infants).
5. **Paralyze:** **Use one:**
Rocuronium 1 mg/kg IV (PEDS: preferable).
or Succinylcholine 2 mg/kg IV (Avoid in hyperkalemia, neuromuscular disease, or ocular trauma)
Wait for relaxation (45-60 sec). Do not bag unless hypoxic.
6. **Position airway:** Head/neck position; BURP, laryngeal manipulation (if needed)
7. **Pass & assess tube:** Maintain in-line cervical immobilization in head/neck trauma
Inflate cuff (Careful not to overinflate) Use esophageal intubation detector; check breath sounds; continuous or colorimetric end-tidal CO₂. **Record depth of tube. Secure the tube. CXR for depth confirmation.**
8. **Pass next tube:** Pass OG or NG. Empty stomach & then intermittent suction
9. **Post-intubation plan:** Drugs/ dosages depend on medications used during intubation.
Titrate all meds and watch for hypotension
and remember sedation/analgesia have
shorter half-lives than paralytics
Sedation: Midazolam 0.1 mg/kg IV.
Paralysis: (if needed) Vecuronium 0.1 mg/kg IV
(if not used for intubation) or Repeat
Rocuronium 1mg/kg IV (if needed)
Analgesia: Fentanyl 1 to 2 MICROgrams/kg IV
or Morphine 0.05 to 0.15 mg/kg IV

If capabilities exist can consider

sedation/analgesia drips for transport
RSI medications may also be given IO. Consider
need for seizure prevention. **Elevate HOB/
reverse trendelenburg if able**

If there is severe facial trauma or traumatic facial swelling with airway compromise

1. Orotracheal intubation may be successful in some circumstances. Try at least once. Otherwise use a supraglottic airway. (**Vol II – AIR SKILLS 3 OROTRACHEAL INTUBATION, AIR SKILLS 6 KING AIRWAY**)
2. In many cases cricothyrotomy (**Vol II – AIR SKILLS 13 CRICOTHYROTOMY**) or (**PEDS**) transtracheal needle ventilation in children < 8 years (**Vol II – AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION**) will be necessary.
3. Oxygenate, hyperventilate, and complete the initial survey if you observe good chest rise.

B stands for breathing.

Look for symmetrical chest rise. Feel for tracheal deviation. Feel for chest wall crepitation that would indicate broken ribs or pneumothorax. Feel for tenderness over the ribs and sternum. Look for chest wounds. If a penetrating wound is found, instruct team members to place an occlusive dressing on it. Do not explore chest wounds or remove impaled objects. Listen over the stomach first to detect esophageal intubation quickly. Listen for breath sounds in the mid-axillary space. Listen for heart tones.

PEDS: Breath sounds in infants are easily transmitted from one side to the other.

If there is a tension pneumothorax indicated by hypotension, tachycardia decreased oxygen saturation, unequal breath sounds, distended neck veins, tracheal deviation on x-ray:

1. Perform needle thoracostomy over the top of the third rib in the midclavicular line. **Depending on the patient's body habitus you may need to instead go to the 4-5 intercostal space on the mid-axillary line**(**Vol II – BREATH SKILLS 5 NEEDLE THORACOSTOMY**)



INITIAL SURVEY

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2. Inflate the lung and keep it inflated by performing positive pressure ventilation. Attach a Heimlich flutter valve to the needle, which will allow egress of air out of the chest while preventing in-flow. This will allow the lung to expand with each breath.
3. Oxygenate, hyperventilate, and complete the initial survey if you observe good chest rise.
4. If the needle is ineffective, immediately insert a large chest tube (28 to 32 F in adults and **PEDS: 8 to 28 F in children**). (**Vol II–BREATH SKILLS 1 CHEST TUBE INSERTION**) Whenever inserting a chest tube in trauma, prepare for the possible collection of blood from the thorax. (**Vol II–BREATH SKILLS 2 CHEST SUCTION AND AUTOTRANSFUSION**)

Sometimes the physical findings of tension pneumothorax are subtle, especially if there are other reasons for shock and hypoxia present. When in doubt, perform a needle thoracostomy because it may be a lifesaving action.

If abdominal distension is interfering with ventilation:

Insert a large bore orogastric tube for stomach decompression. Use a size 16 F to 18 F in adults, (**PEDS**) a size 10 F to 16 F in children, or a size 8 F feeding tube in infants.

A crushing chest injury can result in **severe pulmonary contusion**, necessitating the use of high inflation pressure when a bag-valve-mask is used. Under these circumstances, cricoid pressure may help prevent abdominal distension. (There is a growing body of opinion that this now does not help)

In **severe asthma**, high inflation pressures may also be needed; however, the tidal volumes should be small and each breath 8 seconds apart. This is counter-intuitive, so count the rhythm out loud. Initiate continuous albuterol nebulization immediately. If improvement is not seen and the patient is in a life-threatening condition, perform RSI so that maximum relaxation and airway control can be achieved. (Realize that unless the lungs are improved the patient will die with an endotracheal tube, down their airway)

Other causes of severe respiratory failure include **chronic obstructive pulmonary disease (COPD)** and **pulmonary fibrosis**. These patients may also respond to albuterol nebulization if there is a bronchospastic component to their disease.

PEDS: In infancy, **diaphragmatic hernia** can present with severe respiratory distress. In such cases, an orogastric tube can be lifesaving.

PEDS: Children are prone to swallow large amounts of air when distressed. Gastric distension is very common in injured children to the point that all significantly injured children should receive an orogastric tube as part of their initial resuscitation.

Fulminant **pulmonary edema** secondary to heart failure can result in a patient in severe respiratory distress who cannot lie down or cooperate with his or her care. Pink, frothy sputum is evident. Such patients will usually refuse positive pressure ventilation or the application of an oxygen mask because they are afraid of suffocation. If this is the case, consider administering a benzodiazepine or ketamine to help facilitate BiPAP/CPAP. If an IV can be established, initiate a nitroglycerine drip at 20 µg/min; rapidly titrate to effect. Consider administering furosemide IV if patient appears fluid overloaded. Closely monitor oxygen saturation and blood pressure.

The fulminant pulmonary edema patient may need intubation to protect their airway. (**Vol II – AIR SKILLS 3 OROTRACHEAL INTUBATION, AIR SKILLS 12 TRACHEAL FOREIGN BODY REMOVAL**) Once the patient is intubated and undergoing positive pressure ventilation, he or she can lie down in safety, greatly facilitating further care.

C stands for circulation.

While listening for heart sounds, feel for a carotid or femoral pulse. The presence of a central pulse means that at least 50% of the patient's blood volume is still in circulation. **PEDS:** In children < 1 year, check for a brachial pulse. Observe neck veins for distension. Observe for external bleeding. If such is seen, instruct a team member to apply pressure to the site or apply a tourniquet or do wound packing. Check the time it takes for capillary refill (under 2 seconds under normal conditions). Estimate the pulse rate. By this time, the team will have obtained vital signs and applied an ECG monitor.

If the pulse is absent

1. Start CPR. Apply defibrillation monitor pads. If ventricular fibrillation or ventricular tachycardia is present, shock as soon as possible and **(PEDS)** in children, 2 joules/kg, for first shock, then 4 joules/kg. Follow local protocols for further direction.
2. Continue CPR for 2 more minutes. Determine a rhythm. If asystole is present, begin CPR and complete the initial survey. If pulseless electrical activity is present, begin CPR, start a fluid bolus, and complete the initial survey.

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If the pulse is too slow

PEDS: In neonates, begin CPR if the pulse rate is < 60 bpm. In adults with heart block and shock, apply an external pacer and pace at 60 bpm; complete the initial survey.

If the pulse is too fast

Often tachycardia accompanies shock of most etiologies. Give priority to treating the underlying problem. However, if there is a tachyarrhythmia (such as a supraventricular tachycardia) that is causing severe decompensation, prepare for synchronized cardioversion while completing the initial survey. Use an initial setting of 100 J in adults and **(PEDS)** older children and 0.5 to 1.0 J/kg in small children.

Consider cardiac tamponade.

Beck's triad, although not always present in tamponade, is worth remembering. It consists of muffled heart sounds, distended neck veins, and hypotension. Penetrating chest wounds, a misplaced transvenous pacemaker lead, and medical causes such as myocarditis or a dissecting aortic aneurysm are not uncommon causes. Cardiac ultrasound is a lifesaving tool in the ED.

If cardiac tamponade is resulting in life-threatening shock, attempt transcutaneous or transthoracic pericardiocentesis now. **(Vol II – CIRC SKILLS 6 PERICARDIOCENTESIS)** Otherwise, continue the initial survey.

If ventricular fibrillation ensues and surgical back-up is available in the community, perform an emergency thoracotomy. **(Vol II – CIRC SKILLS 4 EMERGENCY THORACOTOMY)** A pericardiotomy will enable internal cardiac massage, staple closure of the cardiac wounds, and internal defibrillation in preparation for operating room management.

If the patient is in the third trimester of pregnancy

Place a rolled blanket under the right hip to move the uterus to the left, or manually displace the uterus to the left.

Feel the abdomen and gently compress the pelvis in an AP direction

The abdomen and pelvis are common areas of occult blood loss. A brief palpation of these areas can help direct future efforts. Palpation or ultrasound examination may reveal an abdominal aortic aneurysm.

If there is evidence of hypovolemic shock or septic shock with hypotension and flat neck veins

In adults, administer a bolus of 1 to 2 L crystalloid (NS or LR) solution IV and complete the initial survey.

PEDS: In children, administer a bolus of 20 mL/kg of crystalloid (NS or LR) solution IV and complete the initial survey.

When IV access is difficult, there are several quick and good solutions to the problem:

1. In adults an IO can be inserted into the tibial plateau, or the humeral head. **PEDS:** In children under about 6 years of age, intraosseous needles can be inserted into the proximal or distal tibias and/or distal femurs. (Vol II – CIRC SKILLS 5 INTRAOSSEOUS NEEDLE PLACEMENT)
2. If a 20 gauge cannula is present, it can be converted to a large bore introducer using guidewire technique. (Vol II – CIRC SKILLS 1 ARTRIAL AND VENOUS CATHETER INSERTION)
3. Saphenous vein or other large vein cannulation is greatly facilitated when guidewire technique is used. (Vol II – CIRC SKILLS 1 ARTRIAL AND VENOUS CATHETER INSERTION)

D stands for disability (neurologic deficit).

The AVPU system is a quick and reliable method of estimating LOC:

		Corresponding GCS score
A	Alert - the patient is awake.	14 to 15
V	Responsive to Vocal Stimuli	12 to 13
P	Responsive to Pain only	8
U	Unresponsive	3 to 4

**INITIAL SURVEY
STEP 3**

Glasgow Coma Scale – Adult, Pediatric, Infant

Eye Opening

Infant (<1 year)	Pediatric (>1 year)	Adult	
Spontaneous	Spontaneous	Spontaneous	4
Voice	Voice	Speech	3
Pain	Pain	Pressure	2
None	None	None	1

Verbal Response

Infant (<2 years)	Pediatric (>2 years)	Adult	
Coos, babbles	Appropriate word/ phrase	Oriented	5
Irritable but consolable	Disoriented/ converses	Confused	4
Persistent cries/ screams	Inappropriate word	Words	3
Moans/grunts to pain; restless	Incomprehensible sounds	Sounds	2
None	None	None	1

Motor Response

Infant (<1 year)	Pediatric (>1 year)	Adult	
Spontaneous	Obeys	Obeys	6
Localizes pain	Localizes pain	Localizes pain	5
Flexion- withdrawal	Flexion- withdrawal	Withdraws	4
Flexion/ decorticate	Flexion/ decorticate	Abnormal flexion (decorticate)	3
Extension/ decerebrate	Extension/ decerebrate	Abnormal extension (decerebrate)	2
None	None	None	1
			3 to 15

Note; Changes made in 2014 such that previously when eye opening or verbal response could not be tested (swelling or intubation), the value was scored as a 1.

This has now been changed, so that those components, that cannot be tested are marked "NT" and the total score is not calculated.

Do the **DONT** for patients with a depressed LOC. **Give: Dextrose** (if hypoglycemia is possible), unless the team has used a glucose monitor to indicate that hypoglycemia is not present. **PEDS: In infants, use 10% dextrose.**

Oxygen, and **Naloxone** when opiate intoxication is possible, and **Thiamine** for any patient in whom chronic alcoholism may be present.

If there is a depressed LOC with lateralization

Check for a dilated pupil and limb weakness on the opposite side. If present, uncal herniation may be occurring. Give mannitol 1 g/kg IV or use Hypertonic Saline either 3% or 5%. **PEDS: In children, give mannitol 0.25 to 0.5 g/kg IV.** Orotracheally intubate, hyperventilate with oxygen to EtCO₂ of 30, and complete the initial survey.

If there is seizure activity

Administer IV or IM versed, 8 to 10 mg in adults, and (**PEDS**) in children, 0.2 mg/kg IV up to 10 mg. Repeat this dose if needed in 5 minutes. Administer oxygen by mask. Orotracheally intubate if there is a depressed gag reflex. Prepare for vomiting, aspiration, and cardiac arrhythmias; complete the initial survey.

If the patient is posturing

Check for decorticate (arm flexion) or decerebrate (arm and leg extension) posturing. If present, give mannitol, 1 g/kg IV or Hypertonic Saline 3% or 5%, in adults and (**PEDS**) in children, give mannitol 0.25 to 0.5 g/kg IV. Orotracheally intubate and hyperventilate with oxygen to EtCO₂. Continue on.

If there is evidence of a spinal cord lesion or injury

Look for paralysis, sensory deficit, and priapism. A more thorough mini neurologic exam can be done later. Although, if RSI must be done quickly, it should be preceded by a mini neuro exam.

In brief, a mini neuro exam should consist of

1. **Pupils and Vision**
Conjugate or disconjugate gaze
Size, equality, and reactivity
Finger counting
2. **Tympanic membranes**
Hemotympanum
3. **Neck**

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- Midline tenderness posteriorly
4. **Extremities**
Movement and strength on command or to pain
Ankle, patellar, and brachial reflexes; clonus, Babinski reflexes
Sensation and position sense
 5. **Trunk**
Priaprism, saddle sensation, anal sphincter tone
Sensation level

E stands for exposure.

By now the patient should be completely undressed. Observe quickly for problems that need to be addressed immediately. Look for extremity deformity. If there are ominous skin changes (such as the purpura of meningococemia), the red rash of toxic shock syndrome, and/or the bronzing of gas gangrene, order the preparation of IV antibiotics appropriate for the infection.

SAMPLE history

Before going on, be certain to obtain the **SAMPLE** history before the opportunity is lost.

S	Signs and symptoms	P	Past history, pregnancy
A	Allergies	L	Last meal
M	Medications	E	Events, environment

REFERENCE

1. American Society of Anesthesiologists, Inc. Wolters Kluwer Health, Inc., 2017

January 2019

STEP 3: SIMULTANEOUS TEAM ACTION BY TEAM MEMBERS

Immediately after the 10 seconds of silence, the team members work rapidly to obtain exposure, measure vital signs, obtain venous access, and draw blood. They apply monitor leads. They begin fluid resuscitation as directed by the team leader.

The flow of the resuscitation is such that the team leader cannot direct every move of the team. To do so would delay the initial survey and the subsequent pathway. During this phase, the team members work quickly, proceeding from task to task without waiting for orders from the team leader, but asking for direction whenever a choice must be made.

Exposure and Vital Signs

Expose enough of the patient's body to obtain pulse and BP for determining circulatory status.

PEDS: A Broselow Pediatric Emergency Tape may be used to quickly estimate the weight of a small child. Relay this information to the team leader. The tape, chart, or dosage book may be used to help select equipment sizes and medication dosages.

Completely undress the patient. In trauma patients, use scissors to cut clothing. Do not roll or lift the trauma patient to remove clothing. Be careful not to cut through bullet or knife holes because they are important as evidence. Remember to keep patient warm. Use either warming lights or warm blankets.

Remove jewelry, money, and other personal belongings. Store these in a secure location. Check for bracelets or locket that contain medical information. Check to see if the patient is wearing contact lenses. If so, inform the team leader. A clerical team member should inspect the patient's wallet for phone numbers and identification.

Observe for the presence of blood on the underwear and at the tip of the penis. Report the presence or absence of blood to the team leader. Complete exposure is needed in all emergencies. Towels or sheets may be used to provide privacy during most of the resuscitation. Since exposure often contributes to hypothermia, warmth provided by an infrared lamp mounted on the ceiling above the cart can help to prevent hypothermia in adult patients. **PEDS:** Place all small children under warming lights.

Venous Access

Provide access for medications and fluids by placing 2 large bore IVs in the patient's arms. Ideally, insert two 16 or 14 gauge IVs. However, do not ruin veins by trying to insert needles too large for them. Small IVs may be dilated up to large size by using guidewire technique. **(Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS)**

If veins are not visible or palpable, inform the team leader, you may need to resort to intraosseous needle or the provider may need to place a central venous access, (such as a peripheral IJ insertion) or do a vein cutdown. **(Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS, CIRC SKILLS 5 INTRAOSSEOUS NEEDLE PLACEMENT, CIRC SKILLS 8 SAPHENOUS VEIN CUTDOWN)** **PEDS: In infants and children < 7 years of age, the intraosseous approach may be best to use initially.**

Laboratory Studies

In adults, draw sufficient blood for lab tests. In pediatric use small tubes, especially in infants, due to the lower circulatory blood volume.

The individual hospital decides which tests are available and useful in its setting.

Use a glucose monitor to obtain an immediate blood glucose level. Report the results immediately.

Monitor Leads and Oxygen

High-flow oxygen is applied unless the team leader orders low-flow oxygen.

ECG leads are applied as soon as possible. Place them so they will not interfere with subsequent central venous access attempts or defibrillation.

Place the monitor so that the team leader can easily see the tracing.

Oxygen saturation monitors are often unable to detect oxygenation in shocky patients. Also, carboxyhemoglobin will register as oxyhemoglobin, giving a false number. If a patient's finger nail polish interferes with monitoring, remove it with acetone or polish remover.

Automatic blood pressure cuffs and monitors should be compared to pressures obtained in the standard way. Many times these monitors give false numbers when the patient is in shock.

Temperature probes should be checked to be sure they can measure temperatures down to 13°C (55°F). Temperatures this low may be seen in severely hypothermic patients.

Fluid Resuscitation

Unless the team leader instructs otherwise, begin crystalloid (NS or LR) as the initial IV fluid. Too much NS (several liters in an adult) can result in hyperchloremic acidosis. Ringer's lactate solution is more physiologically correct. However, because it contains calcium, it can cause blood clotting in the tubing

SIMULTANEOUS TEAM ACTION

STEP 3

when administered with blood. When a patient has suffered blood loss, it is best to replace it with blood rather than just electrolyte solution. If too much electrolyte solution is used, the blood becomes hemodiluted, increasing the bleeding problem. Take great care to avoid air embolism via IV lines. Also closely monitor the amount of fluid given.

Another important measure is to administer fluids and blood through a high-flow fluid warmer. Hypothermia decreases the ability of the patient's blood to clot.

Tubes

Ask the team leader about the advisability of inserting an orogastric or nasogastric tube. Use an appropriate sized tube.

Ask the team leader about the need for a urinary catheter. In males, you may anesthetize the urethra by injecting about 5 mL of 2% lidocaine gel into the urethra prior to passing the catheter. Also in males, do not inflate the balloon until the catheter is inserted all the way to its hub and urine appears. This is because urine may appear in the catheter before it has entered the bladder. Do not pass a catheter in traumatized males when there is blood at the tip of the penis (urethral meatus) because this signifies a urethral injury.

In a head-injured patient with a possible urethral injury, bladder decompression using a suprapubic cystostomy may be needed. ([Vol II – TRAUM SKILLS 4 SUPRAPUBIC CYSTOSTOMY](#))

Learn how to secure ET tubes in adults and ([PEDS](#)) infants. See [Vol II – AIR SKILLS 1 AIDS TO INTUBATION](#).

PEDS: Achieve a high comfort level caring for children by familiarizing yourself with the tools and equipment that they require. Know where to look when questions arise.

X-rays

Order imaging as indicated by the patient's signs and symptoms. Obtain chest x-ray for almost all trauma patients. In cases of blunt trauma, use a low threshold for obtaining a pelvic x-ray. Acquire additional imaging according to secondary survey findings. In patients who require rapid transfer, limit studies to only those that impact immediate care.

In a rural setting, the roles of team leader and team members may be carried out by different individuals than in an urban setting. For example, a nurse may assume the role of team leader until a provider is available. Specialists may not be available. Nevertheless, the required tasks that must be accomplished for

successful emergency care are not significantly different. The difference lies in how the workload is divided.

January 2019

STEP 4: PRELIMINARY CLINICAL IMPRESSION

The team leader develops a preliminary impression of the patient's condition by using the historical and clinical data obtained thus far. This impression causes the leader to select a pathway of action that will address the patient's problem(s). A clinical impression may be as simple as recognizing the need for neonatal resuscitation or as complex as recognizing that the patient is obtunded with no clear cause. The team leader must remain flexible and willing to start over again if the initial impression begins to seem incorrect.

The presence of clinical conditions that need immediate attention (such as cardiac arrest or status epilepticus) cause the team leader and team members to travel down a pathway leading through numerous, potential obstacles, both seen and unseen. Measures are taken to overcome the obstacles so that the patient's condition is stabilized.

This step of the process is often the most difficult of all. Step 4 is dynamic and fraught with hard choices. Sometimes it is not possible to stabilize a patient. For instance, a patient with a ruptured abdominal aortic aneurysm may be impossible to stabilize short of a laparotomy and replacement of the aorta.

When it is apparent that the patient is not getting better, the team leader starts back through the ABCs again. Perhaps something has been overlooked or, perhaps an intervention is not working. Maybe another intervention would work.

Performing a focused evaluation (secondary survey) will assist in formulating a working diagnosis. Here are the pathways (as presented in [The First 30 Minutes](#) of the CALS text) down which the team might proceed:

PATHWAY 1: Altered Level of Consciousness (Adult and Pediatric)

PATHWAY 2: Cardiovascular Emergencies (Adult and Pediatric)

PATHWAY 3: Abdominal Emergencies (Adult and Pediatric)

PATHWAY 4: Neonatal Emergencies

PATHWAY 5: Obstetrical Emergencies

PATHWAY 6: Respiratory Emergencies (Adult)

PATHWAY 7: Respiratory Emergencies (Pediatric)

PATHWAY 8: Trauma (Secondary Survey for Adult)

PATHWAY 9: Trauma (Secondary Survey for Pediatric)

When in doubt about which pathway to follow, the depressed LOC pathway will most likely assist with developing the working diagnosis.

STEP 5: WORKING DIAGNOSIS AND DISPOSITION

By this time, the team leader has enough information from clinical observations and initial laboratory results to form a working diagnosis. The leader continues to keep an open mind. Once the working diagnosis is clear, Volume III Diagnosis/Treatment and Transition to Definitive Care Portals help to assist the team leader when additional information is needed. Patient disposition is arranged. The team makes sure that transfer information is complete and checks to be sure that, as much as possible, the patient's medical safety has been provided for.

The information learned from the completion of a patient evaluation (physical examination, diagnostic tests, and observation) plus working through pertinent clinical pathways should result in the development of a working diagnosis. When this is attained, ongoing care or continued resuscitation can continue with some confidence that the patient is stabilized or at least getting better. The team can now take the time to refer to **Volume III** for more information or details.

The health care team must remain vigilant, looking for clues to additional critical problems that may either alter the treatment plan or indicate the need for additional treatment. Tunnel vision at this stage can be catastrophic to the patient as critical abnormalities may be overlooked during the resuscitation process.

Disposition of the patient may include:

1. Discharge home following resolution of the problem.
2. Admission to a local hospital for further evaluation and treatment.
3. Transfer to a tertiary care center for continuing care.

Consult liberally, particularly if the patient is not responding to the initial resuscitative actions. If transfer is anticipated, the patient needs to be packaged appropriately to facilitate a safe transfer.

Packaging up the patient is a team effort that includes pulling together data; securing tubes, monitors, and the patient; assuring that optimal fluid resuscitation has taken place; and maintaining vital signs. Packaging up should include consultation with a tertiary care physician and/or physician-to-physician and nurse-to-nurse contact in preparation for transfer to another facility.

See VOLUME I, ACUTE CARE PORTALS, PATIENT TRANSPORT ALGORITHM.

Who to admit and who to transfer

Base the decision to transfer a patient on probability of severe clinical deterioration (ie, death or significant morbidity) if interventions available only at the receiving facility are delayed. Transfer agreements established ahead of time help to smooth the transfer process and to enhance continuity of patient care.

WORKING DIAGNOSIS

STEP 5

Selecting a mode of transport

Using the appropriate team and mode of transport is vital to positive patient outcomes. Basic considerations include whether or not the patient is stable as well as an individual facility's capabilities at a particular time. Some presumably stable patients are at high risk of sudden decompensation. Consider these patients unstable.

Transport of unstable patients is time sensitive. The sicker the patient, the shorter the transport time. Be mindful of the maxim *time is tissue*.

Goals of Transport

- To minimize out-of-hospital time
- To keep care moving in the right direction

Ideally, transport is dispatched from the emergency scene, prior to patient arrival at a facility. If a patient has arrived and the decision to transport has been made, do not delay transport because of diagnostics.

Suggested Criteria for Consideration of Transfer

Central Nervous System

- Penetrating injury/open fracture, with or without cerebrospinal fluid leak
- Depressed skull fracture
- GCS <14 or deterioration
- Spinal cord injury or major vertebral injury

Chest

- Major chest wall injury or pulmonary contusion
- Wide mediastinum or other signs suggesting great vessel injury
- Cardiac injury
- Patients who may require prolonged ventilation

Pelvis/Abdomen

- Unstable pelvic ring disruption
- Pelvic fracture with shock or other evidences of continuing hemorrhage
- Open pelvic injury
- Solid organ injury

Major Extremity Injuries

- Fracture/dislocation with loss of distal pulses or neurological compromise
- Suspected compartment syndrome

- Open long-bone fractures
- Extremity ischemia

Multiple-System Injury

- Head injury combined with face, chest, abdominal, or pelvic injury
- Burns with associated injuries
- Multiple long-bone fractures
- Injury to more than two body regions

Co-morbid Factors

- Age >55 years
- Children ≤5 years of age
- Cardiac or respiratory disease
- Insulin-dependent diabetes, morbid obesity
- Pregnancy
- Immunosuppression

Secondary Deterioration (Late Sequelae)

- Mechanical ventilation required
- Sepsis
- Single or multiple organ system failure (deterioration in central nervous, cardiac, pulmonary, hepatic, renal, or coagulation systems)
- Major tissue necrosis

Mechanism of Injury Risk Factors that Increase the Risk of Unfavorable Outcome from Trauma

Death in same vehicle

Age < 5 years or > 60 years

Consider Air Transport from the Scene Directly to a Trauma Center if:

1. Ground transport time to local hospital is > air transport time to a trauma center; **or**
2. Ground transport leaves the 911 Primary Service Area coverage compromised.

WORKING DIAGNOSIS

STEP 5

Levels of Care

Three levels of care are available when transporting a patient from the scene of an accident or critical access facility to tertiary care: Basic Life Support (BLS), Advanced Life Support (ALS), and Critical Care.^b

Basic Life Support

According to the Minnesota Statutes, “BLS means rendering basic-level emergency care, including, but not limited to, basic airway management, cardiopulmonary resuscitation, controlling shock and bleeding, and splinting fractures” EMTs are trained in initial and focused surveys. Many BLS services have protocols to dispatch Advanced Life Support (ALS) ground crew and flight teams after making an initial survey.

Components of Basic Life Support

- Airway Management
 - Administer oxygen.
 - Other skills include mouth-to-mouth resuscitation, BVM, or oxygen-powered ventilation, as well as use of adjuncts.
- Automatic External Defibrillator (AED)
- Spinal immobilization techniques
- Keep-open IV administration

Advanced Life Support

According to the Minnesota Statutes, the definition of Advanced Life Support (ALS) is “..... rendering basic life support and rendering intravenous therapy, drug therapy, intubation, and defibrillation”

Components of Advanced Life Support

- All the components of BLS apply.
- Manual defibrillation/cardiac monitoring
- Advanced airway management
 - Intubation
 - Needle jet insufflation
 - Some may perform cricothyrotomy.
 - Ventilator (rate and volume only)
- IV therapy
 - Treatment of hypovolemia
 - Continuation of IV drips but usually do not titrate
- Medications

^bThough Minnesota uses Critical Care transport (which may include specialty teams such as a neonatal or high-risk obstetrics), Critical Care transport is not in the Minnesota Statutes. (BLS and ALS are in the Minnesota Statutes. Other states’ statutes may have different definitions.)

- As described in ACLS courses
- Analgesias
- Glucose and bicarbonate
- Some may have RSI capacity

Critical Care

Critical care teams are staffed (minimally) with one nurse and one paramedic. Teams may consist of two nurses or one nurse and a nurse practitioner or physician's assistant. This service is available by both air and ground.

Specialized teams are sometimes available, including neonatal and high-risk obstetrics teams. A crew may include a variety of specialists to suit patient care. (For example, a patient with a balloon pump may include ICU staff or a perfusionist.)

Components of Critical Care

- Usually two care providers in patient care area
- Initiate and titrate drips
- RSI
- Ventilator
 - Rate/Volume
 - PEEP
 - Various oxygen concentration
 - Various I:E ratios
- Pericardiocentesis
- PRN medications without on line medical control
- Blood administration
- Central lines
- Pacemakers
- Invasive monitoring

Modes of Transport

Three types of transport are ground ambulance, helicopter (rotor wing), and airplane (fixed wing). Each mode of transport carries risks and benefits.

Ground Transport

Use ground transport when time is not critical to patient survival. Transferring a patient by ground might be necessary when weather limits the ability of aircraft to fly or when aircraft are simply unavailable.

Though ground transport is appropriate for many patients, since the advent of helicopters, it is often not considered. One advantage of ground transport is the

WORKING DIAGNOSIS

STEP 5

availability (upon request) of all three levels of care. Another advantage is that there is no weight limit for patient or personnel (as there is with air transport). For bariatric patients, ground transport may be more comfortable as well as the only suitable type available. Ground ambulances contain more working space and can accommodate patients with special needs, such as additional personnel or equipment. Ground transport is beneficial to stable pediatric patients who are generally accompanied by caregivers. Ground transport can also accommodate extremely tall patients (> 6'6") who may be too long to fit in average-sized aircraft.

Helicopter (Rotor Wing)

Though helicopters are undeniably expensive, the best standard of care remains that which minimizes time between incident and definitive care (sometimes called the *golden hour*). Critical care flight crews offer the highest level of care in less time.

Another advantage of helicopter transport is maneuverability. Search and Rescue helicopters are often able to access patients in places ground ambulances are unable to reach, thus reducing patient extrication times at the scene. The required 100-foot diameter landing area makes virtually every rural hospital a temporary heliport. Early activation of a helicopter to a scene is key to reduced transport times to an appropriate facility.

Airplane (Fixed Wing)

Fixed-wing aircraft have different capabilities than helicopters. Because of the flying altitude they can attain, they may be able to fly above inclement weather conditions. Fixed-wing transport offers the same levels of care as ground and rotor wing. Consider fixed-wing transport, which is often faster and less expensive, when a patient must travel over 150 to 200 miles. The disadvantage of fixed-wing aircraft is accessibility to rural areas. Additionally, fixed-wing aircraft must land at an airport, making response times considerably longer.

Selecting the transporting team preparedness level

Identify and survey intercept points for patient and transport team safety. Some questions to consider might be: Would it be best if the critical care expertise came to the rural facility rather than risk decompensation during transport? Could close communication between the referring physician and the consultant provide the time needed to bring a specialized transport team to the facility?

In certain cases, the transporting team may need to take along special equipment, such as an intra-aortic balloon pump or a neonatal ventilator. Special fluids and

medications may need to be brought to the rural facility. Critical care providers may also accompany the transport team.

Communication

Communication is key to the efficiency and effectiveness of any complex system of transportation. Make telephone, radio, and telemedicine (desktop computer platform or dedicated line) communications available at the rural facility for ready and easy communication between referral centers and nearby rural facilities. During severe weather, nearby rural facilities may be able to pool equipment and skilled personnel for certain cases (such as obstetric emergencies) until transfer becomes an option. This requires planning and open communication.

Early notification of the possible need for transfer of certain cases – such as a multiple trauma or burn patients – may save many minutes of resuscitation and transportation time. Prehospital personnel can be given the option to notify distant transporting systems of a possible need to transport, even before the patient reaches the rural facility.

Physician Responsibilities in Planning Patient Transfer

In addition to the role of team leader, the responsibility of the physician (or designee – physician's assistant or nurse practitioner) is to stabilize the patient to the level of the facility's capability and plan for the appropriate mode/level of transport. This includes contacting an accepting physician and consulting on treatments given prior to transport and en route.

Often physicians in rural communities are placed in the role of medical director of the local ambulance service. The two types of medical direction include direct and individual. Direct medical direction is communication that transpires by voice either via a radio system or cell phone. EMS staff report clinical findings and receive orders to intervene and continue patient care. Individual medical direction is given by a physician through written protocols and involves the development and ongoing monitoring of protocols and procedures. This involves reviewing prehospital reports to ensure compliance with predetermined procedure. High quality emergency care is dependent on all prehospital personnel understanding, complying with, and applying all treatment protocols consistently, including those protocols beyond primary and secondary assessment. Protocols include transport methods and destination facilities in a geographic region.

WORKING DIAGNOSIS

STEP 5

“Weather” or Not to Transport

- **Don’t make a transport decision based on weather.** Weather may clear a short distance from you, and intercepting ground to air may be in the patient’s best interest.
- **Dispatch transport early.** The majority of air transfer requests are viable transports. There are no negative consequences to calling and then canceling a transport. Keep in mind when dispatching air transport that weather may not be forecasted between reporting stations or change rapidly. If transport is arranged early enough, alternate transport plans may be made with minimal negative impact to the patient.

How Best to Help the Transport Team

- **Communication.** Know that each transport is unique. Dispatcher and transport personnel are trained and skilled at orchestrating the most efficient transport plan. Be open to conferring with teams in order to choose the best option.
- **ABCs.** It is not necessary to have a patient’s ABCs stabilized before you call. Flight crews are highly trained at advanced or difficult airways and line placements.
- **Transfer forms.** Send all documents with the patient.
- **Diagnostic results.** It is helpful for transport teams to have copies of pertinent diagnostic findings. Minimally, provide a verbal account of abnormal findings to help aid in the appropriate treatment regimen. [See Vol I, ACUTE CARE PORTALS, PATIENT TRANSPORT ALGORITHM.](#)
- **Secure a landing zone when utilizing a helicopter.** For safety’s sake, designate a specific person to secure a landing zone throughout the entire interface (about 10 minutes prior to aircraft landing to liftoff and clearing of helipad).

Stable patients may usually be moved by ground transport staffed by appropriately trained personnel. If the distance is long, air transport may also be used.

Caveats for Transport

Once the need for transport is recognized, do not delay the process for lab or diagnostic procedures that have no impact on the transfer process or immediate resuscitation.

The probability of positive outcome can be improved by minimizing the time from injury to appropriate definitive care.

Health care providers in community hospitals and regions of the state should develop specific guidelines based on local resources that will help to identify patients who will benefit from early transfer to a trauma center.

The purpose of triage/transport procedures is to facilitate early transport of critical trauma patients to the most appropriate health care facility.

STEP 6: TEAM PROCESS AND REVIEW

It is important for the team (including the team leader) to review the emergency experience, including what went right, what went wrong, whether needed supplies were easily available, and whether communications were optimal. Following a difficult case, team members may need emotional support.

We must keep each other willing and able to continue.

An optimistic attitude toward emergency care keeps the team vital. The whole team must nurture this attitude. Team members must have confidence in their abilities and know that their level of knowledge will ensure the survival of patients, if survival is at all possible. The team leader must recognize oversights and mistakes as human error, not as moral lapse or stupidity. However, to ignore mistakes is a mistake in itself. The team should strive to find a way to correct problems through education, protocols, and supportive communication. An ability to communicate effectively and pleasantly is essential, even under stressful circumstances. When a consultant is rude or overbearing, let him or her know at a later time. A letter is an effective communication tool. Consider communications with prehospital personnel. Always make sure you have the facts straight before making assumptions, interpretations, and judgments.

Equipment storage, shortage, and malfunction are common problems. Finding improved ways to order and store instruments is important. The operation of monitors and defibrillators can be problematic for physicians and nurses who seldom use them. Designate specific team members to troubleshoot problems as they arise.

Encourage team members to vent their feelings and frustrations; they must also be encouraged to respect the feelings and frustrations of fellow team members and others. Observe confidentiality.

Severe stress reactions to traumatic emotional events may occur. These may be avoided if a sympathetic ear at an unhurried time is made available to team members. Each medical community should develop a plan for such events. Clergy and staff support specialists may be enlisted to counsel and support team members and others who develop symptoms of severe stress reaction, such as anxiety, sleeplessness, flash backs, difficulty concentrating, nausea, and depression or sadness. National and state critical incident stress management programs are available to assist.

TEAM PROCESS AND REVIEW

STEP 6

Having a form available for team members to jot down their ideas, concerns, and suggestions at the end of the case may be a method of obtaining useful information that would otherwise be lost. Please note that each facility should develop its own system for using this form. Use the form below to review emergency cases, positively or negatively. The form may be adapted as needed.

Emergency Case Review

Name _____ Today's date _____

Date and time of the case _____

Your role in the case (team member?) _____

Areas of function for comment or suggestion:

- * **Team activation and preparation**
- * **Immediate placement and control of the patient**
- * **The initial survey: MARCH**
- * **Team actions**
- * **Equipment availability and function**
- * **Availability of supplies**
- * **Documentation**
- * **Pharmacy, laboratory, and x-ray services**
- * **Preparation for admission or transfer**
- * **Communication with the patient**
- * **Communication with others**
- * **Universal precautions and personnel safety**

Please continue on an added sheet of paper if more space needed.

Comments:

SECTION 4 FOCUSED CLINICAL PATHWAYS

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**PATHWAY 1: ALTERED LEVEL OF CONSCIOUSNESS
(ADULT AND PEDIATRIC)**

The team continues the resuscitation along the pathway suggested by the initial clinical impression. Each pathway includes a complete, thorough, and rapid physical examination with additional history taking. The team leader is wary of conditions that may not be apparent. To obtain additional clinical data or correct a missed or newly developed condition, the team leader repeats the initial survey if patient is not responding satisfactorily.

This section applies to situations of neurological impairment in which the cause and/or treatment are not immediately apparent. Many of the topics covered here may apply to confusion, delirium, coma, and/or seizures.

Clinical Consideration in Decreased Level of Consciousness/Coma/Delirium

Text Number	Diagnosis/Condition	Related Materials
TIPS FROM THE VOWELS		
T – TRAUMA, TEMPERATURE, TUMOR		
1	Traumatic Head Injury	Vol I – PATHWAY 8 ADULT TRAUMA, PATHWAY 9 PEDIATRIC TRAUMA, Vol III – TRAU CARE 1, TRAU CARE 2
2	Hypothermia	Vol III – ENV1 HYPOTHERMIA
3	Hyperthermia	Vol III – ENV2 HYPERTHERMIA, SED 3 MALIGNANT HYPERTHERMIA
4	Tumor of the Brain	
I – INFECTION		
5	Meningitis/Encephalitis	Vol III – IN2 MENINGITIS
6	Sepsis	Vol III – IN3 SEPSIS IN ADULTS, Vol I – PATHWAY 6 ADULT RESPIRATORY
7	Pneumonia	
8	Urosepsis	
P – POISONING, PSYCHOGENIC POISONING (DRUG OVERDOSE, TOXIC EFFECTS)		
9	Acetaminophen Overdose	Vol III – TOX3 ACETAMINOPHEN OVERDOSE
10	Aspirin Overdose	Vol III – TOX4 ASPIRIN OVERDOSE
11	Tricyclic Overdose	Vol III – TOX 5 TRICYCLIC ANTIDEPRESSANTS

**ADULT AND PEDIATRIC
ALTERED LEVEL OF CONSCIOUSNESS
PATHWAY 1**

12	Anticholinergic Drug Overdose	
13	Benzodiazepine Overdose	Vol III – TOX8 BENZODIAZEPINE OVERDOSE
14	Hypnotic Drug Overdose	
15	Methamphetamine Intoxication	Vol III – TOX13 AMPHETAMINE ANALOG INTOXICATION
P – POISONING (TOXIC SUBSTANCE INGESTION)		
16	Carbon Monoxide Poisoning	Vol III – TOX15 CARBON MONOXIDE POISONING
17	Cyanide Poisoning	Vol III – TOX17 CYANIDE POISONING
18	Organophosphate (Insecticide)	Vol III – TOX18 ORGANOPHOSPHATES TOXICITY
19	Iron Ingestions	Vol III – TOX14 IRON INGESTION
20	Hydrocarbon Ingestions	
21	Psychogenic Coma (Catatonic State)	
S – STROKE, SHUNTS, SHOCK (CNS INSULTS)		
22	Thrombotic or Embolic Strokes	Vol III – NEU2 STROKE
23	Intracerebral Hemorrhage	Vol III – CV13 HYPERTENSIVE CRISES, NEU5 INCREASED INTRACRANIAL PRESSURE
24	Subarachnoid Hemorrhage	Vol III – NEU5 INCREASED INTRACRANIAL PRESSURE
25	Subdural Hematoma	
26	Epidural Hematoma	Vol II – DISAB SKILLS 1 SKULL TREPHINATION
27	Increased Intracranial Pressure	Vol III – NEU5 INCREASED INTRACRANIAL PRESSURE
28	Shock (from any source)	Vol I – ACUTE CARE PORTALS SHOCK ACRONYM – SHRIMPCAN; Vol III – CV 17 SHOCK
29	Shunt Malfunction	
A – ALCOHOL (INTOXICATION, WITHDRAWAL, TOXIC EFFECTS)		
30	Ethyl Alcohol Intoxication	
31	Ethyl Alcohol Withdrawal	Vol III – TOX9 ALCOHOL WITHDRAWAL
32	Methanol Intoxication	Vol III – TOX10 TOXIC ALCOHOLS

ADULT AND PEDIATRIC
ALTERED LEVEL OF CONSCIOUSNESS
PATHWAY 1

33	Ethylene Glycol Intoxication	
34	Isopropanol Intoxication	
E – EPILEPSY, ENCEPHALOPATHY		
35	Status Epilepticus/Postictal	Vol I – ACUTE CARE PORTALS; Vol III – NEU1 STATUS EPILEPTICUS;
36	Hypertensive Encephalopathy	Vol III – CV13 HYPERTENSIVE CRISES
37	Hepatic Encephalopathy	
I – INSULIN AND OTHER HORMONES		
38	Diabetic Ketoacidosis	Vol III – END/M2 DIABETIC KETOACIDOSIS
39	Non-Ketotic Hyperosmolar Coma	Vol III – END/M5 HYPEROSMOLAR STATE
40	Hypoglycemia	
41	Ketotic Hypoglycemia	
42	Addisonian Crisis	Vol III – END/M2 DIABETIC KETOACIDOSIS
43	Thyroid Storm (Hyperthyroidism)	Vol III – END/M4 THYROID STORM
44	Myxedema Coma	Vol III – END/M3 MYXEDEMA COMA
O – OXYGEN, OPIATES, AND OTHER DRUGS OF ABUSE		
45	Hypoxia	Vol I – PATHWAY 6 ADULT RESPIRATORY, PATHWAY 7 PEDIATRIC RESPIRATORY
46	High Altitude Illness	Vol III – ENV5 HIGH ALTITUDE ILLNESS
47	Opiate/Heroin/Narcotic Overdose	Vol III – TOX12 NARCOTIC OVERDOSE
48	Cocaine Ingestion	Vol III – TOX 11 COCAINE INGESTION
49	Amphetamine Intoxication	Vol III – TOX 11 COCAINE INGESTION
50	Phencyclidine Intoxication	Vol III – TOX 11 COCAINE INGESTION
U – UREMIA AND OTHER METABOLIC ABNORMALITIES		
51	Acute Renal Failure	
52	Electrolyte Abnormalities	Vol III – END/M7 DISORDERS OF ELECTROLYTE CONCENTRATION
53	Acid Base Abnormalities	Vol III – END/M6 ACID-BASE

**ADULT AND PEDIATRIC
ALTERED LEVEL OF CONSCIOUSNESS
PATHWAY 1**

The guiding memory phrase for diagnosing altered levels of consciousness is “**DONT** forget the **TIPS** from the vowels: **AEIOU**.”

The first step (after airway management and stabilization of life threats) is to assess for problems that may be immediately diagnosed and treated. **DO the DONT: Dextrose, Oxygen, Naloxone (Narcan), and Thiamine.**

Dextrose: Assess blood sugar by bedside testing or if not available, give 1 amp of D₅₀ IV for presumed hypoglycemia. (See thiamine for precautions.) **PEDS:** child: 2 mL/kg D₂₅; neonate: 2 to 4 mL/kg D₁₀.

Oxygen: Clinical hypoxia is often apparent as an airway/breathing problem, but monitor O₂ saturation whenever possible. Use liberally if O₂ status is in doubt.

Naloxone (Narcan): Consider administering whenever narcotic intoxication cannot be ruled out. The dose is 0.4 to 2.0 mg IV every 2 to 3 min prn or 0.1 mg/kg IN – max 2 mg, if Carfentil was used then max dose is up to 10 mg (2 mg/2 mL concentration). **Note; Due to the ever changing Opioids being used realize that in certain cases it may be necessary to give multiple doses of Narcan, and then even the need to start a Narcan drip, and titrate it to effect, and continue it till the effects of the Opioid used has resolved.** **PEDS:** Initial dose is 0.01 mg/kg to 0.03 mg/kg IV; increase the dose to 0.1 mg/kg IV. Or give 0.1 mg/kg IN – max 2 mg (2 mg/2 mL concentration). Neonatal dose is 0.1 mg (0.25 mL of 0.4 mg/mL)/kg IV, IO. Problems with Narcan are usually minimal and rare but include precipitation of the withdrawal syndrome, which can include seizures in addicted neonates.

Thiamine: Administer this critical vitamin at a dose of 100 mg IV to patients who are debilitated or may be chronic alcoholics, preferably before giving D₅₀ to adults.

A positive response to an intervention suggests which clinical direction to pursue. After completing the DONT sequence, if an altered level of consciousness (LOC) persists and its cause is still puzzling, continue through the rest of the mnemonic **TIPS from the vowels: AEIOU**. Review the list and start with the most likely condition given the patient’s circumstances.

ASSESSMENT AND MANAGEMENT

T – TRAUMA, TEMPERATURE, TUMOR

1 Traumatic Head Injury

Suspect head trauma in all cases until this condition is ruled out or another cause is identified, as the exam may or may not reveal signs of cranial trauma. CT imaging is necessary to diagnose the different types of intracerebral bleeding. **PEDS:** Perform an ophthalmoscopic examination of the retinas in all infants with a decreased level of consciousness (shaken baby syndrome). Keep the possibility of trauma in mind. (Vol I – PATHWAY 8 ADULT TRAUMA, PATHWAY 9 PEDIATRIC TRAUMA)

2 Hypothermia (Vol III – ENV1 HYPOTHERMIA)

Assess temperature in all patients under all conditions. Core temperature measures are needed for management of extreme cases. Note: many thermometers do not register temperatures lower than 90°F, so a low-reading rectal probe thermometer is needed if low body temperature is suspected. Categorize and treat hypothermia as follows:

Mild hypothermia (92°F to 95°F [33.3°C to 35°C]). A patient may be passively or externally rewarmed with blankets or forced-air, commercially available, plastic or paper blankets.

Moderate hypothermia (86°F to 92°F [30°C to 33.3°C]). A patient may be externally rewarmed if cardiovascular function is intact.

Severe hypothermia (below 86°F [30°C]). Patients with severe hypothermia are often in cardiac arrest or severe bradycardia and are at risk for decompensating to ventricular fibrillation. When monitoring is available, chest compressions are recommended only for ventricular fibrillation (VF) or asystole; avoid CPR if any organized rhythm is present, even if pulses are not palpable. Handle gently (VF is easily precipitated in a hypothermic patient) but manage airway as needed, including intubation. If in VF some may make one attempt to defibrillate, while others will wait till the patient is warmed. If unsuccessful, postpone further attempts until temperature is 86°F (30°C) or higher. Generally, avoid cardiac medications until the patient has been rewarmed; these medications don't work at low temperatures and accumulate without being metabolized, causing problems when rewarming occurs. External rewarming may be hazardous in severe hypothermia.

Employ techniques of core rewarming. These include:

- warmed IV fluids (40°C to 43°C [104°F to 109°F]); avoid volume overload

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- warm lavage of bladder (via Foley catheter) and stomach (via NG tube)
- heated humidified O₂ ventilation at 42°C to 46°C (108°F to 115°F) by mask
- warmed saline lavage of the mediastinum through chest tube (left side or bilateral),
- peritoneal lavage with warm saline
- AV rewarming with femoral catheters and blood warming circuit or complete AV bypass (This will be in a tertiary facility)

3

Hyperthermia

Fever is a physiologic response to many conditions, including infection. Hyperthermia is a pathological elevation in body temperature due to inability to lose heat. Heat exhaustion is a fluid/electrolyte problem manifested by symptoms such as weakness and fainting. Heat stroke is defined by hyperthermia (usually > 104°F [40°C]) and change in mental status. Patients at risk for heat stroke are those exposed to heated environments, young children and the geriatric population, and those taking certain medications, especially phenothiazines.

Treatment of heat stroke

Besides the usual resuscitative measures, **rapid cooling is required**. This may take the form of cold packs to axillae, neck, and inguinal areas; the use of tepid water spraying and fans; or ice water immersion (which is less recommended). Shivering interferes with cooling and may be treated with benzodiazepines and/or phenothiazines (even though these drugs may also predispose to hyperthermia).

Malignant hyperthermia is an uncommon but life-threatening disease seen with use of anesthetics, including drugs used for RSI. Malignant hyperthermia usually comes on dramatically after administration of Succinylcholine. (Monitor post-RSI patients who have a temperature.) Classic signs and symptoms include fever, rigidity, tachycardia, tachypnea, metabolic acidosis, or respiratory acidosis. Discontinue any inciting agent(s), institute cooling measures (**Vol III – ENV2 HYPERTHERMIA/HEAT STROKE**), check for acidosis regarding bicarbonate treatment (**Vol III – END/M6 ACID-BASE**), and give dantrolene 1 to 3 mg/kg IV ;(**Note there are now different versions of dantrolene, such as Ryanodex, which have different dosing regimens**) **PEDS: 0.5 to 1 mg/kg IV**. Search for end-organ damages resulting from this insult including respiratory failure, renal failure, DIC, cardiovascular collapse, or rhabdomyolysis.

Malignant hyperthermia may also be caused by various antipsychotics, which is called neuroleptic malignant syndrome. In addition to high temperatures, findings include labile blood pressure and muscular rigidity. To treat, initiate cooling measures, aggressive support care, muscle relaxation with either paralyzing agents (nondepolarizing neuromuscular blocking agents, **Vol III –**

AIR1 RAPID SEQUENCE INTUBATION) or non-paralytic agents such as dantrolene 1 to 3 mg/kg IV; (**Note there are now different versions of dantrolene, such as Ryanodex, which have different dosing regimens**) (PEDS: 0.5 to 1 mg/kg IV). Consider adding bromocriptine 2.5 to 10 mg PO. Again, for more information, see **Vol III – ENV2 HYPERTHERMIA/HEAT STROKE**.

4

Tumor of the Brain

If brain tumor (either primary or metastatic) is suspected, obtain a head CT for diagnosis. Contrast may be needed for definition of a mass. Steroids (Decadron 10 to 20 mg IV/IM. **PEDS: 0.25 to 0.5 mg/kg IV**) may be dramatically helpful in reducing edema and its mass effect.

I – INFECTION

5

Meningitis/Encephalitis

Suspect CNS infection (meningitis, encephalitis, and abscess) in patients with altered LOC, until these conditions are either ruled out or another cause is identified. Fever, nuchal rigidity, and/or focal neurological changes are highly suggestive of CNS infection.

PEDS: Very young children may exhibit subtle signs, such as listlessness, poor feeding, and irritability. There may be grunting respirations. Seizures are common. A bulging fontanelle is a late and inconsistent finding. A child with meningeal irritation may wish to lie in a fetal position. When meningococemia is occurring, you may see a distinctive purpuric rash. Hypoglycemia may be present. Cerebral edema may be present.

The impression of an experienced examiner, while not perfect, is the best single guide to the presence of CNS infection in this age group. In neonates and young infants (age 0 to 3 months), clinical evaluation may be difficult.

Lumbar puncture (LP) is the best diagnostic test for meningitis. Because of the theoretical risk of herniation in the face of elevated intracranial pressure (ICP), many practitioners prefer to perform a CT scan prior to LP. CT scanning has not been proven absolutely necessary nor 100% effective for this purpose. Most experts now recommend obtaining blood cultures and starting antibiotic treatment before either CT or LP in patients where meningitis is strongly suspected.

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Geriatric patients frequently have minimal physical findings, other than a decreased LOC.

See Vol III – IN2 MENINGITIS for further discussion of CNS infection.

Initial Management

1. Administer oxygen and protect the airway.
2. Establish an IV.
3. Draw a blood cultures and routine labs. Begin antibiotic IV therapy as soon as the diagnosis of bacterial meningitis is seriously considered.
4. Septic shock may be present. Initiate volume loading with crystalloid (NS or LR), 1 to 2 liters in adults and **(PEDS) 10 to 20 mL/kg in children**. Take care not to overshoot the mark, which would result in cerebral edema.
5. If seizure activity, papilledema, or any focal neurologic finding is present, obtain a CT scan of the head as soon as possible to rule out mass effect of a brain abscess or severe cerebral edema. If these are not found, **(PEDS) perform a L4-L5 lumbar puncture in neonates** and a L3-L4 lumbar puncture in older children and adults to obtain spinal fluid for culture and examination for cells and glucose.

6

Sepsis

Definitions:

Sepsis – is defined as the presence (probable or documented) of infection together with systemic manifestations of infection. **(Vol III – IN3 SEPSIS IN ADULTS, Table 1)**

Severe sepsis – is defined as sepsis plus sepsis-induced organ dysfunction or tissue hypoperfusion. **(Vol III – IN3 SEPSIS IN ADULTS, Table 2)**

Sepsis-induced hypotension – is defined as a systolic blood pressure <90 mm Hg or mean arterial pressure < 70 mm Hg, or a systolic BP decrease > 40 mm Hg.

Septic shock – Sepsis-induced hypotension persisting despite adequate fluid resuscitation.

Sepsis-induced hypoperfusion – is defined as infection-induced hypotension, persisting elevated lactate, or oliguria.

Multi-organ dysfunction syndrome – Presence of altered function of two or more organs in an acutely ill patient such that hemostasis cannot be maintained without intervention.

Initial Evaluation

1. History and physical exam
2. Laboratory studies
 - a. Blood cultures (at least 2 with one drawn percutaneously and one drawn through each vascular access device if in place > 48 hours)

- b. Cultures of urine, CSF, sputum, wound, or other body fluids
- c. CBC, coagulation, BUN, Cr, HCO₂⁻, liver enzymes, amylase, UA
- d. Lactate (if greater than 4 mmol/L = severe sepsis)

Initial Resuscitation

1. Fluids – **First priority**. Give 30 cc/kg IV crystalloid minimum as the initial fluid resuscitation. More rapid and greater amounts of fluid may be required for some patients. Septic shock patients may require 6 to 10 liters of fluid in the first 24 hours. If after initial fluid bolus, the patient continues to be hypotensive (septic shock) or shows signs of at least one organ dysfunction or elevated lactate (> 4 mmol/L, severe sepsis), initiate **Early Goal-Directed Therapy**. (See below.)
PEDS: In children, give 20 mL/kg of NS.
3. **Antibiotics** – Give broad-spectrum antibiotics within the first hour after diagnosis of sepsis (after cultures obtained).
4. **If after initial fluid bolus**, the patient continues to be hypotensive (septic shock) or shows signs or at least one organ dysfunction or elevated lactate (> 4 mmol/L, severe sepsis), initiate **Early Goal-Directed Therapy**. (See below.)

Early Goal-Directed Therapy

1. Place central line for monitoring central venous pressure (CVP) and central venous O₂ saturation. (Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS)
2. **Fluid Resuscitation**. Administer 500 cc boluses of NS until CVP is 8 to 12 (12 to 15 in mechanically ventilated patients). If unable to monitor CVP, continue fluid resuscitation until there is subtle evidence of intravascular volume overload. Ultrasound of the inferior vena cava may also be used to estimate CVP and fluid responsiveness if CVP monitor is not available. (Vol II – CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT)
3. **Vasopressor therapy**. If the patient remains hypotensive despite adequate fluid resuscitation, place arterial line. If mean arterial pressure (MAP) is < 65, begin vasopressor therapy with norepinephrine. Epinephrine should be used (either added to or substituted for norepinephrine when an additional agent is needed to maintain adequate BP). Note: Give vasopressors through a central line, and place an arterial line in all patients receiving vasopressors. Maintain MAP of 65 to 90. If unable to provide arterial line or CVP monitoring, consider early transfer to tertiary level ICU.
4. **Measure central venous O₂ saturation** (either by continuous monitor or individual sample). If central venous O₂ saturation < 70, check Hgb. If < 10, transfuse 1 unit of PRBC. If > 10, start inotropic therapy with dobutamine (2 to 20 µg/kg/min; titrate so heart rate does not increase by > 10% of baseline – preferred, especially if the patient is already receiving norepinephrine as a vasopressor). Premature use vasoconstrictors may result in renal failure and severe peripheral ischemia; be sure that the optimal effect

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- of volume loading has been achieved before resorting to this treatment. Also consider intubation and mechanical ventilation.
5. **Lactate clearance** – If ScvO₂ is not available, lactate normalization may be a feasible option. (Jones et al found that a lactate clearance¹ [decrease by at least 10%] was non inferior to achieving a ScvO₂ of < 70 %.)
 6. **Steroids (stress dose)**. Corticosteroids should not be used in adult septic shock patients if adequate fluid resuscitation and vasopressor therapy are able to restore hemodynamic stability. If this is not achievable, give intravenous hydrocortisone at a dose of 200 mg/day. Hydrocortisone may be administered in a continuous infusion (which is preferred) or in divided doses of 50 mg IV every 6 hours. This should be tapered when the patient no longer requires vasopressor support.
 7. **Glycemic control**. Maintain blood glucose < 180. This may require a continuous insulin infusion.
 8. **DVT prophylaxis**. Give severe sepsis patients DVT prophylaxis with either low dose unfractionated heparin (5000 units SQ every 12 hours) or low molecular weight heparin (Lovenox 30 mg SQ every 12 hours). If there is a contraindication to heparin use, a mechanical prophylactic treatment such as intermittent compression devices may be used.
 9. **Stress ulcer prophylaxis**. Administer a proton pump inhibitor or H₂ blocker to prevent stress ulcers.
 10. **Mechanical ventilation in acute lung injury (ALI)/acute respiratory distress syndrome (ARDS)**. In patients who are mechanically ventilated, use low tidal volumes (6 mL/kg of predicted body weight) with the goal of maintaining end-expiratory plateau pressures of < 30 cm H₂O. (**Vol III – IN3 SEPSIS IN ADULTS, Vol I – PATHWAY 6 ADULT RESPIRATORY**)

7

Pneumonia

Pneumonia may present as a decreased LOC at the extreme ages of life. Even without abnormal physical findings, it is important that pneumonia be ruled out in all patients with a decreased LOC.

8

Urosepsis

Urosepsis may be the underlying source of the infection causing a decreased LOC. **PEDS: Be highly suspicious of this in young children** and in geriatric patients. A very resistant bacteria may be the cause of urosepsis in institutionalized geriatric patients.

P – POISONING, PSYCHOGENIC POISONING (DRUG OVERDOSE, TOXIC EFFECTS)

Please Note: Many types of poisoning may cause altered LOC. **Some of the common toxins listed here cause altered LOC only as a late finding.** Always be aware of co-ingestants. For more information, [see Vol III – TOXICOLOGY PORTALS \(particularly TOX1 SYSTEMATIC APPROACH\)](#).

Consider contacting the Poison Control Center (1-800-222-1222, available 24 hours a day) for any patient in whom significant poisoning is suspected. Telephone contact is useful for obtaining the most current recommendations and valuable resources in the assessment and treatment of acutely poisoned patients.^a

9

Acetaminophen Overdose

Acetaminophen overdose can result in fatal hepatotoxicity. In adults and adolescents, hepatotoxicity may occur following ingestion of 7.5 gram (24 regular strength or 15 extra-strength pills). **PEDS:** Younger children are less subject to this complication, but it does occur. The patient is usually free of severe symptoms but may exhibit flu-like symptoms.

Initial Management

1. Consult Poison Control Center (1-800-222-1222, available 24 hours a day) and consider administering activated charcoal.
2. Consider this diagnosis in all cases of overdose. The potentially fatal hepatotoxicity can be prevented by administering N-acetylcysteine before symptoms appear. [See Vol III – TOX3 ACETAMINOPHEN OVERDOSE.](#)

10

Aspirin Overdose ([Vol III – TOX4 ASPIRIN OVERDOSE](#))

Patients with significant aspirin ingestions may require a high level of critical care. In such cases, 300 mg/kg or more has been ingested. Nausea, vomiting, epigastric pain, hyperthermia, confusion, coma, and seizures may occur. Hypokalemia, anion-gap acidosis ([Vol III – END/M6 ACID-BASE](#)), and high or low glucose levels may be present. The patient may complain of tinnitus. Tachypnea may be present. Attempt to determine whether the patient is chronically overdosing with aspirin. If this is the case, serum salicylate levels correlate poorly with the degree of toxicity present. Patients who are chronically ill are prone to develop pulmonary edema. Strongly consider contacting the Poison Control Center (1-800-222-1222, available 24 hours a day) or a toxicologic consultation if a patient is symptomatic with aspirin overdose.

^a Poison Center consultation additionally supports clinicians' treatment decisions. Consult calls provide the Poison Control Center with valuable, collectable data about current, common conditions presenting to

EDs.

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Initial Management

1. Administer oxygen and protect the airway as necessary.
2. Start IVs.
3. Treat seizure activity. (**Vol III – NEU1 STATUS EPILEPTICUS**)
4. Consult Poison Control Center regarding gastric lavage.
5. Begin a volume load of 1 to 2 liters normal saline in adults and (**PEDS**) 10 to 20 mL/kg in children.
6. Administer 0.5 to 1.0 mEq/kg sodium bicarbonate IV. Monitor blood gases to maintain a pH of 7.4 to 7.5.
7. Monitor the ECG for signs of hypokalemia: low voltage, flattened T waves, increased QT interval, U waves. Add 20 mEq potassium to each liter of fluid if the potassium levels are low.
8. Place a Foley catheter.
9. Once rehydrated, use D₅ ½ NS and add 100 mEq sodium bicarbonate to each liter bag. Administer at a rate to achieve a urine output of about 1 to 1.5 mL/kg/h.
10. Pulmonary edema due to capillary leak needs to be monitored carefully.

11

Tricyclic Overdose (Vol III – TOX 5 TRICYCLIC ANTIDEPRESSANTS**)**

Tricyclic antidepressants may cause sudden deterioration in LOC, cardiovascular collapse, and seizures. Patients are notorious for their propensity to suddenly crash after appearing well. Any ingestion over 10 mg/kg or of unknown quantity constitutes grounds for concern and immediate action. Widening of the QRS > 0.1 second, sinus tachycardia, torsades de pointes, and anticholinergic effects such as dry mouth, dilated pupils, and hot red skin also point to tricyclic intoxication. **Anticholinergic** symptoms are described as: **Red** as a beet, **Dry** as a bone, **Hot** as a hare, and **Mad** as a hatter. The patient may seize or suffer a dysrhythmia at any time, so prepare for the worst.

Initial Management

1. Administer oxygen and protect the airway with a cuffed ET tube early. RSI may be of great use. (**Vol III – AIR1 RAPID SEQUENCE INTUBATION**)
2. Check ABCs; correct acidosis.
3. Consider Aqueous AC if ingestion has been within 1 hour.
4. Start IVs immediately
5. Apply ECG leads and an O₂ sat monitor. Check the length of the QRS interval. If it is prolonged (> 0.1 sec or 2.5 little squares), administer sodium bicarbonate 1 mEq/kg IV as a bolus/load, followed by a drip to titrate the pH into an alkalotic range of 7.45 to 7.5. **PEDS:** Use the same dose for pediatric patients. If the QRS interval is > 0.16 sec or 4 little squares, seizures and dysrhythmias are likely.

6. If the QRS interval is not prolonged and the patient is cooperative, administer AC.
7. If the patient seizes, intubate the patient (if not already done). Terminate the seizure with lorazepam or diazepam. (**Vol III – NEU1 STATUS EPILEPTICUS**)
8. Dysrhythmias are common. Sodium bicarbonate is the most effective treatment.
9. Treat hypotension with the Passive Leg Raise and IV fluids. The use of Ultrasound can help with the determination of fluid status. The use of vasopressors (e.g., norepinephrine 4 mg/500 cc D₅W at 0.1 to 0.2 µg/kg/min) may be necessary for hypotension unresponsive to fluids. Do not use dopamine because its beta effects may exacerbate the beta-mediated effects of the tricyclic agent on the heart and vasculature. Intra-aortic balloon pumping may be indicated.
10. Consider Intralipid® for refractory cases. Poison Control Center (1-800-222-1222, available 24 hours a day)

12 Anticholinergic Drug Overdose

Anticholinergic drugs such as atropine, scopolamine, and the antihistamines may cause changes in mental status, along with hot red skin, tachycardia, dry mucosa, and dilated pupils. Treatment is usually supportive. But, in cases of seizure or coma, consider physostigmine. The dose is 0.5 to 2 mg IV over 5 to 10 minutes; may repeat in 20 to 30 minutes. **PEDS: Children 0.02 mg/kg over at least 2 min (neonate dose is unknown).** This may be repeated every 10 to 20 minutes until therapeutic effect manifested as the anticholinergic toxic effects resolve. Do not use physostigmine for tricyclic antidepressant overdose.

13 Benzodiazepine Overdose

Benzodiazepine overdose commonly causes altered LOC. Monitor respiratory depression and consider administration aqueous AC (**Consult Poison Control**). Consider using the antidote flumazenil only after complete patient evaluation and consideration of flumazenil's risks. (**Vol III – TOX8 BENZODIAZEPINE OVERDOSE**)

14 Hypnotic Drug Overdose

Hypnotics/sedatives are a broad group of drugs (barbiturates and non-barbiturates) that are commonly abused and/or used for suicide attempts. Co-ingestants are common. ABC management and limiting further absorption are necessary but insufficient interventions. Use drug levels in critical patients to guide selective aggressive therapy for specific interventions, such as alkaline diuresis for symptomatic barbiturate overdoses or hemodialysis for chloral hydrate overdose. Sodium bicarbonate (1 to 2 mg/kg IV over 2 minutes) followed by 100 mg/L D₅ ½ NS at a rate of 2 to 3 cc/kg/h may be used to alkalinize the urine to facilitate barbiturate excretion.

15

Methamphetamine Intoxication (Vol III – TOX13 AMPHETAMINE ANALOG INTOXICATION)

Clinical effects include hallucinations, paranoia, heightened energy level, and severe agitation. Life-threatening adverse effects include arrhythmias, hyperthermia, hypertensive crisis, myocardial infarction, cardiovascular collapse, seizures, and respiratory failure.

Initial Management

1. Use benzodiazepines for control of seizures and agitation, keeping in mind that a very large dose of benzodiazepines may be necessary to control the patient's symptoms.
2. Use a vasodilator (eg, nitroprusside) or an alpha/beta antagonist (eg, labetalol) or an alpha antagonist (eg, phentolamine) for the treatment of hypertensive crisis.
3. Use active cooling measures to treat drug-induced hyperthermia.
4. Administer intubation and paralysis to manage respiratory failure and to help reduce muscle rigidity that contributes to severe hyperthermia. Avoid the use of succinylcholine for intubation of a patient that is hyperthermic due to meth.
5. Carefully monitor for and treat severe electrolyte imbalance, metabolic acidosis, and disseminated intravascular coagulation.
6. Consult early with a clinical toxicologist to help with the management of difficult patients.

16

Carbon Monoxide Poisoning

Consider carbon monoxide poisoning in patients exposed to smoke, fumes, or fire as well as in patients for whom headache is prominent and where companions and/or family members are similarly affected. Diagnosis is by blood levels; start treatment with 100% O₂ by non-rebreather or ET tube pending the return of the CO level. Use hyperbaric treatment for serious situations or high CO levels. (Consult the nearest hyperbaric team.) (Vol III – TOX15 CARBON MONOXIDE POISONING)

17

Cyanide Poisoning (Vol III – TOX17 CYANIDE POISONING)

Cyanide poisoning should be suspected in the following instances: suicide attempts, industrial accidents, or exposure to products of combustion. Suspect cyanide poisoning in smoke inhalation or burn victims whose CO levels do not accurately reflect their clinical picture. Most serious cases of cyanide toxicity are manifested by seizures, coma, shock, and cardiac arrest. Do not wait for lab results before treating cases that you suspect are life-threatening. The first option,

if available, is hydroxocobalamin, the Cyanokit™ (which contains hydroxocobalamin). In the presence of cyanide, hydroxocobalamin takes up the cyanide and becomes a form of vitamin B12. The starting dose of hydroxocobalamin for adults is 5 g administered as an IV infusion over 15 minutes.

If not available, the other common treatment has 3 components (usually found in a cyanide antidote kit): amyl nitrate inhalation from “popper” ampules; 3% sodium nitrite slow IV push (adult and **PEDS**: 0.33 cc/kg up to 10 cc); and sodium thiosulfate 12.5 g IV at 3 to 5 mL/min (**PEDS**: 412.5 mg/kg IV at 3 to 5 mL/min).

18

Organophosphate (Insecticide)

Organophosphates occur in pesticides and nerve gas. Organophosphates inhibit the enzyme acetylcholinesterase resulting in the accumulation of acetylcholine at muscarinic and nicotinic receptors.

Presenting symptoms may occur from within just a few minutes to 12 hours post exposure. Organophosphates are lipid soluble so patients will develop muscarinic symptoms first and then progress to nicotinic and CNS symptoms. Hypersecretion occurs due to hyperactivity of the gut and the bronchial muscles. Patients secrete copious amounts of fluids from every orifice. Effects on the brain may produce staggering gait, severe tremor, and a psychosis that may be mistaken for alcohol intoxication. **See Vol III – TOX18 ORGANOPHOSPHATES TOXICITY.**

Quick recognition of symptoms is necessary to reverse cholinergic effects. Sudden unconsciousness may be attributable to heat exhaustion, but may potentially be a result of organophosphate exposure. Miosis and muscle twitching are symptoms not seen with heat exhaustion but seen with organophosphate exposure.

Initial Treatment

1. Emergency workers are at risk of becoming poisoned themselves when caring for an organophosphate poisoned patient. Team members should wear rubber gloves and protective gowns to avoid contamination. Protect yourself.
2. The first job is to decontaminate the patient. All types of clothing absorb organophosphates, so remove and decontaminate all clothing to prevent further exposure. Place clothing in a container that can be sealed. Wash the patient with soap and water 3 times. If there has been ocular exposure, irrigate the eyes with a copious amount of tap water for 15 minutes.
3. Syrup of ipecac is contraindicated. Perform gastric lavage (**Consult Poison Control**) followed by activated charcoal if ingestion is within an hour. Because some organophosphates are carried in hydrocarbon-based solvents, use a cuffed ET tube to prevent aspiration.
4. Administer oxygen and intubate the patient if there is a decreased level of consciousness or if tracheal and pharyngeal secretions threaten the airway. Support respiratory function.
5. Obtain IV access.

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6. Obtain an ECG.
7. Medications. Atropine blocks the action of acetylcholine. 2-PAM treats the muscle weakness and reverses the poison's activity on the acetylcholinesterase enzyme, but atropine is cheaper and more readily available to start treatment. Atropine may be given up to 1 to 2 grams in severe cases. **Give until mucus membranes are dry and patient is not wheezing.**
 - **Atropinization.** Consider for all patients with significant cholinergic symptoms. Adult Dose: 1 to 2 mg slow IV push and **PEDS Dose: 0.015 to 0.05 mg/kg slow IV push.** Doses may be repeated every 15 minutes as needed, and doses can be doubled (2 to 4 mg). Atropine drips may also be used at 0.5 to 2.4 mg/kg/hour.
 - The endpoint of therapy is drying of secretions (nasal, oral) or respiratory stability. Atropine IO may also be given. For severe poisonings, therapy may be required for 48 hours. **Note:** Atropine does not reverse muscle weakness or respiratory failure.
 - **Pralidoxime (2-PAM).** 2-PAM reverses the phosphorylation of the cholinesterase molecule. It should be given within 24 hours but may be effective up to 36 to 48 hours. If use of atropine indicates an OP exposure, administer 2-PAM concurrently. Adult Dose: 1 gram IV and **PEDS Dose: 25 to 50 mg/kg in 200 mL D₅W or NS over 15 minutes.** Too rapid exposure can result in tachycardia, muscle rigidity, and neuromuscular blockade. The dose may be repeated in 1 hour and every 6 to 12 hours for 24 to 48 hours if symptoms are still present.
 - The endpoint of therapy is resolution of coma and fasciculations. **Note:** Highly fat-soluble compounds may require longer therapy. Continuous infusion of 500 mg/hour or 10 to 25 mg/kg every 8 hours may be used.
8. Terminate seizures with diazepam.
9. Pralidoxime (2-PAM) is the specific antidote for muscle weakness/paralysis, but atropine is cheaper and more readily available to start treatment. Initial dose 1 to 2 g slow IV push (in severe cases) over 15 min. Give until mucous membranes are dry. **PEDS: Initial dose 25 mg/kg IV over 30 min.** The endpoint of therapy is drying secretions. (**Vol III – TOX18 ORGANOPHOSPHATES TOXICITY**)

19

Iron Ingestions

Iron ingestions are notoriously difficult to assess. Shock and hepatic failure may appear after apparent recovery. The pathophysiology of iron toxicity follows four stages:

Stage 1 GI stage: vomiting, diarrhea, abdominal pain, and GI pain caused by a direct injury to the mucosa of the stomach and gut.

Stage 2 Relative stability stage: lasts only a few hours. The patient may be acidemic and poorly perfused.

Stage 3 Shock stage: circulatory failure, profound hypotension, lactic acidosis, acidosis due to the circulating iron, and hypovolemia. This stage may result in death.

Stage 4 Hepatotoxicity stage: appears within 48 hours and may be fatal.

Iron levels < 300 µg/dL drawn 2 to 6 hours after the ingestion predicts a benign course. Levels > 500 µg/dL predict a severe course.

Initial Management

1. When the patient has severe symptoms, aggressive supportive management is needed for all of the manifestations of toxicity. Address hypovolemia and shock aggressively, obtaining frequent blood gases to guide treatment for acidosis. Large amounts of sodium bicarbonate may be needed to treat acidosis.
2. Whole bowel irrigation may be indicated.
3. After blood volume has been restored to normal, begin chelation therapy with deferoxamine. (Vol III – TOX14 IRON INGESTION)

20

Hydrocarbon Ingestions

Hydrocarbon ingestion may occur at any age, but it is (PEDS) most common in children and adolescents.

PEDS: Hydrocarbon ingestions by children vary in seriousness according to the hydrocarbon ingested. Gasoline, kerosene, lighter fluid, and many other hydrocarbons are not likely to cause serious problems, but as little as 1 mL of mineral seal oil can be fatal. The most dangerous effect is the ability of these low surface tension chemicals to spread over the moist surfaces of the airway and into the bronchial tree. This results in severe chemical pneumonitis. Suspect intoxication from the presence of characteristic odors, or in the case of paint sniffing, facial colors.

Initial Management

1. Do not administer Syrup of Ipecac and AC. Also avoid antibiotic and steroid therapy. The only treatment option is to support ventilation. Observe for at least 6 hours for any developing pulmonary problems.

21

Psychogenic Coma (Catonic State)

Psychogenic coma is a diagnosis of exclusion, which is (PEDS) virtually unheard of in small children. Patients may appear comatose with a remarkable ability to deny pain. Common clues include fluttering eyelashes in an attempt to keep eyes

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closed (despite interest in the environment) and feigned seizure activity characterized by thrashing movements rather than tonic-clonic contractions. If you have any doubts, a normal EEG confirms the diagnosis. Psychogenic coma may be an involuntary manifestation of psychiatric illness or willful malingering.

Initial Management

1. Determine that the patient is physiologically intact with normal vital signs and pupil responses.
2. The well-known ploy of dropping the patient's arm while it is positioned over his or her face (thus causing the patient to move it aside) is fairly reliable as an indication that the patient is feigning coma.
3. Over time, the patient is unable to resist "regaining consciousness"; simply waiting usually resolves the problem.

S – STROKES, SHUNTS, SHOCK (CNS INSULTS)

22

Thrombotic or Embolic Strokes (Vol III – NEU2 STROKE)

Thrombotic or embolic strokes present as an acute neurological deficit, usually without severe headache or vomiting. Strokes may cause coma depending on the size and location of the central nervous system damage and the presence and degree of increased intracranial pressure (ICP). If possible, transfer these patients to an Acute Stroke-Ready Hospital. Telemedicine and teleradiology for stroke is making it possible to deliver stroke care in regions without local stroke expertise.

Initial Management

1. Maintain and protect the airway; administer oxygen to keep O₂ sats > 94%.
2. Establish IV access and initiate cardiac monitoring. Obtain appropriate laboratory tests including blood glucose, coagulation studies, electrolytes, renal function, CBC, and cardiac markers. Obtain an ECG and CT. Only assessment of blood glucose should precede CT. **(Note make certain the patient has a patent airway before taking them to CT)**
3. Treat hypertension only if BP sustained at levels > 185/110. (Although some cases of permissive hypertension are reasonable.)
4. Determine time of symptom onset or time of "last known well." Patients with "wake-up" strokes may identify a time when they were last ambulatory. Assess neurological deficits and possible comorbidities. Evaluate patients with the National Institutes of Health Stroke Scale (NIHSS) evaluation, which is a good tool for patients with all types of stroke. **See Vol I – ACUTE CARE PORTALS NIH STROKE SCALE.**
5. A CT brain scan determines if a patient is appropriate for tPA therapy. Administer tPA as soon as possible, unless there are contraindications. The therapeutic window for treatment is up to 4.5 hours from symptom onset,

during which time exam, stabilization, CT scanning interpretation, and therapy contraindications must be assessed. After 4.5 hours, the risks from thrombolytics increase and benefits decrease. Hospital protocols should clearly define which tests must be performed before acute treatment decisions.

6. Carefully observe patients with severe stroke for signs of increased intracranial pressure. Treat cerebral edema with 20% mannitol, 250 to 500 cc IV rapid bolus. Hypertonic saline may also be used. Consult with neurosurgery.
7. If the stroke is embolic, determine source of embolism by performing an echocardiogram (either transthoracic or transesophageal).
8. **“Drip and Ship.”** If appropriate, administer tPA and transfer to an Acute Stroke-Ready Hospital. Do not delay transfer.
9. Patients over the 4.5 hour window need to be assessed if they are a candidate for mechanical retrieval. This time window can be up to 24 hours. Discuss with your referral center.

23

Intracerebral Hemorrhage

Intracerebral hemorrhage tends to produce hemispheric signs. The onset of these neurological changes is sudden and frequently accompanied by severe headache, vomiting, and changes in LOC often leading to coma.

Initial Management

1. Protect the airway (with the patient ventilated if necessary).
2. Severe hypertension may need treatment, but hypotension must be avoided. Lower severe hypertension to about 180/105; do not try to normalize. (**Vol III – CV13 HYPERTENSIVE CRISES**)
3. Obtain a head CT scan. If increased ICP is present, hyperventilate to an EtCO₂ of 30 and administer mannitol 1 g/kg IV or hypertonic saline. Consult early with neurosurgery about using hypertonic saline. (**Vol III – NEU5 INCREASED INTRACRANIAL PRESSURE**)
4. Consider cerebellar involvement if the patient presented with or noted dizziness and truncal ataxia on ambulation at the start of symptoms. This may indicate hemorrhage into the cerebellum. This is a true emergency because unless decompressed, this mass may result in sudden death. Obtain a head CT scan and neurosurgical consult.

24

Subarachnoid Hemorrhage (**Vol III – NEU5 INCREASED INTRACRANIAL PRESSURE**)

Subarachnoid hemorrhage is more common in women overall, but under the age of 40, subarachnoid hemorrhage is more common in men. The onset is gradual or immediate with severe headache and vomiting. Frequently, there is nuchal rigidity. In cases of massive subarachnoid hemorrhage with decreased LOC, diagnose by CT scan. In as many as 10% of patients with smaller bleeds, CT scans may be negative, and a LP or cerebral angiogram is required for diagnosis.

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The most common etiology is due to a ruptured or leaking berry aneurysm. About 6% of the time, the etiology is a leaking arteriovenous malformation.

Initial Management

1. Frequently there is severe hypertension. If the patient has a systolic blood pressure > 220 torr or a diastolic pressure > 120 torr, carefully lower pressure with labetalol 20 mg IV every 10 to 20 minutes. For patients with diastolic pressure >140 torr, use a Nicardipine drip.
2. Seizures must be prevented. Administer a loading dose of Keppra or phenytoin load 15 to 20 mg/kg at a rate no faster than 50 mg/min or administer fosphenytoin 18 mg PE/kg IV over 10 minutes. **(Vol III – NEU1 STATUS EPILEPTICUS)**
3. Treat nausea and vomiting with an antiemetic such as ondansetron 4 mg slow IV push or prochlorperazine 5 mg slow IV push.
4. Nicardipine (a Ca ++ channel blocker) 5mg/hr., titrate up 2.5 mg every 5 - 15 minutes to a max of 15 mg/hr.
5. Consult with a neurosurgeon.

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Subdural Hematoma

Subdural hematoma is usually caused by venous interruption beneath the dura with the blood spreading out under the dura. Typical history is that of trauma with temporary loss of consciousness with residual headache, personality change, or abnormal neurologic findings. The subdural hematoma may be (1) acute with deteriorating LOC and neurologic status within 24 hours of the injury, (2) sub-acute with symptoms apparent in 2 to 14 days of the injury, or (3) chronic form with symptoms recognized > 14 days after the injury. **PEDS: Consider a diagnosis of subdural hematoma in infants and children when abuse is a possibility.**

Initial Management

1. The airway must be carefully managed.
2. If increased ICP is present, determine the cause and treat the ICP.
3. Consult with a neurosurgeon.

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Epidural Hematoma (Vol II – DISAB SKILLS 1 SKULL TREPHINATION)

Epidural hematoma is classically suspected in a patient who sustains head trauma, has a short period of unconsciousness, and then appears normal. After a period of minutes to hours, the patient develops signs and symptoms of increased ICP due to the arterial bleeding causing an extra-dural hematoma, which bulges inward. The patient develops decreased level of consciousness, pupillary changes, and often a hemiplegia.

Initial Management

1. Protect the airway and ventilate if needed.
2. Rapid evacuation of the epidural hematoma with a trephine may be lifesaving.

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Increased Intracranial Pressure

Increased ICP from any origin causes a decrease in cerebral perfusion pressure (the difference between ICP and the mean blood pressure), resulting in a decrease in cerebral blood flow leading to cerebral anoxia. Initially the patient becomes drowsy but with progressive increase in the ICP. If left untreated, herniation syndromes (uncal or transtemporal) lead to irreversible brain stem damage. Suspect increased ICP in comatose patients with hypertension, widened pulse pressure, and abnormal respiration pattern. Third nerve findings with pupillar dilatation and sluggish reactivity progresses to hemiparesis, decorticate posturing, decerebrate posturing, and finally, apnea, hypotension and cardiac arrest.

Initial Management

1. Secure the airway (**Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION**); continue sedation.
2. Consult with a neurosurgeon about whether to use mannitol or hypertonic saline in a patient with increased intracranial pressure. (**Vol III – NEU4 PHENYTOIN AND FOSPHENYTOIN LOADING**)
3. Give prophylactic anti-seizure medication.
4. Treat the underlying cause for the increased ICP.
5. If uncal herniation is occurring despite these measures, consider/ perform emergency trephination on the side of the dilated pupil. (**Vol II – DISAB SKILLS 1 SKULL TREPHINATION**)
6. Obtain CT scan (if feasible/stabilized), and consult neurosurgery. (**Vol III – NEU5 INCREASED INTRACRANIAL PRESSURE**)

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Shock

Shock of any origin results in a decrease in cerebral blood flow, cerebral anoxia, and decreased LOC.

Initial Management

1. Maintain the airway and ventilate with oxygen if needed.
2. Identify and treat the cause for shock. (**Vol I – ACUTE CARE PORTALS SHOCK ACRONYM – SHRIMPCAN; Vol III – CV17 SHOCK**)

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Shunt Malfunction

If a ventriculoatrial or ventriculoperitoneal shunt is present under the scalp in

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the setting of coma or decreased LOC, immediately determine the patency of the shunt. To check for shunt patency in shunts with one palpable chamber, occlude the tubing proximal to the chamber with one finger and press on the chamber. If the chamber empties, the shunt is open distally. Now release finger pressure on the occluded tubing, and the chamber should fill again. If it does not, there is proximal obstruction. Family members can usually describe the procedure used to clear an obstruction.

If two chambers are palpable, occlude the proximal chamber with finger pressure while pressing on the distal chamber. If the distal chamber does not empty, there is distal obstruction of the shunt. Now release finger pressure on the proximal chamber while maintaining pressure on the distal one. If the proximal chamber does not fill, there is a proximal obstruction.

In cases of impending herniation or suspected infection, shunt chambers may be aspirated (and cultured). **PEDS:** In children, the ventricle itself may be aspirated. Consult a neurosurgeon before undertaking these procedures.

If the patient is stable, obtain a CT scan, looking for evidence of acute hydrocephalus/increased ICP.

If a herniation syndrome is in progress with deepening coma and posturing, move ahead and puncture into the dilated ventricle using a 20-gauge needle through or alongside the tubing through the brain. **PEDS:** In a child with a shunt entering through the anterior fontanelle, direct the needle toward the inner canthus of the ipsilateral eye from the edge of the fontanelle. Stop when cerebrospinal fluid (CSF) returns. Leave the needle in place for continued decompression until seen by a neurosurgeon. Ascending infection is a concern for patients with shunts. Obtain neurosurgical consultation whenever patients exhibit signs and symptoms suggestive of meningitis.

A – ALCOHOL (INTOXICATION, WITHDRAWAL, TOXIC EFFECTS)

30 Ethyl Alcohol Intoxication

Ethyl alcohol (ETOH) intoxication may be life threatening because of respiratory depression and airway compromise, particularly with vomiting. While alcohol intoxication is a common cause of decreased LOC, the presence of ETOH does not rule out other coinciding conditions. Chronic alcoholic patients may demonstrate a variety of disorders besides intoxication that decrease their LOC. **PEDS:** In children and adolescents, ETOH may cause hypoglycemia. These patients need careful observation and serial exams throughout their course of stay.

Blood alcohol levels are helpful but are not well correlated with the degree of decreased LOC. Chronic alcoholics may appear relatively normal with high

blood alcohol levels, **(PEDS)** while children and adolescents may be deeply comatose with lower levels. Treatment is generally supportive in pure ETOH intoxication, with special attention to protect the airway and ensure ventilation. Rule out head trauma. A head CT may be indicated.

31 Ethyl Alcohol Withdrawal

A patient in ethyl alcohol (ETOH) withdrawal presents with delirium, tremors, elevated heart rate and/or BP, and sometimes elevated temperature or seizures. Severe ETOH withdrawal can be a life-threatening condition. Benzodiazepines are the chemical mainstay of withdrawal management: Give diazepam 5 to 10 mg IV bolus every 5 to 15 min until sedated. **PEDS:** 0.05 to 0.3 mg/kg/dose IV (not to exceed 5 mg/dose) over 2 to 3 min every 15 to 30 min until sedated. Lorazepam may also be considered. Aggressive supportive measures and adjunctive treatment with magnesium and thiamine replacement are often required. **(Vol III – TOX9 ALCOHOL WITHDRAWAL)**

32 Methanol Intoxication **(Vol III – TOX10 TOXIC ALCOHOLS)**

Methanol is commonly found in windshield washer fluid. Methanol intoxication mimics ethanol intoxication, but abdominal pain and visual disturbances are frequent. Blindness and coma can result. The laboratory test for blood alcohol is specific for ethanol so consider methanol intoxication if the patient seems more inebriated than the blood alcohol level suggests. An osmolal gap and anion gap acidosis may be prominent diagnostic clues.

Initial Management

1. Begin treatment without laboratory confirmation in methanol ingestion.
2. Consider administration of fomepizole (Antizol or 4-MP), if available. Dose: Loading dose 15 mg/kg IV over 30 minutes, then 10 mg/kg every 12 h x 4 doses, then 15 mg/kg every 12 h. **PEDS:** Suggested doses are the same.
3. Administer thiamine 50 to 100 mg IV every 6 hours. **PEDS:** 0.25 to 0.50 mg/kg IV in first 24 h
4. For methanol ingestion administer folate 50 mg IV every 4 hours.
5. As second-line therapy, administer 125 mL of 50-proof (50%) ethanol by nasogastric tube to provide ethanol to compete with the methanol for alcohol dehydrogenase in the liver. This enzyme converts methanol into the toxic metabolites, formaldehyde, and formic acid. Continue running fusion of 20 mL/h. Ethanol IV may be needed. If administered IV, use 10% ETOH in D₅W: 10 cc/kg over 30 to 60 minutes as IV loading dose, then 1 to 2 cc/kg/h to maintain blood level of 100 to 150 mg/dL.
6. Hemodialysis will correct electrolyte abnormalities and clear parent compound of the toxic alcohol. Hemodialysis is recommended for patients

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- who have severe electrolyte derangements, anuria, or those who have a methanol level of >50 mg/dL.
7. Also give pyridoxine 50 to 100 mg IV every 6 h. **PEDS: 1 to 2 mg/kg IV in first 24 h.**
 8. Contact a toxicology consultant or poison control center early.

33 Ethylene Glycol Intoxication

Ethylene glycol intoxication is similar to methanol intoxication in presentation and management. **PEDS: Unfortunately, ethylene glycol is found in antifreeze solution, a sweet-tasting substance that may attract children** as well as adults looking for an ethanol substitute. Like methanol, the metabolites are formaldehyde and formic acid. Ethanol competes for the enzyme system that produces these toxic metabolites.

An osmolol gap and anion gap acidosis are prominent. Antifreeze solutions usually contain fluorescein that can be detected in the urine with ultraviolet light.

Initial Management

1. Treat the patient in exactly the same way as for methanol ingestion.
2. Hemodialysis and IV ethanol may be needed.

34 Isopropanol Intoxication

Isopropyl alcohol (rubbing alcohol) is an intoxicant more potent but similar in effect to ethanol. In large doses or (**PEDS**) young children, isopropyl alcohol may cause coma, acidosis, and hypoglycemia. Isopropanol produces hemorrhagic gastritis with abdominal pain and sometimes bleeding. Treatment is supportive, with emergent hemodialysis for blood levels > 400 mg/dL or hypotension refractory to fluids and pressors.

E – EPILEPSY, ENCEPHALOPATHY (HYPERTENSIVE CRISIS, HEPATIC)

35 Status Epilepticus (Vol III – NEU1 STATUS EPILEPTICUS)

Status epilepticus is defined as a seizure lasting 30 minutes or two seizures occurring sequentially without an intervening period of normalcy. To fit the criteria, the seizures may be grand mal, partial, or even non-convulsive.

Initial Management

1. Apply oxygen. Suction the mouth if needed and insert an oral airway if possible.

2. If the patient is not breathing adequately, ventilate with a BVM. Airway management by intubation, usually with RSI, may be needed either for status itself or for apnea in the post-ictal period. Remember that a patient paralyzed from neuromuscular blockers may still have deleterious electrical seizure discharges occurring in the brain, and needs immediate anticonvulsant therapy.
3. If possible, establish an IV.
4. If IV access is difficult, consider an IO or give midazolam 0.2 mg/kg IM or IN (5 mg/mL concentration) to a maximum of 10 mg or 0.2 mg/kg IV or IN – max 10 mg (5 mg/mL concentration) for both adult and pediatric patients.
5. If an IV or IO has been established, the initial treatment of choice is a benzodiazepine such as lorazepam: 4 mg IV or IO over 2 to 5 min; repeat in 10 to 15 min prn. If ineffective, levetiracetam (Keppra), valproic acid, phenytoin/ fosphenytoin, phenobarbital, or other agents may be used. (**Vol I ACUTE CARE PORTALS, STATUS EPILEPTICUS TREATMENT PLAN.**)
6. If the seizure continues, consider etiologies such as hypoglycemia, hyponatremia, hypernatremia, hypocalcemia, hypomagnesemia, pyridoxine deficiency, and isoniazide toxicity. In adults, administer thiamine 100 mg IV if alcoholism or malnutrition is a possibility.
7. Terminate the seizures of hypoglycemia in adults with 50% dextrose 0.5 to 1.0 g/kg IV. **PEDS:** In infants and small children, give 2 to 4 mL/ kg of D₂₅ IV; Neonatal: 2 to 4 mL /kg of D₁₀; Premature Neonates: 1 to 2 mL /kg of D₁₀
8. Terminate the seizures of hyponatremia with 6 mL/kg of 3% NaCl.
9. Terminate the seizures of hypocalcemia with a 5 to 10 mL slow push of a 10% solution of calcium chloride and (**PEDS**) 0.2 mL/kg up to 10 mL in children.
10. Terminate the seizures of hypomagnesemia with 4 grams of magnesium sulfate IV in adults and (**PEDS**) 50 mg/kg in children up to 4 grams over 10 minutes.
11. If the patient is taking isoniazide (INH) or has access to isoniazide, administer 1 gram of pyridoxine (vitamin B6) IV for each gram of INH ingested. Otherwise, give 50 to 100 mg pyridoxine IV in case the patient is deficient.
12. Paralysis and anesthesia may be required for very resistant cases.
13. A seizure is often a symptom of some other illness, such as meningitis or drug or substance overdose. Once the seizure has stopped, repeat this pathway to bring all possible causes to mind.

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Hypertensive Encephalopathy (**Vol III – CV13 HYPERTENSIVE CRISES**)

Hypertensive encephalopathy (hypertensive crisis) is caused by cerebral hyperperfusion with loss of normal autoregulation of arterial blood flow. The blood-brain barrier is compromised, resulting in fluid movement into the brain. Patients exhibit lethargy, confusion, and even coma. These patients usually have a history of recently untreated hypertension with an onset of headache, nausea, vomiting, visual changes, and obtundation. BP is usually in the 250/150 range,

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although the absolute value of the BP does not determine the emergency approach but rather the elevated BP and the patient's CNS symptoms. Besides altered LOC, there may be retinal changes, neuro findings (focal signs, reflex changes, nystagmus), or seizures. However, it is the evidence of end-organ injury that makes the diagnosis and not the blood pressure measurement per se.

Initial Management

1. Administer oxygen and protect the airway if necessary.
2. Start an IV and obtain an ECG.
3. Begin a nitroprusside drip 50 mg in 250 mL D₅W at a rate of 0.5 µg/kg/min. Increase this rate gradually to achieve the desired blood pressure. Aim to reduce the mean arterial pressure (MAP) by 20% to 25% in 30 to 60 minutes.

$$\text{MAP} = \text{diastolic BP} + \frac{1}{3} (\text{systolic BP} - \text{diastolic BP})$$

Lowering the blood pressure too much and too fast results in cerebral ischemia.

4. Insert a urinary bladder catheter. Monitor urine output. Some hypertensive patients are volume depleted and need fluids to protect the kidneys from pre-renal failure.

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Hepatic Encephalopathy

Hepatic encephalopathy is seen as a precipitating event in patients with severe (usually chronic) liver disease. The liver is unable to detoxify CNS toxins. Signs and symptoms of liver disease are usually present, such as asterixis, ascites, and jaundice. The precipitating events are varied: GI bleed, sepsis, CNS depressants, azotemia, hypoxia, and others. LOC ranges from confusion to coma. Besides the considerable support necessary in these complex patients for coexisting problems, specific initial treatment consists of correcting /eliminating the precipitating factors and giving lactulose: initial dosing 30 to 50 mL PO every hour until diarrhea OR if unable to give PO, give 300 mL in a 700 mL water retention enema. Consider giving Neomycin 1 g PO.

I—INSULIN AND OTHER HORMONES

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Diabetic Ketoacidosis (Vol III—END/M2 DIABETIC KETOACIDOSIS)

Diabetic ketoacidosis (DKA) is usually exhibited by nausea, vomiting, abdominal pain, dehydration, hypotension, tachycardia, hyperventilation (or Kussmaul's respirations), and a decreased LOC. The breath may smell of acetone. The blood glucose level is > 300 mg/dL, and the serum bicarbonate level is < 15 mEq/L with an anion gap. The patient is usually a known diabetic. **PEDS:** This is not necessarily true in children. Presentations may be severe or mild.

Initial Management

1. Administer oxygen and, if the airway is not protected, intubate the patient. If the patient has been dependent on hyperventilation to compensate for the metabolic acidosis, continue that on the ventilator to avoid inducing acidosis rapidly.
2. Begin a fluid infusion of crystalloid (NS or LR), starting with 1 liter in the first 30 minutes in adults. For patients in hypovolemic shock, administer fluid as rapidly as possible. For others, or when shock is resolved, continue fluid replacement at approximately 1 L/hour, following vital signs, electrolytes, and blood sugar levels. **PEDS:** In children, start with 10 mL/kg boluses if patient is in shock. Give these rapidly until the shock is corrected. Afterward, or if patient is not in shock, give 10 mL/kg crystalloid over an hour; then at 1.5 times maintenance. If transferring a patient, contact the receiving endocrinologist for further instructions. If more fluid loading is needed in order to avoid hyperchloremic acidosis, opt for LR. Once patient is hemodynamically stabilized, switch fluids to ½ NS. Once blood sugars reach the 200 to 300 mg/dL range, switch to glucose-containing solutions. Five liters may be required to achieve rehydration in adults. The single most important therapy for DKA is volume restoration.
3. Even if the serum potassium level is normal in the beginning, total body potassium is low. Monitor the serum potassium hourly, and watch the ECG monitor for signs of hypokalemia. Add KCL to IV.
4. After rehydration is underway and hypokalemia is corrected, begin a low-dose insulin drip (add 100 units of regular insulin to 100 mL crystalloid) of 5 to 10 units of regular insulin per hour (**PEDS:** 0.05 units/kg/h in children) and continue until the acidosis and ketonemia have cleared. Note: If you are going to transfer a child immediately, do not start insulin. **PEDS:** In pediatric patients, blood glucose levels will decrease with fluids alone and may fall too low during transport if insulin is given as well.
5. Because infection is a common cause for DKA, conduct a thorough search for signs of infection, including a chest x-ray, urinalysis, and a neurological exam. Myocardial infarction can also precipitate DKA. Please note that pregnancy is also a source for DKA

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Non-Ketotic Hyperosmolar Coma (Vol III – END/M5 HYPEROSMOLAR STATE)

Non-ketotic hyperosmolar coma occurs when the blood glucose level is high in the absence of ketoacidosis. It occurs in diabetics and is much less common than DKA. The blood glucose often is > 800 mg/dL. These high levels produce hyperosmolarity, dehydration, stupor, and focal or grand mal seizures. Total body potassium deficiency is usually severe.

Initial Management

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1. Administer oxygen and, if the airway is not protected, intubate the patient.
2. Begin a fluid infusion of NS with 1 liter in the first 30 minutes in adults and **(PEDS) 20 mL/kg in children**. If shock is present, 5 or more liters may be required to restore an adequate blood volume. **PEDS: Repeated boluses of 20 mL/kg NS may be necessary in children.**
3. As with DKA, begin potassium replacement early and follow the ECG monitor and serum potassium closely.
4. Begin a low-dose insulin drip as with DKA and continue this until the blood glucose is < 300 mg/dL. Begin adding glucose to the IV when the blood glucose is < 250 mg/dL.

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Hypoglycemia

Hypoglycemia may have many causes, but by far the most common cause is insulin reaction. Patients may exhibit coma, seizures, stupor, hemiplegia, sweating, hyperventilation, inappropriate behavior, and hypothermia. Check blood glucose levels in any patient with an altered mental state. The blood glucose is usually < 50 mg/dL in these cases.

Initial Management

1. Administer oxygen and protect the airway.
2. Start an IV and administer 50 mL of a 50% glucose solution in adults and older children. **PEDS: In children, use a 25% solution at 0.5 to 1.0 g/kg (2 to 4 mL/kg).** In infants, use 10% dextrose at 2 to 4 mL/kg IV.

Hypoglycemia has already been addressed under DONT. Search for the underlying cause of hypoglycemia.

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Ketotic Hypoglycemia

Ketotic hypoglycemia (PEDS) in children results from an inborn error of metabolism that affects children in the age range of 18 months to 5 years. The attacks of hypoglycemia are sporadic and tend to occur in the morning.

Initial Management

These usually respond to the oral administration of glucose.

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Addisonian Crisis (Vol III – END/M1 ADRENAL CRISIS)

Addisonian crisis (adrenal deficiency) occurs when infection or trauma stresses a patient with adrenal insufficiency. Commonly these patients are on chronic steroid therapy with a resulting suppression of the adrenal cortex. Other causes of adrenal insufficiency are pituitary malfunction, hemorrhage, and infection (such as tuberculosis, AIDS). The patient may be confused, weak, and hypotensive. Nausea, vomiting, and abdominal pain are common. Delirium, seizures, hyponatremia, hyperkalemia, and hypoglycemia may also occur.

Initial Management

1. When a patient's history suggests this diagnosis, administer 100 mg of hydrocortisone IV (**PEDS: 2 mg/kg in children**). Consider this diagnosis in any patient with unexplained hypotension. Repeat this dose every 8 hours under stressful circumstances.
2. Give a 1 to 2 liter NS bolus to adults and (**PEDS**) 10 to 20 mL/kg to children.

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Thyroid Storm (Vol III – END/M4 THYROID STORM)

Thyroid storm is a rare, life-threatening event in untreated or partially treated thyrotoxic patients with a precipitating event or physiologic stress. Altered LOC ranges from mania to coma. Other clinical aspects include hypertension, tachycardia, fever, nausea, vomiting, diarrhea, dehydration, tremor, and thyroid enlargement. No laboratory tests are immediately available to help in the diagnosis. The physician has to base treatment solely on clinical grounds.

Initial Management

1. Administer oxygen and protect the airway.
2. Rehydrate with glucose-containing fluids.
3. Initial management is cooling. (See hyperthermia; do not use salicylates.)
4. Give sedation, electrolyte replacement, and hydrocortisone 100 mg IV
5. As initial therapy, give the beta blocker propranolol IV or PO, depending on severity, at 0.5 to 1.0 mg/min with continuous cardiac monitoring. Repeat subsequent doses of 2 to 3 mg IV every 15 minutes until the patient is adequately beta blocked as indicated by reduction of symptoms or until 5 to 10 mg have been given. The cardiac effects should be apparent in 10 minutes with slowing of the heart rate. When there is high-output cardiac failure, be careful not to depress myocardial contractility to the point that cardiac failure worsens despite the slower heart rate.
6. Give propylthiouracil 200 to 400 mg PO/NG/PR followed by iodide 250 to 500 mg PO or IV 1 h after propylthiouracil. Aggressively search and treat the precipitating factor(s).

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Myxedema Coma (Vol III – END/M3 MYXEDEMA COMA)

Myxedema coma occurs in chronically hypothyroid patients who are subjected to stress. Exposure to a cold environment, pneumonia, and heart failure are the most common stressors. When a history of hypothyroidism is not available, diagnosis may be difficult. The characteristic thin hair, puffy face, large tongue, and edematous skin may or may not be present. Lab tests are not immediately helpful. Myxedema coma is more common in women than in men, and half of the patients are between the ages of 60 and 70 years. Hypothermia is a common finding. Core temperature may be 29.5°C (85°F) or less. Respiratory failure, hypotension, bradycardia, cardiomegaly, seizures, coma, psychosis, paralytic ileus with acquired megacolon, and dilutional hyponatremia may be present.

Because the diagnosis is often one of exclusion, attempt to correct each aspect of the patient's problems using conventional therapies before administering thyroid hormone replacement.

Initial Management

1. Administer oxygen and protect the airway. Ventilate the patient if respiratory failure is present.
2. Administer hydrocortisone 100 mg IV to compensate for possible adrenal insufficiency.
3. Seizures due to hyponatremia can be terminated with 6 mL/kg of 3% NS solution.
4. Hypotension may be resistant to vasopressors, even when blood volume is expanded.
5. Re-warm the patient using non-invasive means.
6. When conventional measures fail to improve the patient, consider thyroid hormone replacement. The dose of thyroxine used can be fatal if given to a euthyroid patient. Give initial loading dose of L-T4 hormone 200 to 300 µg IV followed 24 hours later with a second dose (100 µg) IV.
7. Concomitantly, give L-T3 hormone 10 µg IV every 8 hours until the patient is stable and conscious.
8. Consult an endocrinologist regarding this dosing and subsequent doses.

O – OXYGEN, OPIATES, AND OTHER DRUGS OF ABUSE

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Hypoxia

Always consider hypoxia in the comatose or stuporous patient. Oxygen administration should be an automatic response. Oxygen saturation monitoring is an invaluable aid in identifying the hypoxic patient, bearing in mind that oxygen saturation may be normal in patients suffering from carbon monoxide

poisoning. For a discussion of respiratory failure, see [Vol III – PATHWAY 6 ADULT RESPIRATORY, PATHWAY 7 PEDIATRIC RESPIRATORY](#).

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High Altitude Illness

High altitude illness includes acute mountain sickness, high altitude pulmonary edema, high altitude cerebral edema, transient cortical blindness, and high altitude edema. ([Vol III – ENV5 HIGH ALTITUDE ILLNESS](#))

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Opiate/Heroin/Narcotic Overdose ([Vol III – TOX12 NARCOTIC OVERDOSE](#))

Opiate/heroin/narcotic overdose patients exhibit constricted pupils, coma or stupor, and respiratory depression. Needle tracks are usually present on the forearms of chronic abusers.

Initial Management

1. Naloxone: Give 0.4 to 2 mg IV, IM, IO; or give 0.1 mg/kg IN. Naloxone may precipitate withdrawal, so restrain the patient prior to administration
2. The patient may need resuscitative measures (such as airway management or IVs), despite the administering naloxone.
3. The duration of action of naloxone is only about 2 hours, so further doses of naloxone may be needed, and may need to consider the need for a naloxone drip. (**Consult Poison Control**)

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Cocaine Ingestion ([Vol III – TOX 11 COCAINE INGESTION](#))

Cocaine ingestion produces mental status changes, pulmonary edema, tachycardia, hypertension, and hyperthermia. You may also observe mydriasis and diaphoresis. Coronary artery vasoconstriction may result in myocardial ischemia and infarction. Dysrhythmias are common. Intracranial hemorrhage and seizures are sometimes seen. Rhabdomyolysis may result in acute renal failure.

Initial Management

1. Administer oxygen and protect the airway if needed. ET intubation may be required. Be wary of using succinylcholine because of possible potassium release, secondary rhabdomyolysis.
2. Volume loading with NS restores normal blood volume and increases urine output, protecting the kidneys from damage secondary to the rhabdomyolysis. If the urine does not clear rapidly, add sodium bicarbonate at 1 mg/kg IV to alkalinize the urine.
3. Treat hypertension and tachycardia with diazepam, 5 to 10 mg IV prn, titrated for effect. Diazepam at a dose of 10 to 20 mg IV may also be used to terminate seizures.

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4. Consider activated charcoal for oral ingestions.

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Amphetamine Intoxication

Amphetamine intoxication results in the same clinical picture as does cocaine intoxication. The toxic effects are virtually the same, so follow the guidelines listed for cocaine. (**Vol III – TOX 11 COCAINE INGESTION**)

50

Phencyclidine Intoxication

Phencyclidine (PCP) intoxication produces frightening and threatening hallucinations. Agitation and violent behavior are major problems. Vertical and horizontal nystagmus is common. Seizures, coma, muscle rigidity, hypertension, rhabdomyolysis, and hyperthermia are present in severe cases. Treat these problems as described for cocaine intoxication. (**Vol III – TOX 11 COCAINE INGESTION**)

Initial Management

1. Administer oxygen and, if necessary, use RSI (**Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION**) to obtain oral tracheal airway. Avoid the use of succinylcholine if rhabdomyolysis is probable.
2. Use diazepam 5 to 10 mg IV or lorazepam 1 to 2 mg IV to sedate the patient. If this fails, use haloperidol 5 mg IM or IV.

U – UREMIA AND OTHER METABOLIC ABNORMALITIES

51

Acute Renal Failure

Acute renal failure results in coma if a rapid rise in BUN causes an increase in serum osmolality. The brain is unable to adjust to this change.

Initial Management

1. Administer oxygen and protect the airway.
2. Always consider that the patient may have pre-renal failure secondary to hypovolemia. Clinically rule this out by history or examination. When in doubt, administer a fluid bolus to improve urine output and to correct metabolic abnormalities.
3. Hyperkalemia is a great danger and may result in cardiac dysrhythmia. This needs to be checked for and treated if present.

52 **Electrolyte Abnormalities**

Electrolyte abnormalities such as hyponatremia, hypernatremia, hypercalcemia, and hyperkalemia may be manifested as decreased level of consciousness. These must be evaluated for and treated if present. (Vol III – END/M7 DISORDERS OF ELECTROLYTE CONCENTRATION)

53 **Acid-Base Abnormalities**

Acid-base abnormalities especially respiratory acidosis with hypercapnia may result in decreased level of consciousness. This needs to be checked for and treated if possible. (Vol III – END/M6 ACID-BASE)

Complete the thorough physical examination.

Caveats

No one can know everything about toxicology and critical care medicine. Consult early and freely with colleagues.

Do not start vasopressors for hypotension until the maximum effect of volume restoration has been achieved.

The keys to resuscitation are good airway management (Vol II – AIR SKILLS 1 AIDS TO INTUBATION) and good blood volume management. (Vol III – CV17 SHOCK)

**ADULT AND PEDIATRIC
ALTERED LEVEL OF CONSCIOUSNESS
PATHWAY 1**

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August 2017

FOCUSED CLINICAL PATHWAYS

PATHWAY 2: CARDIOVASCULAR EMERGENCIES (ADULT AND PEDIATRIC)

The team continues the resuscitation along the pathway suggested by the initial clinical impression. Each pathway includes a complete, thorough, and rapid physical examination with additional history taking. The team leader is wary of conditions that may not be apparent. To obtain additional clinical data or to correct a missed or newly developed condition, the team leader repeats the initial survey if the patient is not responding satisfactorily.

Text Number	Diagnosis/ Condition	Related Materials
1	CALS Initial Approach	Vol I – ACUTE CARE PORTALS
2	Automated External Defibrillator	Vol I – ACUTE CARE PORTALS
3	Cardiac Arrest	Vol I – ACUTE CARE PORTALS
4	VF/Pulseless VT	Vol I – ACUTE CARE PORTALS; Vol III – CV5 VENTRICULAR FIBRILLATION/ PULSELESS VENTRICULAR TACHYCARDIA
5	Pulesless Electrical Activity	Vol I – ACUTE CARE PORTALS; Vol III – CV6 PULSELESS ELECTRICAL ACTIVITY
6	Asystole	Vol I – ACUTE CARE PORTALS; Vol III – CV7 ASYSTOLE
7	Unstable Rhythms	Vol I – ACUTE CARE PORTALS
8	Bradycardia	Vol I – ACUTE CARE PORTALS; Vol III – CV10 BRADYCARDIA
9	Transcutaneous Pacing	Vol III – CV10 BRADYCARDIA
10	Tachycardia	Vol I – ACUTE CARE PORTALS; Vol III – CV8 TACHYCARDIA
11	Atrial Fibrillation/Flutter	Vol I – ACUTE CARE PORTALS; Vol III – CV8 TACHYCARDIA
12	Narrow Complex Tachycardia	Vol I – ACUTE CARE PORTALS; Vol III – CV8 TACHYCARDIA
13	Wide Complex Tachycardia	Vol I – ACUTE CARE PORTALS; Vol III – CV8 TACHYCARDIA

**ADULT AND PEDIATRIC
CARDIOVASCULAR EMERGENCIES
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14	Ventricular Tachycardia	Vol I – ACUTE CARE PORTALS; Vol III – CV8 TACHYCARDIA, CV5 VENTRICULAR FIBRILLATION/PULSELESS VENTRICULAR TACHYCARDIA
15	Long QT Syndrome (LQTS)	Vol III – CV15 LONG QT SYNDROME
16	Tachycardia in Infants or Small Children	
17	Cardioversion	Vol I – ACUTE CARE PORTALS; Vol III – CV9 ELECTRICAL CARDIOVERSION
18	Undefined Chest Pain	
19	Acute Coronary Syndrome	Vol III – CV11 ACUTE CORONARY SYNDROME
20	Hypotension/Shock	Vol III – CV17 SHOCK
21	CHF/Pulmonary Edema	Vol III – CV12 ACUTE HEART FAILURE
22	Undefined Cardiomyopathy	
23	Digitalis Toxicity	Vol III – CV14 DIGITALIS TOXICITY
24	Hypertensive Crisis	Vol III – CV13 HYPERTENSIVE CRISES
25	Thoracic Aorta Dissection	
26	Cardiac Tamponade	
27	Tension Pneumothorax	Vol I – PATHWAY 6, ADULT RESPIRATORY; Vol II – BREATH SKILLS 5 NEEDLE THORACOSTOMY
28	Pulmonary Embolism	Vol I – PATHWAY 6, ADULT RESPIRATORY, #10
29	Air Embolism	
30	Leaking Abdominal Aortic Aneurysm (AAA)	Vol I – PATHWAY 3 GASTROINTESTINAL/ABDOMINAL EMERGENCIES, #6

31	Implanted Cardioverter/ Defibrillator (ICD)	
32	Congenital and Acquired Heart Defects	
33	Kawasaki Disease	
34	Stroke (CVA)	Vol III – NEU2 TREATMENT OF STROKE
35	Brief Loss of Consciousness - Syncope	Vol I – PATHWAY 1 ALTERED LEVEL OF CONSCIOUSNESS

Please Note: Comments, directions, and instructions specific to pediatric patients are prefaced throughout the manual by PEDS: The pertinent text is underlined.

PEDS: Pediatric considerations. Primary cardiac and vascular emergencies in children are rare. Coronary artery disease, except for in Kawasaki disease, is rare. Congenital heart disease is uncommon, and an emergency case is rare. Cardiac dysrhythmias do occur in children and are discussed wherever appropriate.

Children are not small adults. This needs to be stressed whenever discussing the care of children. Emergency care teams may encounter children needing emergency resuscitation infrequently. The ABCs are still the most important aspect of any resuscitation, but special aspects of the physiology and anatomy of children need to be recognized.

1

CALS Initial Approach to Critically Ill or Injured Patients (Vol I – ACUTE CARE PORTALS)

Follow an organized approach to potential serious cardiovascular illness. The resuscitation team should remain vigilant for possible unrecognized problems that may make resuscitation difficult or impossible. Approaching all patients with the CALS Universal Approach helps the team remain organized and ensures that appropriate resources are utilized in a systematic manner.

2

Automated External Defibrillator (Vol I – ACUTE CARE PORTALS)

When unresponsive patients are encountered and advanced life support equipment and/or personnel are not available, activate the 911 emergency system immediately and obtain an automated external defibrillator (AED), if available. Then check the patient's pulse and signs of circulation (breathing,

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moving, or coughing). If the patient has no pulse, breathing, or signs of life, initiate CPR with a compression rate of 100 - 120/min, 30 compressions to 2 ventilations. **PEDS: For 2-rescuer CPR only, administer 15 compressions to 2 ventilations.** As soon as the AED is available:

1. Turn on the AED.
2. Attach the AED electrodes – right upper sternal border and cardiac apex.
3. Analyze the patient’s rhythm.
4. Follow the directions of the AED and the AED Algorithm.

3

Advanced Life Support - Initial Approach to Cardiac Arrest¹

In the setting of a cardiac arrest, follow basic life support protocols until a defibrillator and advanced airway equipment are available. Once the monitor and defibrillator are available, rapidly assess the patient’s rhythm:

- If the patient is in VF/VT, immediately defibrillate. Proceed with the VF/VT protocol if defibrillation is not successful. **(Vol I – ACUTE CARE PORTALS)**
- If the patient is not in VF/VT, introduce and secure a supraglottic airway or endotracheal airway, being sure to confirm its proper placement by two methods.
- Follow the appropriate non-VF/VT algorithm: Asystole or PEA. **(Vol I – ACUTE CARE PORTALS)**
- Potential reversible causes for the cardiac arrest must be sought following the **5 Hs and 5 Ts:**

Hypovolemia	Tablets/Toxins (drug overdose)
Hypoxia	Tamponade (cardiac)
Hydrogen ion - acidosis	Tension pneumothorax
Hyper-/Hypokalemia (or other metabolic)	Thrombosis (coronary)
Hypothermia	Thrombosis (pulmonary embolism)

4

**Ventricular Fibrillation / Pulseless Ventricular Tachycardia
(Vol I – ACUTE CARE PORTALS; Vol III – CV5 VENTRICULAR FIBRILLATION/
PULSELESS VENTRICULAR TACHYCARDIA)**

The focus of treating VF/VT is the delivery of early and, as needed, repeated shocks to convert the arrhythmia. This is supported by CPR, advanced airway management and selected medications (as needed). Appropriate use of electricity is the most effective treatment for VF/VT. The earlier the attempted defibrillation shock is delivered after the onset of the VF/VT, the greater the chance of success in converting to a perfusable rhythm. While many drugs are listed as potentially helpful in converting VF/VT, no drug has been classified higher than Class Ib, *possibly useful*.

5

Pulseless Electrical Activity (PEA)

(Vol I – ACUTE CARE PORTALS; Vol III – CV6 PULSELESS ELECTRICAL ACTIVITY)

Pulseless electrical activity (PEA) consists of observing an organized rhythm on the cardiac monitor without a detectable pulse. While the initial approach to the patient consists of basic CPR, advanced airway management, IV access, and frequent IV fluids, the ultimate success depends on the early identification and treatment of a reversible cause for the PEA.

The most frequent reversible causes are summarized by the 5 Hs and 5 Ts:

Hypovolemia	Tablets (drug overdose)
Hypoxia	Tamponade (cardiac)
Hydrogen ion – acidosis	Tension pneumothorax
Hyper-/Hypokalemia (or other metabolic)	Thrombosis (coronary)
Hypothermia	Thrombosis (pulmonary embolism)

6

Asystole (Vol I – ACUTE CARE PORTALS; Vol III – CV7 ASYSTOLE)

The initial approach to a patient in asystole consists of CPR, advanced airway management, and IV access. Seek reversible causes of asystole (Hs and Ts), and treat quickly to increase chances of success with the resuscitation. Frequently the asystole is a rhythm consistent with death rather than a treatable condition. The treating team needs to diligently consider if there is evidence that they should NOT be attempting resuscitation – as in situations of DNAR (Do Not Attempt Resuscitation) orders or signs of death. Prolonged attempts at resuscitation of a patient with persistent asystole are rarely indicated except in hypothermia or occasionally with drug overdose.

7

Unstable Rhythms (Vol I – ACUTE CARE PORTALS)

Patients may present with a wide variety of abnormal rhythms that may have varying degrees of instability or risk of deteriorating into an unstable condition (CHF, shock, hypoxia, acute coronary syndrome) or into a full cardiac arrest. Many associated clinical conditions influence the potential seriousness of the arrhythmias and the aggressiveness needed in the treatment. Some of these associated conditions that increase the risk include: the age of the patient (especially the elderly), the presence of an impaired heart (ejection fraction of the left ventricle < 40%), electrolyte abnormalities (hypokalemia, hyperkalemia, hypocalcemia, hypercalcemia), hypoxia, acidosis, hyperthyroidism, drug overdose (tricyclic antidepressants), or cocaine use. Frequently the most appropriate

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treatment of the arrhythmia necessitates the search for and treatment of the associated aggregating conditions.

8

Bradycardia (Vol I – ACUTE CARE PORTALS; Vol III – CV10 BRADYCARDIA)

Key points when evaluating a patient with bradycardia:

1. Treat the patient, not the monitor.
2. A heart rate of 65 may be a relative bradycardia if the patient's BP is low.
3. Consider the normal range of vital signs in pediatric patients.

PEDS: Normal Pediatric Vital Sign Ranges ²			
Age	Normal RR	Normal HR	Blood Pressure
Newborn to 3 months	35 to 60	85 to 205	65-90/45-65
6-12 months	30 to 60	80 to 120	80-100/55-65
1-3 years	20 to 30	70 to 110	90-105/55-70
3-6 years	20 to 25	65 to 110	90-110/60-75
6-12 years	14 to 22	60 to 100	90-120/60-75
Adult	12 to 20	60 to 100	90-120/60-80

4. Look for signs (such as decreased LOC, hypotension, or CHF) and symptoms (such as chest pain or shortness of breath) that indicate adverse clinical manifestations associated with the bradycardia.
5. Make sure that the patient's signs and symptoms are due to the slow heart rate. Shock from any cause, hypovolemia, hypoxia, or myocardial dysfunction may cause hypotension associated with a bradycardia (or a relative bradycardia for the situation), but the patient may not have primary cardiac conduction system abnormality or primary autonomic cause for the bradycardia.

Key Points to Remember in Treating Bradycardia

1. If the patient has a bradycardia or heart block and has signs and symptoms from this bradycardia, initiate and perform treatment quickly and decisively.
2. These patients' conditions may be pre-cardiac arrest and merit aggressive intervention, including a number of interventions together, such as transcutaneous pacing, atropine IV, and preparation for an epinephrine infusion.
3. In a symptomatic patient, give atropine 0.5 mg IV to a maximum dose of 0.03 to 0.04 mg/kg with dosing intervals of 3 to 5 minutes. The severity of the patient's symptoms determines the interval of dosing. Thus, use the 3-minute dosing interval in severe clinical situations. **PEDS: Use epinephrine 0.01**

mg/kg (0.1 mL/kg of a 1:10 000 solution) IV/IO. In children, bradycardias < 60 with poor perfusion are an indication for CPR.

4. Dopamine IV starting at rates of 2 µg/kg/min may be given and increased rapidly up to 10 µg/kg/min. Or epinephrine drip 2 to 10 µg/min may be used to increase the heart rate and blood pressure if other measures are ineffective.
5. In patients with severe signs and symptoms from the bradycardia, consider the immediate use of transcutaneous pacing. Do not wait for IV access or for the atropine to become effective.
6. Transcutaneous pacing may be effective but painful, and the patient may need sedation.
7. Hypovolemia or myocardial dysfunction may cause bradycardia and need to be treated.
8. The prognosis in AV block is related to the site of the infarction (anterior vs. inferior), the site of the block (intranodal [proximal or above the His Bundle] or infranodal [distal or below the His Bundle]), the nature of the escape rhythm, and the hemodynamic consequences.
9. Transplanted (denervated) hearts do not respond to atropine. The treatment of symptomatic bradycardia in these patients requires catecholamine infusion, pacing, or both.

9

Transcutaneous Pacing (Vol III—CV10 BRADYCARDIA)

Transcutaneous pacing (TCP) is a Class I intervention for all symptomatic bradycardia. It is especially useful when other forms of treatment of the bradycardia are either ineffective or will take too long to implement such that the patient becomes progressively unstable. To be effective, TCP must be begun as early as possible in the treatment sequence before the patient develops severe hypoxia or acidosis.

Indications for TCP

1. Treatment of hemodynamic significant bradycardia that has not responded to atropine or epinephrine.
2. TCP is useful as a short interval bridge until transvenous pacing can be initiated.
3. TCP can be a bridge until bradyarrhythmias from hyperkalemia or drug overdose can be reversed.
4. TCP is useful when the patient has received or will receive thrombolytic therapy, and therefore, vascular puncture for transvenous pacing is undesirable.

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Technique of TCP

1. Anterior/posterior application. Place an anterior pacing pad to the left of the sternum centered as close as possible to the point of the maximum cardiac impulse. Place the posterior pad directly behind the anterior pad to the left of the thoracic spine column. An alternative placement would be a sternum-apex position.
2. Sternal-apex chest wall pad placement includes one pad in the right upper chest and a second pad on the left lateral chest wall.
3. Place regular 3- or 4-lead ECG electrodes for monitoring the underlying rhythm.
4. Initiate TCP at a rate of 60 to 70 bpm.
5. With a non-arrest bradycardia, start at a lower power setting and slowly increase the output until achieving capture. Electrical capture usually requires 50 mA to 100 mA.
6. Determination of capture may be difficult on a routine monitor because of the wide electrical activity obliterating the cardiac electrical activity on the monitor. A filter is on the pacing monitor, such that capture can usually be determined. Capture also can be assessed by BP determination. If a peripheral pulse is to be obtained, obtain it at the right carotid or right femoral artery, so as not to confuse the jerking of muscle contraction with that of a pulse.
7. CPR may be administered directly over the insulated pacing pads while pacing is going on, without risk to the CPR operator.
8. Capture success and energy levels are related to pad placement, patient size, and body size. Large pericardial effusions may inhibit capture.
9. TCP is painful, and the patient needs analgesia in the form of narcotics and/or sedation with a benzodiazepine.

Pitfalls of TCP

1. Failure to recognize the presence of underlying treatable VF
2. Failure to recognize that the pacemaker is not capturing
3. Induction of arrhythmia, such as VF
4. Skin damage due to prolonged pacing

10

Tachycardia (Vol I – ACUTE CARE PORTALS; Vol III – CV8 TACHYCARDIA)

Key Points when Evaluating a Patient with Tachycardia

1. Does the patient have signs and symptoms (such as shock, pulmonary edema, CHF, chest pain, or decreased LOC) associated with the tachycardia?

2. Are the patient's signs and symptoms a result of the patient's tachycardia or does the patient have a serious underlying condition (such as septic shock or hypovolemia) that is causing an appropriate sinus tachycardia? Heart rates < 150 bpm usually do not cause serious signs and symptoms unless the tachycardia continues for a prolonged period of time.
3. Is the tachycardia causing such serious signs or symptoms that the patient must be immediately cardioverted, or can the patient's tachycardia be evaluated and considered for treatment with medications?
4. What is the safest way to treat the patient's tachycardia? All antiarrhythmic medications may cause a proarrhythmic effect, regardless of their Vaughn-Williams classification. The use of more than one antiarrhythmic in the same patient exponentially increases the risk of side effects.
5. What is the patient's specific rhythm that is causing the tachycardia?
 - Is the rhythm sinus tachycardia? If so, determine the cause of the sinus tachycardia and treat the underlying cause.
 - Is the basic rhythm atrial flutter or atrial fibrillation?
 - Is the narrow complex supraventricular tachycardia due to abnormal automaticity as in ectopic atrial tachycardia or multifocal atrial tachycardia (MAT) or due to re-entrant circuits as in AV nodal re-entrant tachycardia (AVNRT) or AV re-entry tachycardia (AVRT)?
 - Is the tachycardia of supraventricular or ventricular origin? Treat a wide complex tachycardia as ventricular tachycardia until proven otherwise.
 - Does the patient have an accessory AV pathway as seen in Wolff-Parkinson-White (WPW) syndrome?
 - If the patient is in ventricular tachycardia, is the ventricular tachycardia monomorphic or polymorphic?
 - If the ventricular tachycardia is polymorphic, is it associated with a prolonged baseline QT interval or represent torsades de pointes?
6. Is the patient's left ventricular function significantly impaired (ejection fraction < 40% or signs of CHF)? Antiarrhythmic medications must be used with caution in patients with depressed left ventricular function or overt CHF. Many of the antiarrhythmic medications further depress the left ventricular function and thus worsen or precipitate CHF. Amiodarone is now often the first agent to be used to treat tachycardias due to its lesser negative inotropic effect and its broad antiarrhythmic activity. In many situations, if amiodarone fails to control the tachycardia, early electrical cardioversion should be the next intervention.
7. Does the patient have an identifiable cause for the arrhythmia such as hypokalemia, hypoxia, acute MI, or CHF?

11

Atrial Fibrillation – Atrial Flutter
(Vol I – ACUTE CARE PORTALS; Vol III – CV8 TACHYCARDIA)

For patients with atrial fibrillation/atrial flutter, the clinician must first assess whether the patient is hemodynamically unstable and experiencing serious signs and symptoms (ie, hypotension, angina) from a rapid ventricular rate. If serious signs and symptoms are present, immediate electrical cardioversion is necessary, regardless of the duration of the atrial fibrillation/flutter (Class I).

The initial evaluation of the hemodynamically stable patient with atrial fibrillation/atrial flutter must answer four questions before definitive treatment is begun. These include:

1. Is there an underlying reversible cause for the AF? Acute medical conditions that might cause atrial fibrillation/flutter include hypoxia, CHF, hypokalemia, hypertension, anemia, hypomagnesemia, mitral regurgitation, acute MI, digoxin toxicity, or thyrotoxicosis. If one or more of these are present, they must be treated as part of the AF management.
2. Is the cardiac function impaired? Patients with symptomatic CHF or an ejection fraction < 40% require special precautions to be taken when medications are used to treat the AF. Many antiarrhythmic medications have negative inotropic and proarrhythmic properties that manifest in patients with an ejection fraction < 40%.
3. Does the patient have Wolff-Parkinson-White (WPW) syndrome? Patients with WPW have an accessory conduction pathway between the atria and the ventricle. Many antiarrhythmic medications have the potential of causing a paradoxical increase in the ventricular rate in patients with WPW.
4. Has the patient been in AF for less than or greater than 48 hours? If the patient has been in AF for greater than 48 hours, there is an increased risk of clot formation in the atria and systemic embolization with conversion to sinus rhythm. If stable, the patient requires rate control and anticoagulation for 3 weeks prior to cardioversion.

The management of a patient with hemodynamically stable atrial fibrillation/atrial flutter consists of three primary focuses:

1. The control of the ventricular rate.
2. The determination of the need for anticoagulation.
3. The conversion of the atrial fibrillation/atrial flutter to sinus rhythm.

12

Narrow Complex Supraventricular Tachycardia
(Vol I – ACUTE CARE PORTALS; Vol III – CV8 TACHYCARDIA)

Narrow Complex Supraventricular Tachycardias include:

1. Paroxysmal supraventricular tachycardia (PSVT) caused by a re-entrant circuit, as in AV nodal re-entrant tachycardia (AVNRT) or AV re-entry tachycardia (AVRT).
2. Automatic atrial tachycardia caused by abnormal automaticity in the atria, as in ectopic atrial tachycardia or multifocal atrial tachycardia (MAT).
3. Junctional tachycardia (rare).

While the mechanism causing the tachycardia helps to dictate the most appropriate treatment, frequently the mechanism of the arrhythmia may be difficult to determine. Nevertheless, since the therapeutic approach is different for PSVT vs automatic atrial tachycardia vs junctional tachycardia, it is important that a specific diagnosis be established. The following diagnostic tools may help in this differential:

1. Evaluation of the 12-lead ECG with special attention to the P-waves. Ectopic atrial tachycardia has abnormal P-wave configuration and P-wave axis. MAT is irregular like atrial fibrillation but has three or more different P-wave morphologies identifiable preceding the QRS complexes.
2. Clinical information may also help in the differentiation. PSVT tends to have an abrupt onset and termination, while the automatic atrial tachycardias tend to start and stop more gradually. Many patients with PSVT have previously been diagnosed as having PSVT or an accessory pathway.
3. The initial use of vagal maneuvers can also help to differentiate the type of the tachycardia. Vagal maneuvers cause slowing of conduction through the AV node by an increase in parasympathetic tone. The simplest vagal maneuver is the Valsalva (bearing-down) maneuver. Other vagal maneuvers that may be tried include modified vagal maneuver, coughing, induction of the gag reflex, or facial immersion in ice water.
4. Adenosine IV may also be helpful (provided that there are no contraindications) both in differentiating the type of tachycardia (ie, making the P-waves evident) and possibly in terminating the tachycardia. Remember that adenosine must be used with great caution, if at all, in patients with reactive airway disease because it may precipitate bronchospasm.

13

Regular Wide Complex Tachycardia
(Vol I—ACUTE CARE PORTALS; Vol III—CV8 TACHYCARDIA)

The treatment of patients with regular wide complex tachycardia may be controversial. The accuracy of determining if the tachycardia is ventricular versus supraventricular with aberrant conduction based on ECG criteria is often unreliable. Giving verapamil or diltiazem to a patient with VT may cause hemodynamic collapse. Therefore, treat wide complex tachycardia as if it is VT, unless the team is absolutely certain that the rhythm is PSVT. Most cases of wide complex tachycardia are of ventricular origin.

The initial approach to wide complex tachycardia is to determine if the patient is hemodynamically stable. If the patient is hemodynamically unstable due to the tachycardia (altered LOC, CHF, impaired tissue perfusion), perform immediate electrical cardioversion.

In hemodynamically stable patients, attempt to distinguish if the patient has stable ventricular tachycardia or supraventricular tachycardia with aberrancy. Useful clues to help make this decision include:

- If the patient has a history of coronary artery disease or other structural heart disease, the rhythm is more likely to be ventricular in origin.
- A history of aberrant rhythms, preexisting bundle branch blocks (BBB) or rate-dependent BBB, or accessory pathways suggests supraventricular tachycardia with aberrancy if the QRS from the previous ECG matches that observed with the tachycardia.
- Carefully evaluate a 12-lead ECG for AV dissociation. The loss of the normal 1:1 association between P-waves and QRS complexes is a reliable criterion of ventricular tachycardia.
- Administration of adenosine may be considered in order to identify and treat a suspected aberrant SVT.
- To identify and treat suspected aberrant SVT, consider administering adenosine if the wide complex tachycardia is regular. Adenosine is typically contraindicated in irregular wide complex tachycardia as it may worsen the patient's condition.

If you are able to make a specific diagnosis of the wide complex tachycardia, follow the appropriate algorithm that matches the diagnosis.

14

Ventricular Tachycardia

(Vol I – ACUTE CARE PORTALS; Vol III – CV8 TACHYCARDIA)

Although it is often difficult to be sure if a wide complex tachycardia is VT or not, some clues help in making this determination. Some features that suggest ventricular tachycardia include:

- Presence of AV dissociation
- Fusion/capture beats
- Left axis deviation in the frontal plane on the 12-lead ECG
- QRS width > 140 ms
- Clinical history of coronary artery disease

Key Approaches to Patients with Ventricular Tachycardia

1. The approach to patients assumed to be in VT is largely determined by the hemodynamic stability of the patient. Patients with VT who are in full cardiac arrest are treated the same as patients in VF. (Vol III – CV5 VENTRICULAR FIBRILLATION/PULSELESS VENTRICULAR TACHYCARDIA) Patients with VT who have a pulse but are hemodynamically unstable must be immediately cardioverted. (Vol III – CV5 VENTRICULAR FIBRILLATION/PULSELESS VENTRICULAR TACHYCARDIA)
2. In hemodynamically stable ventricular tachycardia, the form of the VT needs to be differentiated between monomorphic VT and polymorphic VT.
3. Monomorphic VT has QRS complexes that are fairly uniform from beat to beat. In the treatment of monomorphic VT, it is beneficial to know if the patient has a left ventricular hypertrophy with reduced function (ejection fraction <40% or CHF) or normal function (ejection fraction >40%) to help decide on the most appropriate drug therapy.
4. In polymorphic VT, the QRS morphology varies, is irregular in rate and is more unstable, and thus more likely to degenerate into ventricular fibrillation. The polymorphic VT may be found in the setting of a normal baseline QT interval (no evidence of torsades de pointes) or it may be associated with a prolonged baseline QT interval (where torsades de pointes is more common). Torsades de pointes is a special form of polymorphic VT with a characteristic ECG appearance described as *twisting of the points*. This form of VT is seen especially in patients with a prolonged QT interval. Torsades de pointes is often associated with hypomagnesemia, hypokalemia, use of many antiarrhythmic drugs, and tricyclic agents. The recognition of torsades de pointes is important because the treatment approach is different from the usual uniform morphology VT. If the patient is unstable

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hemodynamically, immediate defibrillation needs to be used to attempt conversion. Defibrillation is frequently not successful in converting the torsades de pointes to a normal rhythm; thus, medications (such as magnesium sulfate given at a dose of 1 to 2 g IV over 1 to 2 minutes) and/or overdrive pacing will be needed to attempt to convert this difficult rhythm.

15

Long QT Syndrome (LQTS) (Vol III—CV15 LONG QT SYNDROME)

The long QT syndrome (LQTS) represents a diverse group of disorders that can be inherited (the genetic form) or acquired. The inherited cases are caused either by the more common autosomal dominant Romano-Ward type or the rare autosomal recessive Jervell and Lange Nielsen type that is associated with congenital deafness. These genetic mutations encode for abnormal cardiac ion channels. The acquired forms are caused by a large number of stimuli. The most common causative agents are the Class I and Class III antiarrhythmic agents, but many other drugs and conditions are known to increase the risk of prolonging the QT interval. One important distinction between the inherited and the acquired forms of LQTS is that correcting the underlying disorder or discontinuing the offending drug can often reverse the acquired form. All patients with the LQTS share a prolongation of the QT interval. This is associated with an increased risk of developing a specific type of polymorphic ventricular tachycardia called torsades de pointes or “twisting of points.”

Symptoms Related to LQTS

Many people with a LQTS live for many years without any symptoms from their LQTS, but when symptoms do occur they are often serious. The most common symptoms are syncope or sudden death, typically occurring during physical activity or emotional upset. The most common age to see the symptoms is in the preteen to teenage years, but the symptoms may first appear from the first days of life to middle age. Sudden and unexplained loss of consciousness or cardiac arrest in a child or teenager or a family history of unexplained syncope or sudden death in a young person should raise the suspicion of LQTS.

16

PEDS: Tachycardia in Infants or Small Children

PEDS: Normal Pediatric Vital Sign Ranges ²

Age	Normal RR	Normal HR	Blood Pressure
Newborn to 3 months	35 to 60	85 to 205	65-90/45-65
6-12 months	30 to 60	80 to 120	80-100/55-65
1-3 years	20 to 30	70 to 110	90-105/55-70
3-6 years	20 to 25	65 to 110	90-110/60-75
6-12 years	14 to 22	60 to 100	90-120/60-75
Adult	12 to 20	60 to 100	90-120/60-80

Decompensation in small children with tachycardia is usually an effect of sepsis, fever, and shock from other causes, rather than from the tachycardia. Sinus tachycardia responds to treatment of the underlying cause.

Extreme sinus tachycardia may be difficult to differentiate from SVT because the rates may be similar. The following points can help to distinguish between them:

1. **Heart rate.** Sinus tachycardia is usually at a rate < 200 bpm. The rate in SVT is usually > 230 bpm, but there is overlap.
2. **ECG.** P waves may be difficult to see when the heart rate is more than 200 bpm. If the P waves can be seen, they usually have an abnormal axis in SVT.
3. **Variability.** In sinus tachycardia there may be beat-to-beat variation in rate.

SVT in small children is most often caused by a re-entry mechanism involving an accessory pathway of the AV conduction system.

As opposed to adults, most children tolerate SVT well. If SVT results in shock, it must be reversed emergently.

The heart rate associated with SVT varies according to the child's age. In infants, the rate is commonly 240 bpm. The rate is regular. At very high rates, P waves may not be seen. The QRS duration is usually normal, although a wide QRS can be seen. Distinguishing SVT from VT, AF, and atrial flutter may be difficult. However, treat all of these dysrhythmias in an unstable child with synchronized cardioversion.

Perform synchronized cardioversion without delay for a child in shock. Use an initial setting of 0.5 to 1.0 J/kg. Ideally, intubate and oxygenate the patient prior to synchronized cardioversion.

Attempt vagal maneuvers in stable patients. Place a washcloth soaked in ice water on the patient's face for no longer than 30 seconds. This may be repeated. Older children can perform a Valsalva's maneuver.

Adenosine 0.1 mg/kg rapid IV push with constant ECG monitoring produces a transient AV block that interrupts the re-entrant circuit. If the first dose fails, it

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may be repeated once at twice the dose (not to exceed 12 mg). Use adenosine when the patient is stable enough to allow time to start an IV.

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Cardioversion

(Vol I—ACUTE CARE PORTALS; Vol III—CV9 ELECTRICAL CARIOVERSION)

Strongly consider cardioversion early in the treatment of patients whose tachycardia (usually with ventricular rates > 150) is causing serious hemodynamic effects and when administration of medication is either ineffective, contraindicated, or has too slow an onset of action to correct the tachycardia. Signs and symptoms of CHF – decreased LOC, hypotension/shock, or persistent chest pain – are some of the patient findings that should prompt a team to consider electrical cardioversion. Determine whether the serious signs and symptoms are due to the tachycardia rather than the tachycardia being a response to another condition, such as hypovolemic shock or pain. This is crucial.

The resuscitation team performs many actions (team action) almost simultaneously in preparation for synchronized cardioversion. The team must remember that the seriousness of the patient's signs and symptoms determines how quickly the cardioversion must be accomplished to prevent deterioration in the patient's hemodynamic condition.

Note that cardioversion is a very unpleasant experience, and most alert patients require intravenous sedation. Be prepared to manage the airway in a sedated patient.

18

Undefined Chest Pain

There are numerous potential causes (both cardiac and non-cardiac in origin) for chest pain. The urgency of dealing with chest pain is determined by making a correct working diagnosis for the cause.

Cardiovascular etiologies for chest pain

Acute myocardial infarction	Pericarditis
Dissection of the thoracic aorta	Stable or unstable angina
Hypertensive crisis	Pulmonary embolus
Arrhythmias (significant bradycardia or tachycardia)	

Non-cardiovascular etiologies for chest pain

Costochondritis	Pneumothorax
Cholecystitis	Pneumonia/pleurisy

Esophagitis/spasm-gastroesophageal reflux disease (GERD)	Chest trauma
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19 Acute Coronary Syndrome (Vol III — CV11 ACUTE CORONARY SYNDROME)

Complete the following within the first 10 minutes of the patient’s arrival:

1. Obtain vital signs, including oxygen saturation.
2. Deliver appropriate oxygen.
3. Attach cardiac monitor and observe initial rhythm; determine whether to treat the rhythm immediately.
4. Obtain IV access, including venous blood for initial blood studies.

After the preceding initial steps, continue the rapid evaluation of the patient with chest pain by performing or ordering the following:

5. Complete the targeted **SAMPLE** history. This may include getting information from prehospital care providers, friends, or relatives.
6. **PQRST** is a good assessment tool to obtain information regarding the chest pain. **P**-What provokes the pain? What was the patient doing when the pain presented? **Q**-What is the quality of the pain? **R**-Does the pain radiate? What relieves the pain? **S**-How severe is the pain? **T**-What time did the pain start?
7. Relay all information to the team leader. This information may be critical to initial patient management and determination of fibrinolytic use.
8. Perform a focused physical examination and specifically determine the patient’s eligibility for fibrinolytic therapy.
 - Obtain a 12-lead ECG.
 - Assess the initial 12-lead ECG, history, and information from the focused physical examination to determine if the patient is an appropriate candidate for acute reperfusion with either fibrinolytic therapy or percutaneous coronary intervention (PCI, ie, angioplasty and/or stenting).
 - Obtain the initial laboratory and x-ray tests. A pre-set battery of venous blood studies for acute coronary syndrome (including initial cardiac marker levels, electrolytes, CBC, and coagulation studies) may facilitate the laboratory evaluation.
 - Consider the need for a chest x-ray.
9. From the preceding, the team develops a preliminary impression.

Initial Treatment

Although many treatment protocols may be used simultaneously, for immediate treatment of a patient suspected of having chest pain of ischemic origin, have three drugs readily available: Fentanyl, Aspirin, and Nitroglycerin.

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Quickly weigh the risks and benefits of each.

Fentanyl 1-2 mcg/kg (max 200 mcg), IN/IV/IM/IO, every 5 to 15 min PRN

Aspirin 160 to 325 mg, if not contraindicated (Chewing speeds absorption.)

Nitroglycerin SL or as a spray as many as 3 times, as long as not contraindicated by hypotension, suspected right ventricular infarction, or the patient's use of phosphodiesterase-inhibitor medications (eg, Sildenafil [Viagra] within a time period specific to each drug in this class). **(Note these meds are not only taken by men, they are also used to treat pulmonary arterial hypertension, and as such are known as Adcirca and Revatio, and thus can be taken by men, women and children)**

The treatments that follow must be individualized to the specific patient needs, laboratory findings, clinical setting, and resources available. Some treatment choices include:

- The need for reperfusion therapy – either by fibrinolytics or angioplasty
- The need for anticoagulation therapy with such agents as heparin or IIb/IIIa agents or clopidogrel
- The need for beta blocker therapy
- The need for ACE inhibitor therapy
- The need for management of arrhythmias
- The need for invasive intervention, such as performing a diagnostic angiogram, angioplasty, or coronary artery bypass graft
- The need to manage complications of the acute coronary syndrome, such as CHF or shock

20

Hypotension - Shock (Vol III – CV17 SHOCK)

All shock states result in tissue hypoperfusion, tissue injury, and ultimate tissue damage.

To help determine the cause of SHOCK, in an undifferentiated patient, you can use Ultrasound and follow the Rapid Ultrasound for Shock and Hypotension Algorithm. HI- MAP

H- Heart **I-** IVC **M-** Morrison's pouch **A-**Aorta **P** - Pneumothorax

One way of defining the cause of shock is to consider the potential broad physiological categories that can result in shock:

1. **Cardiac rate problem** – heart rate either too slow or too fast to meet the metabolic needs of the body
2. **Volume problem** – due to an absolute or relative vascular volume deficiency.
 - Vascular volume loss as from hemorrhage or dehydration
 - Distribution problem due to vasodilatation or third space fluid loss as in spinal shock, anaphylaxis, or septic shock
3. **Pump problem** – left ventricular failure due to primary heart abnormalities resulting in inadequate cardiac output
4. **Inadequate preload** – due to obstruction or lack of proper filling of the right ventricle of the heart as occurs with tension pneumothorax, cardiac tamponade, large pulmonary emboli, or right ventricular infarction

Consider the mnemonic **SHRIMPCAN** to help develop a broad differential if the cause is still unknown:

- S** Sepsis – gram-negative infection or other overwhelming infection
- H** Hypovolemia resulting from hemorrhage, dehydration, vomiting, peritonitis, pancreatitis, leaking aneurysm, ectopic pregnancy
- R** Respiratory compromise resulting in hypoxia, tension pneumothorax, massive pulmonary embolus
- I** Ingestion of a toxic substance, drug overdose
- M** Metabolic causes including diabetic ketoacidosis, hyperosmolar coma, myxedema, adrenal insufficiency, electrolyte abnormalities
- P** Psychiatric causes such as water intoxication
- C** Cardiogenic shock, acute myocardial infarction, cardiomyopathy, cardiac tamponade, dysrhythmias, severe cardiac failure, valvular heart disease, atrial myxomas
- A** Anaphylactic shock
- N** Neurogenic causes such as spinal shock, herniation syndromes, and intracranial bleed

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Congestive Heart Failure/Pulmonary Edema (Vol III – CV12 ACUTE HEART FAILURE)

Patients with severe CHF and/or pulmonary edema may exhibit varying degrees of pulmonary congestion, such as tachypnea, labored breathing, rales, jugular venous distention, frothy sputum, and cyanosis. In addition, the reduced cardiac output may result in weak pulses, pallor, diaphoresis, and hypotension. If the cause for the severe congestive heart failure (such as severe bradycardia or tachycardia) can be identified, treat aggressively.

Initial Management

1. Administer oxygen while attaching the patient to the ECG monitor, oxygen saturation, obtaining a rhythm strip and patient vitals.
2. Alert patients may benefit from CPAP or BiPAP.
3. Establish IV access.
4. Give nitroglycerine SL to reduce preload if systolic blood pressure is greater than 100 torr.
5. Other useful medications include ACE inhibitors, nesiritide, dobutamine, inamrinone, and imrinone. (Vol III – CV12 ACUTE HEART FAILURE)
6. Consider administering morphine 2 to 5 mg IV to assist with peripheral dilation to decrease preload and help relieve anxiety. This helps to decrease oxygen demands.
7. Assist ventilation as necessary; early intubation with the use of RSI may be lifesaving.
8. If the patient is normotensive or hypertensive, start a nitroglycerine drip at 10 to 20 $\mu\text{g}/\text{min}$ and titrate blood pressure down to a systolic blood pressure of 100 torr. If the initial blood pressure was high, titrate until the mean blood pressure is 30% lower than on arrival.
9. If signs of fluid overload are present, administer Lasix 40 to 100 mg IV.

22

Undefined Cardiomyopathy

Patients with severe cardiomyopathy can present in cardiac arrest or severe CHF (cardiogenic shock). The cardiomyopathy may be due to end-stage CHF, arteriosclerotic heart diseases, chronic alcoholism, or a wide range of other causes. Such patients who can be stabilized should be considered for intra-aortic balloon pumping as a bridge to heart transplant or other therapy.

23

Digitalis Toxicity (Vol III – CV14 DIGITALIS TOXICITY)

Digoxin is the form of cardiac glycoside used clinically as part of the management of CHF and to assist in slowing ventricular heart rate in patients with atrial fibrillation. Clinical manifestations of digitalis toxicity include gastrointestinal and neurologic symptoms along with a wide variety of cardiac arrhythmias.

Initial Management

1. The most important element of successful treatment of digitalis toxicity is early recognition of the patient's symptoms and/or arrhythmias related to

digitalis toxicity. Temporary withdrawal of the drug along with ECG monitoring (if indicated) often suffices for treatment.

2. Potassium replacement is indicated for patients with hypokalemia or when serum potassium levels are in the low normal range. This is particularly useful if the patient has an ectopic tachyarrhythmia. Potassium replacement must be used cautiously in patients with conduction disturbance since the atrial ventricular conduction may be further slowed by rapid replenishment of serum potassium.
3. Significant arrhythmias need to be carefully managed.
4. Digitalis-specific antibody therapy (Digibind or FAB fragment therapy) may be considered in severe digitalis toxicity.

24

Hypertensive Crises (Vol III—CV13 HYPERTENSIVE CRISES)

Hypertension emergencies are those situations that require immediate reduction of the blood pressure (not necessarily to normal) to prevent or limit target organ damage. Examples include severe hypertension associated with encephalopathy, intracranial hemorrhage, acute left ventricular failure with pulmonary edema, aortic dissection, eclampsia or severe pregnancy-induced hypertension, unstable angina, or acute myocardial infarction. **Hypertensive urgencies** are those situations in which it is desirable to lower the blood pressure within 24 hours. Examples include accelerated or malignant hypertension without symptoms or progressive target organ complications and situations where severe post-operative hypertension occurs.

Initial Management

1. Rapid blood pressure reduction to normal may cause cerebral hypoperfusion.
2. The goal of treatment is to reduce the mean arterial pressure by 20% to 25% over a period of minutes to hours.
3. Most patients are volume depleted, probably due to pressure-induced diuresis.
4. Diuretics and fluid restriction should be reserved for patients who are clinically fluid overloaded.

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Thoracic Aortic Dissection

Dissection of the thoracic aorta occurs when the pulsatile flow of blood breaks through the intimal lining of the aorta into the media of the vessel wall. The arterial blood then separates the intima from the media and adventitia and travels up and down the wall of the aorta. This defect may be caused by arteriosclerosis and hypertension or a congenital weakness of the aortic wall.

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Patients (tall and thin) with Marfan's syndrome are at an increased risk for the disorder.

The typical dissection patient presents with tearing chest pain. If the dissection mainly involves the ascending aorta, the pain is mainly anterior. If the dissection begins in the distal arch, the pain is mainly between the shoulder blades. The pain begins suddenly and tends to occur in males with a history of hypertension.

When the dissection begins in the ascending aorta, distortion of the aortic valve may occur with valvular incompetence. The left coronary artery may become occluded. Frequently, the dissection will rupture into the pericardial sac, producing hemopericardium and tamponade. The dissection can involve the carotid arteries, producing stroke signs. The origin of the left subclavian artery can be involved, resulting in a diminished blood pressure in the left arm as compared to the right.

When the dissection begins more distally, it may dissect into the abdominal aorta and may involve the femoral arteries. Renal arteries and splanchnic arteries may be compromised. Pulses may become reduced in the femoral arteries.

The differential diagnosis includes acute myocardial infarction, so be careful to consider thoracic aortic dissection in the patient who may receive thrombolytic therapy or heparinization.

Initial Management

1. The chest x-ray is seldom diagnostic in establishing the diagnosis. CT of the aorta with IV contrast is the most common diagnostic modality. Transesophageal echocardiography if available is also diagnostic. Ultrasound may be helpful in picking up hemopericardium and tamponade. If such is found, placement of an indwelling catheter may allow intermittent decompression to buy time until surgical correction is possible.
2. Treatment involves decreasing the pulsatile effect of cardiac contraction while also blocking peripheral alpha-adrenergic effects. The alpha and beta blocker, labetalol (Normodyne, Trandate) 20 mg IV every 10 min up to 300 mg accomplishes both of these effects. The effect of the labetalol lasts several hours.
3. Target a systolic blood pressure of about 80 to 100 torr. You may continue an infusion of 2 mg/min. Labetalol is contraindicated in CHF, heart block, and reactive airway disease.
4. If the dissection is in the ascending aorta or results in the occlusion of major branches of the aorta, emergency surgery may be needed. Obtain early consultation.

26

Cardiac Tamponade

Distended neck veins, hypotension, and muffled heart sounds comprise Beck's triad of signs of cardiac tamponade. Unfortunately, these signs are not always present.

Cardiac tamponade may have resulted from myocardial rupture secondary to a myocardial infarction several days previous. Such cases may be salvageable because the site of rupture is usually the thick free wall of the left ventricle and the opening is usually small and repairable.

Cardiac tamponade may also have resulted from dissection of the thoracic aorta with rupture into the pericardial sac, effusion secondary to myocarditis or pneumonia, chest trauma, or **(PEDS) pericarditis in a child**.

Initial Management

1. Use ultrasound through a subxiphoid window to make the diagnosis whenever the possibility arises. **Vol III—UL2 EMERGENCY ULTRASOUND TECHNIQUES**. Alternatively, perform a diagnostic pericardiocentesis. **(Vol II—CIRC SKILLS 6 PERICARDIOCENTESIS)** When significant symptomatic cardiac tamponade is found, place a catheter in the pericardial sac to intermittently decompress the pericardial sac until the patient can be taken to surgery. This tamponade associated with a thoracic aorta dissection is also amenable to surgery if the patient can be stabilized with repeated aspiration and an alpha and beta blocker (labetalol) used to minimize further dissection. **(See Thoracic Aorta Dissection, # 25 this pathway)**

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Tension Pneumothorax **(Vol I—PATHWAY 6, ADULT RESPIRATORY)**

The emergency treatment of tension pneumothorax is addressed in **Vol II—BREATH SKILLS 5 NEEDLE THORACOSTOMY**.

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Pulmonary Embolism

The emergency treatment of pulmonary embolism is addressed in **Vol I—PATHWAY 6, ADULT RESPIRATORY, #10**.

29

Air Embolism

In any patient with a right to left shunt, air embolism is a great danger. Normal patients tolerate small bubbles of air in IV lines without a problem. These small bubbles are trapped in the lungs where they are gradually dissolved by the blood. In shunt patients, the bubbles can cross through the shunt into the arterial tree. They can lodge in the brain or heart and cause serious problems.

Initial Management

1. Place the patient who has air embolism on his or her left side in an attempt to keep the air in the right heart. Place the patient in steep Trendelenburg position to lessen the probability of cerebral embolism.
2. Insert a central venous line to aspirate air from the right atrium and ventricle if the crunching sound of Hamman can be heard over the precordium. Seizures are common and can be resistant to treatment. Hyperbaric oxygenation is the treatment of choice for air embolism.

30

Leaking Abdominal Aortic Aneurysm (AAA)

The emergency treatment of leaking abdominal aortic aneurysm (AAA) is addressed in [Vol I—PATHWAY 3 GASTROINTESTINAL/ABDOMINAL EMERGENCIES, #6](#).

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Implanted Cardioverter-Defibrillator (ICD)

If you are touching a patient as the patient's ICD discharges, you will feel a shock, but you will not be endangered.

Initial Management

1. Avoid placing pacing or defibrillator paddles/pads directly over the implanted device. Otherwise, in all emergencies other than ICD-related problems, treat the patient as if an ICD were not present.
2. Consult the patient's cardiologist regarding management of the ICD post-resuscitation.
3. If an ICD is firing erratically, disarm the ICD by placing a magnet on the skin over the subcutaneous pulse generator. Response will vary with ICD type, but most devices will be disabled as long as the magnet remains on the pulse generator. Magnets designed for disabling pacemakers and ICDs should be available in all emergency departments.

Congenital and Acquired Heart Defects (Tetralogy of Fallot, Valvular Heart Defects)

Tetralogy of Fallot

Fortunately, most congenital heart defects rarely present as emergencies. **PEDS:** Children with right to left shunt heart defects (such as tetralogy of Fallot) may present in shock (tetralogy spells). The most common reason for this decompensation is dehydration. This may occur as a result of exercise or illness.

The cause for these spells is that the left ventricle must have an adequate afterload during systole or its ejection fraction increases. This causes it to accept more blood from the right ventricle via the shunt during diastole. When this happens, as in dehydration, too much blood is diverted from the pulmonary circulation into the systemic circulation. The patient becomes more hypoxic and acidotic, resulting in further decompensation.

PEDS: Children with this defect squat and Valsalva when they feel a spell coming on because squatting causes a rise in peripheral vascular resistance and afterload.

Initial Management

1. The most important method of reversing a tetralogy spell is to administer a fluid bolus. **PEDS:** In children, a 10 to 20 mL/kg fluid bolus is indicated. The fluid bolus restores afterload and corrects the problem.
2. If acidosis and hypoxia have been present too long, a dopamine drip at 5 to 20 µg/kg/min may be used temporarily to increase afterload until a compensated state is reached. Too much vasoconstriction causes more acidosis and further decompensation.

Valvular Heart Defects

Valvular heart defects may result from acute myocardial infarction causing papillary muscle dysfunction, spontaneous papillary rupture, or septal rupture (usually 1 to 7 days after a myocardial infarction). A new cardiac murmur in patients with myocardial infarction may indicate the need for urgent cardiac surgery.

Undiagnosed aortic outlet defects such as aortic stenosis, bicuspid aortic valve, or hypertrophic subaortic stenosis may present as sudden death.

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Initial Management

1. Evaluate patients for cardiac surgery.
2. Others may recover spontaneously from a brief episode of exertional syncope. In all syncope patients, listen for a systolic murmur. In such cases, obtain cardiology or surgical consultation urgently.

33

Kawasaki Disease (Mucocutaneous Lymph Node Syndrome)

This disease syndrome is characterized by a prolonged high fever (at least 5 days), erythema of the palms and soles subsequently resulting in desquamation of the digits, stomatitis, conjunctivitis, lymphadenopathy, and a variety of rashes. Arthralgia or arthritis, pyuria or proteinuria, aseptic meningitis, myocarditis, or pericarditis may also occur. Coronary vasculitis may affect as many as 40% of patients with Kawasaki disease with 1% to 2% of patients dying because of coronary vasculitis. Diagnosis is largely based on the constellation of the clinical syndrome findings.

Initial Management

The inclusion of Kawasaki disease here reflects its potential to cause myocardial ischemia and infarction in children.

The treatment of primary disease includes administration of salicylates and intravenous immune globulin and is outside the scope of this section.

1. Intravenous immunoglobulin stops the process and decreases the risk of coronary disease. This should be given as soon as the diagnosis is clear. Consult pediatric infectious disease if considering this diagnosis and uncertain, then refer for pediatric echocardiography.
2. Salicylate therapy is also helpful in suppressing the fever and discomfort.

34

Stroke (CVA) (Vol III – NEU2 TREATMENT OF STROKE)

Patients with an acute focal neurologic deficit suspected of having sustained a stroke (“brain attack”) require a rapid and accurate clinical evaluation including a careful history and a general and neurologic examination. The team must search for an accurate time of onset, keeping in mind that it is defined as the last time that the patient was “last known well.” Thus, if a patient awakens with a neurologic deficit, it has to be assumed that the injury occurred when he or she was last known to be asymptomatic. The accurate timing of the injury is essential if thrombolytics are to be considered as part of the treatment plan for the patient.

Key Questions in the Evaluation of a Patient with an Acute Neurologic Deficit

1. Is the cause a stroke/TIA or some other diagnosis presenting as an acute neurologic deficit?
2. What type of stroke has the patient had?
3. What is the vascular distribution of the stroke?
4. Is the patient a candidate for thrombolytic therapy?
5. What immediate evaluation and/or tests are necessary to determine the proper treatment of the patient?
6. What early treatments are necessary to minimize the neurologic damage and reduce the associated morbidity associated with the stroke?
7. Is the patient, who is over 4.5 hours a candidate for mechanical retrieval?

Differential diagnosis of a stroke/TIA:

- Ischemic–atherothrombotic stroke due to a narrowed vessel becoming occluded with a thrombus
- Ischemic-embolic stroke due the embolization of a clot from some source outside the brain
- Ischemic stroke of other etiology (vasculitis, venous thrombosis, etc.)
- Intracerebral hemorrhage due to ruptured cerebral vessel, vascular malformation, vasculitis, or other cause
- Subarachnoid hemorrhage due to ruptured aneurysm or other cause
- Traumatic brain injury including subdural or epidural hematoma, subarachnoid hemorrhage, or cerebral contusion
- Chronic subdural hematoma (seen primarily in the elderly population)
- Metabolic condition causing a focal neurologic deficit such as hypoglycemia, hyperglycemia, hyponatremia, encephalopathy, or toxin ingestion
- Rapidly progressive intracranial tumor
- Viral encephalitis with rapid neurologic deterioration (e.g., herpes encephalitis)
- Post-ictal neurologic deficit (most often temporary hemiparesis)
- Migraine with focal deficit
- Central nervous system (CNS) infection (meningitis, encephalitis)
- Epidural abscess

The determination on clinical grounds of the cause for an acute neurologic deficit can be difficult. Some clinical clues include:

- Stroke symptoms are nearly always sudden in onset. Headache may accompany an ischemic stroke, but severe headache increases suspicion of ICH.
- While migraine headache patients may have focal neurologic deficit, for the migraine to be considered a cause of the deficit, the patient should have a migraine history, and other conditions must be excluded.

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- In general, a hemispheric ischemic stroke does not cause a depressed level of consciousness (LOC) unless it is (1) very large and associated with mass effect, (2) multifocal, or (3) compresses or involves the brain stem and reticular activating system.
- An ICH (traumatic or stroke) is more likely to result in a depressed LOC than an ischemic stroke. Thus, when a patient presents with an acute neurologic deficit associated with a depressed LOC, consider the following differential diagnoses:
 1. Large hemispheric stroke with or without mass effect
 - An irregular pulse may indicate atrial fibrillation and thus increase the chance of an embolic stroke.
 2. ICH with or without hydrocephalus
 3. Cerebellar stroke/bleed/tumor with mass effect and brain stem displacement
 4. Intracranial mass (tumor)
 5. Traumatic brain injury
 6. Brain stem stroke involving the reticular activating system
 7. Meningitis/encephalitis
 8. Toxic metabolic encephalopathy
 9. SAH
 - A stiff neck (meningismus) may suggest meningitis or SAH.
 - An additional challenge to the clinical diagnosis of stroke is that stuttering (waxing and waning) stroke symptoms can occur, especially in small vessel strokes, and different symptoms can occur at different times when emboli are showering into multiple vessels.

Consider tPA management of acute ischemic strokes in adults if certain specific criteria are met. These include:

- The patient must be able to receive the tPA within 3 hours to 4.5 hours of the onset of the acute neurologic deficit.
- The baseline brain CT must exclude an intracranial hemorrhage.
- The patient must be free of contraindications for the use of tPA. (**Vol III – NEU2 TREATMENT OF STROKE**)

Diagnostic tests to order depend on the clinical situation but may include:

- Blood glucose
- CBC
- Electrolytes
- Coagulation studies
- Troponin
- Brain CT/non-contrast
- Brain MRI

- Chest x-ray
- 12-lead ECG
- Lumbar puncture (if bleed is suspected, but not seen on CT)

Early treatments that may need to be considered to reduce the risk of death or further neurologic damage in a patient who presents with an acute neurologic deficit include:

- Maintaining and supporting an adequate airway and ventilation. Many patients are either hypoxic or are unable to protect their own airway and thus are prone to further neurologic damage or aspiration.
- Increased ICP from brain swelling or edema must be watched for and if present treated.
- Infections such as pneumonia, especially associated with aspiration, or urosepsis must be carefully watched for and treated if present.
- Non-stroke causes of acute neurologic deficit must be sought and treated if present.

35

Brief Loss of Consciousness - Syncope

Brief loss of consciousness/syncope is a transient loss of self-awareness or loss of consciousness frequently associated with loss of postural tone that lasts from a few minutes to hours from which the patient spontaneously recovers. While the term syncope is most frequently used in cases when the cause for the loss of consciousness is either due to a brief dysfunction of the vasodepressor cardiovascular reflexes or due to a primary cardiogenic cause, it is frequently difficult to tell during the initial evaluation of a patient if their loss of consciousness fits into one of these categories or has another more specific cause. This discussion does not delineate all potential causes for a patient to have a brief loss of consciousness, (**Vol I – PATHWAY 1 ALTERED LEVEL OF CONSCIOUSNESS**) but rather focuses on more common causes to consider.

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**PATHWAY 3: GASTROINTESTINAL/ABDOMINAL EMERGENCIES
(ADULT AND PEDIATRIC)**

The team continues the resuscitation along the pathway suggested by the initial clinical impression. Each pathway includes a complete, thorough, and rapid physical examination with additional history taking. The team leader is wary of conditions that may not be apparent. To obtain additional clinical data or to correct a missed or newly developed condition, the team leader repeats the initial survey if the patient is not responding satisfactorily.

Text Number	Diagnosis/Condition	Related Materials
1	Vomiting Blood	
2	Bleeding Esophageal Varices	Vol III – GI/AB1 ESOPHAGEAL VARICES; Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS, CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT
3	Upper Gastrointestinal Bleeding, Stomach or Duodenum (Peptic Ulcer/Gastritis)	Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS, CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT
4	Rectal Bleeding	
5	Bowel or Gastric Obstruction	
6	Leaking Abdominal Aortic Aneurysm	
7	Ruptured Ectopic Pregnancy	Vol III – OB3 BLEEDING IN EARLY PREGNANCY
8	Abdominal Viscus Perforation (Gastric, Duodenal Ulcer)	Vol II – IN4 ABDOMINAL SEPSIS
9	Abdominal Sepsis/Peritonitis	Vol III – IN3 SEPSIS IN ADULTS, IN4 ABDOMINAL SEPSIS; Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS, CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT
10	Intussusception	
11	Severe Dehydration in Small Children Secondary to Vomiting and Diarrhea	

12	Volvulus in Infants and Children	
13	Volvulus in Adults	
14	Acute Mesenteric Ischemia	
15	Cholangitis	

Gastrointestinal (GI) emergencies can cause abdominal pain, upper or lower GI bleeding, vomiting, or sepsis. Imaging studies to consider include chest x-ray, flat and upright abdominal x-ray, point-of-care ultrasound, and abdominal/pelvic CT scan. The use of IV, PO, or PR contrast with abdominal CT depends on the patient's symptoms, differential diagnosis, age, renal function, and pregnancy status. Various laboratory tests may be considered depending upon the specific symptoms and differential diagnosis based on focused history and physical examination.

1

Vomiting Blood

The source of the hemorrhage may be esophageal, duodenal, or gastric. Examine for the stigmata of liver cirrhosis: spider nevi, jaundice, sclera icterus, ascites, or enlarged liver that may suggest esophageal varices. Ask about a history of alcoholism or hepatitis as a cause of liver disease. Seek a history of food intolerance as well as use of aspirin, NSAIDs, glucocorticosteroids, and anticoagulants, such as warfarin, clopidogrel or NOACs (novel anticoagulants) (Also referred to as DOAC (Directly acting oral anticoagulants)). Ask about epigastric pain typical of gastritis or peptic ulcer disease. Finally, ask about a history of recent vomiting and retching prior to hematemesis, which may suggest a Mallory-Weis tear and a history of aortic graft, raising the possibility of an aortoenteric fistula. Remember that coffee-ground emesis can signify the same problems, but with slower bleeding rate, and prepare for patient deterioration, even if the patient appears stable on arrival. Maintain a low threshold for obtaining GI consultation and ICU admission if hematemesis is persistent. A rectal examination is indicated to detect the presence of blood, either frank or occult.

2

Bleeding Esophageal Varices

Assume that variceal bleeding is likely to be occurring if there is evidence of severe liver disease and upper GI bleeding. If the patient is bleeding massively, approach the problem aggressively. **(Vol III – GI/AB1 ESOPHAGEAL VARICES)**

Initial Management

- Airway protection** is needed to prevent aspiration of blood in patients with severe upper GI bleeding. Prepare to keep the airway clear with suction. Rapid Sequence Intubation may be needed as soon as you establish IVs and the team is prepared. **(Vol III – AIR SKILLS 4 RAPID SEQUENCE INTUBATION)**
- Vascular access:** Establish 2 **large-gauge IV lines**. **(Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS, CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT)**. Central venous access is recommended only if the patient has insufficient peripheral vascular access,

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- requires multiple lines for medications or high volume resuscitation, **and** the provider is trained and comfortable with central line placement. Otherwise, if the patient has insufficient peripheral IV vascular access, obtain intraosseous (IO) access.
- 3. Volume replacement:** Begin volume replacement starting with a 1 to 2 liter bolus of crystalloid (NS or LR) IV in an adult and (**PEDS**) 10 to 20 mL/kg in children. Begin blood replacement early to avoid hemodilution and further hypocoagulopathy. If massive transfusion is anticipated or the patient is taking therapeutic warfarin or otherwise coagulopathic, consider adding fresh frozen plasma (FFP) and other blood clotting components, if available. The use of TXA (tranexamic acid) can be of benefit, especially if FFP and other blood clotting agents are not readily available.
 - 4. Assess coagulation status:** If the patient demonstrates a hypocoagulable state (bleeding from needle puncture sites, etc), assess (PT, PTT, platelet count, bleeding time, and clinical evidence of abnormal clotting) and treat coagulopathies induced by medications, disease states, or bleeding/ transfusions. Treatment may include Vitamin K 10 to 15 U IM or SQ. FFP corrects coagulopathies more rapidly. Platelet transfusions may be needed for thrombocytopenia.
 - 5. Drugs to reduce blood flow to the splanchnic bed:** Octreotide, a synthetic growth hormone, may be administered 25 to 50 µg IV bolus over 3 minutes or diluted in 50 to 200 mL of NS and infused IV over 15 to 30 minutes. Additional 50 µg doses may be given hourly. Or, start an infusion of 25 to 50 µg/hour IV, using lower doses for elderly patients or those with end-stage liver disease. Octreotide selectively limits splanchnic blood flow and thereby reduces portal hypertension. It may be helpful in controlling esophageal variceal bleeding and does not cause coronary artery constriction as vasopressin does. Vasopressin may be considered as a second-line option for cases of severe upper GI bleeding as vasopressin is a potent vasoconstrictor that reduces flow to the splanchnic circulation. Due to the hypertension vasopressin can cause, nitroglycerine or nitroprusside may be needed concurrently, which is the reason it may not be the best first-line option in a resource-limited setting.¹⁻³
 - 6. Obtain an ECG** for signs of ischemia and treat according to ECG findings.
 - 7. Insertion of a nasogastric (NG) tube or large-bore orogastric (OG) tube** to clear the stomach and monitor bleeding was historically recommended for all patients with upper GI bleed to detect active bleeding and help predict need for endoscopy. No studies have demonstrated any outcome benefit for NG or OG tube insertion in the emergency department. Most current recommendations do not support the use of NG or OG tube insertion for all upper GI bleeding cases, but rather support placing NG or OG tubes in patients being prepped for endoscopy to help remove blood, clots, and particulate material from the stomach.⁴
 - 8. Emergency endoscopy**, when available, can be a critical intervention to identify and treat the bleeding source in patients with upper GI bleeding with sclerotherapy. This is dependent upon the availability of a GI or surgical specialist, either of whom can perform the endoscopy.

9. If these treatments do not stop bleeding, or endoscopy is unavailable, **only emergency surgery or balloon tamponade** with a special OG tube (such as a Sengstaken-Blakemore or Minnesota tube) can provide temporary benefit by stopping or minimizing bleeding while the patient is resuscitated and definitive care is sought. Use balloon tamponade as a last resort option because of risks such as esophageal mucosal ulceration, tracheal compression, or gastric rupture due to balloon inflation.¹

3

Upper Gastrointestinal Bleeding, Stomach or Duodenum (Peptic Ulcer/Gastritis)

A history of epigastric pain and the absence of the stigmata of liver disease make a bleeding peptic ulcer or gastritis likely in a patient vomiting large amounts of blood. The amount of blood vomited with bleeding ulcers is usually not as large as with bleeding varices, because the blood tends to go down the gastrointestinal tract.

Initial Management

1. **Airway protection** is needed to prevent aspiration of blood in patients with severe upper GI bleeding. Prepare to keep the airway clear with suction. Rapid sequence intubation may be needed as soon as you establish IVs and team is prepared. (**Vol III – AIR SKILLS 4 RAPID SEQUENCE INTUBATION**)
2. Vascular access. Establish **2 large gauge IV lines**. If bleeding is severe and you are unable to obtain rapid peripheral IV access, place an IO needle for access.
3. **Volume replacement:** Begin a 1 to 2 liter crystalloid (NS or LR) bolus IV or (**PEDS**) a 10 to 20 mL/kg bolus in children. Begin blood infusion as soon as it becomes available to avoid hemodilution and hypocoagulopathy. If massive transfusion is anticipated, consider adding FFP and other blood clotting components, if available. The use of TXA (tranexamic acid) can be of benefit, especially if FFP and other blood clotting agents are not readily available
4. **Insertion of an NG tube or large-bore OG tube** to clear the stomach and monitor bleeding has historically been recommended for all patients with upper GI bleed to detect active bleeding and help predict need for endoscopy. No studies have demonstrated any outcome benefit in the emergency department for NG or OG tube insertion. Most current recommendations do not support the use of NG or OG tube insertion for all upper GI bleeding cases, but rather to place NG or OG tube in patients being prepped for endoscopy to help remove blood, clots and particulate material from the stomach.⁴
5. **Assess coagulation status:** If the patient demonstrates a hypocoagulable state (bleeding from needle puncture sites, etc), assess PT, PTT, platelet count, bleeding time, and clinical evidence of abnormal clotting and treat coagulopathies induced by medications, disease states, or bleeding/ transfusions. Treatment may include Vitamin K 10 to 15 U IM or SQ. Vitamin K may be given IV, but IV administration carries a risk of anaphylactic-type reaction with little additional benefit. FFP corrects coagulopathies more rapidly. Platelet transfusions may be needed for thrombocytopenia.
6. **Suppression of gastric acid secretion:** Medications such as H2 blockers (ranitidine [Zantac] 50 mg IV or famotidine [Pepcid] 20 mg IV) or proton-pump inhibitors

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(pantoprazole [Protonix] 80 mg IV followed by 8 mg/h by continuous drip) are choices to use as adjunctive treatments to suppress gastric secretion. This should be coordinated with GI specialist as these medications are not mandatory to give emergently in ED.

7. Drugs to reduce blood flow to the splanchnic bed may be considered:

Octreotide, a synthetic growth hormone, may be administered IV at 25 to 50 µg bolus over 3 minutes or diluted in 50 to 200 mL of NS and infused IV over 15 to 30 minutes. Additional 25 to 50 µg doses may be given hourly; or start an infusion of 25 to 50 µg/hour IV using lower doses for elderly patients or those with end-stage liver disease. Octreotide selectively limits splanchnic blood flow and thereby reduces portal hypertension. It is frequently effective in controlling esophageal variceal bleeding and does not cause coronary artery constriction like vasopressin. Vasopressin may be considered as a second-line option for cases of severe upper GI bleeding as vasopressin is a potent vasoconstrictor that reduces flow to the splanchnic circulation. Due to the hypertension vasopressin can cause, nitroglycerine or nitroprusside may be needed concurrently which is why it may not be the best first-line option in a resource-limited setting.

8. If bleeding continues, only emergency surgery or emergency gastroscopy with coagulation of bleeders will work.

4

Rectal Bleeding

Bleeding from the upper GI tract usually appears black or tarry (melanotic) at the rectum. However, if the bleeding is brisk, it can appear bright red as with large bowel bleeding (hematochezia).

In order of frequency, the most common sources of lower GI bleeding are diverticular disease, colitis, adenomatous polyps, and malignancies. Approximately 80% of cases of patients with rectal bleeding will resolve spontaneously without intervention.⁵

Initial Management

1. If concerned about active bleeding and/or the patient shows early signs of hemorrhagic shock, start IVs and administer a bolus of 1 to 2 liters of crystalloid (NS or LR) in adults and **(PEDS) a 10 to 20 mL/kg bolus in children**. To avoid hemodilution and hypocoagulopathy, start blood replacement as soon as blood becomes available. If massive transfusion is anticipated, consider adding FFP and other blood clotting components, if available. The use of TXA (tranexamic acid) can be of benefit, especially if FFP and other blood clotting agents are not readily available
2. Consider early transport and telephone consultation with gastroenterologist and/or critical care physician. Consider insertion of an NG tube to decompress the GI tract in cases of massive GI bleed. This may also help localize the source of bleeding.
3. Assess coagulation status and reverse anticoagulation as indicated, similar to step 5 of **#4 of this pathway**, Upper GI Bleeding.

4. If bleeding continues, only emergency surgery or emergency endoscopy with coagulation will result in definitive treatment.

5

Bowel or Gastric Obstruction Present

Examine for abdominal distension with or without respiratory distress. Complete obstruction of the small or large bowel is marked by obstipation (inability to pass stool or flatus) leading to increased abdominal distention, nausea, vomiting, and even pain. Partial bowel obstruction may exist without obstipation. The most common causes of small bowel obstruction include adhesions, hernias, or post-operative strictures, although other causes exist. The most common cause of large bowel obstruction is colonic neoplasms, but may also be due to fecal impaction, ulcerative colitis, volvulus, or diverticulitis. A pseudo-obstruction of the large bowel (Ogilvie Syndrome) can look similar clinically.

Gastric outlet obstruction presents with early satiety, distention, nausea, and vomiting. The most common cause is scarring due to chronic peptic ulcer disease.

Initial Management

1. Establish intravenous fluid. While volume replacement with crystalloid (NS or LR) is the immediate concern, ongoing fluid and electrolyte losses may require potassium replacement and the use of D₅ 0.45 NS.
2. Insert a NG/OG tube for gastric and GI decompression. Consider pre-treating the nasal passage with topical anesthesia (lidocaine) and decongestant (phenylephrine) delivered by nasal spray. If the patient does not have a good gag reflex nor has a depressed LOC, orotracheally intubate (**Vol II – AIR SKILLS 3 OROTRACHEAL INTUBATION**) to avoid aspiration if the patient vomits.
3. If the abdominal distension disappears with gastric aspiration alone, there is probably gastric outlet obstruction. If the aspirate is fecal in appearance, there is probably a small or large bowel obstruction. Leave the gastric tube in place and periodically irrigate it to assure that it is draining the stomach.
4. Manage the airway carefully. Aspiration of feculent material is often a fatal complication of bowel obstruction.
5. Check the flat plate and upright x-rays of the abdomen for the typical appearance of sigmoid or cecal volvulus or air-fluid levels. Consider abdominal and pelvis CT imaging, with oral contrast if tolerated, if diagnosis and cause is uncertain. (**See # 12, #13 this pathway for management of a volvulus.**)

6

Leaking Abdominal Aortic Aneurysm

Symptomatic abdominal aortic aneurysms can present with a variety of symptoms, some of which are non-specific, so misdiagnosis or delayed diagnosis is problematic. The most common presenting symptoms are back or abdominal pain, but it can also present with

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syncope, flank pain, GI bleeding from an aortoduodenal fistula, shock, or even cardiac arrest. If the patient is awake, the abdomen may be tender as the examiner attempts to feel for a pulsatile mass, but the sensitivity of physical exam for aneurysm detection is moderate. A point-of-care ultrasound examination can reliably establish the presence of an aortic aneurysm in many patients, although bowel gas and abdominal tenderness may make the exam difficult in some patients. In hemodynamically stable patients, abdominal CT aortography with IV contrast is the best imaging to establish the diagnosis and prepare the surgical team with the anatomic details of the aneurysm.

Initial Management

1. Administer oxygen and protect the airway if necessary.
2. Establish 2 large bore IVs and order type-specific blood.
3. Surgical consultation and rapid transport to a center capable of performing emergency repair is essential. Some hospitals are equipped for both open surgical repair and endovascular aneurismal repair (EVAR).
4. Obtain an ECG.
5. Insert an oral gastric tube and a Foley catheter.
6. Blood and fluid replacement are in order, but attempt to maintain the systolic pressure at 90 to 100 torr as a goal. Avoid overly vigorous fluid resuscitation. Transfuse O Rh-negative blood as needed to maintain adequate circulation pending type-specific blood.

7

Ruptured Ectopic Pregnancy

Always consider a diagnosis of ruptured ectopic pregnancy in any woman of childbearing age in acute hypovolemic shock and/or with lower abdominal pain, with or without vaginal bleeding or spotting. A pregnancy test is mandatory in this situation. Do not rely on a patient's history to rule out pregnancy with this potentially life-threatening condition, unless circumstances, such as prior hysterectomy, absolutely rule it out. Note that while tubal ligation decreases the overall incidence of pregnancy, it increases the proportion of pregnancies that are ectopic. Severe lower abdominal pain is frequently associated with the rupture; however, the patient may experience only a vague discomfort or an urge to defecate. Besides performing a physical examination, diagnostic tests include urine or serum pregnancy tests and quantitative beta HCG. Blood count, blood type, and blood for crossmatch may also be drawn. Urine pregnancy tests are quite sensitive when the specific gravity is over 1.015, but serum tests may be more convenient in an unstable patient. Serum progesterone levels may also be useful in distinguishing early viable from non-viable pregnancies, but cutoff values have ranged from 5 to 22 ng/mL in various studies and it is not always available urgently.

In the acute setting, quantitative beta HCGs are used in conjunction with ultrasound exam. With quantitative beta HCG over 3000, if the pregnancy is intrauterine, an intrauterine gestational sac should be seen on transvaginal ultrasound examination. This usually occurs by 6 weeks gestation. With quantitative beta HCG greater than 6000, gestational sac should be seen on abdominal ultrasound. This usually occurs by 7 weeks gestation. If an intrauterine gestational sac is seen, the incidence of concurrent ectopic pregnancy (or heterotopic pregnancy) is about 1 in 30 000. **Note:** this rule does not apply in women undergoing

infertility treatment, in which multiple gestations are more common. If no gestational sac is seen, this is consistent with ectopic pregnancy, even if no adnexal mass is seen. If formal OB ultrasound is unavailable, or the physician is not familiar with transvaginal views, a FAST exam finding fluid in the cul de sac, Morrison's pouch, or left upper quadrant, would be consistent with a ruptured ectopic pregnancy or other surgical emergency.

If ultrasound is unavailable, culdocentesis may be performed. Administer mild analgesia as tolerated. Fill a syringe with 2 or 3 cc of local anesthetic. Grasp the "wings" of a 19- or 21-gauge butterfly (scalp vein) needle with a ring forceps and connect the hub of the butterfly's tubing to the syringe. Visualize the cervix through the speculum opened wide. Grasp the cervix with a tenaculum and lift up. Swab the posterior fornix with betadine. Use the butterfly needle grasped by the ring forceps to infiltrate an area on the fornix, and then puncture through this and aspirate with the syringe. (The short length of the butterfly needle prevents penetration past the cul de sac). Return of non-clotting blood is consistent with ruptured ectopic, but a negative aspirate is meaningless. If a butterfly needle is unavailable, some other type of needle/syringe may be used.

Initial Management

1. Establish 2 large bore IVs and begin crystalloid (NS or LR) infusion for volume replacement. Obtain blood for type and cross match. Use O Rh-negative blood if there is a delay.
2. Attempt to stabilize the patient with volume resuscitation including blood transfusion.
3. Arrange for immediate surgical consultation.
4. Administer RhoGAM to Rh-negative women. A dose of 50 µg is effective until 12 weeks of gestation due to the small volume of RBCs in the fetoplacental circulation, although there is no harm in giving the standard dose of 300 µg.

8

Abdominal Viscus Perforation (Gastric Duodenal Ulcer, Appendix, or Colonic Diverticulum)

Examine for signs of severe distress with a rigid abdominal wall that does not relax with ventilation. Lower lobe pneumonia can cause severe pain referred to the abdomen due to irritation of the diaphragms; however, the abdomen is not rigid and relaxes during exhalation.

Initial Management

1. If ruptured viscus is suspected, start an IV and insert an NG tube.
2. Consider pain control with IV opioids.
3. Obtain an upright x-ray of the chest and look for free air under the diaphragm. Free air is not always seen even with perforation. CT, if available, is more sensitive than x-ray for visualizing free air. Water-soluble oral contrast increases sensitivity of detecting persistent leaks and is safe in the setting of possible perforation.
4. **Initial Antibiotics:** Give piperacillin-tazobactam 3.375 g every 6 hours or meropenem 1 g IV every 6 hours (for penicillin-allergic patients). **PEDS:** Give

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piperacillin-tazobactam (weight- and age-adjusted dose). [See also Vol II – IN4 ABDOMINAL SEPSIS](#) for additional recommendations of IV antibiotics.

5. Consult general surgeon immediately as laparotomy to repair the defect is required in most cases. In clinically stable patients, however, conservative management with antibiotics, H2-blockers or proton pump inhibitors, and NG decompression may be considered by the surgeon.

9

Abdominal Sepsis/Peritonitis

Abdominal sepsis with peritonitis is usually accompanied by marked abdominal pain, abdominal distension, or sometimes a palpable mass. Typical signs of peritonitis are usually present including a tender abdomen with guarding, rigidity, and/or percussion tenderness. Temperature elevation is frequently present. Bowel sounds may or may not be present initially. Geriatric patients and (PEDS) small children may show few signs of abdominal sepsis. Conduct a careful abdominal examination of any patient in septic shock. Repeat the exam as necessary. A rectal examination may reveal a mass or localized tenderness. Ruptured viscus may be diagnosed by the presence of free air on upright abdominal or chest x-ray ([see #8 this pathway, Abdominal Viscus Perforation](#)). Pancreatitis, diverticulitis with sepsis and/or perforation, ascending cholangitis, cholecystitis, ureteral calculus, peri-renal or psoas abscess, embolization of splanchnic arteries, arteriosclerosis with bowel infarction, peritoneal dialysis, or primary spontaneous bacteria peritonitis are some of the many causes of abdominal sepsis.

Initial Management

1. Monitor vital signs, ECG, and oxygen saturation carefully. Obtain blood gases and serum lactate.
2. Consider pain control with IV opioids.
3. **Initial fluid resuscitation: Adults:** Give at least 30 cc/kg of crystalloid (NS or LR) IV within the first 3 hours. (PEDS) Give a 20 mL/kg initial bolus in children.
4. Additional fluid administration should be guided by frequent assessment of hemodynamic status, including clinical examination (HR, BP, O₂ sats, respiratory rate, temperature, and urine output), as well as other noninvasive monitoring (eg, echocardiography) or invasive monitoring (eg CVP), if available.
5. If large volumes are needed for persistent hypotension, opt for Ringer's lactate solution to avoid hyperchloremic acidosis.
6. In severe sepsis patients, consider obtaining central venous access ([Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS](#)) for improved access and vasopressors if needed.
7. Maintain a mean arterial pressure (MAP) of at least 65. This may require administration of vasopressors, which may be given concurrently during fluid resuscitation. Norepinephrine is considered the first-line vasopressor for septic shock. Although a central line is ideal for vasopressor administration, a well-functioning peripheral IV may be used initially during resuscitation.

8. Broad-spectrum antibiotics are necessary. (Vol III – IN3 SEPSIS IN ADULTS, IN4 ABDOMINAL SEPSIS)

10

Intussusception

PEDS: Ileocolic intussusception occurs most commonly in the 3-month to 3-year age group. The ileum inverts on itself and pushes distally into the cecum where it becomes edematous, friable, and bloody. Commonly the child has a history of recurrent unexplained bouts of abdominal pain. There may be a history of bloody, mucous-covered stool. This has been called “currant jelly” stool. On physical examination, it may be possible to palpate a thickened and tender cecum. An abdominal x-ray may reveal thickened rings of the distal ilium with proximal obstruction, but these findings are not always present. Ultrasound may also be used in facilities with ultrasound technologists experienced with identifying intussusception. In younger children, the diagnosis can be difficult. In young infants or in late stages, intussusception may present as lethargy, vomiting, poor feeding, or shock.

Initial Management

1. The treatment is an emergent hydrostatic (saline or contrast) or pneumatic (air) enema that pushes the ileum back out of the cecum. This is successful in reducing the intussusception about 80 to 95% of the time and may be guided with either ultrasound or fluoroscopy. Given that these techniques require experience, they should be done in facilities with experience in this procedure, if possible. The patient should have adequate IVF resuscitation prior to reduction attempt if needed.
2. If unsuccessful, surgery is needed. Therefore, the air-contrast enema should be done in facilities with appropriate and available surgical back-up. Surgical approach may also be considered initially if the patient appears critically ill or there are clinical or radiographic signs of perforation.

11

Severe Dehydration in Small Children Secondary to Vomiting and Diarrhea

PEDS: The majority of children with vomiting and dehydration has mild-to-moderate dehydration and can be treated with oral rehydration. Severe dehydration – with associated shock presenting as tachycardia, hypotension, lethargy, and increased respiratory rate – can be a life-threatening emergency. The child may have a depressed level of consciousness and be unable to feed or eat.

Initial Management

1. Obtain IV access and administer a bolus of crystalloid (NS or LR) 20 mL/kg. Reassess for hypovolemia. Hypoglycemia, lactic acidosis, and electrolyte abnormalities may be found. Aggressive restoration of blood volume is the most important action. If IV cannot be obtained quickly, place an IO for fluid resuscitation.

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2. For most patients with acute watery diarrhea, antibiotics are not indicated. Antibiotics should be considered in children < 6-months-old because of a high incidence of bacteremia in this age group. In older children, antibiotics may also be indicated in cases of cholera, bloody diarrhea caused by *Shigella* or *Campylobacter*, or when the diarrhea has been present for 10 to 14 days. Antibiotic therapy for children with *E Coli* strains producing Shiga-toxin may lead to increased risk for hemolytic-uremic syndrome, so use caution if there is risk for acute enterohemorrhagic *E Coli*.
3. Obtain stool and blood cultures before instituting antibiotic therapy. Most cases turn out to be viral, but this is difficult to ascertain initially.

12

Volvulus in Infants and Children

PEDS: In children, volvulus usually involves the cecum and the distal small bowel. The amount of bowel involved varies considerably. It is caused by a failure of the ascending colon and cecum to attach to the posterior abdomen allowing the hypermobile cecum and distal ileum to rotate around the mesenteric pedicle of the ileocecal artery, producing a closed-loop obstruction and a strangulation of the bowel. This can be difficult to diagnose. Bilious vomiting secondary to small bowel obstruction is a clue. If not corrected early, this can lead to septic shock. Abdominal x-rays are rarely diagnostic, but classical findings include a dilated bowel segment. Barium enema may demonstrate the “bird’s beak” characteristic of volvulus.

Initial Management

1. Intravenous fluid therapy is necessary.
2. Consider IV pain control if needed.
3. Surgical intervention is needed. Obtain surgical consult.

13

Volvulus in Adults

Volvulus is the torsion of a segment of the alimentary tract that usually leads to a bowel obstruction. The most common locations for volvulus in adults are the cecum and the sigmoid colon. Other potential locations that may form a volvulus include the transverse colon, splenic flexure of the colon, or a segment of the small bowel. Most patients complain of symptoms similar to a bowel obstruction with nausea, vomiting, abdominal pain, and constipation. Physical examination discloses a distended abdomen representing dilated loops of colon and small bowel. Abdominal x-rays commonly show distended loops of small bowel with a large air-filled loop of colon. CT scan or contrast enema may be diagnostic, but do not perform contrast enema in patients if intestinal gangrene is suspected. The characteristic contrast enema finding of a colon volvulus is the “bird’s beak.”

Initial Management

1. Intravenous hydration is necessary.
2. Pain control with IV opioids may be required.

2. A nasogastric or orogastric tube is necessary to avoid gastric distension and subsequent regurgitation and aspiration.
3. Early surgical consultation is needed.
4. In some cases, decompression can be achieved by a gastroenterologist or surgeon with a rectal tube advanced into an obstructed loop of sigmoid colon under direct vision with a colonoscope.
5. A dilated volvulus (measuring 12 cm across) is in danger of losing its blood supply. Surgical consultation is required to prevent imminent perforation.

14

Acute Mesenteric Ischemia

Acute mesenteric ischemia typically presents as severe but diffuse abdominal pain with minimal tenderness on physical examination. This is often termed “pain out of proportion with examination.” Patients may report recent similar postprandial pain. In about half of cases, occult blood may be present in the stool. As ischemia progresses, the bowel may perforate and produce more classic peritonitis. Combined with the frequent co-morbidities in the patient group, a mortality rate > 70% is expected once the bowel is infarcted. To prevent this outcome, maintain a high index of suspicion and pursue the diagnosis aggressively. While no simple lab test is diagnostic, a persistently normal serum lactate is helpful in excluding the diagnosis, but the test must be repeated over time to help confirm a negative impression. Definitive diagnosis is by CT or angiography or Gadolinium-enhanced MRA (magnetic resonance angiogram).

Initial Management

1. Hydration via IV is needed as part of the initial stabilization.
2. Pain control with IV opioids is often required.
3. Angiographic infusion of papaverine may be effective in patients who do not have bowel tissue necrosis.
3. Rapid transfer for definitive treatment is required, which may be either surgical or endovascular intervention.

15

Cholangitis

Cholangitis is another potentially life-threatening condition most commonly observed in geriatric or immunocompromised patients. In addition to the usual signs and symptoms of biliary disease, the patient may be febrile, shocky, and have an altered mental status. At times, these manifestations may overshadow the biliary pain. Elevated WBC count, bilirubin, and liver function tests are consistent with this diagnosis. Ultrasound may confirm gallstones and a dilated ductal system. CT may also be useful. Nuclear studies, such as HIDA scanning (cholescintigraphy), are sensitive for early obstruction.

Initial Management

1. Hydration via IV is needed as part of initial stabilization.
2. Consider pain control with IV opioids.

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3. Antibiotics need to be initiated early. Recommended antibiotic regimens include ampicillin/sulbactam 3 g IV or ceftriaxone 2 g plus metronidazole 1 g IV.
3. Endoscopic retrograde cholangiopancreatography (ERCP), if available, may be both diagnostic and therapeutic.
4. Surgical consultation is needed.

Caveats

Suspect a leaking abdominal aneurysm in any middle-aged or geriatric patient in shock.

Digitalis toxicity is usually exhibited as a rhythm problem, but prominent gastrointestinal symptoms may be present.

Ischemic bowel can result from embolism, arteriosclerosis, venous thrombosis, or strangulation from a volvulus or internal hernia.

A dilated volvulus (measuring 12 cm across) is in danger of losing its blood supply; perforation is imminent.

Gastric decompression in bowel obstruction reduces morbidity and mortality by preventing vomiting and aspiration.

Keys to resuscitation are good airway management (**Vol II—AIR SKILLS PORTALS**) and good blood volume management. (**Vol III—CV17 SHOCK**)

Of numerous causes of abdominal pain and other GI symptoms—such as vomiting, diarrhea, and rectal bleeding—many are benign. However, cast a wide differential and rule out the worst case scenarios first to quickly identify and treat any life-threatening causes of symptoms.

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PATHWAY 4: NEONATAL EMERGENCIES

The team continues the resuscitation along the pathway suggested by the initial clinical impression. Each pathway includes a complete, thorough, and rapid physical examination with additional history taking. The team leader is wary of conditions that may not be apparent. To obtain additional clinical data or to correct a missed or newly developed condition, the team leader repeats the initial survey if the patient is not responding satisfactorily.

Text Number	Diagnosis/Condition	Related Materials
1	Factors for high-risk neonates	
2	Resuscitation equipment	
3	Newborn resuscitation algorithm	
4	Immediate steps A Initial stabilization B Ventilation C Chest compressions D Medications	Vol III – NEONATAL 1 MECONIUM SUCTIONING, NEONATAL 2 UMBILICAL ARTERY AND VEIN CANNULATION; Vol II – AIR SKILLS 1 AIDS TO INTUBATION, AIR SKILLS 3 OROTRACHEAL INTUBATION; Vol I – ACUTE CARE 41 PEDIATRIC EQUIPMENT SIZES; Vol III – NEU4 PHENYTOIN AND FOSPHENYTOIN LOADING

Be prepared.

Only about 10% of newborns require life support and resuscitation in the delivery room or nursery.¹ However, it is not always possible to predict when an infant will need resuscitation. The probability of the need for resuscitation increases for infants weighing less than 1500 g. At least one person skilled in neonatal resuscitation should be present at every delivery. In many rural EDs, this means that most team members must be skilled to ensure that a qualified individual is always present.

Successful resuscitation depends upon **preparation** for and **anticipation** of a high-risk neonate. The American Academy of Pediatrics defines the neonate period as the first 28 days of life. One may need to readjust the neonate period due to gestational age/prematurity.

**NEONATAL EMERGENCIES
PATHWAY 4**

1

Factors for High-Risk Neonates

Antepartum (fetomaternal)

• Age < 17 or > 38 years	• Multifetal gestation
• Diabetes	• Small fetus for maternal dates
• Hemorrhage	• Postterm gestation
• Fetal malformation identified by ultrasound	• Preterm labor or premature rupture of membranes
• Drug therapy (eg, magnesium, adrenergic-blocking drugs, lithium carbonate)	• Other chronic maternal illness, (eg, cardiovascular, thyroid, or neurological problems)
• Previous fetal or neonatal death	• Immature pulmonary maturity studies
• No prenatal care	• Oligohydramnios or polyhydramnios
• Chronic hypertension or PIH	• Diminished fetal activity
• Anemia or isoimmunization	• Substance abuse
• Bleeding in 2 nd or 3 rd trimester	• Infection

Intrapartum

• Breech or other abnormal presentations	• Indexes of fetal distress (eg, FHR abnormalities)
• Chorioamnionitis or infection	• Operative delivery
• Maternal sedation	• Meconium-stained fluid
• Prolapsed cord	• Prolonged rupture of membranes
• Uterine tetany	• Premature or precipitous labor
• Prolonged 2 nd stage of labor or prolonged labor	• Forceps or vacuum-assisted delivery
• Abruptio placentae or placenta	

2

Resuscitation Equipment in the Delivery Room

A special work surface must be provided with radiant warmers; all necessary equipment must be displayed and ready for use. A drug dosage book is also useful. All delivery rooms should be stocked with the following equipment, which should be checked and replenished periodically.

Suction Equipment

- Bulb syringe
- Adjustable suction source and tubing
- Suction catheters: 5F, 6F, 8F, 10F
- Feeding tube: size 8-, 20-mL syringe

Airway Equipment

- Neonatal bag-valve-mask, 500 cc capacity
- Premie and newborn face masks
- Oral airways: neonate and premie sizes
- Laryngoscope handle (small size) with extra batteries
- ET tube sizes: 2.5, 3.0, 3.5, 4.0
- ET stylets (optional)
- Tape or securing device for ET tube
- Pulse oximeter and probe
- Oxygen source with tubing; portable tank should also be available
- Oxygen face masks
- Laryngoscope blades: No. 0 straight No.1 Macintosh, extra bulbs
- cm water gauge that is attached to or part of the bag-valve-mask (optional)
- Scissors
- CO₂ detector

Circulation Equipment

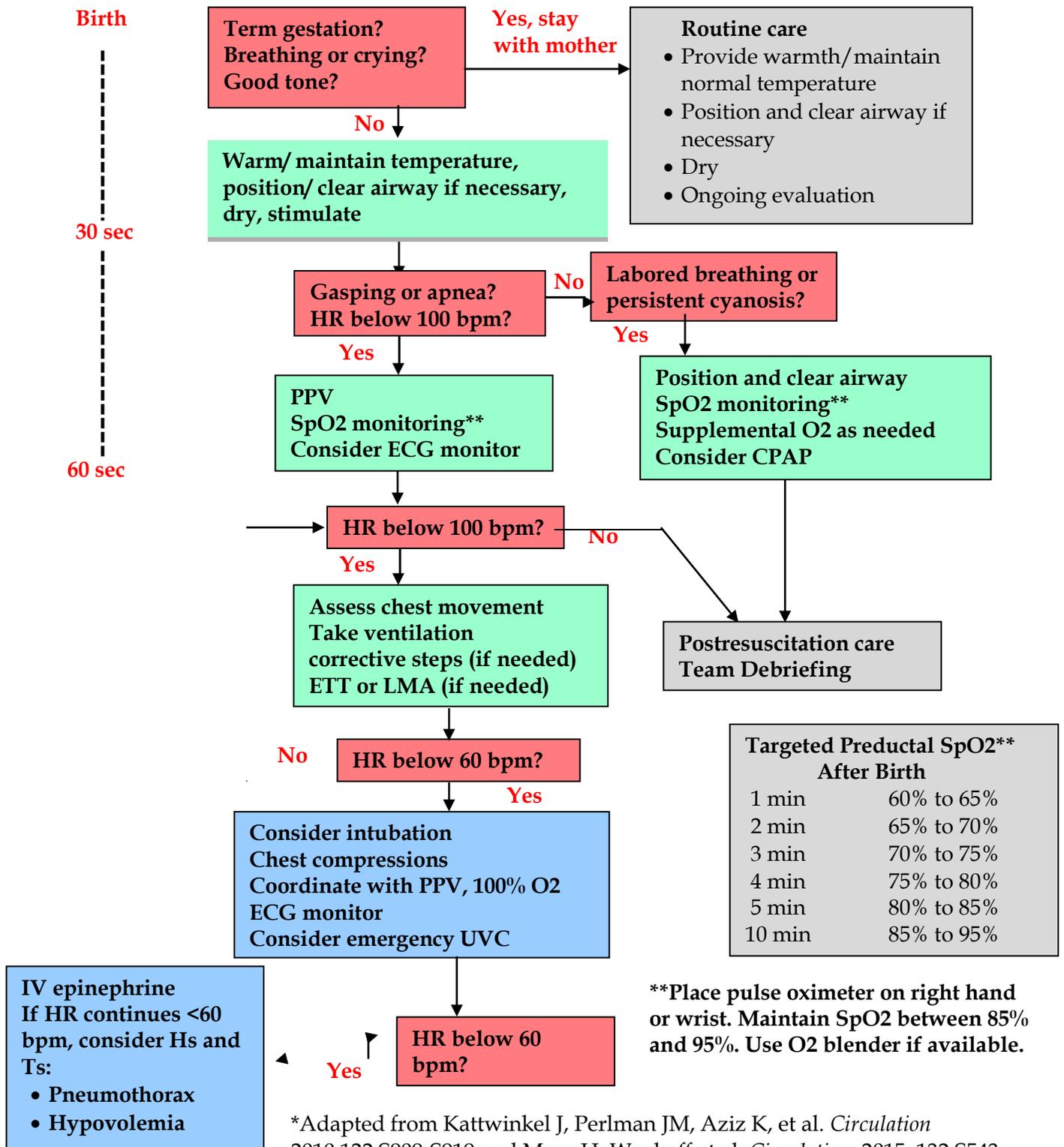
- Cord clamp
- Umbilical kit or tray
- Medications for neonatal resuscitation
- Infant stethoscope
- Needles, syringes, and stopcocks
- Umbilical catheters: sizes 3.5 F and 5.0 F
- Normal saline and Dextrose 10%
- Cardiac monitor and electrodes **OR** pulse oximeter and probe

Miscellaneous

- Radiant warmer
- Blankets
- Heating mattress (optional)
- Covering for infant's head

3

ACUTE CARE 27: NEWBORN RESUSCITATION ALGORITHM*



*Adapted from Kattwinkel J, Perlman JM, Aziz K, et al. *Circulation* 2010;122:S909-S919 and Myra H, Wyckoff et al. *Circulation*. 2015; 132:S543-S560.

4 The Immediate Steps

Place the newborn infant on a work surface with radiant warming lights. Do not take the time to set up the servomechanism (skin probe), whereby the radiant warmer is turned off automatically when the infant's temperature rises. This may be done later. Place the infant on the work surface on his or her back with the neck slightly extended to open the airway. Place a folded towel or small blanket (about 1 inch thick) behind the baby's shoulders, but avoid overextending the neck. If there are copious secretions coming from the mouth, turn the head to the side.

A. Initial Stabilization

Prevent Heat Loss

Critical actions: maintain a warm delivery room, place the infant under a radiant heat source, and quickly dry off the infant. Vigorous towel drying has an added effect of stimulating the infant. Replace the wet linen after drying.



All newborns face increased metabolic stress in cold environments; this is of particular concern in already compromised asphyxiated neonates. Infants who are cold-stressed have an increased metabolic rate and require more oxygen. Minimize the effects of cold stress by providing all newborns with a suitable warm environment. Avoid hypothermia.

These early actions take only a few seconds and have the added advantage of providing some tactile stimulation, which may help in stimulating respirations. Keep the infant's head covered as excessive heat loss may occur due to the size of the infant's head. A heated mattress, commercially made for neonates, may be placed under the infant for added warmth.

NEONATAL EMERGENCIES

PATHWAY 4

Meconium-stained amniotic fluid has the potential to complicate delivery. If the infant is vigorous, endotracheal intubation and suctioning is probably not necessary. These infants display strong respiratory effort, good muscle tone, and HR > 100 bpm. Non-vigorous infants showing signs of distress need suctioning. These infants display poor respiratory effort and poor muscle tone, and HR < 100 bpm. Intrapartum suctioning is no longer routine, especially if shoulder dystocia is anticipated.

If particulate meconium is present at delivery and the infant is non-vigorous, suction the trachea immediately. Suction before the infant is dried, because tactile stimulation may make the infant gasp in more meconium. Adjust the suction bottle on the resuscitation cart to 80 to 100 torr negative pressure. Attach a meconium aspirator to the suction tubing coming from the suction bottle.



Prepare an ET tubes (size 3) with obturators and an ET tubes (size 2.5) in case the size 3 tubes are too large. The intubator visualizes the vocal cords using a laryngoscope with an infant Macintosh or Miller blade and inserts the first ET tube about 3 cm into the trachea. An assistant pulls out the obturator and attaches the meconium suction adaptor to the ET tube. The assistant then removes the ET tube, keeping a finger over the side hole of the aspirator to apply suction and turns the ET tube while removing it. One team member provides free-flow oxygen and checks heart rate throughout endotracheal suctioning. If the infant becomes bradycardic even after suctioning once, begin PPV and consider the possibility of meconium aspiration syndrome.

Meconium suctioning is a team effort that must be accomplished quickly. Rehearse this team effort at every opportunity to maintain the team's skill. **(Vol III – NEONATAL 1 MECONIUM SUCTIONING). NOTE THERE CONTINUES TO BE A MOVE TO NOT DOING ANY SUCTIONING AT ALL, AND JUST BAGGING THE INFANT.**

Opening the Airway

Critical actions: Place the infant on his or her back with the neck slightly extended; suction the mouth and nose.



It is important to prevent overextension or flexion of the neck in order to keep the airway open. Place a blanket or towel (about 1 inch thick) under the infant's shoulders to maintain proper head position. If the infant has copious secretions coming from the mouth, it is helpful to turn the infant's head to one side.

As soon as the infant has been properly positioned, suction first the mouth and then the nose. This will prevent the infant from aspirating material that has collected in the posterior pharynx. The person delivering the neonate can suction the mouth and nose of the infant after the head is delivered, but do not delay delivery of the shoulders for suctioning. It is important to remember that the infant's first respirations will be strong gasps through the mouth before nasal breathing is established. While suctioning, be careful to avoid suctioning deeply within the pharynx, which could stimulate a vagal response and bradycardia; limit suctioning efforts to no longer than 5 seconds at a time.

Tactile Stimulation

Drying and suctioning often provide enough tactile stimulation to induce respirations in most infants. However, if an infant is not breathing immediately, additional stimulation is required. Safe methods of providing tactile stimulation include (1) slapping or flicking the soles of the feet and (2) rubbing the back. Avoid more vigorous methods of stimulation. If the infant does not initiate respirations after 10 to 15 seconds of stimulation, PPV will be required.

NEONATAL EMERGENCIES

PATHWAY 4

Evaluate respiratory effort, heart rate, and color in the next 30 seconds.

Respiratory Effort

Observe the neonate's ventilatory effort. If the infant is obviously gasping or not breathing, go immediately to PPV using a BVM. As in any resuscitation, adequate ventilation is assessed by chest wall motion and auscultation of breath sounds. Lung inflation, following delivery, should be achieved by bringing the pressure up till the lung has inflated. It may require 30 to 40 cm H₂O pressure but this should NOT be with the first breath. Deliver ventilations at a rate of 40 to 60 breaths per minute. Hypoxia in a newborn produces bradycardia, so use heart rate to help determine the need for continued PPV. A 500 cc BVM is ideal for purposes of neonatal resuscitation.

Heart Rate

Count the heart rate for 6 seconds and multiply by 10. You may evaluate the heart rate by stethoscope or by feeling the pulse by lightly grasping the base of the umbilical cord. If the heart rate is < 60 bpm, begin cardiac compressions while continuing PPV. Two techniques are used for chest compressions in the newborn. In the thumb technique, the thumbs are placed on the sternum just below the nipple line, and the fingers encircle the chest for back support. This is the preferred technique. In the two-finger technique, the chest is compressed with the ring and middle finger, and the other hand is used to support the back. This technique is used when placing an umbilical catheter.

The depth of compression should be $\frac{1}{3}$ to $\frac{1}{2}$ of the anterior/posterior dimension of the chest.³ The compression depth should be adequate to produce a palpable pulse. Avoid giving compressions and ventilations simultaneously. There should be a 3:1 ratio: impose one ventilation after every third compression. There should be a rate of 90 compressions and 30 breaths or approximately 120 "events" per minute.²

Prolonged PPV will result in gastric distension, so insert an orogastric tube. After 2 minutes of assisted ventilations, place an orogastric tube. The diaphragm is the main muscle of respiration in infants and may result in regurgitation of a distended stomach.

Check heart rate every 30 seconds to determine whether to continue CPR. Usually a brief application of chest compressions and BVM ventilation results in improvement in heart rate. Palpate the umbilical cord for the pulse rate, or listen to the heart sounds. Tap the rate on the table so that all team members are aware of it.

If the heart rate is > 60, PPV alone is probably sufficient. Check heart rate every 30 seconds to determine whether PPV should continue.

If the heart rate is > 100 bpm, the infant is stabilized. Continue to observe.

Color

Evaluate color for central cyanosis. Acrocyanosis (cyanosis of the extremities only) is common.

Use of Oxygen

Oxygen is essential in critical care resuscitation, but it can also be toxic in newborns, especially premature neonates. Application of oxygen should be guided by pulse oximetry. Place oximeter on the right upper extremity (right hand or wrist) to avoid admixture of venous blood from an open ductus arteriosus.

For most newborns, targeted preductal SpO₂ levels at specified time after birth follow:

- 1 minute=60%-65%
- 2 minutes=65%-70%
- 3 minutes=70%-75%
- 4 minutes=75%-80%
- 5 minutes=80%-85%
- 10 minutes=85%-95%

Note that SpO₂ levels above 90%-95% are probably unnecessary and may put the newborn at risk for oxygen toxicity.

Begin resuscitation with room air and blend in O₂ to achieve these targets if possible.

Exception: If patient is bradycardic/requires CPR, administer 100% O₂ until stabilized.



The majority of infants who require ventilatory support will be adequately ventilated by a BVM. The indications for PPV include:

1. Apnea or gasping respirations
2. Heart rate < 100 /min
3. Persistence of central cyanosis despite 100% oxygen

NEONATAL EMERGENCIES

PATHWAY 4

Perform ventilation of the infant at a rate of 40 to 60 per minute.



As in any resuscitation, adequate ventilation is assessed by increasing heart rate, chest wall motion, and auscultation of breath sounds. Lung inflation, following delivery, should be achieved by bringing the pressure up till the lung has inflated. It may require 30 to 40 cm H₂O pressure but this should NOT be with the first breath.

Prolonged BVM ventilation may produce gastric distention, which can be relieved by insertion of an 8F orogastric tube left open to air and periodically aspirated with a syringe.

Endotracheal intubation is indicated when BVM ventilation is ineffective, when tracheal suctioning is required, and when prolonged PPV is necessary or anticipated (including when a diaphragmatic hernia is suspected). For ET intubation of the neonate, [see Vol II – AIR SKILLS 1 AIDS TO INTUBATION, AIR SKILLS 3 OROTRACHEAL INTUBATION.](#)

C. Chest Compressions

Perform chest compressions if the heart rate is < 60/min, despite adequate ventilation with 100% oxygen for approximately 30 seconds.

There are two techniques for performing chest compressions in a neonate: thumb technique and two-finger technique. In both techniques, the sternum is compressed by placing the thumbs or fingers on the sternum just below the nipple line. In the thumb technique, the two thumbs are placed on the sternum, with the fingers encircling the chest and supporting the back. (Note: this technique cannot be used if another procedure [such as umbilical vein cannulation] needs to be performed.) In the two-finger technique, the ring and middle fingers of one hand are placed on the sternum; the other hand is used to support the infant's back.



The depth of compression of the chest is $\frac{1}{3}$ to $\frac{1}{2}$ the anterior-posterior dimension of the chest.³ The rate of compressions is 90/minute.

The combined rate of compressions and ventilation can be 120 events/minute with a 3:1 ratio of compressions to ventilation. (This would provide 90 compressions and 30 breaths per minute.)

Continued Bradycardia

If PPV with a BVM and chest compressions do not result in a rise in heart rate within about 30 seconds, begin more aggressive measures. Consider more aggressive measures including endotracheal intubation and the use of medications.

Orotracheal Intubation and Ventilation

ET intubation is necessary when prolonged PPV is required, when BVM is ineffective, and with small, premature infants. Prepare necessary supplies and equipment for ET intubation in advance. The approximate size of the ET tube is determined by the infant's weight. Depth of insertion is 6+ the neonate's weight in kilograms. [See Vol II – AIR SKILLS 1 AIDS TO INTUBATION, Pediatric ET Tube Sizes.](#)

D. Medication and Venous Access

Medications are administered if, despite adequate ventilation and chest compressions, the heart rate remains < 60 bpm. Routes of drug administration include umbilical veins, peripheral veins, and intraosseous. Use endotracheal tube for epinephrine only if needed while obtaining other access.

The umbilical veins are the most accessible vascular route of drug administration immediately after birth. A 3.5 or 5 F umbilical catheter is inserted into the vein of the umbilical stump until the tip of the catheter is just below skin level and a free flow of blood is present. If the catheter is inserted further, there is risk of infusing hypertonic solutions into the liver and causing liver damage. Use twill tape to provide hemostasis before and after the cannula is inserted. [\(Vol III – NEONATAL 2 UMBILICAL ARTERY AND VEIN CANNULATION\)](#)

NEONATAL EMERGENCIES

PATHWAY 4



1. Epinephrine

Epinephrine increases the strength and rate of cardiac contractions and causes vasoconstriction. Epinephrine is the first medication administered. Give a dose of 0.1 mL/kg to 0.3 mL/kg (equal to 0.01 mg/kg to 0.03 mg/kg) of **1:10 000** solution, rapid IV push. Epinephrine can be injected directly into the ET tube. If the heart rate remains below 60, consider repeating the dose every 3 to 5 minutes as required. Epinephrine may be given down the ET tube only while obtaining other access. The dose for epinephrine ET is higher (up to 0.1 mg/kg or 1 mL/kg) of 1:10 000 solution. Higher doses in newborns may result in brain and heart damage.¹

2. Dextrose

Sick and stressed newborns are very susceptible to becoming hypoglycemic. Infants at high risk to develop hypoglycemia include:

- Small for gestational age (SGA) infant
- Large for gestational age (LGA) infant
- Infants of diabetic mothers (IDM)
- Premature infants
- Infants with perinatal stress, sepsis, shock, asphyxia, and hypothermia

If blood sugar below 40 mg/dL, give 2 mL/kg of D₁₀ over several minutes. Re-check blood sugar within 15 to 30 minutes after glucose bolus. Infant may need to be re-bolused and have IV infusion of 10% dextrose started to have blood sugar stabilize over 50 mg/dL. Starting infusion rate is 80 mL/kg/day.

3. Volume expanders

Consider volume expanders for any newborn who fails to respond to initial resuscitation. The infant could be hypovolemic because blood was sequestered in the placenta or because of blood loss from the placenta. Signs of hypovolemia in

the newborn include pallor, weak pulse (even with a good rate), and poor response to resuscitative measures.

Commonly used volume expanders include:

1. Whole blood (O-negative crossmatched with the mother's blood or with cord blood)
2. Crystalloid solutions (NS or LR)

The fluid bolus is administered at a volume of 10 mL/kg using a syringe and stopcock over 5 to 10 minutes. This may be repeated if signs of hypovolemia persist.

4. Sodium bicarbonate

The use of sodium bicarbonate has now been dramatically reduced and if used at all, may be used for documented acidosis. Use sodium bicarbonate only after ventilation is established. **Consult with neonatologist before use.**

The adult solution is 8.4% and contains 1 mEq/mL. Dilute this to 4.2% with NS (0.5 mEq/mL). Alternatively, 10 mL syringes at 4.2% are commercially available. Administer sodium bicarbonate 2 mEq/kg IV (4 cc/kg of the 4.2%) slowly over at least 2 minutes.

5. Naloxone hydrochloride

The use of Naloxone hydrochloride (Narcan), which is a narcotic antagonist, has also been greatly reduced, due to the risk of Absence Withdrawal Syndrome. **Always establish and maintain adequate ventilations before administering naloxone. Consult with neonatologist before use.**

The dose is 0.1 mg/kg (0.25 cc/kg of 0.4 mg/mL dosage) and may be repeated every 2 to 3 minutes as needed. Its duration of action is 1 to 4 hours. It may be given IV or IM; IV is the preferred method.

6. Dopamine

Dopamine infusion is an adrenergic drug with mainly beta effects at low doses. Dopamine can be useful in the management of neonates who remain hypotensive despite the previously named aggressive measures. Consultation with a neonatologist is mandatory at this point. In preparation for transfer, a dopamine drip solution may be prepared for continuous infusion to support the neonate's BP. The infusion should start at 2 µg/kg/min and may be increased up to 20 µg/kg/min if necessary.

NEONATAL EMERGENCIES

PATHWAY 4

Other Neonate Medications

Seizure activity in neonates can be subtle. Rhythmic eye movements; facial, oral and lingual movements; apnea; and stertorous breathing may all indicate seizure activity. Treat seizures with lorazepam or diazepam 0.3 mg/kg. If unsuccessful, use Phenobarbital 20 mg/kg slow IV push. Then use Keppra (Consult Neonatologist for dosage). If Keppra is unavailable, administer phenytoin 18 mg/kg over 20 to 30 minutes or fosphenytoin 18 mg/kg IV over 10 minutes. (**Vol III – NEU4 PHENYTOIN AND FOSPHENYTOIN LOADING**) Consider as possible causes: hypoglycemia, pyridoxine deficiency, low sodium, low calcium, and low magnesium.

Treat hypoglycemia with 10% dextrose 2 to 4 mL/kg.

Treat possible pyridoxine deficiency with pyridoxine 100 mg IV.

Treat hyponatremia with 6 mL/kg of 3% NS solution.

Treat hypocalcemia with 50 to 100 mg/kg of calcium gluconate.

Treat hypomagnesemia with 2 to 4 mL of 2% MgSO₄.

Treat seizures with phenobarbital 20 mg/kg slow IV push (1 mg/kg/min); use caution as it may cause respiratory depression.

Consult a neonatologist about these difficult cases. Toxoplasmosis, cytomegalovirus, herpes simplex, and coxsackievirus infections are other possible causes of seizures in newborns. Congenital causes are hydrocephalus and microcephalus.

Finally, calculate the **Apgar Score** at 1 minute, 5 minutes, 10 minutes, and 15 minutes, until the resuscitation is over.

Apgar Score

Sign	Score		
	0	1	2
Heart rate/min	Absent	Slow (< 100)	> 100
Respirations	Absent	Weak cry, hypoventilation	Good, crying
Muscle tone	Limp		Active motion
Reflex irritability	No response	Grimace	Cry or active withdrawal
Color	Blue or pale	Pink body, blue extremities	Completely pink

Caveats

Newborn infants tire easily when breathing is impaired. Do not hesitate to aggressively manage the airway.

Do not resort to vasopressors until full advantage is taken of blood volume restoration.

Any birth can result in the need for neonatal resuscitation. **Be prepared.**

REFERENCES

1. Kattwinkel J, Perlman JM, Aziz K, et al. Part 15: Neonatal Resuscitation: 2015 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015; 122:S909-S919.
2. Kattwinkel J, McGowan, JE, Zaichkin J, eal. *Textbook of Neonatal Resuscitation*. 6th ed. Elk Grove Village, IL and Dallas, TX: American Academy of Pediatrics and American Heart Association; 2011.

January 2019

FOCUSED CLINICAL PATHWAYS

PATHWAY 5: OBSTETRICAL EMERGENCIES

The team continues the resuscitation along the pathway suggested by the initial clinical impression. Each pathway includes a complete, thorough, and rapid physical examination with additional history taking. The team leader is wary of conditions that may not be apparent. To obtain additional clinical data or to correct a missed or newly developed condition, the team leader repeats the initial survey if the patient is not responding satisfactorily.

Text Number	Diagnosis/Condition	Related Materials
1	Physiology of Pregnancy	Vol III – OB1 PHYSIOLOGY OF PREGNANCY
2	Ultrasound	Vol III – OB2 ULTRASOUND USE PORTAL
3	Bleeding in Early Pregnancy/ Miscarriage	Vol III – OB3 BLEEDING IN EARLY PREGNANCY/MISCARRIAGE
4	Dilatation and Curettage	Vol III – OB4 DILATATION AND CURETTAGE
5	FHT Monitoring	Vol III – OB5 FETAL HEART TONE MONITORING
6	Preterm Labor Management	Vol III – OB6 PRETERM LABOR MANAGEMENT
7	Bleeding After First Trimester	Vol III – OB7 BLEEDING IN THE SECOND HALF OF PREGNANCY
8	Hypertension in Pregnancy	Vol III – OB8 HYPERTENSION IN PREGNANCY
9	Trauma in Pregnancy	Vol III – OB9 TRAUMA IN PREGNANCY
10	Emergent Cesarean	Vol III – OB10 EMERGENCY CESAREAN SECTION
11	Imminent Delivery	Vol III – OB11 IMMINENT DELIVERY
12	Prolapsed Umbilical Cord	
13	Malpresentation/Malposition	Vol III – OB12 MALPRESENTATIONS AND MALPOSITIONS
14	Assistance for Delivery	Vol III – OB13 FORCEPS AND VACUUM DELIVERY
15	Shoulder Dystocia	Vol III – OB14 SHOULDER DYSTOCIA

16	Postpartum Complication	Vol III – OB15 THIRD-STAGE AND POSTPARTUM EMERGENCIES
17	Thromboembolic Disease	Vol III – OB16 THROMBOEMBOLIC DISEASE AND PREGNANCY

Obstetrics patients pose unique problems in emergency situations. Both maternal and fetal conditions must be systematically evaluated and treated. The questions in this pathway address a variety of obstetrical problems. Use the preceding table to cross-reference information in this pathway with the diagnostic and treatment portals.

It is important to determine the gestational age of the pregnancy. Determine the date of the patient’s last menstrual period and whether a pregnancy test has been done. When did the patient feel the first movements of the fetus (quickening)? Measure the fundal height to determine the uterine size.

Listen for fetal heart tones (FHTs). If FHTs are not present, use ultrasound to detect cardiac activity. In early first trimester pregnancies, cardiac activity or even a gestational sac on ultrasound may not be apparent. Correlation of ultrasound findings with a quantitative HCG may be helpful.

1 **Physiology of Pregnancy**
(Vol III – OB1 PHYSIOLOGY OF PREGNANCY)

Several physiological changes occur during pregnancy. These changes may be used diagnostically. For example if the uterus is at the level of the umbilicus, the gestational age is about 20 weeks or (5 months). Other physiological changes are important during assessment of other conditions such as shock due to trauma. For example, if the patient has a low blood pressure, this may be due the supine hypotensive syndrome caused by the weight of the uterus pressing on the vena cava when the patient is lying on her back. Placing the patient on her left side should relieve this.

2 **Ultrasound**
(Vol III – OB2 ULTRASOUND USE PORTAL)

Ultrasound may be helpful in addressing a number of important clinical questions that arise during labor and delivery such as gestational age, cardiac activity, multiple gestation, and fetal presentation. Ultrasound can also be useful in assisting with common procedures such as amniocentesis.

3

Bleeding in Early Pregnancy/Miscarriage
(Vol III – OB3 BLEEDING IN EARLY PREGNANCY/MISCARRIAGE)

Vaginal bleeding in the first trimester occurs in 30% to 40% of all pregnancies. Approximately half of this percentage of women miscarry at a gestational age of 12 weeks or fewer.

Obstetrical causes of bleeding include spontaneous abortion, normal pregnancy, embryonic death, blighted ovum, ectopic pregnancy, and trophoblastic disease. Non-obstetrical causes include vaginitis, cervical polyps, cervical erosion, cervicitis, or cervical cancer. Non-uterine causes of bleeding should be apparent during the speculum exam.

Laboratory tests for the stable patient without other medical problems should include a hemoglobin, quantitative beta HCG, and Rh status. In a clinically stable patient, either watchful waiting or surgical intervention is medically reasonable.

4

Dilatation and Curettage
(Vol III – OB4 DILATATION AND CURETTAGE)

If the patient is bleeding heavily or retaining tissue, the patient is considered unstable and may need a dilatation and curettage.

If peritoneal signs are present, a laparoscopy is indicated: laparotomy may be indicated if the patient is unstable. As an alternative, if laparoscopy is not available, culdocentesis may be performed.

5

FHT Monitoring
(Vol III – OB5 FETAL HEART TONE MONITORING)

Intrapartum electronic fetal monitoring is a useful tool in the emergency setting where intervention may result in a live infant (23 to 24 weeks minimum). Fetal heart rate changes are repetitious changes termed accelerations or decelerations. Determine if the FHTs are reassuring, warning, or ominous, and note the relationship to contractions. Review the tracing in a systematic fashion.

6

Preterm Labor Management
(Vol III – OB6 PRETERM LABOR MANAGEMENT)

Palpate the uterus for strength, duration, and frequency of contractions. If gestational age is < 36 to 37 weeks, treat for preterm labor. (Approximately 9% of all neonates in the United States are born prior to 37 completed weeks of gestation.)

7

Bleeding After First Trimester

(Vol III – OB7 BLEEDING IN THE SECOND HALF OF PREGNANCY)

Causes of bleeding range from normal to life-threatening and must be evaluated systematically. Ultrasound evaluation is needed. Examine the patient and consider the possibility of bloody show (blood-stained mucus), placental abruption, placenta previa, and vasa previa.

8

Hypertension in Pregnancy

(Vol III – OB8 HYPERTENSION IN PREGNANCY)

Hypertension in pregnancy can be differentiated into 3 categories: chronic or essential hypertension, gestational hypertension, and preeclampsia/eclampsia. Gestational hypertension is present if the BP is > 140/90 or if there is a systolic rise of 30 mm Hg or diastolic rise of 15 mm Hg during pregnancy.

Preeclampsia is gestational hypertension with facial/hand edema and a proteinuria reading of 1+ on urine dipstick (> 300 mg proteinuria/24 hours) between 20 weeks gestational age and 1 week postpartum. Eclampsia is preeclampsia with either coma or seizure present.

9

Trauma in Pregnancy

(Vol III – OB9 TRAUMA IN PREGNANCY)

The team leader must be aware of the physiology of pregnancy (including consideration of the mother and child as two separate patients). Treat serious maternal or fetal trauma with the same systematic approach as other kinds of trauma. Most maternal deaths are due to head injury and hemorrhagic shock. Most fetal deaths are unexplained or are due to maternal death or placental abruption; however, if maternal abdominal trauma is involved, abruption is the most common cause.

10

Emergent Cesarean

(Vol III – OB10 EMERGENCY CESAREAN SECTION)

The physician may need to perform an emergency cesarean section to save the baby when the mother is near death or has just died. Perform the cesarean section in the ED, unless an operating room is immediately available.

11 **Imminent Delivery**
(Vol III – OB11 IMMEDIATE DELIVERY; VOL I – ACUTE CARE 38 EMERGENCY CESAREAN)

Determine if delivery is imminent. If the patient is crowning or the perineum is bulging, prepare for immediate delivery. The team should be prepared to do this in the ED or other locations as indicated. Have emergency kits available for quick retrieval.

Move the patient quickly to an area where delivery can occur in a controlled manner and the infant can be resuscitated if necessary. (Vol III – NRP2 DRUGS IN NEONATAL RESUSCITATION, NRP3 MECONIUM SUCTIONING)

12 **Prolapsed Umbilical Cord**

If the cord is palpable on vaginal bimanual exam, a cord prolapse is present. Pulsation in the cord is typically palpable if the fetus is viable. Immediately place the patient in the knee-chest position; then, with your hand in the vagina, push the presenting part up as high as possible and hold the head in that position until accomplishing delivery by cesarean section.

13 **Malpresentation/Malposition**
(Vol III – OB12 MALPRESENTATIONS AND MALPOSITIONS)

Breech presentations are most common in preterm fetuses. Other factors that predispose to breech presentation include multiparity and uterine relaxation, hydramnios, hydrocephaly, and previous breech birth, tumors in the pelvis, multiple gestation, oligohydramnios, anencephaly, and uterine anomalies. Breech presentations are classified as frank, complete, or footling. Due to the greater risk of cord prolapse, head entrapment, and brachial plexus injuries, most breech presentations are delivered via Cesarean section. The final choice of birth route depends on physician's delivery room experience.

14 **Assistance for Delivery**
(Vol III – OB13 ASSISTED DELIVERY)

Occasionally it is necessary to deliver the neonate quickly due to abnormal FHTs. Assisted deliveries may also be necessary to avoid maternal exhaustion. Regional analgesia may interfere with voluntary expulsive efforts. Those trained in use of vacuum extractor may be used.

15

Shoulder Dystocia
(Vol III – OB14 SHOULDER DYSTOCIA)

Shoulder dystocia is the impaction of the anterior shoulder against the symphysis pubis after the fetal head has been delivered. Shoulder dystocia is a life-threatening emergency for the fetus and needs to be recognized and treated quickly to avoid morbidity or even mortality. Over 50% of all shoulder dystocias occur in the normal birth weight neonate and are unanticipated. Identify risk factors early to prepare for this complication.

16

Postpartum Complication
(Vol III – OB15 THIRD-STAGE AND POSTPARTUM EMERGENCIES)

Retained placenta is the inability to deliver the placenta within 30 minutes after birth. Postpartum hemorrhage is bleeding in excess of 500 cc in the first 24 hours after completion of the third stage of labor. Do not underestimate the amount of blood loss. Postpartum hemorrhage can be caused by: (1) uterine atony, (2) birth trauma, (3) uterine inversion, (4) uterine rupture, or (5) acquired coagulopathy.

17

Thromboembolic Disease
(Vol III – OB16 THROMBOEMBOLIC DISEASE AND PREGNANCY)

Superficial thrombophlebitis may be treated with elevation, analgesia, elastic stockings, heat, and ambulation. Deep vein thrombosis below the knee is often treated with low molecular weight heparin. Deep vein thrombosis above the knee always requires heparin therapy.

January 2019

FOCUSED CLINICAL PATHWAYS

PATHWAY 6: RESPIRATORY EMERGENCIES (ADULT)

The team continues the resuscitation along the pathway suggested by the initial clinical impression. Each pathway includes a complete, thorough, and rapid physical examination with additional history taking. The team leader is wary of conditions that may not be apparent. To obtain additional clinical data or to correct a missed or newly developed condition, the team leader repeats the initial survey if the patient is not responding satisfactorily.

During the initial survey, the airway was assessed, and appropriate measures were taken to assure an open airway and adequate ventilation. If this has not been done, return to the initial survey and do it now. If it was not necessary to secure the airway at that time but the airway becomes compromised, return to the initial survey and secure the airway, utilizing the approaches outlined.

In this pathway, choose the section(s) most closely corresponding to the problem at hand. For example, fever, cough, and dyspnea are most likely suggestive of pneumonia or pulmonary embolism and may be used as a starting point. In patients with known or suspected co-existing diseases (such as CHF and COPD), go to all pertinent pathways and sections. If after a thorough history and physical exam the nature of the patient's respiratory condition is uncertain (perhaps due to lack of history or associated symptoms), go to the Dyspnea section.

Because of the close anatomic and physiologic relationships between the respiratory and cardiovascular systems, consider the following:

- Cardiac pain tends to be substernal and constant; pulmonary pain tends to be peripheral and associated with respiration.
- All that wheezes is not asthma: Patients with CHF may wheeze.
- Myocardial ischemia may present as painless dyspnea.
- Pulmonary embolism involves both systems and may be insidious in its multiple presentations.

Text Number	Diagnosis/ Condition	Related Materials
1	Dyspnea	Vol I – PATHWAY 2 CARDIOVASCULAR EMERGENCIES
2	Upper Airway: General	Vol II – AIR SKILLS 12 TRACHEAL FOREIGN BODY REMOVAL
3	Upper Airway: Angioedema	Vol III – AIR3 HELIOX TREATMENT, AIR8 ANAPHYLAXIS
4	Upper Airway: Epiglottitis	Vol III – PED3 EPIGLOTTITIS
5	Upper Airway: Foreign Body	Vol II – AIR SKILLS 12 TRACHEAL FOREIGN BODY REMOVAL; Vol III – AIR3 HELIOX TREATMENT
6	Acute Respiratory Distress Syndrome: Non-cardiogenic Pulmonary Edema	Vol II – BREATH SKILLS 1 CHEST TUBE INSERTION, CIRC SKILLS; Vol III – AIR4 VENTILATOR MANAGEMENT
7	Asthma/ Anaphylaxis	Vol III – AIR8 ANAPHYLAXIS; AIR1 RAPID SEQUENCE INTUBATION, AIR3 HELIOX TREATMENT, AIR4 VENTILATOR MANAGEMENT, AIR7 STATUS ASTHMATICUS
8	Chronic Obstructive Pulmonary Disease (COPD)	Vol III – END/M6 ACID-BASE, AIR7 STATUS ASTHMATICUS, AIR5 NONINVASIVE VENTILATORY SUPPORT
9	Pneumonia and Aspiration	Vol III – IN1 ADULT PNEUMONIA
10	Pulmonary Embolism	Vol II – CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT; Vol II – AIR SKILLS; Vol III – AIR4 VENTILATOR MANAGEMENT
11	Pneumothorax and Pneumomediastinum	Vol I – STEP 3 INITIAL SURVEY; Vol II – BREATH SKILLS 1 CHEST TUBE INSERTION
12	Massive Hemoptysis	Vol II – AIR SKILLS 3 OROTRACHEAL INTUBATION; Vol II – BREATH SKILLS 3 ENDOBONCHIAL TUBE

**ADULT RESPIRATORY EMERGENCIES
PATHWAY 6**

1

Dyspnea

Dyspnea is a subjective symptom, a perceived inability to breathe normally, causing anxiety and/or fear, which then amplify the sensation. A vicious cycle ensues, which may mislead caregivers to focus on the anxiety and discount the underlying disease. Both need to be addressed. The goal is to detect and treat life-threatening or serious causes of dyspnea. Think in terms of organ systems to organize the clinical process:

Upper Airway

- allergic reaction
- foreign body
- mass
- stenosis
- tracheomalacia

Pulmonary

- air embolism
- adult respiratory distress syndrome
- aspiration
- asthma
- COPD
- interstitial lung disease
- mass
- pleural effusion
- pneumonia
- pneumothorax
- pulmonary embolism
- pulmonary hypertension

Cardiac

- arrhythmia
- CHF
- pericardial effusion
- myocardial ischemia
- valvular heart disease

Neuromuscular Disorders

- Guillain-Barre syndrome
- myasthenia gravis
- myopathy
- neuropathy

Hematologic/Metabolic

- abnormal hemoglobin: CO, methemoglobin
- acidosis
- anemia
- electrolyte disorders: calcium, phosphate, potassium
- fever/sepsis
- thyrotoxicosis

Psychogenic

- hyperventilation
- panic disorder

Miscellaneous

- de-conditioning
- diaphragm encroachment: massive ascites; pregnancy
- drug withdrawal

If the survey has not explained the underlying cause of dyspnea, consider the assessment of the patient in the approximate order presented. Some assessments are performed simultaneously:

- Evaluate pulse oximetry findings: saturations less than 90% to 92% indicate the need for oxygen, further evaluation, and probable admission. Note that oximetry does not evaluate ventilation/ CO_2 status, and a normal value does not rule out a pulmonary embolism or abnormal hemoglobin.
- In cases of respiratory depression, use non-invasive end-tidal CO_2 monitoring (capnography) if available to assess effective ventilation.
- Perform Peak Expiratory Flow Rate (PEFRs) to help diagnose/exclude reactive airway disease: normal values direct the search toward other causes; improvement of abnormal values with bronchodilator therapy is presumptive evidence of the disease.
- Obtain a chest x-ray. If abnormal, try to obtain prior x-rays for comparison to evaluate whether the findings are new or unchanged. Classic findings (ie, pneumothorax, CHF) suggest the cause of dyspnea. Indefinite findings (atelectasis, effusion, infiltrate, non-classic patterns for CHF/ARDS) need further evaluation. If the x-ray is normal, continue the assessment. Be aware that clinical presentations may be apparent before being visible in the x-ray findings. Obtain delayed x-rays as needed.
- Reconsider the risks for the cardiac diseases in the preceding list, and test appropriately: ECG, cardiac enzymes, echocardiography, stress testing. (**Vol I – PATHWAY 2 CARDIOVASCULAR EMERGENCIES**)

ADULT RESPIRATORY EMERGENCIES PATHWAY 6

- Reconsider the risks for pulmonary embolism and test appropriately: bedside echocardiography, V/Q scanning, helical CT. (See [Pulmonary Embolism, # 10 this pathway.](#))
- Obtain a blood count or hemoglobin for patients at risk of anemia.
- Obtain ABG/VBG for patients with suspected acidosis and/or ventilatory insufficiency.
- Evaluate continued unexplained hypoxia and/or distress. Consider *evolving* ARDS by thinking of its precipitating conditions. For example, is your patient at risk for aspiration?
- Consider psychogenic disorders only in a young healthy person with a normal exam, normal oximetry values, and a prior history of similar attacks. It is a diagnosis of exclusion.
- Note that deconditioning dyspnea – exertional dyspnea in obese, sedentary patients – is also a diagnosis of exclusion.

DOs and DON'Ts of DYSPNEA

DO:

- Assume that anxiety will complicate the evaluation. Deal with anxiety proactively and appropriately. Repeated honest reassurance and explicit explanations help to alleviate anxiety.
- Take explicit histories as directed in the preceding list. Some patients don't volunteer information, don't understand their disease or previous similar experiences, or do minimize symptoms. Example: Distinguish between the work-up for pulmonary embolism/deep vein thrombosis (DVT) and the actual diagnosis. Distinguish between blood clots and what may be mistaken for them, such as hematomas or varicose veins.
- A thorough exam: thrush in the mouth in someone complaining of dyspnea on exertion highly suggests *Pneumocystis jiroveci* (formerly called *Pneumocystis carinii*) pneumonia (and underlying HIV/AIDS).
- Monitor closely those patients on oxygen: while treating hypoxia, oxygen can take away the respiratory drive of COPD patients, leading to CO₂ narcosis.
- Obtain the PA and lateral chest x-ray, unless the patient is unstable and requires a portable x-ray.
- Obtain ABGs/VBGs in patients with altered mental status, suspected CO₂ retention, and suspected acidosis.
- Recognize the constraints of the ABG. The A-a gradient is accurate only on room air, and an abnormal gradient only *suggests* diseases. A normal gradient does not rule out pulmonary embolism.
- Suspect pulmonary embolism in cases with modest or equivocal findings. Unlike other serious causes of dyspnea, which present with severe symptoms, pulmonary embolism may present subtly.

DON'T:

- Assume anxiety is the cause of dyspnea.
- Treat anxiety with sedatives unless you are prepared to undergo full evaluation and full respiratory support, including intubation and mechanical ventilation.
- Obtain ABGs to rule out hypoxia if pulse oximetry is normal on room air.
- Assume infallibility of testing. Helical CTs can miss small peripheral emboli.
- Assume that pulmonary infiltrates are pneumonia. It may be impossible for radiologists to distinguish between pneumonic and embolic infiltrates.
- Assume that this episode has the same cause as the last. New conditions emerge.
- Assume that the absence of pain rules out myocardial ischemia or pulmonary embolism.
- Assume that dyspnea in pregnancy is physiologic. Pregnancy is a risk factor for pulmonary embolism and eclampsia, both of which cause dyspnea.

2

Upper Airway: General

Anatomical predictors of airways difficult to intubate include:

- limited mouth opening
- narrow mouth
- long teeth
- large tongue
- small intra-oral cavity
- poor view of posterior structures
- narrow submental angle
- submental and submandibular swelling
- deviated trachea
- neck swelling and scars

Ask the patient to describe, point to, localize the problem. Ask directed questions about symptoms in relation to breathing, coughing, and swallowing. Important clues of significant upper airway problems include stridor (audible inspiratory noise), drooling, sitting bolt upright, muffled voice or hoarseness (involvement at the level of the cords), and paradoxical movement of the torso with inspiration (the abdomen moves outward while the sternum moves inward).

Carefully inspect all structures involving the airway as many disease processes take place in the head and neck regions. Do not be satisfied with only a look in the throat. Take the extra steps involved to visualize the airway to the level of the cords either directly (laryngoscopy or fiber-optic nasopharyngoscopy) or indirectly (mirror laryngoscopy). Use topical anesthesia if necessary to gain the cooperation of the patient to obtain visualization. ([Vol II – AIR SKILLS 12 TRACHEAL FOREIGN BODY REMOVAL](#))

3 Upper Airway: Angioedema

Rapid sequence intubation may be contraindicated in patients with angioedema. Paralyzing the patient may take away the muscle tone and positioning that is keeping the airway minimally open. Awake intubation (nebulized 4% lidocaine, IV sedation, and laryngoscopy or fiberoptic intubation) is an alternative. In still more urgent situations and depending on the amount of angioedema, a supraglottic airway may be effective. Be prepared for emergent cricothyrotomy.

Angioedema (localized soft tissue swelling involving deeper layers) may be due to several different causes, which are responsive to different treatment regimens. Remember, angioedema from different causes may look the same. It may be impossible to distinguish these causes by history, especially if you are seeing the first episode. **(Vol III – AIR8 ANAPHYLAXIS)** Various kinds of angioedema and their treatments follow:

- **Acquired angioedema.** Manage the patient's airway. There will be variable to no response to epinephrine, antihistamines, and corticosteroids. Give C₁ INH (C₁ esterase inhibitor concentrate) to patients with a deficiency. Use fresh frozen plasma if C₁ INH is not available.
- **Allergic angioedema.** Manage the patient's airway. Administer epinephrine first, then antihistamines (H1 blockers), H2 blockers and corticosteroids.
- **Angiotensin-converting enzyme (ACE) inhibitor angioedema.** Manage the patient's airway. There will be poor or little response to epinephrine, antihistamines, and corticosteroids.
- **Hereditary angioedema.** Manage the patient's airway. Administer intravenous C₁INH (or fresh frozen plasma if C₁INH is not available). This condition will not respond to epinephrine, antihistamines or corticosteroids.

An approach to patients with angioedema includes the following:

- A. Airway management: the same for all entities. **(Vol I – STEP 3 INITIAL SURVEY)**
- B. Utilize O₂ as indicated. Heliox (80% helium, 20% oxygen) may help. **(Vol III – AIR3 HELIOX TREATMENT)**
- C. Unless you know that the patient has acquired or hereditary angioedema, administer the following medications:
Epinephrine IM or IV. Choice of route depends on severity.
 - IM: 0.01 mL/kg (up to 0.3 mL) of 1:1000 solution. Repeat every 5 min prn.
 - IV: 1 mg in 250 cc D5W carefully titrated to response under direct supervision while monitoring BP and cardiac rhythm.

H1 and H2 Antihistamines (give both):

Corticosteroids:

- Methylprednisolone 125 mg IV
- D. If the patient has no response to epinephrine (antihistamines and steroids take longer to act), consider glucagon 1 to 2 mg IV every 5 min titrated to effect; may need a drip at 1 to 5 mg/h. (Helpful if patient is taking B-blockers)
- E. For cases of hereditary angioedema (and those cases of acquired angioedema that have the C₁ INH), administer either:
- C₁-inhibitor concentrate: 500 to 1000 U IV **or**
 - Fresh frozen plasma 2 U IV
- F. Remove the allergen source from the patient:
- Remove the stinger from an insect bite with a blade to avoid possible injection of more allergen.
- G. Monitor patient carefully for response to therapy, adverse outcomes, and disease progression. **Call Poison Control (1-800-222-1222) for further instructions.**

4

Upper Airway: Epiglottitis (Vol III – PED3 EPIGLOTTITIS)

Adult epiglottitis (acute inflammation and swelling of the epiglottis and surrounding tissue) can have a very rapid onset with progression to airway obstruction. Do not underestimate this potential. Epiglottitis is usually caused by bacteria (*Hemophilus influenza*, group A *Streptococcus*, or *Staphylococcus*), but the epiglottis can become inflamed by other agents. How seriously ill a patient appears depends on the degree of illness at the time care is sought. In the past, seeing a thumbprint sign of a swollen epiglottis on a soft tissue lateral cervical x-ray was considered diagnostic; however, direct visualization of the actual area of involvement is now used for diagnosis. A normal appearing epiglottis and surrounding tissue structures rule out this condition.

Start the patient on ceftriaxone 1 to 2 g IV. Take blood cultures, and if the patient is intubated, take cultures of the epiglottal area. Racemic epinephrine, beta-agonists, sedation, and steroids are either not helpful or can make the condition potentially worse. Consult an ENT surgeon. If you have decided not to intubate at this time, observe the patient in intensive care unit conditions under close supervision, and be prepared to do an emergency cricothyrotomy or formal tracheostomy at any time.

5 Upper Airway: Foreign Body/Bodies

For the patient with partial obstruction or passage of the foreign body further down the pulmonary tree, continue to support the patient, closely observe for any deterioration, and arrange to have the foreign body emergently removed by bronchoscopy by a specialist. (Rigid bronchoscopy is done in the operating room under general anesthesia; flexible bronchoscopy may be done in the procedure room under topical anesthesia with or without sedation, depending on the situation.)

- Use Heliox as a temporizing measure to assist oxygenation as needed. ([Vol III – AIR3 HELIOX TREATMENT](#))
- Obtain a PA and lateral chest x-ray and examine for the foreign body and complications, such as atelectasis, mediastinal shift, air trapping, pneumothorax, or compensatory contralateral emphysema. Aspirated foreign bodies commonly lodge in the right main stem bronchus or lower lobe.
- Prepare the patient for the procedure. Pre-bronchoscopy steroids (methylprednisolone 125 mg IV) and a broad-spectrum antibiotic (cefazolin 1 g IV) may serve to reduce the complications of swelling and infection.

For complete tracheal obstruction, [see Vol II – AIR SKILLS 12 TRACHEAL FOREIGN BODY REMOVAL](#).

6 Acute Respiratory Distress Syndrome (ARDS)

ARDS is a hypoxemic respiratory failure syndrome consisting of non-cardiogenic pulmonary edema that is associated with numerous precipitating conditions including:

- infection – sepsis, pneumonia
- shock – especially septic and traumatic
- aspiration – drowning, GI aspiration
- toxic inhalation(s)
- trauma – pulmonary contusion, multiple long bone fractures, pelvic fractures
- multiple transfusions
- disseminated intravascular coagulation
- drug overdose – aspirin, heroin, barbiturates, others

Onset from time of initial insult may vary from hours to days, so most patients will become symptomatic from ARDS while being evaluated for the primary cause after admission. The stage in which ARDS presents will guide evaluation and therapy. A patient who has dyspnea in the early stages of ARDS may be difficult to diagnose, as chest x-ray changes may be initially absent. (Findings

may not indicate the changes occurring in the pulmonary tissue.) Maintain a high index of suspicion for early onset ARDS, and do not send the patient home with a history consistent with developing ARDS. These patients can deteriorate rapidly, and they have high morbidity and mortality.

Dyspnea is the main presenting symptom, and pulse oximetry guides further ABG testing for hypoxia. The chest x-ray is helpful for the diagnosis. Classic findings are bilateral diffuse alveolar/interstitial infiltrates. For patients in obvious pulmonary edema, echocardiography is helpful in differentiating ARDS from cardiogenic pulmonary edema, which is treated differently. If echocardiography is not available, other factors (besides the history of precipitating conditions) that can help differentiate this process from cardiac pulmonary edema include:

- an often normal ECG (other than tachycardia)
- no S3 gallop on cardiac exam
- no hepatojugular reflux
- no peripheral edema
- no cardiomegaly on chest x-ray (although chest x-ray is not a sensitive test for cardiomegaly)

Recent attention has been given to the blood test called brain natriuretic peptide (BNP) in differentiating CHF from other conditions.

Unlike cardiogenic pulmonary edema, there are no medications per se for this disease in its early stages. Treatment is directed at cardiopulmonary support and the underlying condition(s). Intubate and mechanically ventilate those patients with clinical signs of respiratory failure and/or hypoxemia refractory to oxygen therapy. PEEP is often utilized. **(Vol II – BREATH SKILLS 1 CHEST TUBE INSERTION, Vol II CIRC SKILLS; Vol III – AIR4 VENTILATOR MANAGEMENT)** Also utilize those portals that relate to the underlying conditions. Avoid excessive fluid administration. Even with appropriate fluid management, cardiac support may require vasopressors such as dopamine or dobutamine. Admit these high mortality patients to the intensive care unit with critical care consultation.

7

Asthma/Anaphylaxis^{1,2}

Hemodynamically stable patients with anaphylaxis involving the lower airways generally exhibit bronchospasm with accompanying wheezing. Furthermore, some patients with asthma are allergic, and these two conditions interplay. Asthma and anaphylaxis are considered here because of their similarities. (Remember, *all that wheezes is not asthma or allergies*; wheezing may also occur with CHF, foreign body, pulmonary embolism, pneumonia, and other rarer causes.)

ADULT RESPIRATORY EMERGENCIES PATHWAY 6

Anaphylaxis

This section addresses the respiratory manifestations of anaphylaxis. For a patient with anaphylaxis who is hemodynamically unstable, see [Vol III – AIR8 ANAPHYLAXIS](#).

If a patient has no history of asthma, a good history for allergen exposure and/or anaphylactoid response, and a clear upper airway but has dyspnea/wheezing, proceed as follows:

- Begin oxygen therapy if oxygen saturation is < 92%.
- Give 0.3 mL of epinephrine 1:1000IM. This dose may be repeated every 5 to 10 minutes for a total of 3 doses. An adult EpiPen® may be used.
- Treat bronchospasm with inhaled albuterol 2.5 to 5.0 mg and/or use nebulized ipratropium bromide (Atrovent) 0.5 mg if patient is on beta blockers. Repeat albuterol and/or ipratropium every 20 minutes prn for a total of three doses.
- If the patient is not improving, consider continuous nebs ([see Vol III – AIR7 STATUS ASTHMATICUS](#)).
- After initial treatment, give H1 and H2 antihistamines (diphenhydramine 50 mg PO or IV and Zantac 50 mg IV or Pepcid 20 mg IV).
- Decrease the antigen load:
 1. If an oral ingestion precipitated the attack, give activated charcoal 1 g PO or NG. (After administration of activated charcoal, give further oral medications via another route.)
 2. Stinger removal: if the anaphylactic reaction is the result of a bee sting, inspect the site to see if the stinger is still there. Lift the stinger out of the skin with a blade to avoid injecting more venom. Cold packs may decrease absorption.
- Give corticosteroids such as prednisone 60 mg PO or methylprednisolone 125 mg IV.
- Monitor the patient for worsening/no improvement. ([Vol III – AIR8 ANAPHYLAXIS](#))

Asthma

For the patient who has asthma or probably has either asthma and/or anaphylactic bronchospasm (and no other reason to wheeze, such as CHF, COPD, pulmonary embolism, foreign body exposure, pneumonia) proceed as follows:

For the potentially unstable patient who is not yet intubated, prepare for intubation while performing the following:

- Oxygen or Heliox therapy to keep saturation > 90% to 92% ([Vol III – AIR3 HELIOX TREATMENT](#))
- Continuous nebulized beta agonist therapy with albuterol, 10 to 15 mg/hour. ([Vol III – AIR7 STATUS ASTHMATICUS](#))

- Nebulized ipratropium bromide, 0.5 mg; may repeat every 20 minutes as needed for a total of three doses.
- Methylprednisolone 125 mg IV

While not routine, the following interventions may be used in an attempt to avoid the need for intubation:

- Terbutaline 0.25 mg SQ or 0.3 cc epinephrine 1:1000 IM/SQ
- Magnesium 2 g IV over 5 to 10 minutes (if no renal failure)
- Consider BiPAP (Bi-level Positive Pressure Ventilation -- [see Vol III – AIR5 NONINVASIVE VENTILATORY SUPPORT.](#))
- Proceed with rapid sequence intubation (RSI)/airway management if the patient is not improving ([Vol III – AIR1 RAPID SEQUENCE INTUBATION, AIR7 STATUS ASTHMATICUS, AIR4 VENTILATOR MANAGEMENT](#)) (**Realize that just because the upper airway is now controlled with an ETT, does not mean the patient will survive. The patient will die unless the lungs are opened.**)

When ventilating a patient with asthma, allow extra time for exhalation. Patients with severe asthma have a problem with hyperinflation (“auto-PEEP”). Ventilate the patient slowly, at a rate of 8 to 10 times/minute. Attach a PEEP valve to help to keep the lungs open and help with exhalation.

For the stable patient:

- Oxygen to keep saturation > 90% to 92%.
- Inhaled albuterol, 2.5 to 5.0 mg; repeat every 20 minutes prn for a total of three doses.
- Inhaled ipratropium bromide, 0.5 mg; repeat every 20 minutes prn for a total of three doses.
- If the patient is not improving, consider continuous nebs ([see Vol III – AIR7 STATUS ASTHMATICUS](#))
- Consider pulmonary function tests (PEFR or FEV₁) to help guide further management.
 - Asymptomatic/resolved symptoms and PEFR/FEV₁> 70% predicted: Evaluate for outpatient management. ([Vol III – AIR7 STATUS ASTHMATICUS](#))
 - Continued symptoms and PEFR/FEV₁>70% predicted: Continue inhalation therapy while reassessing for alternative conditions ([See Dyspnea, #1 this pathway.](#))
 - Continued moderate symptoms and PEFR/FEV₁ from 40% to 70% predicted: Continue inhalation therapy. Consider terbutaline 0.25 mg SQ or 0.3 cc epinephrine 1:1000 IM/SQ. Add methylprednisolone 125 mg IV and continue assessment. ([Vol III – AIR7 STATUS ASTHMATICUS](#))
 - Continued severe exacerbation and PEFR/FEV₁ from 40% to 70% predicted: Continue inhalation therapy. Consider terbutaline 0.25 mg SQ or 0.3 cc

**ADULT RESPIRATORY EMERGENCIES
PATHWAY 6**

epinephrine 1:1000 IM. Add methylprednisolone 125 mg IV. Consider magnesium 2 g IV over 20 minutes followed by a 2 g/hour drip. Consider Heliox inhalation and terbutaline IV. Closely assess response and [see Vol III – AIR7 STATUS ASTHMATICUS](#) for other considerations.

8

Chronic Obstructive Pulmonary Disease (COPD)

COPD patients often have other diseases, which precipitate and/or interact with acute exacerbations. Further, other causes of dyspnea can co-exist in the COPD patient: asthma/wheezy bronchitis, pulmonary embolism, myocardial infarction, CHF, pneumonia, lobar atelectasis, pneumothorax, and volume overload. A thorough history and associated symptoms help to focus management; previous records help to establish baseline status. Utilize other pathways and portals as necessary. As in many emergency situations, evaluation and treatment may have to proceed in tandem. Monitoring oxygenation and ventilation status is an important aspect of management of COPD exacerbations. Patients with saturation values less than 92% on pulse oximetry need treatment; however, some COPD patients are chronically hypoxic. COPD patients who retain CO₂ may be dependent on their hypoxic drive as a means to ventilate. Necessary oxygen treatment can take this hypoxic drive away, leaving the patient to become lethargic/obtunded from further CO₂ retention. Pulse oximetry does not determine the ventilation/CO₂ status. Closely observe COPD patients on oxygen. Use ABGs/VBGs to evaluate pH and ventilatory status in patients who are in severe distress, have altered mental status, and may have acidosis. ([Vol III – END/M6 ACID-BASE](#)) Whereas COPD patients may have a baseline compensatory respiratory acidosis (elevated pCO₂ with a normal pH), acute respiratory acidosis reflects decompensation from their baseline, and metabolic acidosis can signal impending cardio-respiratory failure. Recent guidelines suggest that acute spirometry does not have a role in the *emergency* treatment of COPD (unlike its role in a primary care setting).

Routine testing besides ABGs/VBGs that is helpful in the acute setting of COPD includes the chest x-ray and the ECG. Multifocal atrial tachycardia (MAT) is commonly associated with COPD and is often confused with atrial fibrillation. However, this dysrhythmia is often caused by hypoxia and is not treated with anti-arrhythmics but by reversing the hypoxia. Otherwise, laboratory testing is directed to specific needs, such as a theophylline level if patients have tremors, have arrhythmias while on aminophylline, or are vomiting.

Initial treatment of acute exacerbations of COPD in patients not intubated includes:

- Oxygen to keep saturations > 90%; closely observe for obtundation.
- Inhaled albuterol: 2.5 to 5 mg via nebulizer or MDI with spacer; may repeat every 20 min or sooner prn. (For continuous nebulization, [see Vol III – AIR7 STATUS ASTHMATICUS.](#))
- Inhaled ipratropium: 0.5 mg via nebulizer or MDI with spacer; may repeat every 20 min prn.
- Corticosteroids: prednisone 40 to 60 mg PO or methylprednisolone 125 mg IV.
- Early use of non-invasive ventilation support (NIV) in moderate-to-severe dyspnea and tachypnea may prevent the need for intubation and mechanical ventilation. ([Vol III – AIR5 NONINVASIVE VENTILATORY SUPPORT](#))

9

Pneumonia and Aspiration

Pneumonia

1. Triage the patient with cough and fever appropriately for the safety and well being of the staff (and the community at large). Place a mask on the patient. Minimize exposure to others.
2. Obtain a chest x-ray.
3. For a patient with history of cough longer than 2 weeks associated with night sweats, weight loss, fever, or a cough associated with HIV/AIDS, IV drug use, recent incarceration, homelessness, immigration from a developing country, or history of tuberculosis / tuberculosis exposure, take appropriate precautions: Place patient in an isolation room with respiratory precautions until the chest x-ray is read for tuberculosis or, when HIV/AIDS is suspected, until sputum culture results are read. (The chest x-ray can be normal in HIV/AIDS patients with active tuberculosis.)

Many diseases can mimic pneumonia, as many conditions present with a combination of respiratory symptoms, fever, and infiltrate on chest x-ray. Common conditions include CHF, pulmonary embolism, COPD exacerbation, and aspiration. Ascertain risk factors for pulmonary embolism and investigate accordingly. Furthermore, symptoms of pneumonia, including elevation of the temperature, may be absent in geriatric or debilitated patients. Patients with suspected pneumonia and normal oral temperatures should have a rectal temperature taken. The history and physical should be conducted with HIV/AIDS in mind, as these patients are at risk for tuberculosis and *Pneumocystis jiroveci* pneumonia (formerly called *Pneumocystis carinii*), which require a different approach to treatment. Oral thrush in this setting is highly suggestive of HIV-related *Pneumocystis* pneumonia.

ADULT RESPIRATORY EMERGENCIES PATHWAY 6

In the face of pneumonia, two main management decisions face the team:

1. Which patients to admit and where, and
2. Which antibiotic(s) to use initially (empirically).

Admission Considerations

History is paramount in the management of pneumonia. Criteria for patients at high risk and needing admission include:

- geriatric patients
- debilitated patients/poor nutritional status
- patients with alcoholism
- IV drug abusers (Also, investigate for endocarditis.)
- patients with underlying diseases such as diabetes, CHF, renal failure, liver disease, CVA, anemia, neuromuscular disease
- immunosuppressed/compromised patients: steroid use, immunosuppression agents, chemotherapy agents, HIV/ AIDS, sickle-cell disease, asplenia/splenectomy, connective tissue disorders, neoplastic disease, organ/bone marrow transplants
- resistant microorganisms (recent hospitalization, nursing home residence, recent antibiotic use)
- social situation: caregiver, transportation, communication, financial considerations
- altered mental status
- hypoxia
- tachypnea (> 30 breaths per minute)
- hypotension
- vomiting/ unable to keep food down
- chest x-ray with multi-lobar disease, effusions, and/or cavitations
- extrapulmonary infection
- metabolic acidosis

Consider using an objective clinical prediction scale, such as the CURB-65 score³:

- Confusion (based upon a specific mental test or new disorientation to person, place, or time)
- Urea (blood urea nitrogen in the United States) > 7 mmol/L (20 mg/dL)
- Respiratory rate \geq 30 breaths/minute
- Blood pressure (BP, systolic <90 mm Hg or diastolic \leq 60 mm Hg)
- Age \geq 65 years

Each item is assigned 1 point. Patients with a total score of 0 to 1 can usually be treated as outpatients; patients with scores of 2 or higher should be admitted, with ICU care considered for scores of 4 or 5.

Direct your testing to specific situations, and don't delay antibiotic treatment in acutely ill patients with pneumonia because of testing.

- Chest x-ray is essential for diagnosis and management. Chest x-ray uncovers not only the pneumonia but also high-risk findings such as multi-lobar disease, effusions, and cavitations. Therefore, do not treat without one. The chest x-ray has its limitations. Clinical findings may be apparent before, and even a radiologist may be unable to distinguish between the infiltrates of pneumonia and pulmonary embolism. (Note that a V/Q scan is also unlikely to make this distinction.)
- ABGs are useful in evaluating hypoxic patients with underlying pulmonary disease, patients with altered mental status, patients suspected of having metabolic acidosis, and patients with *Pneumocystis* pneumonia.
- The white blood cell count may be misleading, especially for patients who are immunosuppressed.
- Patients who are seriously ill need at least a hematocrit, glucose, sodium, and pH.
- Blood cultures are recommended on hospitalized patients (although they have limited yield). Sputum culture and urine testing for Legionella and pneumococcal antigens have also been recommended for more seriously ill patients and those with co-morbidities.

Use your judgment in discharging low-risk patients with pneumonia to outpatient management. Factor in their psychosocial situation. Home IV therapy may be appropriate in certain situations.

Admit acutely ill patients to the appropriate level of care using your clinical judgment. Patients with HIV/AIDS who are admitted need to be placed in isolation until tuberculosis has been ruled out.

When dealing with immunocompromised patients in rural or community hospital settings, it is imperative to consult as early as possible with the patient's oncologist, transplant center, or other specialty service.

Treatment Considerations

- Use oxygen to keep saturations > 92%.
- Treat bronchospasm with inhaled albuterol: 2.5 mg via nebulization or MDI with spacer.
- Watch closely for worsening respiratory status.
- Monitor hydration status carefully. Treat dehydration with IV fluids. If a patient is in shock, give a bolus of crystalloid (NS or LR). Guide fluid resuscitation based on patient response, urinary output, and inferior vena cava (IVC) measurement if ultrasound available. (**Vol II – CIRC SKILLS 1 ARTERIAL AND VENOUS CATHETER INSERTION**). Avoid over-hydration and possible pulmonary edema.

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- If a pleural effusion is present, consider performing a thoracentesis. Empyemas (pus in the pleural space) need placement of a chest tube and ongoing drainage. Note: this is not an emergency procedure, should not interfere with ongoing evaluation and treatment, and can be done hours after antibiotics have been started.
- Promptly administer empiric antibiotic therapy. No combination of criteria available in the initial investigation period can reliably establish the cause of pneumonia with perhaps the exception of the findings of anaerobic lung abscess on chest x-ray (cavity with an air-fluid level) or a classic tuberculosis chest x-ray (scarring, apical cavitation, hilar adenopathy). One consequence is that the terms *typical* and *atypical* as applied to pneumonia are being abandoned except when they refer to antibiotic susceptibility. Empiric therapy covers microorganisms for both. Account for where the patient resides, disposition, and the antibiotic resistance patterns within your locale. (Note that the distinction between community-acquired and healthcare-associated pneumonia may be open to debate, depending on how long the patient has been away from a hospital or nursing home setting.)
- Outpatient setting antibiotic selection (non-immunocompromised): choose between a macrolide, a fluoroquinolone, or doxycycline. Optimal duration of coverage remains unclear.
- Inpatient setting antibiotic selection: see antibiotic management in [Vol III – IN1 ADULT PNEUMONIA](#).

Aspiration

Aspiration is foreign material entering the respiratory system. What happens after that depends on the nature of the aspirate, how much has been aspirated, and how the body reacts to this insult. Micro-aspiration is a common event, usually handled well by bodily mechanisms. Those micro-aspiration events that are not handled well, along with macro-aspiration events, give rise to pneumonic reactions generally referred to as pneumonia or pneumonitis. This usually occurs in patients with impaired airway protection.

The two main categories of aspiration are infectious and chemical pneumonitis. If distinguishable, they are treated differently:

Infectious pneumonitis in a community setting is usually caused by aerobic and anaerobic oropharyngeal flora. In a hospital or nursing home setting, gram-negative and resistant *Staphylococcus* organisms also need to be considered. Complications include lung abscess and/or empyema. Treatment is covered under pneumonia above and in [Vol III – IN1 ADULT PNEUMONIA](#).

Chemical pneumonitis is caused by aspirating gastric contents. This may or may not include particulate matter. Large volumes and/or high acidity worsen the reaction and consequences, which can include mechanical obstruction, bronchospasm, ARDS (see #6 ARDS in this pathway), respiratory failure, and nosocomial bacterial pneumonia. Mortality may be high.

Treatment consists of:

- removal of obstructions by tracheobronchial suction/bronchoscopy
- bronchodilator agents for bronchospasm
- respiratory support with oxygen; CPAP and intubation are utilized if needed. Mechanically ventilated patients may benefit from PEEP.
- antibiotics appropriate to the setting and clinical presentation

Ventilator-Associated Pneumonia Prevention

Protect patients at risk for gastric aspiration.

- Intubate the airway if it can't be maintained and protected by the patient.
- Elevate the head of the bed to a 30- to 45-degree angle, or reverse trendelenburg if possible.
- Suction as needed. Place OG tube and empty stomach and then low intermittent suction.
- Maintain proper oral care.
- Decrease gastric activity with antacids/H2 blockers.
- Gastric ulcer prophylaxis.
- Feed through an NG or gastric tube, if necessary. Check residual gastric volume regularly.

10

Pulmonary Embolism

Pulmonary embolism is common and highly variable in its presentation. While diagnosing pulmonary embolism may be straightforward, diagnosis may also be difficult as pulmonary embolism may mimic other diseases. Pulmonary embolism may be fatal in healthy people who don't appear critically ill.

Pulmonary embolism may be a co-morbid illness. The patient may appear well or be in full cardiac arrest. Blood clots may be difficult to detect both at their source (often the deep veins of the legs) and in their final destination, clogging the pulmonary vascular system and interfering with gas exchange. Maintain a high index of suspicion for pulmonary embolism and take unexplained and/or isolated dyspnea seriously.

Risk factors are important in the diagnostic process. Risk factors include recent immobilization, lower extremity immobilization/cast, a recent trip in a sitting position longer than 4 hours, history of DVT or pulmonary embolism, family

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history of thromboembolic disease, recent surgery or trauma, indwelling venous catheters, history of cancer, advanced age, heart failure, and obesity. These factors along with the acute history and examination form the basis for the diagnosis.

Symptoms associated with pulmonary embolism include dyspnea, anxiety, cough (may be bloody), syncope, new wheezing, chest pain (usually pleuritic in nature), and others.

Physical findings associated with pulmonary embolism include: fever (although a rectal temp higher than 102°F is unlikely in pulmonary embolism), tachycardia, Kussmaul's sign (neck vein distension with inspiration, signifying an increase in right-sided CVP), an accentuated second heart sound, pulmonary rales, wheezing, unilateral leg swelling, calf tenderness, palpable venous cords (especially in the popliteal fossa), or a leg in a cast. Pulmonary embolism is a cause of obstructive shock. Use of Ultrasound to check for right heart strain. Check for an elevated CVP (>12 cm H₂O) in the ominous context of hypotension. **(Vol II – CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT)**

Even with all these factors absent, the patient may still have a significant pulmonary embolism, which can quickly progress to death. Patients may even be therapeutically anticoagulated and still have pulmonary embolism. With such a wide variation in its presentation, the differential diagnosis list is extensive. Clinical variables alone cannot guide a treatment decision. Because of this and the fact that pulmonary embolism is a common cause of unexpected death in most age groups, diagnostic testing for patients at risk for pulmonary embolism continues until it is ruled in or out or another diagnosis is clear.

Diagnostic Testing Considerations

- Form the best clinical impression from the history and physical examination and consider the patient to be in one of two categories: low or high probability of having pulmonary embolism. Diagnostic tools such as the Wells criteria¹ (reproduced at the end of this section on pulmonary embolism) are useful for assessing the pre-test probability of pulmonary embolus. While this may be useful in interpretation of results, many patients fall into the moderate risk category in which a definitive diagnosis is almost entirely dependent on laboratory and imaging results.
- No blood test changes the clinical likelihood category of the patient. Quantitative D-dimer testing has a reported sensitivity of > 90% in detection of venous thrombosis and pulmonary embolus.² Used in conjunction with imaging studies (CT, V/Q scan, and/or venous ultrasound and Doppler), a quantitative D-dimer level may be helpful in interpreting equivocal results.

While some advocate performing this test prior to deciding on imaging studies,⁴ it may be more practical to perform the test concurrently with imaging studies.

- While ABGs may be helpful, the pO₂, oxygen saturation levels, and the alveolar-arterial gradient have no predictive value for pulmonary embolism in the typical population of patients suspected to have pulmonary embolism. These values can all be normal in pulmonary embolism.
- Chest x-ray is mandatory but non-diagnostic for pulmonary embolism. (Even radiologists cannot distinguish a pulmonary embolism infiltrate from pneumonia.) Chest x-ray helps to rule in or out other causes and interpret other findings. Common pulmonary embolism findings are infiltrate and effusion. If an infiltrate is seen, a subsequent V/Q scan will be non-diagnostic, so go directly to another diagnostic test such as an enhanced helical CT scan or angiography.
- Helical (spiral) CT with contrast or CT angiography has become the most widely used modality for the diagnosis of pulmonary embolus. Contraindications are limited to contrast allergy, inability to tolerate a contrast load due to renal insufficiency, or concerns about radiation exposure in early pregnancy. As there are a number of chest CT scanning protocols, make certain that the CT technician and radiologist know that the scan is being performed for diagnosis of pulmonary embolism.
- The ventilation-perfusion scan (V/Q scan) is an important diagnostic modality. (Note that the scan cannot distinguish pulmonary embolism from pneumonia.) To use this test, you must categorize the patient's risk into either high or low probability of having pulmonary embolism before getting the test. Test results should be reported to you as either diagnostic or non-diagnostic.
- **Diagnostic scans** come in one of two forms, both of which may be used to guide clinical management if they are consistent with your pretest clinical risk assessment.
 - **Normal scan** means there are no perfusion defects noted. If the pretest probability was low, chances are good that the patient does not have pulmonary embolism. (Note that a small number of patients can have pulmonary embolism, even with a normal test). If the pretest probability was high, you need to investigate further for pulmonary embolism.
 - **High probability scan:** One of numerous defect patterns that highly suggest pulmonary embolism. If the pretest probability was low, get another confirmatory test (D-dimer and/or venous ultrasound), especially if the patient is not an ideal candidate for fibrinolytics. If the pretest probability was high, begin treatment for pulmonary embolism.
- **Non-diagnostic scan:** One of numerous defect patterns that is equivocal. (This category includes the former categories of low and intermediate

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probability of PE.) An important concept is that a non-diagnostic scan is not a diagnostic endpoint. A definitive test must be performed for either pulmonary embolism or an alternative diagnosis.

- Duplex ultrasound, which means concurrent imaging and Doppler flow recording, of the lower extremities is useful when positive: Demonstrating the presence of DVT virtually proves the diagnosis of pulmonary embolism. A negative study does not rule out pulmonary embolism or markedly reduce its probability.
- Pulmonary angiography remains the most definitive test for pulmonary embolism diagnosis; however the advent of CT scanning for pulmonary embolism has to a large extent rendered angiography unnecessary. A positive study provides certainty; a negative study properly done provides greater than 90% certainty that pulmonary embolism doesn't exist. (A negative study can't visualize the really small emboli.)
- Echocardiography – transthoracic or transesophageal – can identify right heart strain (RV hypokinesis and/or dilatation, tricuspid regurgitation), pulmonary hypertension, or large clots in the primary branches of the pulmonary tree. Bedside ultrasound may show evidence of right ventricular enlargement due to a major occlusion of a pulmonary artery. As a rule, the right ventricle should normally appear smaller than the left on apical 4-chamber view.⁵
- ECG testing is rarely helpful. The classic right heart strain pattern shows up infrequently and is only suggestive, not diagnostic, of pulmonary embolism. Massive pulmonary embolism may be associated with symmetric T wave inversion in V1-4.

Management Considerations

- A. Administer oxygen therapy to all patients suspected of having pulmonary embolism, as increased alveolar oxygen may promote pulmonary vasculature vasodilatation.
- B. Intubate and mechanically ventilate if there are signs of respiratory failure. **(Vol II – AIR SKILLS; Vol III – AIR 4 VENTILATOR MANAGEMENT)**
- C. Support circulation using CVP measurements as a guide. **(Vol II – CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT)**
 1. Without invasive monitoring or a cautious fluid trial and measurement, you can't know whether the preload of additional fluid will help or harm a heart struggling to pump against high outflow pressures caused by an obstruction. If the patient's condition worsens, stop immediately. Note that improvement with fluids does not mean that the patient is stabilizing; the primary problem hasn't changed.
 2. Bedside ultrasound or echocardiography is useful in the hemodynamically unstable patient whose heart might be failing from obstructive shock generated by pulmonary embolism.

3. Patients in cardiac arrest because of pulmonary embolism will not respond to CPR and treatment suggested by the ACLS protocols. Obstruction to oxygenated blood flow to the heart (and brain) is the cause. Full dose fibrinolytics given as a bolus, and then continuous CPR (use of a mechanical device would be helpful), may be helpful. (Emergency cardiopulmonary bypass can provide a bridge to operative embolectomy. Short of that, bilateral thoracotomy with pulmonary artery massage may be of some benefit. Though both would be futile in a rural setting).
- D. Medication mainstays in the treatment of pulmonary embolism are anticoagulants and fibrinolytics. Be familiar with the precautions, risks, and contraindications of these agents before using them.
1. Upon suspicion of DVT or pulmonary embolism, start anticoagulation with one of the following drugs in order to inhibit clot extension. (Note that the drugs do not dissolve the clot.)
 - Full-dose low molecular weight heparin (LMWH) enoxaparin (Lovenox): 1 mg/kg SQ every 12 hours. (Alternatives are dalteparin or fondaparinux SQ.) No need for coagulation studies.
 - Full dose heparin IV (different from cardiac dose heparin) can be used but can be difficult to titrate. (Note that heparin is used differently in certain clinical states involving fibrinolytics.) It may be given either concurrently with fibrinolytic therapy for pulmonary embolism, or it can be held and restarted after fibrinolytic has been given.
 2. Consider fibrinolytics in the following circumstances (assuming there are no contraindications to fibrinolytics).
 - hemodynamically unstable patients with proven pulmonary embolism (Give immediately.)
 - pulmonary embolism patients with right heart strain and exhausted cardiopulmonary reserves
 - empiric treatment in unstable/deteriorating patients with highly suspected pulmonary embolism (and unlikely aortic dissection) who won't survive long enough to get a confirmatory study
 - proven massive pulmonary embolismQuicker-acting agents such as alteplase or reteplase are preferred. (Others that have been used include urokinase and streptokinase.)
 - **Alteplase:** 100 mg IV over 2 hours. (An accelerated, 90-minute schedule using the same amount but based on body weight has also been used.)
 - **Reteplase:** 10 units IV bolus and repeat in 30 minutes. In cardiac arrest situations, the 20 units have been given at once.Anticoagulants are discontinued during fibrinolytic administration.

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E. Consultation should not delay diagnosis or therapy. The team leader ultimately makes the decision to start fibrinolytic and/or anticoagulation therapy. Interventional radiologists may be helpful for directed fibrinolysis. Emergency surgical thromboembolectomy is indicated in those cases where fibrinolysis fails, is not tolerated, or is contraindicated.

Pulmonary Embolism Caveats

- When diagnosed, DVT is believed to signify pulmonary embolism. DVT below the knee is just as important as above the knee.
- Pulmonary embolism is common during pregnancy and post partum for several months. Diagnose and treat a patient with pulmonary embolism who is pregnant the same as you would a non-pregnant patient.
- Pulmonary embolism is more common as age increases. Do not dismiss respiratory symptoms in the geriatric patients.

Wells Clinical Prediction Rule for Pulmonary Embolism (PE)

Clinical Feature	Points
Clinical symptoms of DVT	3
Other diagnosis less likely than PE	3
Heart rate >100 bpm	1.5
Immobilization/surgery within past 4 weeks	1.5
Previous DVT or PE	1.5
Hemoptysis	1
Malignancy	1

Total points

PE=pulmonary embolism; DVT=deep vein thrombosis.

Risk score interpretation (probability of PE):

- >6 points: high risk (PE incidence 78.4%)
- 2 to 6 points: moderate risk (PE incidence 27.8%)
- <2 points: low risk (PE incidence 3.4%)

11 Pneumothorax and Pneumomediastinum

Tension pneumothorax is addressed in **Vol I – STEP 3 INITIAL SURVEY**.

Traumatic pneumothorax treatment is straightforward: Insert a chest tube. (**Vol II – BREATH SKILLS 1 CHEST TUBE INSERTION**)

Nontraumatic (or spontaneous) pneumothorax occurs when a bleb on the surface of the lung ruptures and air escapes into the negative pressure pleural space. The gas pressure equilibrates across the tear, which may or may not seal. Vital capacity and PaO₂ tend to decrease. This situation tends to be more serious or life threatening when the underlying lung is not normal (such as in COPD), when the pneumothorax is large, and/or when the patient is geriatric. Usually the patient is at rest when this happens. Symptoms may vary, and the patient may not come in for days. Therefore, this clinical condition may vary considerably on presentation. Symptoms may include ipsilateral chest pain and dyspnea.

The chest x-ray is usually diagnostic. Expiratory films may help to see a small pneumothorax. It may be difficult to estimate the size of the pneumothorax. Base therapy on the overall clinical status of the patient.

Management includes interventions to re-expand the lung. Patients who have waited a number of days before seeking treatment may be at increased risk for pulmonary edema when the lung is re-expanded. (How suction is used may play a role in re-expansion pulmonary edema.)

1. Give oxygen: increased O₂ saturation speeds the re-absorption of the free pleural air.
2. Ensure hemodynamic stability before proceeding with expansion attempts.
3. Consider sedation and analgesia needs before placing a chest tube. (**Vol III – SED1 PROCEDURAL SEDATION**)
4. Re-expansion treatment varies depending on factors such as the baseline status of the underlying lung prior to the pneumothorax, whether the patient is symptomatic, and the size of the pneumothorax.
 - If lungs are normal, there are no symptoms, and the pneumothorax is < 15%, consider observation without invasive intervention.
 - If lungs are normal, the pneumothorax is <15%, but the patient is symptomatic, consider needle thoracostomy.
 - If the lungs are normal, the pneumothorax is >15%, and the patient is symptomatic, proceed with tube thoracostomy. (**Vol II – BREATH SKILLS 1 CHEST TUBE INSERTION**)

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- If lungs are abnormal, proceed with tube thoracostomy. (**Vol II – BREATH SKILLS 1 CHEST TUBE INSERTION**)
5. Connect the chest tube to an underwater-seal drainage apparatus or a Heimlich valve (rather than a suction device) in order to avoid re-expansion pulmonary edema.
 6. Obtain a repeat chest x-ray to confirm placement and lung re-expansion.
 7. Arrange for pleurodesis by a consultant in cases with underlying abnormal lungs.
 8. Consider prophylactic antibiotics depending on underlying condition(s).

Nontraumatic pneumomediastinum usually occurs when intrathoracic pressures become elevated due to conditions such as coughing, asthma, vomiting, Valsalva maneuvers, smoking crack cocaine, childbirth, angioedema, and seizures. Perivascular alveoli rupture and air dissects into connective tissues, travels to the mediastinum, and then tracks superiorly. Once the air reaches the neck spaces, it can spread diffusely through the body's subcutaneous tissues (crepitus may be felt subcutaneously). Patients may or may not have symptoms such as dyspnea or chest pain that radiates to the back and neck and is worsened by cough or deep breathing. It is usually benign and self-limited in the absence of serious comorbid disease; however, most patients are admitted for observation of signs of serious complications. These follow:

- pneumothorax (free air can rupture through the mediastinal parietal pleura)
- mediastinitis
- malignant pneumomediastinum (pressure high enough to cause cardiopulmonary failure)

In contrast, pneumomediastinum due to esophageal rupture has high mortality. This may occur under similar conditions, such as vomiting, forceful swallowing, straining, and weight lifting. Also ask about any recent medical procedures involving the upper GI system, suggesting iatrogenic perforation with delayed symptoms. Presentations vary from mild to acutely ill and septic. Symptoms include dyspnea, chest pain, abdominal pain, and vomiting blood. Physical findings include fever, tachycardia, tachypnea, decreased breath sounds, crunching heart sounds, and palpable subcutaneous emphysema. Chest x-ray findings are suggestive but usually non-diagnostic and can include pneumomediastinum, pleural effusion, pneumothorax, mediastinal widening, air-fluid levels, and mediastinal emphysema. Early diagnosis and treatment is crucial. Emergency treatment consists of:

- Airway and ventilation management
- Hemodynamic stabilization considerations
- Early consultation during the diagnostic work-up to ascertain esophageal perforation, which may include staging gastrografin swallows, endoscopy, non-contrast CT, and/or barium esophagography.

- Once the diagnosis is established, antibiotics and an emergency cardiothoracic surgical referral for esophageal repair are indicated.

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Massive Hemoptysis (Vol II – BREATH SKILLS 3 ENDOBRONCHIAL TUBE)

Quantifying the amount of blood that a patient coughs up can be somewhat arbitrary. For the patient with active/massive/gross bleeding from the respiratory system, or respiratory bleeding with respiratory insufficiency, or respiratory bleeding and unstable hemodynamics:

1. Orotracheally intubate with largest ET tube possible. (Vol II – AIR SKILLS 3 OROTRACHEAL INTUBATION) If left-sided bleeding is suspected, do not place ET tube in the right main stem bronchus because of risk of occluding the right upper lobe bronchus. In right-sided bleeding, it may be beneficial if possible to place the ET tube into the left main stem bronchus for selective ventilation.
2. Suction blood from the tracheobronchial tree to improve oxygenation.
3. Resuscitate with crystalloid (NS or LR) 1 to 2 L IV; order and use blood as necessary.
4. Call for stat pulmonary/thoracic surgery consult.
 - Bronchoscopy is performed to maintain ventilation and direct endobronchial blockade. (Rigid bronchoscopy may be better for massive bleeding.)
 - When bleeding has been isolated, choices for definitive therapy include embolization, surgical resection, or both.
 - Prepare the patient with these end goals in mind.
5. Chest x-ray after intubation.

For a patient who is stable in whom you suspect pulmonary embolism as a cause of bleeding, [see #10 this portal](#). For the other stable patients, obtain a chest x-ray and continue to evaluate for the possible cause/site of bleeding.

Many underlying disorders can cause blood from the respiratory tract:

- cancer
- coagulopathy
- drugs (anticoagulants, others)
- iatrogenic causes
- idiopathic causes
- infection (bronchitis, tuberculosis, pneumonia, fungal, others)
- heart problems (mitral stenosis, others)
- lung disease (bronchiectasis, COPD, others)
- trauma
- vascular disorders (pulmonary embolism, pulmonary AV malformation, pulmonary infarction, others)

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Further work-up may include CT scan or pulmonary angiography.

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PATHWAY 7: RESPIRATORY EMERGENCIES (PEDIATRIC)

The team continues the resuscitation along the pathway suggested by the initial clinical impression. Each pathway includes a complete, thorough, and rapid physical examination with additional history taking. The team leader is wary of conditions that may not be apparent. To obtain additional clinical data or to correct a missed or newly developed condition, the team leader repeats the initial survey if the patient is not responding satisfactorily.

PEDS: Because this entire pathway pertains only to pediatric patients, the convention of underlining has been omitted.

For the purposes of this survey, the pediatric age group ranges from infancy to the attainment of adult size.

Children are not small adults. For further general discussion of this issue, [see Vol III – PED1 PHYSIOLOGIC AND ANATOMIC CONSIDERATIONS.](#)

Topics not covered here but found in Pathway 6 Adult Respiratory (Vol I – PATHWAY 6 ADULT RESPIRATORY) may provide immediate insight into respiratory management, especially for older/larger pediatric patients.

Number	Diagnosis/ Condition	Related Materials
1	Foreign Body/Obstructed Airway	Vol II – AIR SKILLS 12 TRACHEAL FOREIGN BODY REMOVAL, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION
2	Retropharyngeal or Peritonsillar Abscess	Vol II – AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 4 RAPID SEQUENCE INTUBATION, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS
3	Diphtheria	Vol II – AIR SKILLS 1 AIDS TO INTUBATION, AIR SKILLS 4 RAPID SEQUENCE INTUBATION, AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS

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4	Epiglottitis	Vol III – PED3 EPIGLOTTITIS; Vol II – AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION
5	Laryngeal Edema Post Extubation	Vol III – AIR3 HELIOX TREATMENT; Vol II – AIR SKILLS 1 AIDS TO INTUBATION, AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS
6	Anaphylaxis and Angioedema	Vol III – AIR8 ANAPHYLAXIS; Vol II – AIR SKILLS 1 AIDS TO INTUBATION, AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION
7	Laryngotracheobronchitis (Croup)	Vol III – PED4 LARYNGOTRACHEL BRONCHITIS; Vol II – AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION; Vol III – AIR3 HELIOX TREATMENT
8	Bacterial Tracheitis	Vol III – PED5 BACTERIAL TRACHEITIS; Vol II – AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION; Vol III – AIR3 HELIOX TREATMENT
9	Bronchiolitis	Vol III – PED6 BRONCHIOLITIS; Vol II – AIR SKILLS PORTALS
10	Pneumonia	Vol III – PED7 PNEUMONIA; Vol II – AIR SKILLS PORTALS

11	Asthma	Vol III – AIR7 STATUS ASTHMATICUS, AIR3 HELIOX TREATMENT, AIR4 VENTILATOR MANAGEMENT; Vol II – AIR SKILLS
12	Drowning	Vol I – PATHWAY 9 PEDIATRIC TRAUMA, PATHWAY 1 ALTERED LEVEL OF CONSCIOUSNESS; Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION; Vol III – AIR4 VENTILATOR MANAGEMENT
13	Acute Respiratory Distress Syndrome	Vol I – PATHWAY 6, #6

1 Foreign Body/Obstructed Airway

Children constantly put objects in their mouths, leading to frequent occurrence of foreign body (FB) aspiration. Strongly consider FB airway obstruction with a history of sudden onset of respiratory difficulty, especially in a previously healthy child. Also consider this diagnosis in a non-resolving respiratory disorder.

If the situation is life threatening or there is a complete obstruction, removal of the object must be attempted. An aggressive approach may be necessary to establish an adequate airway.

Initial Management

1. Initially follow BLS guidelines (Heimlich maneuver/abdominal or chest thrusts, back blows for infants, CPR if patient becomes unresponsive), while preparing for FB removal. (Vol II – AIR SKILLS 12 TRACHEAL FOREIGN BODY REMOVAL)
2. Perform laryngoscopy and attempt removal of the foreign body using a Magill or other long forceps. If the FB is not visible, it may be past the cords.
3. If laryngoscopy and FB removal is unsuccessful, try removing the FB using a meconium aspirator, wall suction set at the maximum setting, and a cut-off ET tube. Insert the ET tube until you feel a slight pressure. A meconium aspirator is attached to the cut-off ET tube and to the suction tubing connected to the wall suction. Suction is applied as the ET tube is removed. Even if the FB is not extracted, reattempt ventilation as the FB may have been dislodged or pushed into the right main stem bronchus. If the FB is now in the right main stem bronchus, the patient's left lung can still be ventilated.

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4. If these measures fail, a surgical airway may be necessary. These include TTNV and tracheotomy. (**Vol II – AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION**) The success or failure in removing a FB utilizing a surgical airway will depend on the location of the FB in the airway.

If the FB is not removed but the patient is able to breathe (because of partial obstruction or passage of the FB further down the pulmonary tree), closely observe the patient while arranging for emergent rigid bronchoscopy in the operating room. Though flexible bronchoscopy doesn't need general anesthesia and is useful for diagnosis of FB, it doesn't have as much capability for FB removal as rigid bronchoscopy. Continue to support the patient, closely observing for any deterioration:

- Use Heliox as a temporizing measure to assist oxygenation as needed. (**Vol III – AIR3 HELIOX TREATMENT**)
- Obtain a PA and lateral chest x-ray to identify the presence of an FB or complications such as atelectasis, mediastinal shift, air trapping, pneumothorax, or compensatory contralateral emphysema. Aspirated FBs commonly lodge in the right main stem bronchus or lower lobe.
- Prepare the patient for the procedure. Administering a broad-spectrum antibiotic may not be indicated for infection prior to the procedure, but the antibiotic may be given post procedure, depending on what is visualized. Steroids to limit edema from the FB and the procedural manipulations are debatable; discuss this with an endoscopist.

If the FB has caused substantial breathing difficulty, observe the child after FB removal for development of pulmonary edema or ARDS (**see #13 this portal, ARDS**).

2

Retropharyngeal or Peritonsillar Abscess

Patients may present with pain, fever, difficulty swallowing, trismus, drooling, fetid breath, neck stiffness/swelling, and voice changes. On exam, the mouth may not fully open. Red and swollen posterior soft tissue structures may be fluctuant and may be encroaching on or occluding the upper airway. Do not palpate a retropharyngeal bulge. The uvula may be displaced. The patient may be dehydrated due to decreased oral intake and fever-related losses.

Diagnosis may be complicated by patients being unable to open their mouths. Lateral soft tissue cervical x-rays may not definitively show evidence of retropharyngeal abscess as demonstrated by widening prevertebral space, gas, or air fluid level. In a stable patient, if extension of infection is suspected, the diagnosis may be facilitated by either intraoral ultrasound or CT scan.

Management Considerations

- The primary goal is to maintain a secure airway. If intubation is indicated, take care not to rupture the abscess. Rupture may cause possible aspiration, dissemination, and/or increased mortality. (**Vol II – AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 4 RAPID SEQUENCE INTUBATION, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS**)
- Replace fluid deficits with crystalloid (NS or LR).
- Start antibiotic coverage prior to drainage procedures: in both of these conditions the microorganism flora is mixed aerobes and anaerobes. Antibiotic regimens will vary depending on your location and local resistance of organisms in you locale. Refer to local infectious disease specialist or a reference such as “The Sanford Guide to Antimicrobial Therapy”.
- In penicillin-allergic patients, the antibiotic will be different.
- The definitive treatment for an abscess is incision and drainage, which should be performed in a controlled setting such as the operating room. (Before treating, be sure the abscess is present.) An exception may be the cooperative, non-toxic/non-septicemic patient with a peritonsillar abscess that has no deep neck extension of the infection and can be needle aspirated using a 19-gauge, depth-protected needle on a 10 cc syringe following topical anesthetic spray and submucosal lidocaine injection. The aspirate should be sent for culture.

3

Diphtheria

Unvaccinated children are at risk for diphtheria, a toxin-mediated disease (caused by *Corynebacterium diphtheriae*), which generally presents as infection of the tonsils and pharynx. A gray-brown, adherent, leather-like pseudo-membrane (a dense coagulum of necrotic debris) can form and obstruct the airway. This membrane can also form lower in the larynx. Peripharyngeal swelling can be significant. The exotoxin affects many different parts of the body; of particular importance during an emergency situation is its paralytic effect on the palate and hypopharynx (further compromising the airway), its effect on the myocardium (inflammation/myonecrosis, dysrhythmias, heart block, CHF), and various neurologic complications.

If diphtheria is suspected, begin treatment. Confirmation is by swab and special culture techniques. (Alert the lab that special cultures are being sent.) Testing is also recommended for toxigenicity.

Initial Management

1. Establish control of the airway. (**Vol II – Air Skills 1 Aids to Intubation, Air Skills 13 Cricothyrotomy, Air Skills 4 Rapid Sequence Intubation**)
With laryngeal involvement, tracheostomy may be needed. (**Vol II – Air Skills 14 Tracheotomy, Air Skills 15 Tracheotomy in Infants**)
2. Replenish fluids.
3. Determine cardiac involvement (ECG, monitoring, serum troponin) and potential for circulatory collapse.
4. Give antitoxin (diphtheria antitoxin) (obtain from CDC +1 404-639-2889) to neutralize free toxin. Empiric IV treatment. Dose depends on the severity of the disease.
5. Administer erythromycin to eradicate the bacterium: consult CDC or infectious disease specialist for dosing.
6. Consult with cardiology, neurology, and ENT as needed.
7. Implement strict isolation.

4

Epiglottitis

Acute inflammation of the epiglottal area (usually by the bacterium *Haemophilus influenzae* type B) can cause airway obstruction, respiratory arrest, and death. The incidence of this disease has greatly decreased with the introduction of *Haemophilus influenzae* type B vaccine.¹

Onset is acute, with pain, fever, restlessness/anxiety, sore throat, muffled voice, nasal flaring, drooling, stridor, and tachypnea. Patients look toxic; the child may sit forward with neck extended and mouth open.

With this presentation and suspected diagnosis, do not attempt an exam. Hold an oxygen mask near the child's face without frightening him or her. (Note: racemic epinephrine, other nebulizers, steroids, or sedation are not recommended.) Prepare all airway equipment and make sure the team members are ready in case the first attempt at intubation fails. The best setting may be the operating room. When preparation has been completed, lay the child down, BVM with oxygen, and have an airway sequence prepared. Consider using ET tubes a size smaller than normal to adapt for airway swelling. If the first attempt at intubation is unsuccessful, perform transtracheal needle ventilation. Definitive airway control may be done once the transtracheal needle is successfully placed. The team leader can now retry to intubate, which may be successful now as the airflow is moving in both directions. If this is still unsuccessful, perform a tracheotomy. (**Vol II – Air Skills 1 Aids to Intubation, Air Skills 13 Cricothyrotomy, Air Skills 14 Tracheotomy, Air Skills 15 Tracheotomy in Infants, Air Skills 16 Transtracheal Needle Ventilation**)

To rule out epiglottitis in stable, cooperative patients, use direct visualization. This may be done several different ways. One way is to anesthetize the nares with lidocaine and visualize with a fiberoptic laryngoscope. Note that the soft tissue lateral neck x-ray is helpful only if the epiglottis shadow is enlarged (the 'thumbprint' sign). An indeterminate or negative x-ray does not rule out epiglottitis.

Once the airway is secured, sedate appropriately, and begin mechanical ventilation. Obtain epiglottal area and blood cultures. Begin antibiotics. One option is cefotaxime 50 mg/kg/ q8h IV. (There are other antibiotic options available, consult with local specialists.) Assess fluid status and treat appropriately. Admit to an appropriate ICU setting.

Remember to arrange to give rifampin chemoprophylaxis to persons who have been exposed.

5

Laryngeal Edema Post Intubation

An intubated child may develop laryngotracheal edema post intubation. (Corticosteroids may prevent this edema prior to extubation.) Clinical signs include stridor, cough, and respiratory distress. Depending on the severity of the case, mist therapy and/or aerosolized epinephrine may be beneficial. Heliox therapy may be of some benefit. [\(Vol III – AIR3 HELIOX TREATMENT\)](#) Closely observe for improvement or progression. Establish IV access. Fiberoptic nasopharyngoscopy may be helpful in assessing the severity of the upper airway edema or other upper airway problems and the need for re-intubation or other approaches. Be aware that tracheal edema (and cricoid narrowing) won't be seen but can be inferred if the upper airway isn't edematous. These patients need a smaller tube than normally used for reintubation. In progressive or severe cases, prepare airway equipment for all possible scenarios, gather team members, and establish control of the airway, starting with intubation. Cricothyrotomy or tracheotomy may be needed. Note that RSI may not be indicated in cases where the patient's edema-compromised airway is being maintained by their muscle tone/positioning. [\(Vol II – AIR SKILLS 1 AIDS TO INTUBATION, AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS\)](#)

6

Anaphylaxis and Angioedema

Anaphylaxis (literally meaning 'without protection') is a multi-systemic allergic reaction to an inciting agent or event. This is a clinical diagnosis.

Anaphylactoid reactions are similar in nature and treatment to allergic reactions.

Angioedema (localized soft tissue swelling involving deeper layers) of the upper aero-digestive tract is a common local manifestation of anaphylactic/anaphylactoid reactions. Angioedema because of other mechanisms, such as hereditary angioedema or acquired angioedema are responsive to different

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treatment regimens. However, it looks the same, and angioedema may be impossible to distinguish by history, especially if it is the first episode. (**Vol III – AIR8 ANAPHYLAXIS**)

A common sense approach to the respiratory aspect of these conditions is to treat these patients as if the episode is an anaphylactic/anaphylactoid reaction while keeping in mind that hereditary angioedema or acquired angioedema will respond poorly or not at all to epinephrine, antihistamines, and steroids. Therapy is directed at reversing current effects and preventing further progression. Like many emergency conditions, assessment and treatment occur simultaneously in this setting. Epinephrine (a physiologic antagonist to the effects of the biochemical mediators) is the drug of choice and should be given as soon as the diagnosis is suspected, whatever system(s) is involved.

Upper Airway Considerations

- A.** Examine the airway for signs of angioedema. If immediate airway intervention is not indicated, give epinephrine while mobilizing the team and equipment for intervention if the situation worsens. Initially, use a BVM with oxygen, then attempt to intubate. If the first attempt at intubation is unsuccessful perform transtracheal needle ventilation. Definitive airway control can be done once the transtracheal needle is successfully placed. The team leader can now retry to intubate, which may be successful now as the airflow is moving in both directions. If you are still not successful, then perform a tracheotomy. Fiberoptic exam can assist in determining the severity of the edema, especially in patients who are not responding or progressing rapidly and whose laryngeal involvement is not readily apparent. RSI may not be indicated in cases where the patient's edema-compromised airway is being maintained by their muscle tone/positioning. (**Vol II – AIR SKILLS 1 AIDS TO INTUBATION, AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION**)
- B.** Utilize O₂ as indicated. Heliox (80% helium and 20% oxygen) may also be helpful. (**Vol III – AIR3 HELIOX TREATMENT**)
- C.** Unless the patient has hereditary angioedema or acquired angioedema for certain, give the following medications:
- Epinephrine IM, IV, or IO.** Choice of route depends on the severity:
- **For mild-to-moderate cases:** Administer epinephrine IM: 0.01 mL/kg (up to 0.3 mL) of 1:1000 solution. Repeat every 10 to 20 minutes (up to 3 doses prn). Epinephrine IM is now the preferred route over SQ.^{2,3} EpiPen Jr[®] is available.
 - **In life-threatening situations or when patient is unresponsive:** Administer epinephrine IV/O: 1 mg in 500 mL NS (2 µg/mL) given by IV drip at rate determined by the seriousness of the situation and patient's response. Usual doses are from 2 to 10 µg/minute IV (or 1 to 5 mL/minute of the solution of 1 mg of epinephrine/500 mL of NS).

Higher doses may be needed if the patient is clinically unresponsive to lower doses but the dose must be carefully titrated under direct supervision while monitoring BP and cardiac rhythm. If continuous infusion is necessary, switch to a pump-controlled regimen when feasible/available.

H1 and H2 Antihistamines (Give both; choice of route depends on severity.)

- H1 antagonist: diphenhydramine (Benadryl), 1 mg/kg PO/IM/IV/IO
- H2 antagonist: Zantac 50 mg IV
Pepcid 20 mg IV

Corticosteroids

- Methylprednisolone, 1 to 2 mg/kg PO/IV/IO

D. Hereditary angioedema and acquired angioedema treatment options (C₁ esterase inhibitor concentrate, fresh frozen plasma, nebulized heparin, androgen steroids) have unclear/unestablished pediatric doses/indications. Consult with a specialist when the airway is secure and/or stable.

Lower Airway Considerations

Examine the lungs for bronchospasm (wheezing, increased expiratory phase, difficulty breathing). If symptoms persist despite epinephrine, give nebulized albuterol: 2.5 mg in 2 to 3 mL NS every 15 to 20 minutes; continuous nebs may be indicated at 2 to 3 mg/kg/h.

Other Considerations

- A. Treat shock and/or cardiovascular collapse with epinephrine, fluids, and pharmacologic vasopressor support if hypotension does not respond to fluid therapy. **See Vol III – CV17 SHOCK; Vol II – CIRC SKILLS 1 ARTERIAL AND VENOUS CATHETER INSERTION if the patient is hemodynamically unstable.**
- B. Remove allergen source from patient:
- Remove stinger from insect bite with a blade to avoid injection of more allergen.
 - If an ingested substance has caused the reaction, give activated charcoal 1 g/kg PO/NG. (If a patient has charcoal in the stomach, give medications via a non-oral route.)
- C. Monitor the patient carefully for response to therapy, adverse results, or disease progression.

7

Laryngotracheobronchitis (Croup)

Laryngotracheobronchitis (also called croup) is a viral disease that commonly affects infants and children from 6 months to 3 years of age. Laryngotracheobronchitis is associated with a barking (seal-like) cough, hoarseness, fever, and mild wheezing. The majority of patients recover well. In severe cases, the patient exhibits stridor, retractions, tachypnea, tachycardia (out of proportion to fever), and various degrees of respiratory distress. This is due to luminal narrowing of the larynx and trachea

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secondary to edema and/or fibrin exudate. In more severe cases, respiratory failure may ensue. Epiglottitis and/or tracheitis may mimic or complicate laryngotracheobronchitis; fiberoptic exam helps to distinguish between these and other entities. X-rays are not diagnostic but may provide guidance for treatment.

Management Considerations

Assess severity of airway and carefully monitor to detect early hypoxia. Severe croup that is unresponsive to therapy and/or progressive may require intubation with a smaller ET tube than is normally used. (**Vol II – AIR SKILLS 1 AIDS TO INTUBATION, AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION**)

- Humidified O₂ delivery. Heliox (80% helium and 20% oxygen) may help. (**Vol III – AIR3 HELIOX TREATMENT**)
- Racemic epinephrine nebs: 0.05 mL/kg/dose (of 2.25% solution) in 3 mL NS; repeat every 20 min (up to 3 doses prn). Observe any patient given this treatment for at least 3 hours.
- Dexamethasone 0.6 mg/kg IM/IV (max dose 10 mg). In mild cases, corticosteroids can also be given orally.
- Rehydration
- Assess toxicity. Administer empiric antibiotics if bacterial tracheitis is suspected. Obtain blood and tracheal cultures.
- Admit severe cases to an ICU. Severe cases include those patients who continue to have respiratory distress, have a relapse of signs and symptoms of respiratory distress, and/or require administration of multiple epinephrine nebs.

8

Bacterial Tracheitis (Vol III – PED5 BACTERIAL TRACHEITIS)

Bacterial tracheitis is an occasional complicating factor of an upper respiratory infection such as croup, signified by toxicity, high fever, and severe respiratory distress/failure. Prompt diagnosis is essential. At this time it seems that the only means to distinguish this entity from other pediatric upper airway diseases is direct laryngo-tracheo-bronchoscopy. Therefore, maintain a high index of suspicion. Endoscopy is diagnostic (direct visualization and cultures) and therapeutic (removal of secretions and airway control). Treatment priorities include aggressive airway management and antibiotics.

Airway

Managing and securing the airway of children in respiratory distress is of paramount importance (and should not be delayed while waiting for endoscopy).

Management Considerations

1. Assess severity of airway difficulty and carefully monitor to detect early signs of hypoxia. Severe croup that is unresponsive to therapy and/or progressive may require intubation using endotracheal tubes that are smaller than recommended. (Vol II – AIR SKILLS 1 AIDS TO INTUBATION, AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION)
2. Humidified O₂ delivery. Heliox (80% helium and 20% oxygen) may be helpful. (Vol III – AIR3 HELIOX TREATMENT)
3. Racemic epinephrine nebs: 0.05 mL/kg/dose (of 2.25% solution) in 3 mL NS; repeat every 20 min prn as many as three times. Observe any patient given this treatment for at least 3 hours.
4. Dexamethasone 0.15 to 0.6 mg/kg IM/IV
5. Rehydration
6. Assess toxicity. Empiric antibiotics should be administered if you suspect bacterial tracheitis. Obtain blood and tracheal cultures.
7. Admit severe cases to ICU. Severe cases may include those patients who continue to have respiratory distress, have a relapse of signs and symptoms of respiratory distress, and/or require administration of multiple epinephrine nebs.

Antibiotics

Polymicrobial flora may be present; *Staphylococcus aureus* and *Hemophilus influenzae* most likely predominate. A reasonable empiric choice is ampicillin/sulbactam (Unasyn) 25 to 75 mg/kg IV every 6 h or ceftriaxone 50 to 75 mg/kg/d. (Note that these do not cover resistant *Staphylococcus aureus*. Vancomycin may be used for *Staphylococcus* in this situation, 10 to 15 mg/kg every 6 h.)

9

Bronchiolitis (Vol III – PED6 BRONCHIOLITIS)

Bronchiolitis is a viral infection (usually caused by the respiratory syncytial virus, RSV) of the 2- to 24-month-old age group, which starts as an URI and progresses to the lower and smaller airways (bronchioles). Mucous, debris, and edema cause obstruction, airway resistance, hyperinflation, and hypoxia, which is evidenced by dyspnea, wheezing, and tachypnea. Progression leads to respiratory failure, hypercapnia, acidosis, and apnea. Other signs include low-grade fever, grunting, poor feeding, irritability, and post-tussive vomiting. Chest x-ray is necessary for assessment of other diseases, but isn't diagnostic for bronchiolitis. Nasal washing may be done for RSV antigen testing (rapid turnaround) and/or cultures.

Initial Management

1. Isolation from other children (RSV is very contagious.)
2. Humidified O₂

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3. Rapid identification of those at severe risk: underlying cardiopulmonary disease, prematurity, bronchopulmonary dysplasia, immunodeficiency, age < 6 weeks.
4. Beta agonists (not proven to help but not thought to be harmful and still a standard of care) for wheezing: nebulize albuterol 2.5 mg in 2 to 3 mL NS every 15 to 20 min (up to 3 doses prn). May switch to continuous nebulization if needed.
5. Ipratropium 250 µg may be added to the albuterol neb.
6. Assessment of need to secure airway for respiratory support of (near) failure (hypoxia not responding to O₂, tiring child, worsening vital signs, etc.): **See Vol II – AIR SKILLS PORTALS, AIR SKILLS 3 OROTRACHEAL INTUBATION, AIR SKILLS 4 RAPID SEQUENCE INTUBATION.**
7. Fluid management: restoration and maintenance.

Steroids are not proven to help. Ribavirin therapy shows no clinical benefit but may still be used in selected/severe inpatient cases.

10

Pneumonia

The causes and presentations of infection of the lower respiratory tract are variable, depending on age and underlying medical condition(s). Newborns may be hypothermic, and they don't cough. Pneumonia may present in the very young infant with nonspecific signs and symptoms, such as poor feeding, vomiting, and/or low-grade temperature. Signs such as grunting, severe tachypnea, cyanosis, nasal flaring, and retractions are ominous. Auscultation is difficult; however, until proven otherwise, focal inspiratory crackles indicate pneumonia in a febrile child with previously normal lungs. Obtain a rectal temperature (more accurate in this setting). A chest x-ray is necessary for work-up but not necessarily diagnostic, as pneumonia may lag in appearance on film.

Initial Management Considerations

1. Use oxygen to keep saturations > 92%. Treat bronchospasm with inhaled albuterol: 2.5 mg in 2 to 3 mL NS every 15 to 20 min (up to 3 doses prn). May switch to continuous nebulization, if needed. Ipratropium (Atrovent) 250 µg may be added to the albuterol neb.
2. Watch closely for worsening respiratory status. Severe respiratory distress unresponsive to oxygen therapy needs intubation and mechanical ventilation. **(Vol II – AIR SKILLS PORTALS, AIR SKILLS 3 OROTRACHEAL INTUBATION, AIR SKILLS 4 RAPID SEQUENCE INTUBATION)** Intubated patients may benefit from tracheal suction; send the secretions for culture.
3. Monitor hydration status carefully; fluid management is a critical aspect of care. Pediatric patients become dehydrated easily. However, overaggressive fluid overload may cause pulmonary edema and worsen the patient's respiratory status. **(Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS, CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT)** For hypotensive patients, administer a bolus with 20 mL/kg of crystalloid (NS or LR) and monitor

closely. Patients not responding may be in septic shock and require invasive monitoring of fluid replacement therapy. (Vol III – IN3 SEPSIS IN ADULTS, CV17 SHOCK)

4. Arrange to have a thoracentesis done if a pleural effusion is present. Empyemas need ongoing drainage. Note: this procedure is not an emergency procedure, should not interfere with ongoing evaluation and treatment, and can be done hours after antibiotics have been started.
5. Purulent pericarditis may occur as a complication of pneumonia. Perform echocardiography and tap any significant accumulation of fluid (send for culture). (Vol II – CIRC SKILLS 6 PERICARDIOCENTESIS)
6. Certain viruses are common causes of pneumonia at different ages: CMV, rubella, and herpes in neonates; RSV and others in infants; adeno, influenza, and parainfluenza in children/adolescents.
7. Start empiric antibiotics once the working diagnosis of bacterial pneumonia has been established; diagnostic testing should not delay antibiotic administration. If possible, draw blood cultures before starting antibiotics. Culture the tracheo-bronchial secretions of intubated patients. The following drug choices are from *The Sanford Guide to Antimicrobial Therapy 2011*⁴:

Outpatient treatment of stable, nontoxic, well-hydrated, and nourished patients:

- **3 months to 5 years:** Consider *Streptococcus pneumoniae*, chlamydia, mycoplasma. Choose between erythromycin, clarithromycin, or azithromycin.
- **5 years to 18 years:** Consider mycoplasma, chlamydia, *Streptococcus pneumoniae*. Choose between clarithromycin or azithromycin. Doxycycline is acceptable if the patient is > 8 years old.
- If drug-resistant *Streptococcus pneumoniae* is suspected, look up local susceptibility patterns in your community and treat accordingly.

Initial inpatient treatment regimens. (See doses immediately following.)

- **Neonates:** Consider group B *Streptococcus*, *Listeria*, coliforms, *Staphylococcus aureus*, *P aeruginosa* (also chlamydia). Treatment: Ampicillin plus gentamicin or ampicillin plus cefotaxime. Add vancomycin if resistant *Staphylococcus aureus* is of concern.
- **1 month to 5 years:** Consider *Streptococcus pneumoniae*, mycoplasma, chlamydia. Treatment: Non-ICU: ceftriazone or cefotaxime plus azithromycin; ICU setting: cefotaxime or ceftriazone plus azithromycin plus vancomycin.
- **5 to 18 years:** Consider *Streptococcus pneumoniae* and mycoplasma. Treatment: Non-ICU: ceftriazone or cefotaxime plus azithromycin. Consider monotherapy with a fluoroquinolone (levofloxacin, moxifloxacin) for the older age group, if penicillin-allergic. ICU setting: cefotaxime or ceftriazone plus azithromycin plus vancomycin.

Initial Dosages

Ampicillin: 50 mg/kg IV; see age and weight for specific interval (every 6/8/12 h).
Azithromycin: 10 mg/kg PO for first day (up to 500 mg)
Cefotaxime: 66 mg/kg IV every 8 h
Ceftriaxone: 50 to 75 mg/kg IV every 24 h (to max of 2 g/d)
Cefuroxime: 50 mg/kg IV every 8 h
Clarithromycin: 7.5 mg/kg PO q 12 h (max 1 g/d)
Cloxacillin: 50 mg/kg IV every 6 h
Erythromycin: 10 mg/kg IV every 6 h (every 8 to 12 h in neonates)
Gentamicin: 2.5 mg/kg IV; see age and weight for interval (every 6, 8, or 12 h)
Levofloxacin (over age 5): 10 mg/kg dose once per day, maximum 750 mg/day
Nafcillin: neonates, 25 to 37 mg/kg IV (dose and interval depends on neonatal age and size); 1 month and older, 37 mg/kg IV every 6 h
Vancomycin: neonates, 12.5 to 22 mg/kg IV (dose and interval depends on age and size); 1 month and older, 10 to 15 mg/kg every 6 h

11

Asthma^{5,6}

There are numerous reasons for a pediatric patient to wheeze besides asthma, including anaphylaxis, foreign bodies, bronchiolitis, CHF, pneumonia, acute respiratory distress syndrome, or any other cause of thick secretions. If you suspect any of these, proceed to the appropriate sections. A chest x-ray may be invaluable in determining other causes and/or complications. Suspect an increase in mortality risk in patients who have steroid dependence, syncope, history of ICU admission/mechanical ventilation, more than two asthma hospitalizations per year, more than three asthma emergency visits per year, or are worse at night.

For the potentially unstable asthma patient who is not yet intubated: Prepare for intubation while performing the following:

Oxygen or Heliox therapy to keep saturation > 95%. (**Vol III – AIR3 HELIOX TREATMENT**)

- Continuous beta agonist therapy: albuterol neb 0.5 mg/kg/h not to exceed 15 mg/h.
- Nebulized ipratropium bromide, 0.25 to 0.5 mg. May repeat every 20 minutes prn to a total of three doses.
- Epinephrine (1:1000 solution) 0.01 mg/kg (up to 0.3 mg) IM every 20 min up to 3 doses or terbutaline 0.01 mg/kg SQ (up to 0.3 mg) every 20 minutes prn for a total of three doses. (Epinephrine IM is the preferred route over SQ.³)
- Magnesium 50 mg/kg IV over 5 to 10 minutes (not to exceed 2 g).
- Methylprednisolone 2 mg/kg IV.

- Proceed with RSI/airway management, if the patient is not improving. Consider ketamine as the sedative for its bronchodilator properties. **(Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION; Vol III – AIR4 VENTILATOR MANAGEMENT, AIR7 STATUS ASTHMATICUS)**

If ventilating the patient, allow extra time for exhalation. Patients with severe asthma have a problem with hyperinflation. Ventilate slowly, 8 to 10 times/minute.

For a stable patient:

- Oxygen to keep saturation > 95%.
- Albuterol neb, 0.15 mg/kg/dose (up to 5 mg; minimum dose 2.5 mg). Repeat every 20 minutes prn for a total of three doses.
- Ipratropium bromide neb, 0.25 to 0.5 mg. Repeat every 20 minutes prn for a total of three doses.
- Start steroids as soon as evaluation has been completed. Oral prednisone (or prednisolone) 1 to 2 mg/kg; methylprednisolone 2 mg/kg IV.⁷
- Consider pulmonary function tests (PEFR/FEV₁) in older children to help guide further management:
 - **Asymptomatic/resolved symptoms and PEFR/FEV₁ > 70% predicted:** Evaluate for outpatient management. **(Vol III – AIR7 STATUS ASTHMATICUS)**
 - **Continued symptoms and PEFR/FEV₁ > 70% predicted:** Continue inhalation therapy while reassessing for alternative conditions. **(Vol I – PATHWAY 6 RESPIRATORY EMERGENCIES, ADULT)**
 - **Moderate symptoms and PEFR/FEV₁ from 40% to 70% predicted:** Continue inhalation therapy, Consider continuous beta agonist therapy: albuterol neb 0.5 mg/kg/hour not to exceed 15 mg/h. **(Vol III – AIR7 STATUS ASTHMATICUS)**
 - **Severe symptoms and PEFR/FEV₁ < 40% predicted:** Continue inhalation therapy.
 - Consider magnesium 25 mg/kg IV (not to exceed 2 g) over 20 minutes. Consider Heliox. **(Vol III – AIR3 HELIOX TREATMENT)** Closely assess response, and see **Vol III – AIR7 STATUS ASTHMATICUS** for other considerations.

12

Drowning

Pertinent history includes the temperature of the water (cold water immersion carries a better prognosis), submersion time, type of water (although salt water versus fresh water carries little significance), water contaminants, and other scene factors. Determine any secondary causes of drowning such as events around the incident (use of alcohol/drugs, exhaustion, hypothermia, suicide, abuse, trauma, rescue attempts) or underlying disease (seizures, hypoglycemia, cardiac). These can play an important role in immediate and ongoing treatment. For example, a common scenario would be a diving accident with possible head

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and neck/spine injuries. Unless the patient is reliable in being able to recount the incident or there is a reliable eyewitness account of all details, suspect concomitant trauma. ([Vol I – PATHWAY 9 PEDIATRIC TRAUMA](#))

Hypoxia is the main factor contributing to tissue injury, organ failure, and death. Immediate term effects include:

- Increased vascular permeability, leading to loss of fluid from the intravascular compartment and hypovolemia/hypotension
- Myocardial dysfunction, cardiac arrhythmias, asystole
- CNS neuronal injury

Aspiration (usually low volumes due to laryngospasm) causes injury to the alveolar/capillary unit with resultant impaired gas exchange, decrease in functional volume, and possible pulmonary edema.

Acidosis may result from both of these primary insults.

Clinical presentation of submersion injuries can range from no symptoms to death. History and exam guide diagnostic studies. Altered mental status makes complete assessment much more complex. Do not assume its cause but systematically rule out all serious potential and reversible causes. (If the patient has hypoxic neuronal damage, intoxication, or occult closed head trauma, [see Vol I – PATHWAY 1 ALTERED LEVEL OF CONSCIOUSNESS](#).) Remember that normal c-spine x-rays do not rule out a spinal injury; continue to utilize appropriate protective measures.

Management Considerations

Rapid assessment for and treatment of hypoxia and acidosis while protecting the cervical spine:

1. If supplemental oxygen isn't reversing hypoxia, intubation and mechanical ventilation is indicated in order to use PEEP. ([Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION](#); [Vol III – AIR4 VENTILATOR MANAGEMENT](#))
2. If you are intubating and using paralytics/sedatives for management purposes and your patient has altered mental status, use anti-seizure medication unless seizures can be ruled out. ([Vol III – NEU1 STATUS EPILEPTICUS](#))
3. Obtain early ABG for assessment and treatment of acidosis: most acidosis responds to correction of hypoxia and dehydration. [See Vol III – END/M6 ACID-BASE](#) for use of bicarbonate in severe acidosis only after ventilation has been assured.
4. Early establishment of core temperature with aggressive rewarming measures if patient is hypothermic, as hypothermia prolongs/causes organ dysfunction and changes (and hinders) treatment priorities and efforts. ([Vol I – PATHWAY 1 ALTERED LEVEL OF CONSCIOUSNESS](#))
5. Cardiac monitoring for dysfunction and dysrhythmias. Inotropic support may be required if hypotension doesn't respond to fluid treatment. ([Vol I – PATHWAY 2 CARDIOVASCULAR EMERGENCIES](#))

6. Treatment of hypovolemia/hypotension while avoiding overhydration and pulmonary edema: consider initial fluid bolus of 20 cc/kg crystalloid (NS or LR) IV and invasive monitoring such as US of IVC or CVP and arterial line. **(Vol II—CIRC SKILLS 1 ARTERIAL AND VENOUS CATHETER INSERTION, CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT)**
7. Decompression of the stomach with NG tube.
8. Assessment for bronchoscopy for FB, debris, or aspirated food.
9. Early consultation with an intensivist for appropriate care level. ARDS and multi-system failure can occur and require a high level of intensive care. **See #13, Acute Respiratory Distress Syndrome.**
10. Close monitoring for progression and complications. Observe a patient who is completely asymptomatic for at least 3 to 6 hours.
11. Adjunctive measures: empiric antibiotics are controversial and probably not indicated unless the patient was submerged in contaminated water. Early steroid use is not indicated.

13

Acute Respiratory Distress Syndrome

Acute Respiratory Distress Syndrome (ARDS) is a hypoxemic respiratory failure syndrome of non-cardiogenic pulmonary edema that is associated with numerous precipitating conditions and/or critically ill patients. Treatment for the pulmonary system alone is intensely complex and meticulously supportive. There is no definitive treatment per se. For further discussion, **see Vol I—PATHWAY 6, #6.** Respiratory and multi-organ system failure are both common in these patients. If the team is caring for a child who fits into the category of someone likely to develop ARDS, consider early transfer to a facility with pediatric ICU capabilities and a readily available pediatric intensivist/ pulmonologist.

**PEDIATRIC RESPIRATORY EMERGENCIES
PATHWAY 7**

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**PATHWAY 8: TRAUMA EMERGENCIES
(SECONDARY SURVEY FOR ADULT)**

The team continues the resuscitation along the pathway suggested by the initial clinical impression. Each pathway includes a complete, thorough, and rapid physical examination with additional history taking. The team leader is wary of conditions that may not be apparent. To obtain additional clinical data or to correct a missed or newly developed condition, the team leader repeats the initial survey if the patient is not responding satisfactorily.

The team leader performs a head-to-toe, rapid but thorough, physical examination. Some possible abnormalities¹ encountered are:

Text Number	Diagnosis/ Condition	Related Materials
1	Compromised Airway	Vol I – AIRWAY SKILLS
2	Abnormal LOC	Vol I – PATHWAY 1 ALTERED LEVEL OF CONSCIOUSNESS
3	Facial and Skull Fracture/Scalp Laceration	Vol I – ACUTE CARE PORTALS; VOL II – DISAB SKILLS 2 RANEY SCALP CLIPS; VOL III – NEU5 INCREASED INTRACRANIAL PRESSURE
4	Direct Eye Trauma	
5	Soft Tissues of the Neck	Vol II – AIRWAY SKILLS
6	Cervical Spine Injury	VOL II – XRAY SKILLS 1 C-SPINE RULES, XRAY SKILLS 2 CERVICAL SPINE XRAY INTERPRETATION
7	Chest Wall and Pulmonary Injury	VOL II – BREATH SKILLS 1 CHEST TUBE INSERTION, BREATH SKILLS 2 CHEST SUCTION/ AUTOTRANSFUSION, BREATH SKILLS 5 NEEDLE THORACOSTOMY
8	Cardiac Tamponade	VOL II – CIRC SKILLS 6 PERICARDIOCENTESIS
9	Hypovolemic Shock	Vol I – ACUTE CARE PORTALS; VOL II – CIRC SKILLS 1 ARTERIAL AND VENOUS CATHETER INSERTION; VOL III – CV17 SHOCK
10	Abdominal Injury	Vol I – ACUTE CARE PORTALS; VOL III – CV17 SHOCK

**ADULT TRAUMA EMERGENCIES
PATHWAY 8**

11	Pelvic Fracture	VOL II – TRAU SKILLS 3 PELVIC FRACTURE STABILIZATION, XRAY SKILLS3 INTERPRETATION OF PELVIC XRAY
12	Trauma in Pregnancy	VOL III – OB9 TRAUMA IN PREGNANCY
13	Major Joints, Femurs, and Amputations	Vol I – ACUTE CARE PORTALS; VOL II – TRAU SKILLS 1 COMPARTMENT PRESSURE MEASUREMENT, TRAU SKILLS 2 FEMUR FRACTURE SPLINTING; VOL III – CV17 SHOCK
14	Burns	Vol I – ACUTE CARE PORTALS; VOL III – ENV3 BURNS MANAGEMENT
15	Heat Stroke	VOL III – ENV2 HYPERTHERMIA
16	Severe Hypothermia	VOL III – ENV1 HYPOTHERMIA
17	Drowning	VOL III – ENV4 DROWNING
18	Caustic Substance Ingestion	

Imaging

For patients who require rapid transfer, limit studies to those that impact immediate care.

Chest x-ray is mandatory to reveal pneumo/hemo thorax as well as proper placement of lines and tubes. Order pelvic x-rays on the basis of clinical suspicion and mechanism of injury.

Order imaging tests immediately and continue the examination as the films are being obtained. If the patient deteriorates during this survey, repeat the initial survey, checking for airway, breathing, circulation, and so on.

Obtain additional imaging based on secondary survey findings. These may include plain films and/or CT of the head, spine, chest, abdomen, and pelvis.

Hidden back injury or wound

Carefully log roll the patient, while maintaining c-spine immobilization, just enough to expose the back. Palpate the entire length of the spine checking for deformity, hematoma, lacerations, and pain. Palpate the flanks and the buttocks. Palpate the posterior thorax and scapulae. The log roll maneuver in the presence of an unstable pelvic fracture may stimulate more bleeding. The team should carefully lift the patient straight up instead.

Remove glass shards and any other harmful materials before laying the patient back down. If you suspect spinal injury, immobilize the patient securely. If the patient is to remain on the extrication board, take this opportunity to pad the board or use a cushion device such as the Backraft.

1

Compromised Airway

During the initial survey, the airway was opened and the patient was ventilated pending this step of the process. If there was failure to ventilate or to open the airway, invasive methods were used to accomplish this. If this has not been done, return to the initial survey and do it now. There is no point in continuing until the patient is breathing or can be ventilated.

If the patient is breathing or being BVM ventilated, a decision regarding the need for emergent or elective intubation is necessary. Before intubation, be sure to obtain a **SAMPLE** history and perform both mini neurological and rectal exams. [\(See Abnormal LOC, #2 this portal.\)](#)

Reasons to intubate:

- Gag reflexes are absent.
- The patient is difficult to ventilate as in severe asthma or severe pulmonary contusion.
- A burn victim has soot or erythema in the mouth with stridor.
- The patient needs to be paralyzed for studies, such as CT scan or angiography.
- The patient needs to be paralyzed for safe transport.
- There is a flail chest with oxygen desaturation.
- A tension pneumothorax was or is present.
- The patient is in extreme pain, and analgesia is needed, which could lead to respiratory depression.
- The patient is unable to handle secretions because of swelling, bleeding, etc.
- The patient is weak or tiring, and respirations are becoming less effective.
- Hyperventilation is needed because of CNS deterioration.
- There is poor oxygenation because of pulmonary problems.
- There is an open chest wound.
- An invasive emergency procedure requires anesthesia.
- The patient is combative or unable to tolerate needed procedures.

If the patient is deeply obtunded, RSI may not be required, unless airway reflexes and muscle tone are present. If a difficult intubation is anticipated, have a supraglottic airway or cricothyrotomy kit available. Always have a back-up plan in case intubation fails.

ADULT TRAUMA EMERGENCIES PATHWAY 8

Check to be sure that the needed equipment is at hand. Equip the airway cart ([See Vol II – AIR SKILLS PORTALS](#)) with rescue airways as well as standard orotracheal intubation equipment. Familiarize yourself with its contents. If physical findings or a mechanism of injury suggest the possibility of a spinal injury, perform orotracheal intubation with in-line immobilization of the head and neck. A surgical airway may be indicated.

Open the cervical collar temporarily: it can limit your ability to lift the chin. A team member may provide cervical immobilization by standing at the bedside, resting the forearms on the patient's clavicles and placing the palms of the hands over the patient's ears. Fingers and thumbs should be extended so as to provide maximum control of the head while leaving the mandible free. This method works best if the team member performing cricoid pressure stands on the opposite side.

RSI greatly facilitates orotracheal intubation. RSI eliminates laryngospasm. The risk of RSI is greatly reduced if the intubator is prepared to secure the airway with another procedure. A supraglottic airway, such as the iGEL, may often be easily inserted.

An elevated serum potassium level makes the use of succinylcholine risky. (Succinylcholine raises the serum potassium level.) Chronic or acute renal failure, a crush injury or burn more than 24 hours old, muscular dystrophy, muscle wasting, and extreme muscular exertion as in cocaine or amphetamine overdose may elevate the serum potassium level. Rocuronium is an alternative that may be used. It is longer acting than succinylcholine, but takes effect nearly as quickly. Vecuronium may be used. The disadvantage of vecuronium is that its effects last 30 minutes in comparison with succinylcholine, the effects of which last less than 10 minutes. Succinylcholine also takes effect in less than one minute, while vecuronium may take more than 3 minutes to take effect.

Other forms of securing the airway instead of RSI include:

- **Ororotracheal intubation** may be accomplished with or without topical anesthesia. An **intubating laryngeal-mask airway (ILMA)** may be used effectively with topical anesthesia. ([Vol II – AIR SKILLS 9 NASOTRACHEAL INTUBATION](#), [AIR SKILLS 8 INTUBATING LARYNGEAL MASK AIRWAY](#))
- **Nasotracheal intubation** is rarely used in trauma resuscitation because it raises ICP and may cause bleeding. A retropharyngeal hematoma is a contraindication. ([Vol II – AIR SKILLS 3 OROTRACHEAL INTUBATION](#))

- A **surgical airway** may be the best option if anatomic problems are severe. **Transtacheal needle ventilation (TTNV)** and **retrograde intubation** may be good options if time is not critical. (Vol II – Air Skills 10 TOPICAL ANESTHESIA, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION) When time is critical, cricothyrotomy is fastest and most easily accomplished. (Vol II – Air Skills 13 CRICOTHYROTOMY) If there has been trauma to the larynx, a **tracheotomy** is indicated. (Vol II – Air Skills 14 TRACHEOTOMY)

Unfortunately, the myriad of airway injuries and problems that may arise in trauma occurring in patients of all sizes and configurations make recommendations applicable to all situations impossible. An algorithm quickly becomes a tangle of possibilities. Another way of looking at treatment choices is to consider the tools best suited to a specific problem. Here are some generalities about the tools that may help.

Orotracheal intubation is still the optimal choice for airway management. The disadvantages of causing harmful reflexes and of being painful and difficult in an aware patient are overcome by the use of RSI. The ETI and the EID also make intubation less difficult.

The **King airway** is a supraglottic airway that is inserted blindly. The King airway consists of a curved tube with ventilation apertures located between 2 inflatable cuffs. A single valve/pilot balloon is used to inflate both cuffs. The distal cuff seals the esophagus; the proximal cuff seals the oral pharynx. A 15-mm connector attaches to the proximal end of the tube for attachment to a standard breathing circuit or resuscitation bag. The King airway is contraindicated for patients who have a gag reflex, known esophageal disease (eg, cancer, varices, stricture), laryngectomy with a stoma, and/or caustic ingestion or airway burns.

I-gel[®] is a non-inflatable supraglottic airway that makes an anatomic seal and comes in the 3 adult sizes. Consider patient's weight when fitting the I-gel[®].

Size	Weight kg/lbs
Small – 3	30-60 kg/ 65-130 lbs
Medium – 4	50-90 kg/ 110-200 lbs
Large – 5	90+ kg/ 200+ lbs

Cricothyrotomy is fast and reliable; however, it requires surgical skill. A tracheal hook and an ETI are helpful.

Tracheotomy is more difficult than a cricothyrotomy; however, it is needed if there has been trauma to the larynx.

**ADULT TRAUMA EMERGENCIES
PATHWAY 8**

Intubating laryngeal mask airway (ILMA) is effective in obese patients and may be used with topical anesthesia. It is possible to remove the ILMA while leaving the patient intubated, but it requires several steps.

Transtracheal needle ventilation (TTNV) is an excellent temporary airway. The patient may be oxygenated using this technique, allowing time for more invasive procedures to be accomplished.

2

Abnormal LOC

If a patient is belligerent, unable to cooperate, or delirious, use RSI to gain control of the patient's airway, allowing the resuscitation to continue. (**Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION**) Before paralyzing the patient, be sure that the SAMPLE history has been obtained, the LOC has been determined (using the AVPU scale), and both mini neuro and rectal exams have been documented. The DONT therapy should have been considered during the initial survey.

A **mini neurologic exam** may be performed quickly prior to RSI or sedation. It is useful to have a list available for review.

1. **Level of Consciousness**
AVPU and Glasgow Coma Scale
2. **Pupils and Vision**
Conjugate or disconjugate gaze
Size, equality, and reactivity
Finger counting
3. **Tympanic Membranes**
Hemotympanum
4. **Neck**
Midline tenderness
5. **Extremities**
Movement and strength on command or to pain
Check ankle, patellar, Babinski, brachial reflexes, and clonus
Sensation and position sense
6. **Trunk and Perineum**
Priapism, saddle sensation, anal sphincter tone
Sensation level

Level of Consciousness	
AVPU Scale	Corresponding GCS
A Awake	14 to 15
V Responds to voice	12 to 13
P Responds to pain	8

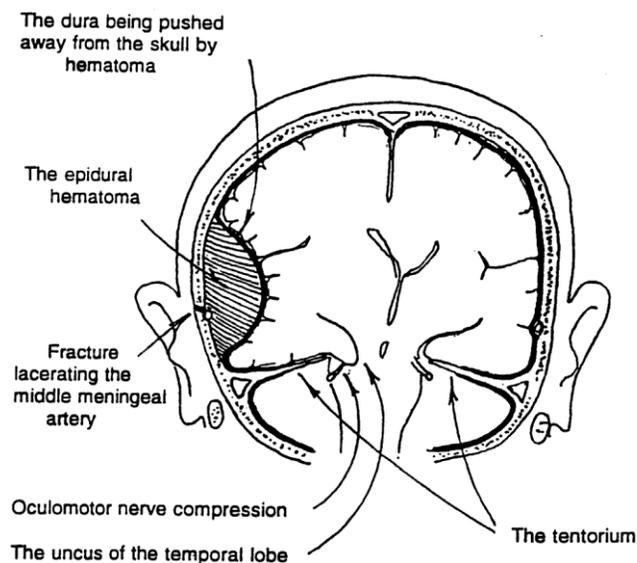
U	Unresponsive to pain	3 to 4
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If uncal herniation is occurring (dilated pupil and weak opposite extremity) or if the patient is posturing (extensor or flexor), perform the following:

- Maintain mean arterial pressure (MAP) > 90 mm Hg throughout treatment, which usually maintains the cerebral perfusion pressure >70 mm Hg. The goal is not to allow MAP to fall below 50 mm Hg.
- Avoid prophylactic hyperventilation therapy following injury. However, for impending herniation, hyperventilation therapy (PCO₂ 30 mm Hg) is appropriate.
- Administer mannitol 1 g/kg IV or hypertonic saline; consult with neurosurgery.
- If time allows, consider giving anti-epileptic; consult neurosurgeon for drug of choice.
- Elevate head of the bed.

If the patient had an initial lucid interval and continues to herniate despite the measures previously described and transport to a trauma center is delayed, perform the following:

- Obtain a head CT to identify an epidural hematoma and location. About 80% of the time, the epidural is located on the side of the dilated pupil. If a CT scan is unavailable, skull trephination should be performed on the side of the dilated pupil. **See Vol II – DISAB SKILLS 1 SKULL TREPHINATION** for procedural information. Consult with a neurosurgeon if possible. If the epidural is solid white on CT scan, it is clotted and may not extrude through an emergency trephine. An acute epidural typically looks like a mixture of black and white on CT, indicating continuing bleeding. Emergency trephines for epidural hematomas in areas of the skull other than the typical location are often not practical because of skull thickness and the presence of venous sinuses.



An epidural hematoma in the typical location

- If the patient has a seizure, administer lorazepam 4 mg IV. Repeat this dose in 5 minutes if needed. If there is suspicion that the patient has cerebral contusion or blood in the cranium, load with Keppra or phenytoin or Fosphenytoin after RSI. (Paralysis will mask seizure activity.)
- In the context of multiple trauma, acceptable systemic BP may be variable. For instance, a patient with a ruptured thoracic aorta may be stable and not actively bleeding. Assess carefully the amount of fluid used. It may be advisable to keep the BP low. If the patient has a head injury, a BP of 90 mm Hg systolic is acceptable in terms of cerebral perfusion. Do not use hypotonic solutions.
- The Glasgow Coma Score incorporates the 15 point scale, and now has added the Pupil Reactivity Score. The pupil score is based on if the pupils DO NOT react to light. If both pupils react to light the score is 0, if only one react to light the score is 1 and neither react to light the score is 2. This score should be subtracted from the 15 point scale. Further if the patient is intubated the patient should not be scored a 1, but should instead be scored as not testable.

- Calculate the **Glasgow Coma Scale**. It is important in terms of estimating prognosis.

Glasgow Coma Scale

Eye Opening

Infant (<1 year)	Pediatric (>1 year)	Adult	
Spontaneous	Spontaneous	Spontaneous	4
Voice	Voice	Speech	3
Pain	Pain	Pressure	2
None	None	None	1

Verbal Response

Infant (<2 years)	Pediatric (>2 years)	Adult	
Coos, babbles	Appropriate word/ phrase	Oriented	5
Irritable but consolable	Disoriented/ converses	Confused	4
Persistent cries/ screams	Inappropriate word	Words	3
Moans/grunts to pain; restless	Incomprehensible sounds	Sounds	2
None	None	None	1

Motor Response

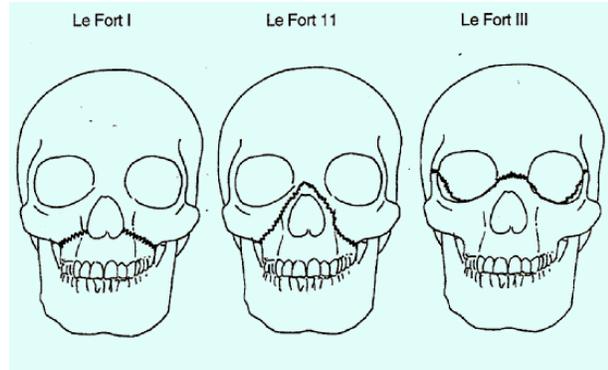
Infant (<1 year)	Pediatric (>1 year)	Adult	
Spontaneous	Obeys	Obeys	6
Localizes pain	Localizes pain	Localizes pain	5
Flexion- withdrawal	Flexion- withdrawal	Withdraws	4
Flexion/ decorticate	Flexion/ decorticate	Abnormal flexion (decorticate)	3
Extension/ decerebrate	Extension/ decerebrate	Abnormal extension (decerebrate)	2
None	None	None	1
			3 to 15

If the primary (initial) assessment is complete, begin the secondary (focused) evaluation.

3

Facial and Skull Fractures/Scalp Lacerations

Palpate the patient's face and jaw for abnormal step-offs and movement. It is not important to diagnose fracture type; however, Le Fort fractures are frequently mentioned in trauma manuals. See illustration below:

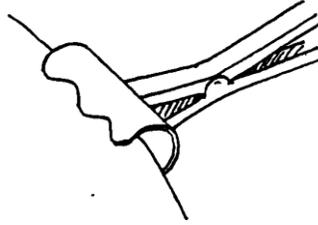


Check for loose or missing teeth that the patient may have aspirated in the patient's mouth and then on x-rays if obtained. Tongue injuries with swelling may endanger the airway. Be aware of the possibility of either tongue rings, or other jewelry in the mouth. When facial trauma endangers the airway, cricothyrotomy may be necessary. **(Vol II – AIR SKILLS 13 CRICOTHYROTOMY)**

Severe nasal hemorrhage may result from facial injuries, especially in hypocoagulable patients. A nasal balloon or Foley catheter may be used to tamponade bleeding and protect the airway.

If there is a scalp wound, palpate the skull with a gloved finger to identify open or depressed skull fractures. A widely open skull fracture is not necessarily a fatal injury. Cover fractures with a sterile dressing. If the wound edges are bleeding, apply Raney clips to stop the bleeding. These clips do not close the wound. Another technique is to use a large running suture along the wound edges to stop bleeding. Another method is to use injectable lidocaine with epinephrine. **BE AWARE THAT A LARGE AMOUNT OF BLOOD CAN BE LOST FROM A SCALP WOUND.**

Raney clips are tubular plastic jaws that can grip the full thickness of the scalp, providing hemostasis. They are applied with a Raney clip applying forceps as shown.



Posterior access to the scalp is limited. When the patient is lifted or rolled for back inspection, apply the clips. In-line immobilization of the head and neck is necessary. Consider IV antibiotics.

4

Direct Eye Trauma

CNS injury, direct eye trauma, previous eye surgery as well as nasal decongestants such as phenylephrine may cause dilated or unequal pupils. Topical ophthalmologic anesthetic drops may be used in the initial evaluation and care of any painful eye injury except ruptured globe. Do not remove impaled foreign bodies from eyes. Remove contact lenses unless there is a possible globe rupture.

Scleral rupture from blunt trauma is accompanied by peripheral scleral hemorrhage. If such is possible, avoid touching the eye. Cover it with a firm plastic or metal shield. Or, use a non-depolarizing paralytic agent instead of succinylcholine. Fasciculations may cause the muscles of the orbit to squeeze out the vitreous.

If the patient is awake at this time, ask him or her to count fingers as a vision check.

Lid lacerations involving lid margins or tear ducts require repair by an ophthalmologic surgeon.

When there has been eye contamination with acidic or basic chemicals, use copious irrigation with sterile NS using a Morgan lens to prevent continuing injury. Check pH of the tears with pH paper or you could use a urine dipstick. The resulting pH should be 6 to 8. If there is hyphema of the anterior chambers, try to prevent the patient from struggling as it results in further bleeding.

Bilateral fixed and dilated pupils are ominous for brain death unless secondary to a reversible problem. Hypothermia is an example as well as anticholinergic poisoning.

**ADULT TRAUMA EMERGENCIES
PATHWAY 8**

Disconjugate gaze and nystagmus may be associated with head injury or drug ingestion. Subconjunctival hemorrhage and petechiae of the face occurs with traumatic asphyxia with compression of the superior vena cava.

5

Soft Tissues of the Neck

Do not explore lacerations and penetrating wounds deeper than the platysma muscle in the ED. Massive hemorrhage can result. Explore these wounds in the operating room, with or without prior angiography. Deep lacerations of the neck may divide the trachea. The trachea may often be directly intubated in such cases.

Laryngeal fracture commonly results from a blow to the neck. A hematoma forms within the larynx, producing airway obstruction. Try gentle orotracheal intubation (if not already performed), but tracheotomy is probably needed. (**Vol II – AIR SKILLS 14 TRACHEOTOMY**)

The trachea may be displaced to the opposite side of a tension pneumothorax or by a hematoma.

Carotid pulses may be absent secondary to hypotension, direct injury, and aortic injury.

If the external jugular veins can be seen, they provide valuable information regarding the volume status of the trauma patient. Jugular venous distension may indicate tension pneumothorax, cardiac tamponade, volume overload, pulmonary embolism, traumatic asphyxia (compression of the superior vena cava), and heart failure.

6

Cervical Spine Injury

If cervical spine injury is a possibility, take cervical spine precautions with a C-collar, head blocks, and a long spine board. If the patient has multiple injuries and is likely to be transferred within 30 minutes, maintain immobilization and make no attempt to rule out fracture radiographically. If the patient will be in the emergency department for more than 30 minutes, attempt to get the patient off the spine board by log rolling the patient off the board and requiring him or her to lie supine with continued cervical immobilization until cleared. Remember that a non-boarded patient still needs to be log rolled and moved as a unit.

PEDS: In children and uncooperative patients who require prolonged immobilization, should still be gotten off the extrication board.

A CT scan is preferable to plain films, but if imaging the cervical spine with plain films, carefully review the lateral c-spine x-ray (**Vol II – XRAY SKILLS 1 CERVICAL SPINE RULES AND USE OF IMAGING, XRAY SKILLS 2 CERVICAL SPINE X-RAY**)

INTERPRETATION) for bony or ligamentous injury. Obtain a swimmer's view if necessary to see the C7-T1 connection. Doing this routinely in large or muscular patients saves time. Look for hematoma formation with airway displacement. Check the sphenoid sinus, if visible, for an air-fluid level signifying a basal skull fracture. For more definitive information about the integrity of the cervical spine.

When a patient who is breathing but obtunded needs intubation and the probability of cervical spine injury is high, consider methods that require little neck motion.⁷ Retrograde intubation (**Vol II – AIR SKILLS 10 TOPICAL ANESTHESIA**) is a method that may be used. Standard orotracheal intubation⁸ with RSI has also been shown to be safe and effective in patients with c spine injury *when performed with careful manual in-line stabilization of the head and neck.*

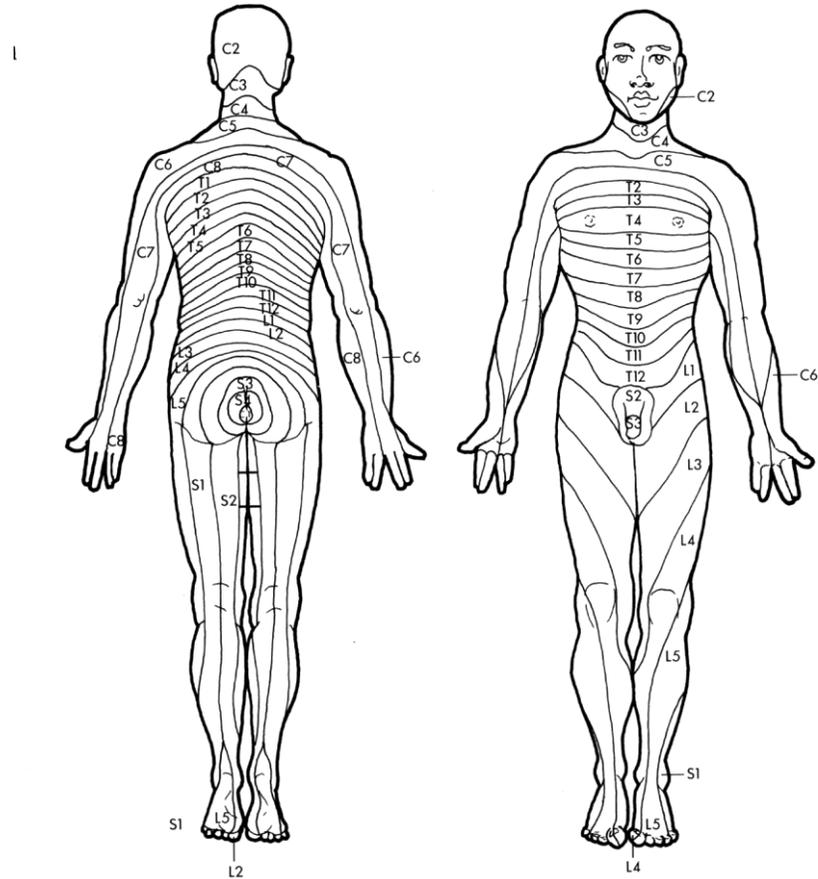
If not already performed, conduct both mini neuro and rectal exams as described under **Abnormal LOC**.

Spinal cord injury syndromes follow:

1. **Complete injury.** Total loss of sensation and movement below level of injury.

Level	Function	Sensory level
C2	Breathing	Occiput
C3	Breathing	Neck
C4	Breathing	Top of shoulders
C5	Arm abduction	Anterior of arm
C6	Elbow flexion	Lateral arm and thumb
C7	Elbow extension	Posterior arm and index finger
C8	Finger flexion	Medial arm and small finger
T1	Finger abduction	Just below shoulders

ADULT TRAUMA EMERGENCIES PATHWAY 8



Dermatomes corresponding to the levels of the spinal cord. From *Emergency Management of Skeletal Injuries*. Ruiz E, Cicero JJ, editors. Mosby-Yearbook, 1995. Reprinted with permission.⁹

- Incomplete injury.** This is an important finding. Surgical intervention or aggressive reduction may result in recovery. Variable loss of sensation and movement below the level of injury indicates an incomplete injury. An example is sacral sparing with preserved anal sphincter tone. Another is the presence of an **anterior cord syndrome** in which there is muscle paralysis but preservation of position sense.
- Brown-Sequard syndrome.** Occurs with penetrating trauma with hemitransection of the cord. It results in loss of motor function below and on the same side as the lesion. This is coupled with loss of pain and temperature sensation on the opposite side of the lesion.
- Central cord syndrome.** Damage to the central area of the cord predominantly resulting in marked weakness of the arms relative to the legs. Hand grip is tested by shaking hands with the patient. Flexion-extension injuries

result in ischemia and edema of the center of the spinal cord, producing this syndrome even without fracture.

5. **Cauda equina syndrome.** Damage to the cauda equina as it descends through the sacrum or lumbar spine. This damage produces anesthesia in a saddle distribution of the perineum with loss of anal sphincter tone.

For All Spinal Cord Injuries:

Neurogenic shock (hypotension with bradycardia) is a diagnosis of exclusion. Neurogenic shock responds to volume loading. Vasopressors may be added after volume loading is well under way.

Consult with a neurosurgeon early to ensure that all treatment options are pursued to preserve neurologic function.

7

Chest Wall and Pulmonary Injury

Palpate the clavicles and the chest wall again for crepitation and fracture. If there are wounds to the chest, do not explore them. Pneumothorax may result. Wounds that enter the chest should be covered with an occlusive dressing with one corner or edge left free to prevent development of tension pneumothorax.

Do not remove impaled objects because they may be providing tamponade to a pulmonary or cardiac wound. Observe for asymmetrical chest movement that may occur with pneumothorax, flail chest, and spinal cord injury with abdominal breathing.

If a needle thoracostomy has been performed during the initial survey for tension pneumothorax, insert a 28 - 32 French chest tube on that side now. (**Vol II – BREATH SKILLS 1 CHEST TUBE INSERTION**) Connect it to chest suction with preparations made to collect blood for possible auto-transfusion, note that auto-transfused blood has none to minimal clotting factors. (**Vol II – BREATH SKILLS 2 CHEST SUCTION AND AUTOTRANSFUSION**) Chest wall crepitation means pneumothorax on that side. Insert a chest tube.

The immediate return of 1500 mL of blood means that emergency surgery is probably needed. The immediate return of < 1500 mL of blood that continues to come out at a rate of about 200 mL/h is also ominous. If the BP falls precipitously with drainage of a large volume of blood from the chest, clamp the chest tube because the blood in the chest may have had a tamponade effect. Using an autotransfuser allows the return of the patient's own blood, only if there is no possibility of gastric contents contaminating the chest cavity.

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If a bronchus rupture has occurred, there may be a massive air leak from the chest tube with inability to inflate the lungs. If this occurs, attempt to place the tip of the endotracheal tube into the left or right main bronchus, into that side that does not have the massive air leak.

Use a BVM to ventilate the patient. The ventilatory rate for adults is 10 to 12 breaths per minute (or 1 every 5 to 6 seconds) with a tidal volume of 500 to 600 mL (6 to 7 mL/kg). An orogastric tube will also help prevent gastric distension and subsequent elevation of the diaphragm. Palpate the ribs to detect rib fractures. Multiple rib fractures on the same side may result in paradoxical chest wall motion with breathing signifying a flail chest. Not all patients with flail chest need ET intubation. Monitor the patient carefully and intubate at the first sign of fatigue or hypoventilation.

Chest wall bruising or sternal tenderness – as with steering wheel impact – may indicate that the aorta has been injured. Carefully review the chest x-ray for mediastinal widening or other clues, such as tracheal deviation to the right and an apical cap. If thoracic aortic rupture is being considered, attempt to keep the BP at about 100 mm Hg. A helical CT scan of the chest with IV contrast is now considered to be adequate screening for this condition. However, this is technique dependent. Be certain your radiologist and CT technician understand the purpose of the scan.

Examine the chest x-ray for fractured ribs, pneumothorax, hemothorax, scapular fractures, elevated diaphragm, abnormal cardiac silhouette, lung contusion, position of the ET tube and other tubes, and thoracic spine. With bronchial injury, tracheal injury, and esophageal injury, mediastinal air is visible. If a pneumothorax is present, insert a 28 - 32 French chest tube and connect to chest suction.

If the patient has been tracheally intubated, listen carefully for breath sounds. If the tube is in too far, the left lung will be inadequately ventilated. Also check for this on the chest x-ray. When a patient is intubated, obtain a follow-up x-ray to check for correct tube placement.

Pulmonary contusion gets worse as the patient's hemodynamic status improves. Blood oxygen saturation will decrease. Transtracheal pressures increases as the lungs become less compliant. Consider using 5 to 10 cm H₂O PEEP if pulmonary contusion is present and hypoxia develops. This may be problematic for a patient in a helicopter, so consider inserting a prophylactic chest tube so that if a pulmonary bleb ruptures, the lungs will still inflate.

8

Cardiac Tamponade

Muffled heart sounds associated with shock and distended neck veins (Beck's triad) may indicate cardiac tamponade. Cardiac tamponade with blunt trauma is uncommon, but does occur with atrial rupture. The majority of patients with this injury have suffered penetrating trauma.

Perform echocardiography from the subxiphoid window or along the left sternal border. **(Vol III – ULTRASOUND 2 EMERGENCY ULTRASOUND TECHNIQUES)**

Hypovolemic shock and spinal shock are associated with a vigorously pumping heart; significant myocardial contusion is associated with a weakly pumping heart. Cardiac tamponade is associated with a ring of hypoechoic fluid surrounding the heart. A compressed right ventricle with a weak beat may also be detected. Ultrasound is a valuable tool in the management of trauma.

In such a case, insert a guidewire-assisted catheter device from the left subxiphoid position directed at the left scapula. **(Vol II – CIRC SKILLS 8 SAPHENOUS VEIN CUTDOWN)** Use repeated aspirations to keep the patient stable pending surgical repair.

If this procedure is unsuccessful, consider an emergency thoracotomy if your facility has surgical backup. The blood in the pericardial sac may be clotted. Perform the thoracotomy as described in **Vol II – CIRC SKILLS 4 EMERGENCY THORACOTOMY**. If the heart is beating, make a small incision into the sac creating a "window" through which the clot can extrude. A sterile suction catheter may be used to extract the clot. Use a finger to cover this window pending surgical repair.

If the heart is not beating, the sac needs to be widely opened to facilitate internal massage and staple repair of the wound. Extend the pericardial incision longitudinally to avoid injuring the phrenic nerve, which also runs longitudinally along the posterior margin of the pericardial sac. If possible, repair the cardiac wound using a skin stapler. Do this before restarting the heart. **(Vol II – CIRC SKILLS 4 EMERGENCY THORACOTOMY)** The staples will be removed and replaced with sutures in the operating room.

If myocardial contusion is possible, observe for ECG changes and evidence of heart failure. Lidocaine 1 mg/kg IV followed by an infusion of 1 to 4 mg IV/min may be indicated for significant ventricular arrhythmias. Amiodarone 150 mg infused over 10 minutes may also be used. Ventricular arrhythmias in this situation are rare.

9

Hypovolemic Shock

CPR is not as effective when hypovolemia is involved. Volume replacement is one important aspect of resuscitation from traumatic cardiac arrest. The others are oxygenation and relief of any tension pneumothorax.

Use ultrasound for cardiac view as patient can be in a Low Cardiac Output state, and as such not have a palpable pulse.

If IVs have not been established, insert two large bore IVs. An intraosseous method, such as the EZ-IO, may be used. A lower extremity IV may not be effective if there is a vena cava laceration. Number the IV bags to keep track of the amount of fluid being infused.

Always be careful to avoid air embolism. Track vital signs and review the physical examination for continuing evidence of hypoperfusion (prolonged capillary refill, pallor, coolness, absent or decreased pulses). BP readings alone are not enough. Check the accuracy of your automatic BP machine by comparing it to manual auscultation measurements. These machines are not always reliable in shocky patients. Observe the ECG monitor. Head injuries in and of themselves do not cause hypotension.

Warm crystalloid (NS or LR) solution (1 L) is the preferred fluid during the initial few minutes of resuscitation. To avoid hemodilution, switch to blood transfusion after 1 liter crystalloid (NS or LR) if patient remains in shock. Blood is usually administered as packed RBCs. While typed and crossmatched blood is preferred, type-specific blood may also be used. If blood is required immediately, Type O blood is also acceptable. Use Type O Rh-negative for any female with current or future childbearing potential; for all others, use O Rh-positive.

If blood is required immediately, give TXA as 1 gm IV over 10 minutes, for an adult, and 15 mg/kg to max of 1 gm in a child. Anticipate massive transfusion. Along with PRBCs, consider giving fresh frozen plasma (FFP) and platelet packs (if available) in a 1:1:1 ratio.

Another common cause of hypocoagulation is hypothermia. Immediately take measures to keep the patient warm. An overhead warming light is useful. Use blood and fluid warmers to warm the IV fluids. Keep the patient covered whenever appropriate.

If blood is not available and continuing fluid infusion is necessary, switch from NS to Ringer's lactate to avoid hyperchloremic acidosis.

Consider early use of tranexamic acid (TXA, Cyklokapron) to reduce bleeding in a patient in hemorrhagic shock. Give 1 g IV over 10 minutes as an initial dose.

Geriatric patients are at great risk of volume overload and pulmonary edema. If the jugular veins are not clearly visible, insert a central venous line in such patients so that central venous pressure (**Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS**) can be monitored to titrate volume resuscitation. Head-injured patients and spinal cord-injured patients also benefit from this titration. To rule out the complication of pneumothorax as well as to check placement, always obtain a chest x-ray after insertion of a central line.

To tamponade abdominal or pelvic bleeding, use commercially available pelvic wraps or a sheet and towel clips.

Consider other causes of hypocoagulopathy.

1. Patients on aspirin or Plavix have poorly functioning platelets. An average-size adult who takes aspirin needs about 4 platelet packs to restore adequate platelet function.
2. Measure INR in patients on Coumadin. Give Kcentra® if available; otherwise give FFP units to correct. Under most circumstances, administer Vitamin K.
3. Draw a fibrinogen level in patients with poor liver function, as in chronic alcoholism. Cryoprecipitate units may be needed.
4. If patient is on a Directly acting Oral AntiCoagulants (DOAC), previously called novel oral anticoagulants (NOAC), such as the direct thrombin inhibitors or direct factor Xa inhibitors, see **VOL I – ACUTE CARE 47 REVERSE ANTICOAGULATION THERAPY**.

Inform the referral center of these concerns. Even though your facility may not have these blood products in the laboratory, considerable time can be saved by alerting the referral hospital about your concerns.

10

Abdominal Injury

If the stomach is filled with air, blood, or food, insert a large orogastric tube and connect to suction. If the patient is intubated, an 18-gauge nasogastric tube may be inserted orally. Pregnant women are especially prone to gastric distension and should almost always have a gastric tube placed when traumatized.

Abdominal tenderness may mean peritoneal irritation secondary to free blood

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from bowel injuries, splenic rupture, liver laceration or disruption, vena caval or aortic laceration, mesenteric laceration, penetrating wounds, and renal injuries. Patients may minimize abdominal tenderness when distracted by other painful injuries, such as pelvic or femur fractures. Examine for linear bruising caused by seat belts. These are associated with devascularizing injuries to the bowel. Retroperitoneal organs may be injured without signs of peritoneal irritation. Duodenal rupture (or hematoma) and pancreatic lacerations are examples that may be difficult to detect on physical examination.

Lower rib cage injuries may produce abdominal tenderness, but this is a diagnosis of exclusion. Penetrating abdominal knife wounds may be explored down to the first layer of fascia to detect peritoneal penetration. Beyond the first fascial layer, exploration is unreliable.

Flank wounds may involve the colon, kidneys, vena cava, and aorta. When intra-abdominal wounds are suspected, administer a broad-spectrum antibiotic, such as cefoxitin 2 g IV. Penicillin-allergic patients (anaphylactic reaction) may be treated with gentamicin 2 mg/kg IV and clindamycin 600 mg IV.

In most instances, CT scans and/or ultrasound have replaced peritoneal lavage in abdominal trauma. Peritoneal lavage may not be appropriate in the rural setting unless (1) immediate surgical intervention is available and (2) the surgeon must choose between embolization of pelvic bleeding in the radiology suite or surgery in the operating room.

Rapid bedside ultrasound examination (FAST-Focused Assessment Sonography in Trauma) performed by the clinician has become routine at many institutions. Sensitivity and specificity are variable, but identification of intraperitoneal fluid/blood greater than 700 cc, and detection of pericardial tamponade are quite reliable and may guide subsequent management in selected cases. Morrison's pouch view, subxiphoid view of the heart, and trans-vesical views are the easiest to perform.

11

Pelvic Fracture

Compress the pelvis in a horizontal and AP plane. Movement or pain with this test signifies pelvic fracture. Movement signifies an unstable pelvic fracture. Review the supine AP view of the pelvis. (**Vol II – XRAY SKILLS 3 INTERPRETATION OF PELVIC X-RAY**) Pelvic fractures may be difficult to see, but some clue is almost always present. Consider any pelvic fracture dangerous. The major danger is occult hemorrhage.

Follow a protocol when evaluating pelvic trauma:

1. Examine for perineal lacerations. Do not explore perineal lacerations because external hemorrhage may result. Check for blood in the underwear.
2. Perform a rectal exam to feel for the position and consistency of the prostate gland in males (including adolescent males) and to detect frank blood in the rectum as well as to determine sphincter tone. Perform this exam before attempting to insert a Foley catheter because a boggy or high-riding prostate indicates urethral transection. Blood in the rectum means rectal penetration or laceration. Begin antibiotics, such as cefoxitin 2 g IV. Treat penicillin-allergic patients (anaphylactic reaction) with gentamicin 2 mg/kg IV and clindamycin 600 mg IV.
3. Blood at the urinary meatus in males also indicates urethral injury. Insertion of a Foley catheter is contraindicated at this time. However, an urologist may elect to use a Foley to treat a partial dissection of a urethra. In females, gentle insertion may still be performed.
4. Pelvic fractures, with or without instability, can be associated with massive internal hemorrhage. Circumferential compression may help reduce bleeding. Apply circumferential pressure with a sheet or commercially available binder. **(Vol II – TRAU SKILLS 3 PELVIC FRACTURE STABILIZATION)**
5. If the bladder is distended to palpation or ultrasound examination and a urethral disruption or injury has occurred (blood at the meatus), perform a percutaneous suprapubic cystostomy using guidewire technique. **(Vol II – TRAU SKILLS 4 SUPRAPUBIC CYSTOSTOMY)** This is especially important for head-injured patients because bladder distension can raise ICP. If the patient is in shock, do not perform this procedure.
6. Check for femoral pulses because some pelvic fractures are associated with iliac artery injury.
7. Perform a bimanual vaginal examination feeling for laceration and bone spicules. If positive, begin antibiotics, such as cefoxitin 2 g IV. Treat penicillin-allergic patients (anaphylactic reaction) with gentamicin 2 mg/kg IV and clindamycin 600 mg IV.

If the patient has an unstable pelvic fracture, take care not to move the pelvis, causing more hemorrhage. Instead of log rolling the patient to view his or her back, lift the patient straight up. If the patient is large, simply slide one's hands under the patient to feel the back.

12

Trauma in Pregnancy

Palpate the abdomen for the presence of a pregnant uterus. If the uterus is palpable above the umbilicus, turn the patient 30° toward the left lateral decubitus position to take the weight of the uterus off of the inferior vena cava. In a spine-boarded patient, towels or blankets can be placed under the right edge of the board to achieve this. In the non-boarded patient, blankets or pillows

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can be placed under the right thigh, buttock, flank and shoulder. The use of Ultrasound can be useful when the cause of hypotension is not clear. Do not forgo essential x-ray studies in pregnant patients. Be aware of the blood volume changes of pregnancy and the placental sensitivity to circulating catecholamines when the mother's blood volume is decreased. Evaluate fetal heart rate with a Doppler stethoscope or ultrasound. Fetal heart rate (FHR) is a sensitive indicator of the mother's volume status.

Placental abruption is common with significant abdominal trauma and may not be accompanied by physical findings. Even small abruptions can result in fetomaternal hemorrhage with isoimmunization. If the mother is Rh-negative, administer Rh-immune globulin for abdominal trauma: 50 µg in the first trimester and 300 µg during the second and third trimesters of pregnancy. Placental abruption may or may not result in external vaginal bleeding. Ultrasound should be preformed to assess for possible placenta abruption or other causes of vaginal bleeding but frequently will not reveal small abruptions. If bleeding is present sterile speculum exam of the cervix is permissible, but digital exam should not be preformed until the possibility of placental previa has been ruled out. ([Vol III – OB7 BLEEDING IN THE SECOND HALF OF PREGNANCY](#))

If no serious injuries are detected in a pregnant patient but abdominal trauma has been sustained, monitor the patient for at least 4 hours for signs of abruption using FHT tracings, contractions, uterine tenderness, and vaginal bleeding. ([Vol III – OB9 TRAUMA IN PREGNANCY](#)) Obtain obstetric consultation.

If the patient has soaked fewer than 3 pads and has no positive findings after 4 hours of monitoring, she may be discharged with instructions to contact her physician or return to the ED should any of the following symptoms occur: vaginal bleeding, abdominal pain, contractions, fainting or dizziness, rupture of membranes, and decrease in fetal movement.

Amniotic fluid embolism resulting from abruption results in respiratory failure and disseminated intravascular clotting (DIC) consuming fibrinogen. Treat according to the results of blood clotting tests and clot lysis. Consider perimortem cesarean section if fetal heart beats are present on ultrasound and the level of the uterus is well above the umbilicus. ([Vol III – OB10 EMERGENCY CESAREAN SECTION](#))

See also [Vol III – OB1 PHYSIOLOGY OF PREGNANCY](#) for more information about the physiology of the pregnant patient and the emergency management of labor.

13

Major Joints, Femur Fractures, and Amputations

Reduce major joint dislocations (such as hips, knees, and ankles) using sedation,

relaxation, and traction with counter-traction. Short-acting sedatives such as etomidate and propofol work well for this. Reduce Etomidate dose to 0.1- 0.2 mg/kg to avoid respiratory depression. Add an opioid for pain relief. **(Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION)** Take airway precautions. Before the reduction, check for pulses/motion. Absent or decreased pulses can result from deformity or direct trauma to arteries and systemic hypotension. Reduction often results in return of pulses. Remove jewelry from extremities prior to manipulation.

When a bone end or fragment is protruding from a wound, rinse any gross contaminant (eg, pebbles or dirt) with normal saline-soaked sponges then apply traction to reduce the fracture.

If a penetrating wound is in proximity to a major vessel or nerve, special imaging and/or surgical exploration may be indicated, even in the presence of an apparently good pulse.

Check the legs, thighs, and arms for tense swelling of the muscle compartments. Alert your surgeon about any area suspicious for a compartment syndrome. A decreased pulse is not a necessary component of the syndrome. Fasciotomy may be needed if the patient's transfer is delayed.

If life-threatening problems are ongoing, simply splint the limb in its current position, deferring reduction until the clinical situation permits. Reduce dislocated shoulders and elbows and splint when time permits. **Avoid taking x-rays of the extremities for injuries that you will not have to definitively manage.**

Fractured femurs. Significant blood loss (as much as 1500 mL) can occur into the soft tissues of the thighs. Traction splints may reduce this loss. Apply traction splints (Sager, Hare, Kendrick, Slishman or one of the other femur traction devices) to reduce femur fractures. **(Vol II – TRAU SKILLS 1 COMPARTMENT PRESSURE MEASUREMENT)** The helicopter service may prefer one type of splint because of space issues.

If **amputation** has occurred and the body part is available, rinse in NS solution and place in a plastic bag (in case it can be implanted later or serve as a source for skin grafting). Place the bag on ice, but do not freeze.

14

Burns

Perform orotracheal intubation **(Vol II – AIR SKILLS 3 OROTRACHEAL INTUBATION)** with in-line immobilization for the following indications: stridor, soot in the pharynx, edema of the uvula, significant burns of the neck, and depressed LOC. If an explosion or closed space fire has occurred, be especially alert to the need to intubate. Also consider possible cyanide exposure. The RSI

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protocol may be advisable if the patient has muscle tone and reflexes. (**Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION**) Ventilate with oxygen.

Completely undress the patient. Be careful of smoldering clothing. Remove jewelry and contact lenses. Immediately cool all burns with NS if <10% of TBSA is burned. (If NS is not available, use water.) If >20% TBSA is burned, cover patient with dry gauze dressings. Patients with burns >20% TBSA are at greater risk for developing hypothermia if wrapped in wet gauze. If <20% TBSA is burned, keep burns moist, but cover patient with blankets to prevent hypothermia.

Start 2 IVs in unburned skin, but use burned skin sites if other sites are not available. Obtain pain relief with morphine IV. **Do not administer IM.** Titrate from 4 mg to whatever dose provides relief. (Note that you may need much higher doses than normal.) Ventilatory support may be needed.

Administer crystalloid (LR) solution IV to restore normal BP if possible. Insert a Foley catheter and monitor urine output to provide a guide to the adequacy of fluid administration. A urine output of 50 to 70 mL/h is the goal. (The gold standard for fluid resuscitation in burn victim is to maintain urine output at 50 to 70 mL/hour.)

Don't treat tachycardia as all burn patients are prone tachycardic.

For any patient with a total body surface burn of 20% deep partial or full thickness burns or greater, administer IV fluids. A Formula that can be used to calculate the estimated fluid requirements of burn patients:

Percentage of Body Surface Area burned X weight in kg X 2 mL crystalloid (LR) solution = 24 hour fluid requirement.

For pediatrics use 3 ml.

For electrical burns use 4 ml

Monitor urinary output hourly and titrate fluids accordingly. Aim for 0.5cc/kg/hr. in and adult and 1cc/kg/hr. for a child.

Half will be needed during the first 8 hours.

The other half will be needed during the following 16 hours.*

***New ATLS Guidelines 2018 Fluid resuscitation for burn patients**

Consider a H2 blocker or PPI. Insert an NG tube if the burn is significant. Using the **Rule of Nines**, grossly estimate the percentage of body surface burned: the head is 9%, each arm is 9%, the front of the torso is 18%, the back of the torso is 18%, each whole leg is 18%, and the perineum is 1%.



The Rule of Nines is used to estimate percentage of body burned.

Monitor the patient's core temperature with a rectal probe. Do not use cool NS soaks for pain relief. A dry occlusive dressing will suffice. Do not apply antiseptic creams. Do not break intact blisters. Do not administer antibiotics. Consider tetanus status.

Circumferential third-degree burns of the extremities or the chest (torso) can produce a tough eschar that can compromise blood flow or chest wall movement. Escharotomies may be necessary. Contact a burn consultant emergently regarding how and where to perform these escharotomies. Fortunately, these areas will be anesthetic as the nerve endings have been destroyed.

Treat patients with the following indications at a burn center:

- Burns > 10% of body surface in patients < 10 or > 50 years
- Burns > 20% of body surface in all age groups
- Full thickness (third-degree) burns > 5% of body surface - all ages
- Burns involving the hands, feet, eyes, perineum
- Inhalation, electrical, or chemical burns
- Burns associated with major trauma or preexisting disease

Electrical burns

The extent of electrical burns may be difficult to ascertain immediately. The injury is typically to the deeper structures of the body, such as muscle and vascular (not superficial or cutaneous) tissue. Thus, the damage is not easily observable to the naked eye on physical exam. For this reason, cardiac dysrhythmias may result from electrical burns. ECG monitoring is important.

When dysrhythmias occur, [see Vol I – PATHWAY 2 CARDIOVASCULAR EMERGENCIES](#) for treatment options. Another common finding is muscle necrosis

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resulting in myoglobinuria. Clinically, this is observed as a reddish hue to the urine. Increase the infusion of fluids to result in a urine output of 100 mL/h. add a bicarbonate drip and titrate to urine pH. If the urine does not rapidly clear, add 25 g mannitol IV and add 12.5 g to each subsequent liter bag of crystalloid (NS or LR).

Many patients with electrical injury have suffered injuries from seizures and falls, including cervical spine injuries and head trauma. Carefully evaluate the whole patient.

Lightning strike

Cardiac arrest with VF or asystole is the most serious effect of lightning strike. Treat any dysrhythmia as described in **Vol I—PATHWAY 2 CARDIOVASCULAR EMERGENCIES**. Burns from lightning strike are often inconsequential. Wet clothing may protect the patient by causing the current to flow over the patient rather than through him or her. This is referred to as flashover phenomenon.¹⁰ Under unusual conditions, an explosive or implosive effect from a lightning bolt can cause blunt injury. Initial management is as with all trauma patients.

15

Heat Stroke

Heat exhaustion is accompanied by weakness, faintness, and diaphoresis. Core body temperature does not reach dangerous levels. Cool liquids and rest usually suffice.

Heat stroke is accompanied by weakness, faintness, or coma and (classically) dry skin, with dangerous core temperatures of 40.5°C (105°F) or higher. In athletes and others well accommodated to heat, the skin may be diaphoretic.

A patient usually presents with prostration and a depressed LOC. Seizures may occur. Orotracheal intubation may need to be performed. (**Vol II—AIR SKILLS 3 OROTRACHEAL INTUBATION**)

When vital signs reveal severe hyperthermia, immediately begin cooling the body to reduce core temperature before permanent CNS damage occurs. An effective method is to undress the patient and spray tepid water on the body while fans blow air over the body. The resulting evaporation lowers body temperature without causing shivering that can slow the cool-down. Ice packs may be placed in the axillae and groin. If the core temperature fails to respond, gastric and bladder irrigation with cool NS may also be used. Lower the body temperature to about 40°C (104°F) then remove the water and fans because the core temperature will continue to decrease. **Geriatric patients taking psychotropic medications and/or living in settings without air conditioning are**

especially prone to heat stroke. Monitor such patients carefully.

Many complications may ensue including cerebral edema, heart failure, myonecrosis, and pulmonary edema. The patient may be severely dehydrated on arrival. Hydrate vigorously but monitor carefully so as not to overshoot the mark. Central venous access ([Vol II – CIRC SKILLS 3 CENTRAL VENOUS PRESSURE MEASUREMENT](#)) may be advisable, especially in geriatric patients. Most patients will require monitoring in an intensive care unit post resuscitation.

16

Severe Hypothermia

Many patients with hypothermia have also suffered trauma. Evaluate hypothermia patients as trauma patients.

Definition According to Core Temperature

Mild hypothermia	34°C to 36°C (93°F to 96°F)
Moderate hypothermia	30°C to 34°C (86°F to 93°F)
Severe hypothermia	< 30°C

Mildly hypothermic patients may be rewarmed with the external application of warmth and warm fluids. Moderately hypothermic patients must be closely monitored because of cardiac irritability at this temperature. However, external warmth and warm IV fluids will suffice as rewarming methods.

Core temperature is best measured with an esophageal probe with a monitor capable of reading very low temperatures, but this is invalid if gastric lavage is used. Rectal temperature is the most practical method.

The severely hypothermic patient may appear to be clinically dead. In the absence of other factors incompatible with life, aggressively attempt resuscitation until the core temperature is above 34°C (93°F). Asystole above this temperature should not be attributed to hypothermia and other causes should be considered. ([Vol I – ACUTE CARE PORTALS, ASYSTOLE](#))

Patients have survived total immersion in very cold water for 30 to 45 minutes and core temperatures of about 15°C to 16°C (60°F). Use clinical judgment. The American Heart Association recommends attempting to rewarm every patient before pronouncing death, as astounding cases of survival from apparent death do occur.

Aggressive airway management is necessary. RSI is usually not needed. Cold bronchorrhea occurs and demands good tracheal toilet. Heated humidified oxygen ventilation is also needed. Gross movement – as in moving from an

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upright to supine position – can precipitate VF. Perform CPR. If the patient is in VF, attempt defibrillation one time, but do not persist.

The natural history of VF arrest in hypothermia in a patient with a normal heart is as follows:

At a core temp of about 21°C (75°F), the rhythm spontaneously converts to atrial fibrillation with a slow ventricular response. At a core temp of about 27°C (80°F) the ventricular response becomes faster. At a core temp of about 30°C (86°F) the rhythm spontaneously converts to normal sinus rhythm.

These are estimates only, and individual cases vary. Continue CPR until the pulse is palpable. If the patient has not converted at a temp of about 32°C (90°F), administer appropriate cardiac medications to restore sinus rhythm. (**Vol I – PATHWAY 2 CARDIOVASCULAR EMERGENCIES**) Cardiac medications are not effective in severely hypothermic patients and may accumulate with adverse effects when the patient is warm. Hypothermic patients will have abnormal electrolytes, acid/base components, and glucose. Measure these frequently during the rewarming process.

Hypothermic patients are hypovolemic because of cold diuresis produced when the cold kidneys fail to concentrate urine. Establish large bore IVs for administering a bolus of warm NS. The warm IV fluids will correct the hypovolemia, but do not have enough kilocalories of energy to warm the patient.

Cold causes a shift of the oxyhemoglobin dissociation curve to the left. Some degree of acidosis (which causes a shift to the right) is protective. A low PCO₂ can cause alkalosis; do not hyperventilate these patients.

Blood gases may be corrected for temperature or not. One line of reasoning is that the whole body is cold, not just the blood, so measuring blood gases at 37°C is appropriate because the cold body temperature changes the body's enzyme systems and chemistries in the same way as it does the blood gases. Normal blood gases, under these circumstances, indicate that a physiologic balance of oxygen, hydrogen ions, and carbon dioxide are present in a hypothermic patient.

To rewarm severely hypothermic patients, avoid using external methods because increasing the oxygen demands of the periphery before the central circulation is capable of delivery is counter-productive. Peripheral vasodilation may return cold blood to the core with a resulting paradoxical drop in core temperature. Use internal methods if the patient's core temperature is less than 30°C (86°F). Expect a rate of rewarming of about 4°C (7°F) per hour.

Internal Rewarming Methods That May Be Used:

1. Heated, humidified oxygen ventilation at 42°C to 46°C (108°F to 115°F).
2. Warm IV (or IO) fluids at 40°C to 43°C (104°F to 109°F) Again, warm IV fluids correct hypovolemia, but do not have enough kilocalories of energy to warm the patient.
3. Warm gastric lavage using tap water at 40°C to 43°C (104°F to 109°F). Use a standard gastric lavage set up
4. Warm urinary bladder lavage using sterile NS at 40°C to 43°C (104°F to 109°F). Connect the Foley catheter to a standard gastric lavage set up that has been sterilized. Use a volume of about 5 mL/kg.
5. Closed peritoneal lavage if the patient does not have a surgical incision on the abdomen. Use NS at 40°C to 43°C (104°F to 109°F).
6. Closed left chest lavage using a chest tube connected to chest suction (**Vol II – BREATH SKILLS 1 CHEST TUBE INSERTION**) and a large needle thoracostomy catheter inserted over the 3rd rib in the midclavicular line (**Vol II – BREATH SKILLS 5 NEEDLE THORACOSTOMY**). Warm NS at 40°C to 43°C (104°F to 109°F) is infused into the chest through the needle thoracostomy catheter and allowed to bathe the heart and left lung before evacuation through the chest tube.
7. Cardiopulmonary bypass is the quickest and most effective way of rewarming severely hypothermic patients. Trauma centers are prepared to do this. Patients who have solidly frozen extremities or are in cardiac arrest are most likely to benefit with this form of rewarming. Consult freely about these cases.

17

Drowning

Some drowning patients are actually diving accident victims. Always work up patients with possible trauma in mind.¹¹ Patients should be immobilized. In the ED, evaluation and treatment follows the usual trauma patient protocol.

Patients rarely inhale much fluid into the lungs because of laryngeal spasm and breath holding. The occurrence of hemolysis in fresh water drowning and hemoconcentration in salt water drowning is largely theoretical. A significant admonition, however, is that patients may develop acute pulmonary edema hours after the event. All drowning patients require a period of observation, even if asymptomatic upon presentation. If the patient remains entirely asymptomatic with normal respiratory function at 6 to 8 hours, he or she may be safely discharged. Any abnormality that occurs during the observation period such as cough, difficulty breathing, abnormal lung sounds, oximetry, or blood gasses in an indication for further observation and intervention as needed. Recognize that hypothermia may be present. Measure rectal temperature.

If hypothermia, trauma, and significant aspiration are not present, hypoxic brain injury is probably the cause of continuing coma. (**Vol I – PATHWAY 1 ALTERED LEVEL OF CONSCIOUSNESS**)

18

Caustic Substance Ingestion

Caustic ingestions usually involve the intentional ingestion of drain cleaners containing concentrated alkaline powders or solutions. These strongly basic substances, usually sodium or potassium hydroxide, produce liquefaction necrosis. In powder form, they likely result in more proximal tissue destruction and more airway compromise. In liquid form, they likely destroy tissue in the distal esophagus and stomach.

The potential for airway compromise is great. If there is any question about swelling, stridor, edema, or burning about the posterior pharynx, orotracheally intubate the patient. (**Vol II – AIR SKILLS 3 OROTRACHEAL INTUBATION**) Perform RSI to ensure the gentlest intubation. (**Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION**) Nasotracheal intubation is contraindicated because it may result in more injury. If orotracheal intubation is not successful, perform emergency cricothyrotomy. (**Vol II – AIR SKILLS 13 CRICOTHYROTOMY**) Administer oxygen and establish IVs. Pain control may be needed. Support the patient's vital signs.

Beyond airway control and general support, little can be done in the rural setting. Gastric lavage and dilution may do more harm than good. Consult the otolaryngologist and prepare to transfer as quickly as possible. The consultant may favor steroid therapy and antibiotics. Subsequent gastric and esophageal perforation may occur a week or two after the ingestion.

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January 2019

PATHWAY 9: TRAUMA EMERGENCIES (SECONDARY SURVEY FOR PEDIATRIC)

The team continues the resuscitation along the pathway suggested by the initial clinical impression. Each pathway includes a complete, thorough, and rapid physical examination with additional history taking. The team leader is wary of conditions that may not be apparent. To obtain additional clinical data or to correct a missed or newly developed condition, the team leader repeats the initial survey if the patient is not responding satisfactorily.

PEDS: Because this entire pathway pertains only to pediatric patients, the convention of underlining has been omitted. For purposes of this survey, the pediatric age group ranges from infancy to the attainment of adult size.

Children are not small adults.

This obvious admonition needs to be stressed whenever discussing care of children. The ABCs are the most important aspects of any resuscitation, but certain aspects of the physiology and anatomy of children need to be considered. These differences are clarified in this pathway.

The team leader performs a head to toe, rapid but thorough, physical examination. Some possible abnormalities are:

Number	Diagnosis/Condition	
1	Compromised Airway	Vol I – AIRWAY SKILLS
2	Abnormal LOC	Vol I – PATHWAY 1 ALTERED LEVEL OF CONSCIOUSNESS
3	Facial and Skull Fracture/Scalp Laceration	Vol I – ACUTE CARE PORTALS; VOL II – DISAB SKILLS 2 RANEY SCALP CLIPS; VOL III – NEU5 INCREASED INTRACRANIAL PRESSURE
4	Direct Eye Trauma	
5	Soft Tissues of the Neck	Vol I – AIRWAY SKILLS
6	Cervical Spine Injury	VOL II – XRAY SKILLS 1 C-SPINE RULES, XRAY SKILLS 2 CERVICAL SPINE XRAY INTERPRETATION
7	Chest Wall and Pulmonary Injury	VOL II – BREATH SKILLS 1 CHEST TUBE INSERTION, BREATH SKILLS 2 CHEST SUCTION/ AUTOTRANSFUSION, BREATH SKILLS 5 NEEDLE THORACOSTOMY

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8	Cardiac Tamponade	VOL II–CIRC SKILLS 6 PERICARDIOCENTESIS
9	Hypovolemic Shock	Vol I– ACUTE CARE PORTALS; VOL II–CIRC SKILLS 1 ARTERIAL AND VENOUS CATHETER INSERTION; VOL III– CV17 SHOCK
10	Abdominal Injury	Vol I– ACUTE CARE PORTALS; VOL III– CV17 SHOCK
11	Pelvic Fracture	VOL II– TRAU SKILLS 3 PELVIC FRACTURE STABILIZATION, XRAY SKILLS 3 INTERPRETATION OF PELVIC XRAY
12	Major Joints, Femurs, and Amputations	Vol I– ACUTE CARE PORTALS; VOL II– TRAU SKILLS 1 COMPARTMENT PRESSURE MEASUREMENT, TRAU SKILLS 2 FEMUR FRACTURE SPLINTING; VOL III– CV17 SHOCK
13	Burns	Vol I– ACUTE CARE PORTALS; VOL III– ENV3 BURNS MANAGEMENT
14	Heat Stroke	VOL III– ENV2 HYPERTHERMIA
15	Severe Hypothermia	VOL III– ENV1 HYPOTHERMIA
16	Drowning	VOL III– ENV4 DROWNING
17	Caustic Substance Ingestion	

Imaging

For patients who require rapid transfer, limit studies to those that impact immediate care.

Chest x-ray is mandatory to reveal pneumo/hemo thorax as well as proper placement of lines and tubes. Order pelvic x-rays on the basis of clinical suspicion and mechanism of injury.

Order these immediately and continue the examination as the films are being obtained. If the patient deteriorates during this survey, repeat the initial survey, checking for airway, breathing, circulation, and so on.

Obtain additional imaging based on secondary survey findings. These may include plain films and/or CT of the head, spine, chest, abdomen, and pelvis.

Hidden back injury or wound

Carefully log roll the patient, while maintaining c-spine immobilization, just enough to expose the back. Palpate the entire length of the spine checking for deformity, hematoma, lacerations, and pain. Palpate the back of the head, posterior thorax, scapula, flanks, and buttocks. The log roll maneuver in the presence of an unstable pelvic fracture can stimulate more bleeding. The team should carefully lift the patient straight up instead. If spinal injury is suspected, securely immobilize the patient. Perform the rectal exam when the patient is lying flat on their back, not when the patient has been rolled to the side or lifted, as they can arch their back, and possibly cause aggravation of any spinal injury. Remove glass shards and any other harmful materials before laying the patient back down. If you suspect spinal injury, immobilize the patient securely.

If the patient is to remain on the extraction board, take this opportunity to pad the board or use a cushion device such as the Backraft.

Compromised Airway

1

During the initial survey, the airway has been evaluated. Re-evaluate the airway, checking for patency, airway protection, correct ET tube placement, breath sounds, stridor, hypoxia, respiratory effort, soot, or burns. If possible, complete a mini-neuro exam before doing RSI.

The Mini-Neuro Exam

1. **Level of Consciousness**
AVPU and Glasgow Coma Scales
2. **Pupils and Vision**
Size, equality, and reactivity to light
Conjugate or disconjugate gaze
Count fingers
3. **Tympanic Membranes**
Hemotympanum
4. **Neck**
Posterior midline tenderness
5. **Extremities**
Movement and strength on command or to pain
Ankle, patellar, and brachial reflexes
Babinski reflexes and clonus
Sensation and position sense
6. **Trunk and Perineum**
Priapism, saddle sensation, anal sphincter tone
Sensory level

**PEDIATRIC TRAUMA EMERGENCIES
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A child's airway is significantly different from an adult's.

1. The cartilages of a small child's larynx and trachea are soft and easily compressed by hyperextension and hyperflexion of the neck, resulting in airway obstruction. Because of the softness of the trachea, the esophageal intubation detector (EID) (**Vol II – AIR SKILLS 1 AIDS TO INTUBATION**) is not useful. The operator cannot make a distinction between the trachea and the esophagus in children less than 1 year of age. End-tidal CO₂ is a reliable method in all but the smallest infants (< 2 kg).
2. A standard ET tube introducer (ETI) (**Vol II – AIR SKILLS 1 AIDS TO INTUBATION**) is 15 French in diameter. The smallest ET tube that fits over the ETI is size 4.0. For pediatric airway equipment sizes, see **Vol II – AIR SKILLS 1 AIDS TO INTUBATION**. Though useful, the ETI is fortunately rarely needed in small children.
3. Relative to the position of the larynx in most adults, the larynx of a small child lies more superiorly and anteriorly. The cricoid pressure maneuver (Sellick's maneuver) used to be recommended during bagging, to prevent air from entering the stomach, but has now lost favor. If the intubator is having difficulty visualizing the tracheal opening a maneuver, applying pressure backward, upward, and to the right (BURP) as described in **Vol II – AIR SKILLS 5 CRICOID PRESSURE AND THE BURP TECHNIQUE**, may help bring the glottic opening within view.
4. To prevent air leaks, low-pressure cuffed tubes are being increasingly used in children. Low-pressure cuffs are now being used in pediatric patients on ventilators in ICU settings. For ET tube sizes appropriate for children, see **Vol II – AIR SKILLS 1 AIDS TO INTUBATION**.
5. The length of the trachea is not proportional to that of an adult: The trachea of a small child is surprisingly short. Inadvertent right main stem intubation is a common problem in small children.
6. Infants can be easily extubated simply by head movement. Securing the ET tube after placement is of critical importance. (**Vol II – AIR SKILLS 1 AIDS TO INTUBATION**.)
7. The upper airway is very reactive in small children, at times making intubation difficult. Stimulation of the hypopharynx may cause a vigorous gagging motion and laryngeal spasm. Rapid sequence intubation (RSI) negates these effects. (**Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION**)
8. The intercostal muscles are poorly developed in early childhood, the result being that a child relies heavily on diaphragmatic movement to

breathe. For this reason, gastric and abdominal distension may severely interfere with respirations. Thus, insert an orogastric or nasogastric tube. Another effect is an inability to sustain increased effort when the work of breathing is increased for any reason.

9. The compliant chest wall retracts easily when there is airway obstruction. In small children with upper and lower airway obstruction, rib and supraclavicular retractions are early signs of an increase in the work of breathing. This is useful for identifying respiratory distress and obstruction.
10. In children, barotrauma is easily produced in the lungs by positive pressure ventilation (PPV). Tidal volume is small, and the respiratory rate is fast. Well-meaning team members may easily hyperinflate the lungs using mechanical devices or mouth-to-mouth breathing. Pneumothorax results.
11. Pneumothorax in children is especially likely to result in tension pneumothorax because the mediastinum may shift easily with resulting decreased filling of the heart and compression of the opposite lung. All those who care for children should be familiar with the technique of needle thoracostomy. **(Vol II – BREATH SKILLS 5 NEEDLE THORACOSTOMY)**
12. Due to the ribs being quite compliant, any rib fracture should raise suspicions of severe intrathoracic trauma.

Many different airway injuries and problems can develop in trauma situations. A variety of presentations in patient anatomy and size make absolute recommendations difficult. An algorithm quickly becomes confusing. A good way of approaching any clinical situation is to consider the tools best suited for a specific problem. A small number of tools are available:

Bag-Valve-Mask (BVM) Ventilation – This tool can be effective in many situations. Critical to success is skillful use of the BVM and oral and nasal airways. Additionally, pay careful attention to tidal volume, rate, and pressure. A BVM can keep a patient ventilated until other means of airway control are available. **(Vol II – AIR SKILLS 2 BAG-VALVE-MASK USE)**

Orotracheal intubation – This tool is still the preferred choice for airway management. Though oro-tracheal intubation can cause harmful reflexes as well be painful and difficult in an aware patient, the use of RSI removes these disadvantages. **(Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION, AIR SKILLS 1 AIDS TO INTUBATION)**

Transtracheal needle ventilation (TTNV) – TTNV is an effective temporizing airway. TTNV increases the time available for more invasive procedures or even oro-tracheal intubation and is useful in all ages, especially below 8-10 years of age when the anatomy is too small for cricothyrotomy. **(Vol II – AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION)**

**PEDIATRIC TRAUMA EMERGENCIES
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Cricothyrotomy – Cricothyrotomy is fast and reliable and requires surgical skill. A tracheal hook and an ETI are helpful. A child must be at least 8 to 10 years of age. (**Vol II – AIR SKILLS 13 CRICOTHYROTOMY**)

Tracheotomy - The ultimate airway. More difficult than a cricothyrotomy but may be used in all ages. Transtracheal needle ventilation may be used as a temporizing technique while being performed. (**Vol II – AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS**)

Not every trauma patient needs to be intubated. Most seriously injured patients should be intubated, however, for safe ongoing care. Intubation allows control of the airway and of ventilations, affords the ability to administer pain medications and sedatives without fear of reducing ventilation, and makes transfer safe for many reasons. RSI has made performing orotracheal intubation possible without the former negative effects of tracheal intubation. On the negative side, neuromuscular blockade masks seizure activity and makes clinical neurological evaluation difficult.

If physical findings or a mechanism of injury suggest the possibility of a spinal injury, perform orotracheal intubation with in-line immobilization of the head and neck. Open the cervical collar because it can limit the ability to lift the chin. A method is to lean across the child's chest, rest your hands on the clavicles, and secure the head. **See Vol II – AIR SKILLS 5 CRICOID PRESSURE AND THE BURP TECHNIQUE, AIR SKILLS 3 OROTRACHEAL INTUBATION** for a description of the team approach to tracheal intubation and adjuncts, such as an ET tube introducer (ETI) and the EID.

If an already intubated patient is making cough attempts hindering ventilation, administer topical tracheal lidocaine 1 mg/kg through the ET tube or sedate and paralyze the patient.

Some rescue airways used in adults when orotracheal intubation is unsuccessful are not possible or available for use in children. Pediatric-sized laryngeal mask airways (LMA) and pediatric-sized King airways are available. A pediatric intubating LMA (ILMA) is not available.

I-gel[®] is a non-inflatable supraglottic airway that makes an anatomic seal and comes in 4 pediatric sizes. Consider patient's weight when fitting the I-gel[®].

Size	Weight kg/lbs
Neonate – 1.0	2.5 kg/5-11 lbs
Infant – 1.5	5-12 kg/11-25 lbs
Small – 2.0	10-25 kg/22-55 lbs

Large – 2.5	25-35 kg/55-77 lbs
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Nasotracheal intubation is rarely used in trauma resuscitation because it raises ICP and can cause bleeding. During infancy, adenoid tissue is prone to bleeding and the anteriorly placed larynx makes placement difficult, so this method is not used. In children, this should be done only by someone experienced.

A surgical airway may be the best option if anatomic problems are severe. In children younger than about 8 years of age, transtracheal needle ventilation (TTNV) is probably the best option. The tight cricoid ring of small children can make tracheal cannulation through the cricothyroid membrane difficult, for example, as in cricothyrotomy. If the larynx is easily palpable, the percutaneous insertion of the needle is safe. However, if the larynx is very moveable in an infant or toddler, the best approach may be to use a small skin incision over the larynx to assure accurate placement of the needle. (**Vol II – AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION**)

In children older than about 8 to 10 years, a cricothyrotomy can be quickly and reliably accomplished with the insertion of a cuffed 6.0 ET tube over an ETI as in adults. (**Vol II – AIR SKILLS 13 CRICOTHYROTOMY**)

When there is trauma to the larynx and airway obstruction occurs, tracheotomy is indicated. Tracheal stenosis may occur if the procedure is performed incorrectly in small children. In small children it is safest to keep the patient oxygenated using TTNV through the cricothyroid membrane while the tracheotomy is performed using meticulous technique. (**Vol II – AIR SKILLS 14 TRACHEOTOMY, AIR SKILLS 15 TRACHEOTOMY IN INFANTS**)

Whichever method of airway management is used, carefully assess for correct tube placement and function. Monitor breath sounds, oxygen saturation, and blood gases. A post-intubation chest x-ray is also important.

2

Abnormal LOC

Continuing from the initial survey, parts of this evaluation and treatment have already been done. If the patient is unable to cooperate or is disoriented, use RSI to gain control of the airway, allowing the resuscitation to proceed. Again, be aware of the anatomic differences between children and adults. (**Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION**) However, before paralyzing the patient, obtain the SAMPLE history, determine LOC (using the AVPU scale or the Pediatric Glasgow Coma Scale), and document a mini neuro exam. During the initial survey, always check Dextrose, administer Oxygen as needed, and consider Narcan.

Level of Consciousness

AVPU Scale		Corresponding GCS
A	Awake	14 to 15
V	Responds to voice	12 to 13
P	Responds to pain	8
U	Unresponsive to pain	3 to 4

- The Glasgow Coma Score incorporates the 15 point scale, and now has added the Pupil Reactivity Score. The pupil score is based on if the pupils **DO NOT** react to light. If both pupils react to light the score is 0, if only one react to light the score is 1 and neither react to light the score is 2. This score should be subtracted from the 15 point scale. Further if the patient is intubated the patient should not be scored a 1, but should instead be scored as not testable

Glasgow Coma Scale – Adult, Pediatric, Infant

Eye Opening

Infant (<1 year) Pediatric (>1 year) Adult

Spontaneous	Spontaneous	Spontaneous	4
Voice	Voice	Speech	3
Pain	Pain	Pressure	2
None	None	None	1

Verbal Response

Infant (<2 years) Pediatric (>2 years) Adult

Coos, babbles	Appropriate word/ phrase	Oriented	5
Irritable but consolable	Disoriented/ converses	Confused	4
Persistent cries/ screams	Inappropriate word	Words	3
Moans/grunts to pain; restless	Incomprehensible sounds	Sounds	2
None	None	None	1

Motor Response

Infant (<1 year) Pediatric (>1 year) Adult

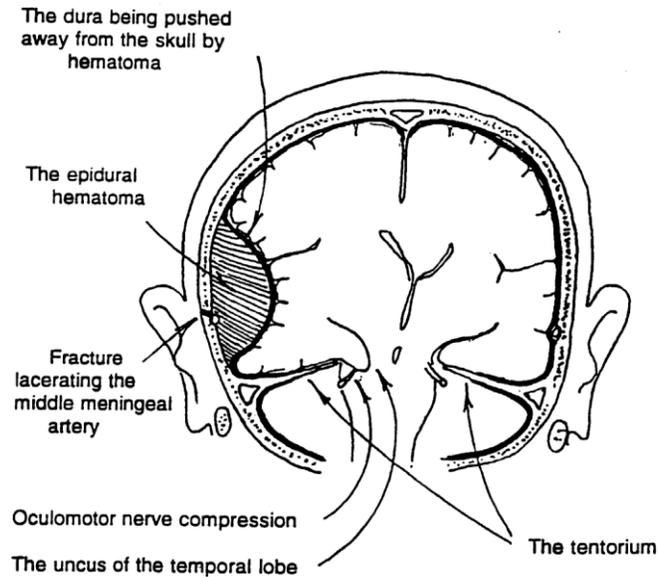
Spontaneous	Obeys	Obeys	6
Localizes pain	Localizes pain	Localizes pain	5
Flexion- withdrawal	Flexion- withdrawal	Withdraws	4
Flexion/ decorticate	Flexion/ decorticate	Abnormal flexion (decorticate)	3
Extension/ decerebrate	Extension/ decerebrate	Abnormal extension (decerebrate)	2
None	None	None	1
			3 to 15

PEDIATRIC TRAUMA EMERGENCIES PATHWAY 9

In infants younger than about 18 months, the fontanelle may be palpable. Bulging indicates increased ICP and signifies a severe injury. With decreasing or low level of consciousness, airway control is needed. Premedication with lidocaine, to prevent a rise in ICP is no longer recommended nor is pretreatment with atropine ([as described in this portal, #8 Compromised Airway](#)) to prevent bradycardia; unless the child is bradycardic, before performing RSI.

If uncal herniation is occurring (dilated pupil and weak opposite extremity) or if the patient is posturing (extensor or flexor), carry out the following:

- Hyperventilate to a CO₂ level of about 30 to 35 mm Hg. Ventilate at a rate of 16 to 20 breaths per minute; however, ventilate infants at a rate of 20 to 30 breaths per minute. Tidal volume should be about 10 to 15 mL/kg. To avoid producing a pneumothorax, limit the amount of pressure applied to the ventilating bag to less than about 30 cm of H₂O.⁶
- Administer mannitol IV 1 g/kg or hypertonic saline 3 or 5%. Consult with neurosurgeon if they would want an antiepileptic agent.
- Elevate the head of the bed.
- If the patient had an initial lucid interval and continues to herniate despite these measures and transport to a trauma center has been delayed, obtain a head CT to identify epidural hematoma and location. A lucid interval followed by a herniation syndrome usually indicates the presence of an epidural. About 80% of the time, the epidural is located on the side of the dilated pupil. So, if a CT scan is unavailable, perform trephination on the side of the dilated pupil. ([Vol II – DISAB SKILLS 1 SKULL TREPHINATION](#)) Consult with a neurosurgeon.



An epidural hematoma in the typical location

Epidural hematoma is less common in children than in teenagers or adults. The clinical picture of an epidural hematoma can be mimicked by a subdural hematoma. A subdural hematoma can possibly be decompressed through the trephination if the dura is opened. Unless the patient went through a lucid interval, trephination will probably not help. If on CT, the clot is all white, it means that it is clotted and probably will not extrude through a trephination hole, typically only 0.5 inch in diameter. If the hematoma is located in an unusual location, it is generally not feasible to perform a trephination in the ED. Skull thickness and the presence of venous sinuses in the skull can be problematic.

Cerebral edema is common in head-injured children; take care not to overdo volume resuscitation. On the other hand, hypovolemic shock is also detrimental. Do not use hypo-osmolar fluids.

Bilateral fixed and dilated pupils are ominous for brain death unless they are secondary to a reversible problem, such as hypothermia or anticholinergic poisoning. Disconjugate gaze and/or nystagmus may be associated with head injury or drug ingestion.

CNS and autonomic nervous system immaturity may mask seizure activity in small children. Any repetitive muscle motion, including eye and diaphragmatic motion, may represent seizure activity. Terminate seizure activity with diazepam (Valium) 0.3 mg/kg IV or IO or lorazepam (Ativan) 0.1 mg/kg IV or IO.

PEDIATRIC TRAUMA EMERGENCIES PATHWAY 9

A seizure in a head-injured patient is a severe complication. The injured but viable brain tissue may be permanently damaged. Avoid seizures in these patients at all costs.

Patients who are paralyzed and have a head injury are at risk for undetected seizure activity. If the patient has a probability of cerebral contusion, strongly consider seizure prophylaxis with Keppra.

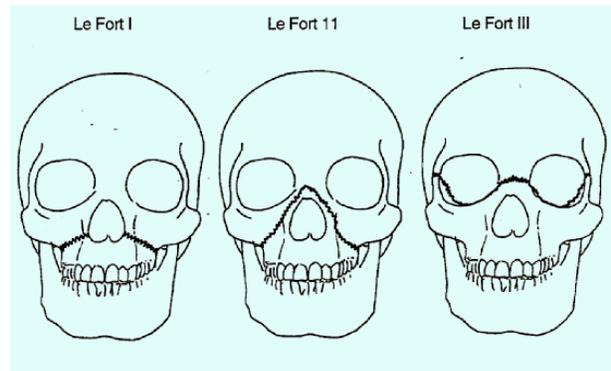
If the primary (initial) assessment is complete you can begin the secondary (focused) evaluation.

3

Skull Fracture/Scalp Laceration

Palpate the patient's face and jaw for abnormal step-offs and movement. It is not important to diagnose type of fracture, but Le Fort fractures are frequently mentioned in trauma manuals.

See right:



Examine the tympanic membranes for rupture or hemotympanum. Whenever basal skull fracture is possible, do not insert nasal tubes to avoid intubating the cranial vault. To avoid the cribriform plate, insert nasal tubes directly posterior over the top of the palate.

Check for loose or missing teeth that the patient may have aspirated in the patient's mouth and then on x-rays if obtained. Tongue injuries with swelling can endanger the airway. When facial trauma endangers the airway, cricothyrotomy or TTNV may be necessary. **(Vol II – AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 16 TRANSTRACHEAL NEEDLE VENTILATION)**

Palpate the skull. In small children, a subgaleal hematoma can result in hypovolemic shock. Battle's sign or discoloration referred to as *raccoon eyes* occurs after basal skull fracture, but usually after the immediate resuscitation phase.

Check scalp wounds for excessive bleeding. If present, use pressure, staples, or Raney clips to stop the bleeding. These clips do not close the wound. (**Vol II – DISAB SKILLS 2 RANEY SCALP CLIPS**) Another technique is to use a large running suture along the wound edges to stop bleeding. Access to the scalp posteriorly is limited. When the patient is lifted or rolled for back inspection, there is an opportunity to address this. Of course, in-line immobilization of the head and neck is needed. . BE AWARE THAT A LARGE AMOUNT OF BLOOD CAN BE LOST FROM A SCALP WOUND.

With a gloved finger, gently explore scalp wounds to detect depressed skull fractures. If a depressed skull fracture is present, inform the trauma consultant.

4

Eye Trauma

Topical ophthalmologic anesthetic drops may be used in the initial evaluation and care of any painful eye injury except ruptured globe.

CNS injury, direct eye trauma, prior eye surgery or injury, as well as nasal decongestants such as phenylephrine may cause dilated or unequal pupils.

Remove contact lenses unless there is a possible globe rupture.

Globe rupture from blunt trauma is accompanied by peripheral scleral hemorrhage. If such is possible, avoid touching the eye. Cover it with a firm plastic or metal shield.

Blood in the anterior chamber of the eye is called hyphema. To prevent further bleeding into the chamber with subsequent severe corneal scarring, keep the patient still and calm. Keep the head elevated.

If the patient is awake currently, ask him or her to count fingers as a vision check.

Lid lacerations involving the lid margins, or the tear duct ultimately require repair by an ophthalmologic surgeon.

When there has been eye contamination with acidic or basic chemicals, use copious irrigation with sterile NS to prevent continuing injury. Check pH of the tears using pH paper or a urine dipstick. The resulting pH should be 6 to 8. Children may tolerate a Morgan lens; if not, a nasal cannula over the bridge of the nose can drip fluid into both eyes.

**PEDIATRIC TRAUMA EMERGENCIES
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Bilateral fixed and dilated pupils are ominous for brain death unless secondary to a reversible problem. Hypothermia is an example as well as anticholinergic poisoning. Disconjugate gaze and nystagmus may be associated with head injury or drug ingestion.

Retinal hemorrhage

Retinal hemorrhage is evidence of the shaken baby syndrome and should alert the team to the possibility of child abuse. Unfortunately, child abuse must be considered in almost all situations. Clues include unusual bruising, unexplained fractures, burns, and an unusual reaction to strangers and caregivers. If child abuse is suspected, remember to perform a thorough head-to-toe examination, as there may be other injuries. An ophthalmologist should be consulted to document the injuries for follow-up and for legal matters.

5

Soft Tissues of the Neck

Do not explore lacerations and penetrating wounds deeper than the platysma muscle in the ED. Massive hemorrhage can result. These may need to be explored in the operating room, with or without prior angiography. If the physical exam does not show evidence of severe injury, CT angiography may be sufficient. Deep lacerations of the neck can divide the trachea. The trachea can often be directly intubated in such cases.

Laryngeal fracture commonly results from a blow to the neck. A hematoma forms within the larynx, producing airway obstruction. Try gentle orotracheal intubation (if not already performed), but tracheotomy is probably needed. (**Vol II – AIR SKILLS 14 TRACHEOTOMY**)

The trachea can be displaced to the opposite side of a tension pneumothorax, or it can be displaced by a hematoma.

Carotid pulses can be absent secondary to hypotension, direct injury, and aortic injury.

If the external jugular veins can be seen, they provide valuable information regarding the volume status of the trauma patient. Jugular venous distension can indicate tension pneumothorax, cardiac tamponade, volume overload, pulmonary embolism, traumatic asphyxia (compression of the superior vena cava), and heart failure.

6

Cervical Spine Injury

If cervical spine injury is a possibility, take cervical spine fracture precautions

with a hard collar, head blocks, and a spine board. In small children, place a pad behind the shoulders because the large posterior skull causes flexion of the neck. Clearing the c-spine **by plain films only is not recommended.**

When a patient who is breathing but obtunded needs intubation and the probability of cervical spine injury is high, be sure to maintain excellent c-spine control during intubation.

Spinal immobilization of children often requires a somewhat different approach than in the adult. Very young infants without evident critical injuries may be immobilized in an infant car seat with towel rolls and tape to immobilize the head. Seriously injured infants should be immobilized supine in the routine manner. Children under 8 or 9 have a prominent occiput that flexes the neck in the supine position. A pad or towel should be placed under the shoulders to maintain the head in neutral position. Color-coded cervical collars are available. When maintenance of spinal immobilization is critical in an uncooperative child, sedation with airway management may be necessary.

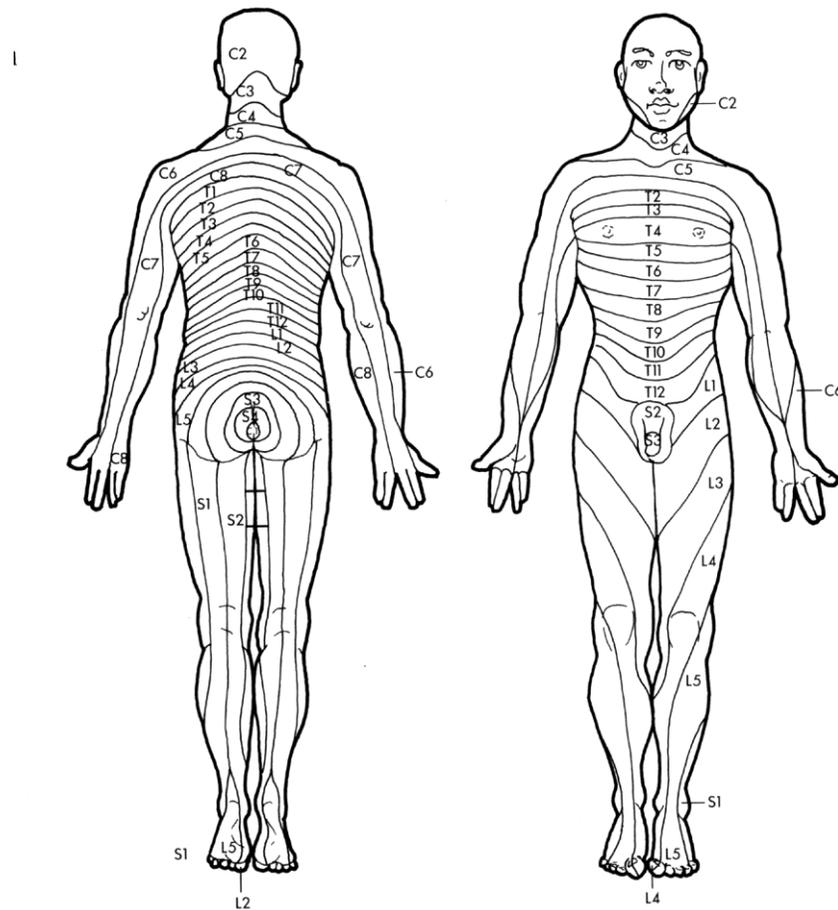
If not already performed, conduct a mini neuro exam. [\(See #2, this pathway, Abnormal LOC.\)](#)

Spinal cord injury syndromes follow:

1. **Complete injury.** Total loss of sensation and movement below the level of injury.

Level	Function	Sensory level
C2	Breathing	Occiput
C3	Breathing	Neck
C4	Breathing	Top of shoulders
C5	Arm abduction	Anterior arm
C6	Elbow flexion	Lateral arm and thumb
C7	Elbow extension	Posterior arm and index finger
C8	Finger flexion	Medial arm and small finger
T1	Finger abduction	Just below shoulders

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Dermatomes corresponding to the levels of the spinal cord. From *Emergency Management of Skeletal Injuries*, Ruiz E, Cicero JJ, eds. Mosby-Yearbook, 1995. Reprinted with permission.⁷

2. **Incomplete injury.** This finding is extremely important because recovery may result from surgical intervention or aggressive reduction. Variable loss of sensation and movement below the level of injury indicates an incomplete injury. An example is sacral sparing with preserved anal sphincter tone. Another is the presence of an **anterior cord syndrome** in which there is muscle paralysis but preservation of position sense.
3. **Brown-Sequard syndrome.** Occurs with penetrating trauma with hemitransection of the cord. It results in loss of motor function below and on the same side as the lesion. This is coupled with loss of pain and temperature sensation on the opposite side of the lesion.
4. **Cauda equina syndrome.** Damage to the cauda equina as it descends through the sacrum or lumbar spine. This damage produces anesthesia in a saddle distribution of the perineum with loss of anal sphincter tone.

5. **Central cord syndrome.** Damage to the central area of the cord predominantly resulting in marked weakness of the arms relative to the legs. Test patient's hand grip by shaking hands. Flexion-extension injuries result in ischemia and edema of the center of the spinal cord, producing this syndrome even without fracture.

If a ligamentous injury is suspected, even with a negative CT then do an MRI Spinal cord injury without radiographic abnormality (SCIORWA) was described as occurring in children because their soft vertebral column and lax ligaments allowed considerable displacement without fracture.

Spinal shock (hypotension with bradycardia) is a diagnosis of exclusion. Spinal shock responds to volume loading. Vasopressors may be added after volume loading is well under way.

7

Chest Wall and Pulmonary Injury

The sternum and ribs of a small child are soft and compliant. They are not easily broken, but the viscera of the chest are at risk for compression injury, even without rib fractures.

Palpate the clavicles and the chest wall again for crepitation and fracture. If there are wounds to the chest, do not explore them because a pneumothorax may result. Wounds that enter the chest should be covered with an occlusive dressing. Do not remove impaled objects because they may be providing tamponade to a pulmonary or cardiac wound. Observe for asymmetrical chest movement that may occur with pneumothorax, flail chest, and spinal cord injury with abdominal breathing.

If a needle thoracostomy has been performed during the initial survey for tension pneumothorax, insert a chest tube of appropriate size on that side now. **(Vol II – BREATH SKILLS 1 CHEST TUBE INSERTION)** Connect it to chest suction with preparations made to collect blood for possible autotransfusion. **(Vol II – BREATH SKILLS 2 CHEST SUCTION AND AUTOTRANSFUSION)** Chest wall crepitation means pneumothorax on that side. Insert a chest tube.

The immediate return of about 10 mL/kg of blood means that emergency surgery may be needed. The immediate return of blood that continues to come out is also ominous. If the BP falls precipitously with drainage of a large volume of blood from the chest, clamp the chest tube because the blood in the chest may have had a tamponade effect.

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If a bronchus rupture has occurred, there may be a massive air leak from the chest tube with inability to inflate the lungs. If this occurs, intubate and insert the ET tube into the right or left main stem bronchus so that at least one lung can be ventilated.

Use a BVM to ventilate the patient. The ventilatory rate for a 1-year-old is about 25 breaths/min and about 15 breaths/min for an older child. The tidal volume should be 10 to 15 mL/kg. An orogastric tube also helps to prevent gastric distension and subsequent elevation of the diaphragm.

Palpate the ribs to detect rib fractures. Multiple rib fractures on the same side may result in paradoxical chest wall motion with breathing signifying a flail chest. Not all patients with flail chest need ET intubation. Monitor the patient carefully and intubate at the first sign of fatigue or hypoventilation.

Chest wall bruising or sternal tenderness – as with dashboard impact – may indicate that the aorta has been injured. Carefully review the chest x-ray for mediastinal widening or other clues such as tracheal deviation to the right and an apical cap. If thoracic aortic rupture is being considered, attempt to keep the BP at about 100 mm Hg.

If available, a helical CT scan of the chest with IV contrast is now considered to be adequate screening for this condition. However, this is technique dependent. Be certain your radiologist and CT technician understand the purpose of the scan.

Also examine chest x-ray for fractured ribs, pneumothorax, hemothorax, scapular fractures, elevated diaphragm, abnormal cardiac silhouette, lung contusion, position of the ET and other tubes, and thoracic spine. With bronchial, tracheal, and esophageal injury, mediastinal air is visible. If pneumothorax is present, insert appropriate-sized chest tube; connect to chest suction. If the patient has been tracheally intubated, listen for breath sounds carefully. If the tube is in too far, the left lung will be inadequately ventilated. Also check for this on the chest x-ray. When a patient is intubated, a follow-up x-ray is indicated to check for correct tube placement.

Pulmonary contusion worsens as the patient's hemodynamic status improves. Blood oxygen saturation decreases. Transtracheal pressures increase as the lungs become less compliant. Consider using 5 to 10 cm H₂O PEEP if pulmonary contusion is present and hypoxia develops. This may be problematic for a patient in a helicopter, so consider inserting a prophylactic chest tube so that if a pulmonary bleb ruptures, the lungs will still inflate.

8

Cardiac Tamponade

Muffled heart sounds associated with shock and distended neck veins (Beck's triad) may indicate cardiac tamponade. Cardiac tamponade with blunt trauma is uncommon, but does occur with atrial rupture. A majority of patients with this injury have suffered penetrating trauma.

Perform echocardiography from the subxiphoid window or along the left sternal border. (**Vol II – UL2 EMERGENCY ULTRASOUND TECHNIQUES**) Hypovolemic shock and spinal shock are associated with a vigorously pumping heart; significant myocardial contusion is associated with a weakly pumping heart. Cardiac tamponade is associated with a ring of hypoechogenic fluid surrounding the heart. A compressed right ventricle with a weak beat may also be detected. Ultrasound is an invaluable tool in the management of trauma in many ways.

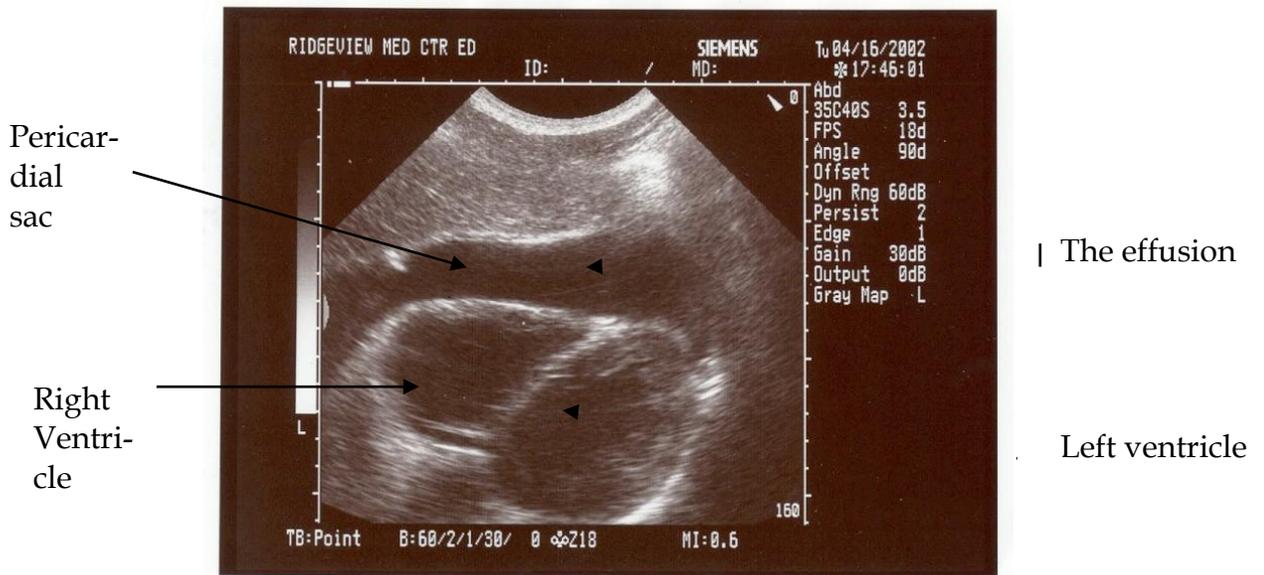
In such a case, insert a guidewire-assisted catheter device from the left subxiphoid position directed at the left scapula. (**Vol II – CIRC SKILLS 6 PERICARDIOCENTESIS**) Use repeated aspirations to keep the patient stable pending surgical repair.

If this procedure is unsuccessful, consider an emergency thoracotomy if your facility has surgical backup. The blood in the pericardial sac may be clotted. Perform the thoracotomy as described in **Vol II – CIRC SKILLS 4 EMERGENCY THORACOTOMY**. If the heart is beating, make a small incision into the sac creating a "window" through which the clot can extrude. A sterile suction catheter may be used to extract the clot. Use a finger to cover this window pending surgical repair.

If the heart is not beating, the sac will have to be widely opened to facilitate internal massage and staple repair of the wound. Extend the pericardial incision longitudinally to avoid injuring the phrenic nerve, which also runs longitudinally along the posterior margin of the pericardial sac. If possible, repair the cardiac wound using a skin stapler. Do this before restarting the heart. (**Vol II – CIRC SKILLS 4 EMERGENCY THORACOTOMY**) The staples will be removed and replaced with suture in the operating room.

If myocardial contusion is possible, observe for ECG changes and evidence of heart failure. Lidocaine IV 1 mg/kg followed by an infusion at 2 mg/min may be indicated for significant ventricular arrhythmias. Amiodarone may also be used.

Because the chest wall is thin and soft in small children, penetration can occur with minimal force.



This heart ultrasound is from a small child shot at close range with a toy BB gun, which resulted in cardiac tamponade.

9 Hypovolemic Shock

CPR is not as effective when there is hypovolemia. Volume replacement is the most important aspect of resuscitation from traumatic pulselessness. Conduct CPR while volume is restored. (This is now being questioned as if there is no blood in the heart, there is nothing to squeeze. It is better to give blood as a priority)

If IVs have not been established and the child is younger than about 6 years, insert an intraosseous needle in the proximal tibia if other access cannot be obtained quickly. (**Vol II – CIRC SKILLS 5 INTRAOSSEOUS NEEDLE PLACEMENT**) Do not use an intraosseous needle in a fractured extremity. Another choice would be the distal femur. A femoral venous line at the groin may also be used. If that is not successful, perform a saphenous vein cutdown at the ankle. (**Vol II – CIRC SKILLS 8 SAPHENOUS VEIN CUTDOWN**) For accurate and quick administration, administer fluid boluses using a syringe and stopcock.

Small children are unable to maintain body temperature without support because of their large surface area compared to their body mass. Make every effort to keep the child warm. Hypothermia results in hypocoagulability and further bleeding. Be aware of normal BP and pulse rate in children.

- **Infants** have a systolic BP of 70 to 80 torr and a pulse rate of up to 160 bpm.
- **Toddlers** have a systolic BP of 80 to 90 torr and a pulse rate of up to 140 bpm.

- **Older children** have a systolic BP of 80 torr plus twice their age in years.

The vascular responsiveness of children potentially masks marked hypovolemia, so careful clinical monitoring is needed. By the time hypotension appears, more than 30% of blood volume has been lost, and the patient is in severe shock.

The blood volume of children is small in absolute terms, but their cardiac output is high relative to adults. This results in a small circulating blood volume reserve. The cardiac chambers are small and relatively stiff, with little reserve for increasing stroke volume. They respond to a need for increased cardiac output with an increase in heart rate. When bradycardia or extreme tachycardia (> 200 bpm) occurs, cardiac output falls dramatically.

Track vital signs and review physical examination for evidence of hypoperfusion, (prolonged capillary refill [> 3 seconds], pallor, coolness, tachycardia, depressed LOC, and decreased pulse pressure). Observe capillary refill centrally by applying pressure to the forehead or sternum. Observe the ECG monitor.

Head injuries do not cause hypotension except in infants with large subgaleal hematomas (cephalohematomas).

A Broselow pediatric measuring tape can estimate weight, but be aware that the increasing weight of children, may not be reflected on the tape. There are other resources as well, such as the Pediatric Emergency Drug Book, by Hennepin County Medical Center or the Children's Drug book by Children's Hospital, or one of the many phone apps now available.

Central venous access is not commonly used in children in emergency situations because volume overload is usually easier to detect and predict than in adults. Central venous access routes are similar to those used in adults. (**Vol II – CIRC SKILLS 2 CENTRAL VENOUS ACCESS**) However, steep Trendelenburg positioning at 30 degrees is advisable.

Warm crystalloid (NS or LR) is the preferred fluid during the initial resuscitation. Use a 10 mL/kg bolus administered with a 60 mL syringe and stopcock. If the patient's BP falls again or the pulse does not slow, the patient may be continuing to hemorrhage. To avoid hemodilution, switch to blood transfusion after one 20 cc/kg boluses if patient remains in shock. Blood is usually administered as packed RBCs. While typed and crossmatched blood is preferred, type-specific blood may also be used. If blood is required immediately, type O blood is also acceptable. Use type O Rh-negative for any female with current or future childbearing potential; all others may get type O Rh-positive. Administer blood at 10 mL/kg. This 10 ml/kg also applies to FFP and Platelets.

Also, Tranexamic Acid should be administered to the pediatric trauma patient with

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the first bolus at 15mg/kg in 100 ml NS given IV, once over 10 minutes, and the second infusion of 15mg/kg in 100 ml NS given over 8 hours. Especially if FFP and Platelets are not readily available.

If blood is required immediately, anticipate massive transfusion. Along with PRBCs, consider giving fresh frozen plasma and platelet packs (if available) in a 1:1:1 ratio at 10 cc/kg. Continuing hemorrhage is ominous; obtain surgical consultation as soon as possible. When blood is not available then allow permissive hypotension at 70 plus 2 times their age and do what is necessary to stop the bleeding.

Maintenance fluids for children after normal volume is restored are as follows:

Infants < 1 year (< 10 kg)	4 mL/kg/h
Child 1 to 5 years (10 to 20 kg)	(40 mL plus 2 mL/kg)/h
Child 5 years or older (> 20 kg)	(60 mL plus 1 mL/kg)/h

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Abdominal Injury

If the stomach is filled with air, blood, or food, insert a large orogastric tube and connect to suction. Children are prone to swallow air after trauma and should almost always have a gastric tube inserted and connected to low suction.

Abdominal tenderness may mean peritoneal irritation secondary to free blood from bowel injuries, splenic rupture, liver laceration or disruption, vena caval or aortic laceration, mesenteric laceration, penetrating wounds, and renal injuries. Examine for the linear bruising caused by seat belts.

Patients who are distracted by painful injuries such as pelvic or femur fractures may minimize symptoms when reporting abdominal tenderness.

Retroperitoneal organs may be injured without signs of peritoneal irritation. For example, duodenal rupture (or hematoma) and pancreatic laceration may be difficult to detect on physical examination.

Lower rib cage injuries can produce abdominal tenderness, but is a diagnosis of exclusion.

Flank wounds can involve the colon, kidneys, vena cava, and aorta. When you suspect intra-abdominal wounds, administer a broad-spectrum antibiotic such as cefotetan 25 mg/kg IV. Treat penicillin-allergic patients (those with anaphylactic reaction) with gentamicin 2.5 mg/kg IV and clindamycin 10 mg/kg IV.

In most instances, CT scans (or ultrasound) have replaced peritoneal lavage in abdominal trauma. Peritoneal lavage should not be performed in the rural setting,

unless immediate surgical intervention is available. The surgeon must choose between embolization of pelvic bleeding in the radiology suite and exploratory laparotomy in the operating room.

Rapid bedside ultrasound examination FAST (Focused Assessment Sonography in Trauma) performed by the clinician has become routine at many institutions. Sensitivity and specificity are variable, but identification of intraperitoneal fluid/blood greater than 700 cc and detection of pericardial tamponade are quite reliable and may guide subsequent management in selected cases. Morrison's pouch view, subxiphoid view of the heart, and trans-vesical views are the easiest to perform.

11 Pelvic Fracture

As part of the initial survey, the pelvis was gently compressed in a horizontal and AP plane as part of the circulation evaluation. This is because a pelvic fracture can be a site of massive occult hemorrhage. Movement or pain with this test signifies pelvic fracture. Do not repeat this exam because it may stimulate further bleeding. Review the supine AP x-ray of the pelvis. ([Vol II – XRAY SKILLS 3 INTERPRETATION OF PELVIC X-RAY](#)) Pelvic fractures may be difficult to see, but some clue is almost always present. Any pelvic fracture must be considered dangerous. The major danger is occult hemorrhage, although children have less of a tendency for hemorrhage than adults.

Follow a protocol when evaluating pelvic trauma.

1. Examine for perineal lacerations, genital injury, and blood at the urinary meatus. Prepubertal children are at less risk for genital injuries.
2. Do not explore perineal lacerations because external hemorrhage may result.
3. Perform a rectal exam to detect frank blood in the rectum and to determine sphincter tone. In adolescent males, feel for the position and consistency of the prostate gland. Perform this exam before attempting to insert a Foley catheter because a boggy or high-riding prostate indicates urethral transection. Blood at the urinary meatus in males also indicates urethral injury. Blood in the rectum means rectal penetration or laceration. Begin antibiotics, such as cefotetan 25 mg/kg IV. Treat penicillin-allergic patients (those with anaphylactic reaction) with gentamicin 2.5 mg/kg IV and clindamycin 10 mg/kg IV.
4. Pelvic fractures, with or without instability, may be associated with massive internal hemorrhage. To provide external stabilization, you can fold a sheet to an appropriate size and wrap it tightly around the pelvis. This technique is

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called the Sheet Wrap Alternative. **(Vol II—TRAU SKILLS 3 PELVIC FRACTURE STABILIZATION)** Commercially available pelvic stabilization devices are also available.

5. If the bladder is distended to palpation or ultrasound examination and a urethral disruption or injury has occurred (blood at the meatus), perform a percutaneous suprapubic cystostomy using guidewire technique. **(Vol II—TRAU SKILLS 4 SUPRAPUBIC CYSTOSTOMY)** This is especially important in head-injured patients because bladder distension causes a rise in intracranial pressure (ICP). If mannitol is indicated because the patient's neuro status is deteriorating, providing bladder drainage is especially important.
6. Check for femoral pulses because some pelvic fractures are associated with iliac artery injury.
7. If there is blood at the introitus, perform a gentle digital vaginal examination feeling for laceration and bone spicules. If positive, begin antibiotics, such as cefotetan 25 mg/kg IV. Penicillin-allergic patients (those with anaphylactic reaction) may be treated with gentamicin IV 2.5 mg/kg and clindamycin IV 10 mg/kg.
8. Scrotal swelling and discoloration can indicate testicular injury. Obtain urologic consultation if such is found.
9. Hip dislocation is often associated with pelvic fracture. Look for this when reviewing the pelvis x-ray. Also, in children one often sees much of the abdomen on the pelvis film. Look for spine injury and evidence of intra-abdominal blood and air. **(Vol II—XRAY SKILLS 3 INTERPRETATION OF PELVIC X-RAY)**

If the patient has an unstable pelvic fracture, take great care not to move the pelvis, causing more bleeding. Instead of log rolling the patient to view the back, lift the patient straight up. If the patient is large, simply slide one's hands under the patient to feel the back. Be careful of glass shards.

12

Major Joints, Femur Fractures, and Amputations

Reduce major joint dislocations (such as hips, knees, and ankles) using sedation, relaxation, and traction with counter-traction. Short-acting sedatives such as ketamine or propofol work well for this purpose.

Add an opioid to propofol for pain relief. **(Vol II—AIR SKILLS 4 RAPID SEQUENCE INTUBATION)** Take airway precautions. Before the reduction, check for pulses and motion. Absent or decreased pulses can result from the deformity or direct

trauma to arteries as well as systemic hypotension. Reduction often results in the return of pulses. Remove all jewelry from extremities prior to manipulation.

When a bone end or fragment is protruding from a wound, rinse off any gross contaminant (such as pebbles or dirt) with saline-soaked sponges; then, apply traction to reduce the fracture. Cover site with sterile dressing. Begin antibiotics such as cefotetan 25 mg/kg IV. Treat penicillin-allergic patients (those with anaphylactic reaction) with gentamicin IV 2.5 mg/kg and clindamycin IV 10 mg/kg.

If there is a penetrating wound in proximity to a major vessel or nerve, special imaging and/or surgical exploration may be indicated, even in the presence of an apparently good pulse. Check the legs, thighs, and arms for tense swelling of the muscle compartments. Alert your surgeon about any area suspicious for a compartment syndrome. A decreased pulse is not a necessary component of the syndrome. Fasciotomy may be needed if the patient's transfer is delayed. (**Vol II – TRAU SKILLS 1 COMPARTMENT PRESSURE MEASUREMENT**)

If life-threatening problems are ongoing, simply splint the limb in its current position, deferring reduction until the clinical situation permits. Reduce dislocated shoulders and elbows and splint when time permits. Avoid taking x-rays of the extremities for injuries that you will not have to definitively manage. If amputation has occurred and the body part is available, rinse in NS and place in a plastic bag (in case it can be implanted later or serve as a source for skin grafting). Place the bag on ice, but do not freeze.

Fractured femurs. Significant blood loss (with as much as 1500 mL) can occur into the soft tissues of the thighs. Traction splints can reduce this loss. Apply traction splints (Sager or Hare or Slishman or Kendrick or other device.) to reduce femur fractures. The helicopter service may prefer one type of splint because of space issues. (**Vol I – TRAU SKILLS 2 FEMUR FRACTURE SPLINTING**)

13

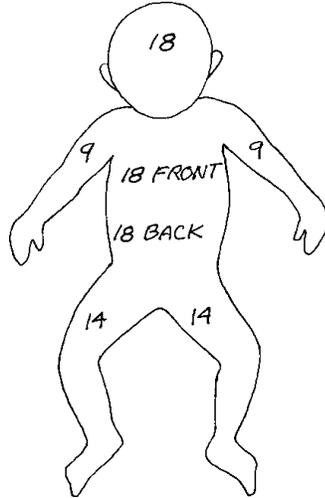
Burns

Continuing from the initial survey, perform orotracheal intubation with in-line immobilization, if not already done, for the following indications: stridor, soot in the pharynx, edema of the uvula, significant burns of the neck, and depressed LOC. If an explosion or closed space fire has occurred, be especially alert to the need to intubate. Ventilate with oxygen. Consider possible cyanide and carbon monoxide exposure if patient has been involved in closed space fire.

Completely undress the child. Be careful of smoldering clothing. Remove jewelry and contact lenses.

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Using a pediatric burn chart, grossly estimate the percentage of body surface burned. In children older than 10 years, use the adult rule of nines chart.



Immediately cool all burns with NS if <10% of TBSA is burned. (If NS is not available, use water.) If > 20% TBSA is burned, cover patient with dry gauze dressings. Patients with burns > 20% TBSA are at greater risk for developing hypothermia if wrapped in wet gauze. If < 20% TBSA is burned, keep burns moist, but cover patient with blankets to prevent hypothermia.

Start 2 IVs in unburned skin, but use burned skin sites if other sites are not available. Obtain pain relief with morphine IV. **Do not administer IM.** Titrate from 0.1 mg/kg to whatever dose provides relief. Ventilatory support may be needed.

Administer crystalloid (LR) solution IV as a 20 mL/kg bolus to restore normal BP. Follow by another bolus if needed.

Insert a Foley catheter and monitor urine output to provide a guide to adequate fluid administration. A urine output of 1 mL/kg/h is the goal.

Monitor the child's core temperature with a rectal probe. Do not use cool saline soaks for pain relief. Use morphine instead. A dry occlusive dressing suffices. Do not apply antiseptic creams. Do not break blisters. Do not administer antibiotics. Consider tetanus status. A patient with a significant burn needs an NG tube. The Parkland Formula for estimating fluid requirements for adults is not accurate for small burned children. The Parkland Formula for children is:

Lactated Ringer's 3 mL X weight in kg X percentage of body surface burned over

the first 24 h plus maintenance.

Half is given over the first 8 hours; the second half over the following 16 hours.

Urine output is the most practical guide for ongoing fluid management.

Monitor small children closely for the development of hypoglycemia.

Circumferential third-degree burns of the extremities or the chest may produce a tough eschar that may compromise blood flow or chest wall movement.

Escharotomies may be necessary. Contact a burn consultant regarding how and where to perform these escharotomies. Fortunately, these areas are anesthetic.

Send patients with the following indications to a burn center for treatment:

Burns > 10% of body surface for patients under 10 years

Burns > 20% of body surface in all age groups

Full thickness (third-degree) burns

Burns involving the hands, feet, eyes, perineum

Inhalation, electrical, or chemical burns

Burns associated with major trauma or preexisting disease

Electrical burns

The extent of damage from electrical burns may be difficult to ascertain immediately. The injury is typically to the deeper structures of the body, such as muscle and vascular (not superficial or cutaneous) tissue. Thus, the tissue is not easily observable to the naked eye on physical exam. For this reason, cardiac dysrhythmias may result from electrical burns. ECG monitoring is important. When dysrhythmias occur, see [Vol I – PATHWAY 2 CARDIOVASCULAR EMERGENCIES](#).

Another common finding is muscle necrosis resulting in myoglobinuria. Clinically, this is observed as a reddish hue to the urine. Increase the infusion of fluids to raise urine output to 1.5 mL/kg/h. If this does not rapidly clear the urine, give mannitol IV 0.25 g/kg and add 12.5 grams to each subsequent liter bag of Ringer's solution. Many children with electrical injury have also suffered injuries from seizures and falls, including cervical spine injuries and head trauma. The whole child needs careful evaluation.

Lightning strike

Cardiac arrest with VF or asystole is the most serious effect of lightning strike.

Treat any dysrhythmia as described in the [Vol I – PATHWAY 2 CARDIOVASCULAR EMERGENCIES](#). The term *suspended animation* has been ascribed to the recovery of lightning victims from cardiac standstill.

Burns from lightning strike are often inconsequential. Wet clothing may protect the patient by causing the current to flow over the patient rather than through him or her. This is referred to as flashover phenomenon.⁸ Under unusual conditions, an explosive or implosive effect from a lightning bolt can cause blunt injury. Initial management is as with all trauma patients.

14 Heat Stroke

Heat exhaustion is accompanied by weakness, faintness, and diaphoresis. Core body temperature does not reach dangerous levels. Cool liquids and rest usually suffice.

Heat stroke is accompanied by weakness, faintness, or coma and (classically) dry skin, with dangerous core temperatures of 40.5°C (105°F) or higher. In athletes and others well accommodated to heat, the skin may be diaphoretic.

The patient usually presents with prostration and a depressed LOC. Seizures may occur. You may need to perform orotracheal intubation to protect the airway. (**Vol II – AIR SKILLS 3 OROTRACHEAL INTUBATION**)

When vital signs reveal severe hyperthermia, immediately begin cooling the body to reduce core temperature before permanent CNS damage occurs. An effective method is to undress the patient and spray tepid water on the body while fans blow air over the body. The resulting evaporation lowers body temperature while not inducing shivering that is counter-productive. Ice packs may be placed in the axillae and groin. If the core temperature fails to respond, gastric and bladder irrigation with cool NS may also be used. Lower the body temperature to about 40°C (104°F) then remove the water and fans because the core temperature will continue to decrease.

Many complications may ensue including cerebral edema, heart failure, myonecrosis, and pulmonary edema. The patient may be severely dehydrated on arrival. Hydrate vigorously but monitor carefully so as not to overshoot the mark. Most patients will require monitoring in an intensive care unit post resuscitation.

15 Severe Hypothermia

Many children with hypothermia have also suffered trauma. Evaluate these patients as trauma patients.

Definition According to Core Temperature

Mild hypothermia	34°C to 36°C (93°F to 96°F)
Moderate hypothermia	30°C to 34°C (86°F to 93°F)
Severe hypothermia	<30°C

Mildly hypothermic patients may be rewarmed with the external application of warmth and warm fluids. Moderately hypothermic patients will require warm IV fluids and careful monitoring for possible dysrhythmias. Rectal temperature is the

most practical method of measuring core temperature. Esophageal and urinary bladder probes are ineffective when gastric and bladder lavage are used as rewarming methods.

The severely hypothermic child may appear to be clinically dead. In the absence of other factors incompatible with life, aggressively attempt resuscitation until the core temperature is above 34°C (93°F). Asystole above this temperature should not be attributed to hypothermia and other causes should be considered. **(Vol I— ACUTE CARE PORTALS, ASYSTOLE)**

Pronounce patient's death if there is no response or if the history indicates a length of exposure incompatible with survival. Patients have survived total immersion in very cold water for 30 to 45 minutes and core temperatures of about 15°C to 16°C (60°F). Use clinical judgment. Not every patient needs to be completely rewarmed, to 98.6°F, before pronouncing death, but astounding cases of survival from apparent death have occurred.

Aggressive airway management is necessary to provide heated, humidified oxygenation. Cold bronchorrhea occurs and demands good tracheal toilet.

Although gross movement of a hypothermic patient (such as going from an upright to a supine position) may precipitate VF, the benefit of ET intubation for oxygenation and airway protection far outweighs the risks of precipitating VF. If the child is in VF, attempt defibrillation 1 time at 2 J/kg, but do not persist. The natural history of VF arrest in hypothermia is as follows:

At a core temp of about 21°C (75°F), the rhythm spontaneously converts to atrial fibrillation with a slow ventricular response; At a core temp of about 27°C (80°F), the ventricular response will become faster; At a core temp of about 30°C (86°F), the rhythm will spontaneously convert to normal sinus rhythm.

Continue CPR until the pulse is palpable. If the child has not converted at a temp of about 32°C (90°F), administer appropriate cardiac medications to restore sinus rhythm. **(Vol I— PATHWAY 2 CARDIOVASCULAR EMERGENCIES)** Cardiac medications are not effective in severely hypothermic patients and may accumulate with adverse effects when the child is warm. Hypothermic children may have abnormal electrolytes, acid/base components, and glucose. Measure these often. Hypothermic children are hypovolemic because of cold diuresis produced when the cold kidneys fail to concentrate urine. Establish large bore IVs for administering a bolus of warm NS. Cold causes a shift of the oxyhemoglobin dissociation curve to the left. Some degree of acidosis (which causes a shift to the right) is protective. A low PCO₂ can cause alkalosis; do not hyperventilate these patients.

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Blood gases may be corrected for temperature or not. One line of reasoning is that the whole body is cold, not just the blood, so measuring blood gases at 37°C (98.6°F) is appropriate because the cold body temperature will change the body's enzyme systems and chemistries in the same way as it does the blood gases. Normal blood gases, under these circumstances, indicate that a physiologic balance of oxygen, hydrogen ions, and carbon dioxide are present in the hypothermic patient.

To rewarm severely hypothermic children, avoid using external methods before the body's central circulation is ready to supply the oxygen demands of the warmed periphery. Use internal methods if the patient's core temperature is less than 30°C (86°F). Expect a rate of rewarming of about 4°C (7°F) per hour.

Internal Rewarming Methods That May Be Used:

1. Heated, humidified oxygen ventilation at 40°C (104°F)
2. Warm IV fluids at 40°C
3. Warm gastric lavage using tap water at 40°C. Use a standard gastric lavage set up.
4. Warm urinary bladder lavage using sterile NS. Connect the Foley catheter to a standard gastric lavage set up that has been sterilized. Use a volume of about 10 to 15 mL/kg.
5. Closed peritoneal lavage if the patient does not have a surgical incision on the abdomen. Use NS or LR at 40°C. (**Vol II – CIRC SKILLS 7 REWARMING TECHNIQUES**)
6. Closed left chest lavage using a chest tube connected to chest suction (**Vol II – BREATH SKILLS 1 CHEST TUBE INSERTION**) and a large needle thoracostomy catheter inserted over the 3rd rib in the midclavicular line. (**Vol II – BREATH SKILLS 5 NEEDLE THORACOSTOMY**) Warm NS at 40°C is infused into the chest through the needle thoracostomy catheter and allowed to bathe the heart and left lung before evacuation through the chest tube. You may not consider this method unless a chest tube is needed for other reasons.
7. Cardiopulmonary bypass is the most effective way of rewarming severely hypothermic patients. Trauma centers are prepared to do this. Patients that have solidly frozen extremities are most likely to benefit. Consult freely.

16

Drowning

Some drowning patients are actually victims of diving accidents. Always keep the possibility of trauma in mind during the assessment of these patients.⁹ Patients should be immobilized. Place C-collar if not already done. In the ED, patient evaluation and management follows the usual trauma patient protocol.

These children rarely inhale much fluid into the lungs because of laryngeal spasm and breath holding. The occurrence of hemolysis (in fresh water drowning)¹⁰ and hemoconcentration (in salt water drowning) is largely theoretical.

A significant admonition, however, is that these patients may develop acute pulmonary edema hours after the event. Observe drowning patients for 24 hours.

Consider hypothermia as possibly being present. Measure rectal temperature.

If hypothermia, trauma, and significant aspiration are not present, hypoxic brain injury is probably the cause of continuing coma. ([Vol I – PATHWAY 1 ALTERED LEVEL OF CONSCIOUSNESS](#))

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Caustic Substance Ingestion

Caustic ingestions usually involve the intentional ingestion of drain cleaners containing concentrated alkaline powders or solutions. These strongly basic substances, usually sodium or potassium hydroxide, produce liquefaction necrosis. In powder form, they more likely result in more proximal tissue destruction and more airway compromise. In liquid form, they more likely produce most of their destruction in the distal esophagus and stomach.

The potential for airway compromise is great. If there is any question about swelling, edema or burning about the posterior pharynx, or stridor, orotracheally intubate the child. Perform RSI to ensure the gentlest intubation. ([Vol II – AIR SKILLS 4 RAPID SEQUENCE INTUBATION](#))

If orotracheal intubation is not successful and the airway is compromised, perform emergency cricothyrotomy. In children younger than about 8 years, a tracheotomy will be necessary. ([Vol II – AIR SKILLS 13 CRICOTHYROTOMY, AIR SKILLS 14 TRACHEOTOMY](#)) Administer oxygen and establish IVs. Pain control may be needed. Support the patient's vital signs.

Beyond airway control and general support, little can be done in the rural setting.

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Gastric lavage and dilution may do more harm than good. Consult an otolaryngologist and prepare to transfer as quickly as possible. Your consultant may favor steroid therapy and antibiotics. Subsequent gastric and esophageal perforation may occur a week or two after the ingestion.

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