The Emergency Health Care Clinical Guidelines for Physicians

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THE HASHEMITE KINGDOM
OF JORDAN

MINISTRY OF HEALTH

المملكة الأردنية الهاشمية

وزارة الصحة

Over the last two decades, Jordan's health care system has improved dramatically, placing it among the top ten countries with regard to infant mortality. Across Jordan, emergency health services, particularly public hospital emergency departments, face many challenges. As guardians of our nation's health, it is of the utmost importance that we ensure high-quality emergency health services for the Jordanian population.

This publication is part of the Evidence-Based Clinical Guideline series that was developed by the Ministry of Health. It is intended for public sector Jordanian health care professionals. These guidelines have been developed through an integrated and coordinated collection of reference protocols, manuals, guidelines, and other materials. Consistent use of these guidelines will aid health care professionals and result in the enhance provision of high-quality health services to Jordanians. The goal is to decrease disability, improve the quality of care provided, and reduce mortality rates resulting from trauma, obstetric complications, and non-traumatic surgical conditions.

The information contained in these guidelines should be disseminated to all relevant health professionals, so that patients may benefit from their increased knowledge and skill.

All personnel who have worked so diligently to produce this series, Ministry of Health and their technical counterparts, deserve praise and appreciation.

Minister of Health

Dr. Nayef Hayel Al-Fayez

Acknowledgements

The HSS II acknowledges and gives special thanks to the clinical specialists who have contributed to the development of the ***Emergency Health Care Clinical Guidelines.***

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Acronyms

A

AAA	Abdominal Aortic Aneurysm
ABCs	Airway, Breathing, Circulation
ABCDE	Airway Breathing Circulations Disability Exposure
ABG'S	Arterial Blood Gases
ACC	American College of Cardiology
ACEP	American College of Emergency Physicians
ACLS	Advanced Cardiac Life Support
ACS	Acute Coronary Syndromes
AED	Automatic External Defibrillator
AF	Atrial Fibrillation
AG	Anion Gap
AHA	American Heart Association
ALS	Advanced Life Support
AMI	Acute Myocardial Infarction
AP	Anterior Posterior
ARDS	Adult Respiratory Distress Syndrome
AS	Aortic Stenosis
ATLS	Advanced Trauma Life Support

B

BSA	Body Surface Area
BSL	Blood Sugar Level
BVM	Bag-Valve-Mask

C

C+S	Culture and Sensitivity
Ca	Calcium
CABG	Coronary Artery Bypass Graft
CAD	Coronary Artery Disease
CBC	Complete Blood Count
CBG	Complete Blood Gases
CHD	Coronary Heart Disease
CHF	Chronic Heart Failure
CK MB	Creatinine Kinase Myocardial Bands
CNS	Central Nervous System
COHB	Carboxyhemoglobin

COPD	Chronic Obstructive Pulmonary Disease
CPK	Creatine Phosphokinase
CPR	Cardiopulmonary Resuscitation
CroFab	Polyvalent Crotalidae Immune Fab
CSF	Cerebrospinal Fluid
CSSD	Central Sterile Sanitizing Department
CT	Computerized Tomography
CV	Cerebrovascular
CVP	Central Venous Pressure
CXR	Chest X-Ray

D

DIC	Disseminated Intravascular Coagulation
DKA	Diabetic Keto-acidosis
DM	Diabetes Mellitus
DPL	Diagnostic Peritoneal Lavage
DVT	Deep Vein Thrombosis

E

ECG	Electrocardiogram
ED	Emergency Department
EEG	Electro- Encephalogram
ET	Endotracheal
ETC	Esophageal Tracheal Combitube
ETI	Endotracheal Intubation
EVM	Eye, Verbal, Motor

F

F.B	Foreign Body
FAST	Focused Abdominal Ultrasound
FBAO	Foreign Body Airway Obstruction
FEV	Forced Expiratory Ventilation
FRC	Functional Residual Capacity

G

GCS	Glasgow Coma Scale
GERD	Gastroesophageal Reflux Disease
GHB	γ-Hydroxybutyrate
GI	Gastrointestinal
GIIB/IIIA INHIBITORS	Glycoprotein IIB/IIIA Inhibitors
GTD	Gestational Trophoblastic Diseases

H

HCO3	Bicarbonate
HDCV	Human Diploid Cell Vaccine
HDU	High Dependency Unit
HHS	Hyperosmolar Hyperglycemic State
HR	Heart Rate
HTN	Hypertension

I

ICP	Intracranial Pressure
IO	Impedance Plethysmography
ISS	Injury Severity Score
IUGR	Intrauterine Fetal Growth Retardation
IV	Intravenous
IVF	Intravenous Fluid
IVU	Intravenous Urogram
IWMI	Inferior Wall Myocardial Infarction

K

K+	Potassium
KFT	Kidney Function Test

L

LBBB	Left Bundle Branch Block
LFT	Liver Function Test
LDH	Lactate Deydrogenase
LMA	Laryngeal Mask Airway
LMP	Last Menstrual Period
LMWH	Low Molecular Weight Heparin
LOC	Level of Consciousness
LV	Left Ventricular

M

MI	Myocardial Infarction
MRI	Magnetic Resonance Image

N

NG	Naso-Gastric
NIS	Noninvasive Study of the Lower Extremities

NSTEMI	Non –ST Segment Elevation Myocardial Infraction

P

P.O	Per Oral (By Mouth)
PA	Pulmonary Angiography
PAO2	Partial Pressure of Oxygen in Arterial Blood
PCI	Percutaneous Coronary Intervention
PE	Pulmonary Embolism
PEA	Pulseless Electrical Activity
PEF	Pulmonary Expiratory Force
PEFR	Peak Expiratory Flow Rate
PFT	Pulmonary Function Test
PRN	Pro Re Nata and Means As Needed
PTCA	Percutaneous Transluminal Coronary Angioplasty

R

R/O	Role Out
RAS	Reticular Activating System
RBG	Random Blood Glucose
RL	Ringer Lactate
RR	Respiratory Rate

S

SBP	Systolic Blood Pressure
SC	Subcutaneous
SGOT	Serum Glulamic Oxaloacetic Thrausaminase
SGPT	Serum Glutamate Pyruvate
SLNTG	Sublingual Nitroglycerin
SOB	Shortness of Breath
START	Simple Triage and Rapid Treatment
STEMI	ST Segment Elevation Myocardial Infarction

T

TB	Tuberculosis
TBV	Total Blood Volume
TEE	Transesophageal Echocardiogram
TFTs	Thyroid Function Test

U

U/S	Ultrasound
UA	Unstable Angina

UFH	Unfractionated Heparin
UTI	Urinary Tract Infection

V

VQ	Ventilation Quotient
VF	Ventricular Fibrillation
VT	Ventricular Tachycardia

W

W/ OR W/O	With or Without
WBC	White Blood Cells

Introduction

It is a priority for the Jordanian government to improve health services. The Ministry of Health (MOH) continues to improve its readiness to meet the increasing number of Emergency Department (ED) patients in MOH hospitals (Amman and other main cities). Across Jordan, emergency medical services (EMS) face numerous management, workforce, and infrastructure challenges; these challenges include fragmentation of care and variability in quality care delivery. Due to the increasing influx of trauma related patients (specifically motor vehicle accidents), the MOH is taking active steps to improve the status and infrastructure (personnel and physical plant improvements) of selected hospital emergency departments.

The Health Systems Strengthening II project (HSS II), in partnership with the MOH, will continue to support programs that expand and institutionalize high quality health care services in Jordan. Through the development of The Emergency Health Care Clinical Guidelines for MOH Emergency Department physicians, the HSSII and MOH partnership is working toward high quality emergency care.

These guidelines intend to ensure early and appropriate management of life threatening conditions, and to relieve pain and suffering for hospitalized patients. They reflect best clinical practice and offer practical, clearly written protocols, for the diagnosis and management of commonly encountered ED health problems. These in-depth clinical guidelines are going to be utilized as an easy-to-use resource, scientific reference, and clinical tool to support performance improvement.

The latest available trauma-related scientific evidence delineated the guideline's topics and information. The guidelines consist of four emergency-related categories (general, surgical, medical, and obstetric); each categorical section includes multiple guidelines related to the management of a trauma-specific patient.

These guidelines are designed for the non-specialist and specialist clinician working in the ED, including:

- General medical practitioners,
- Junior and senior resident doctors, and
- Emergency medicine specialists.

Part 1: General Emergencies

Chapter 1: Disaster Management

GUIDELINE OBJECTIVES:

- Define a disaster.
- Utilize disaster levels.
- Understand the stages of management during a disaster.
- Understand and be able to categorize patients during disaster triage, utilizing the simple triage and rapid treatment (S.T.A.R.T) methodology.
- Recognize important tasks that should be accomplished during the field (pre-hospital) phase.
- Understand and implement the hospital phase of disaster management.

1. INTRODUCTION

A disaster is any event (man-made or natural) that causes devastation or destruction, which cannot be managed by usual resources. A disaster is not defined by the number of injuries, deaths, or destructive damage. If the resources are overwhelmed, the event is categorized as a disaster. "Disaster Management", is the activities instituted before, during, and after a disaster that attempt to maintain control, lessen the impact, and aid in recovery.

2. CLASSIFICATION

2.1. Types of Disasters

A. Natural:

- Weather (hurricane, drought, typhoon, cyclone).
- Topographic (landslide, avalanche, flood, mud slide).
- Underground (earthquake, tsunami, volcanic eruption).
- Biological (communicable disease outbreak).

B. Man-Made:

- Warfare (nuclear/biologic/chemical terrorism, blockade, siege).
- Civil disturbance (riot, demonstration).

C. Accidental:

- Transportation accident (airplane crash, train wreck, sinking ship, traffic accident).
- Structural collapse (building, mine, dam).
- Explosion (fire, hazardous materials release, nuclear accident).
- Biological (inadequate sanitation).

2.2. Disaster Levels

A. Level I:

Local resources adequate.

B. Level II:

Requires regional aid.

C. Level III:

Requires national aid.

3. MANAGEMENT

3.1. Four Stages of Disaster Management

A. Preparedness:

Being prepared includes having equipment and supplies in place, in addition to a well thought-out, and rehearsed plan. Teaching disaster response and basic first aid to the public is invaluable.

B. Hazard Mitigation:

Building stability and safety, code enforcement, safety measures (e.g., bolting down bookcases in earthquake areas), and possession of insurance policies to cover disaster-related damage.

C. Response:

- Provision of emergency medical care, which includes basic first aid or more advanced care, depending on the number of injuries and the availability of staff and supplies.
- Provision of psychological support.
- Provision of basic human needs including food, water, and shelter.

D. Recovery:

Recovery occurs after initial stabilization is ensured, and includes continued medical care, cleanup, and rebuilding.

3.2. Disaster Triage

- The primary goal of disaster triage is to do the most good for the most number of victims. Triage is designed to give maximal medical results with available resources.
- *The main function of the triage team is to sort the victims, not treat them.* Therefore, the triage team should not spend an inordinate amount of time on any single victim.

A. Disaster Triage Consists of Two Phases:

- Phase one is used to initially categorize the victims as “immediate”, “delayed”, “dead”, or “walking wounded”.

- Phase two is implemented if treatment begins immediately on scene. A second triage will be instituted to determine which of the victims will be seen first, and in what order.

B. Triage Categories:

- Immediate
  - Airway compromise.
  - Suspected internal bleeding.
  - Severe uncontrolled external bleeding.
  - Serious fractures (pelvis, femur, neurovascular compromise).
- Delayed
  - Stable fractures.
  - Spinal cord injuries.
  - Minor burns.
- Dead/Expectant
  - Obvious dead.
  - Severe burns (80-100% full thickness).
  - Cardiac arrest.
  - Severe head injury (e.g., brain matter showing or evidence of increased intracranial pressure (ICP)).

3.3. The Simple Triage and Rapid Treatment (START) System

This is a widely accepted system developed by a Californian hospital. It is a simple step-by-step method, employed by the first qualified person who arrives on the disaster scene. Although designed for the pre-hospital setting, it can be used in the hospital as well (see figure 1.1).

START allows first responders to triage victims based on three assessments:

- Is ventilation adequate?
- Is perfusion adequate?
- Is the brain injured?

A. How (START) Is Implemented:

- Assess the scene to make sure it is safe to enter and begin triage.
- Identify the mechanism of disaster (e.g., building collapse, fire, electrocution, etc).
- Separate the "walking wounded" by announcing, "If you can walk, move over there" (designated area). These victims are considered minor and are designated with green tags.
- Triage and tag the remaining victims.
  - Immediate: Red tag.
  - Delayed: Yellow tag.
  - Dead: Black tag.

  Once all the victims have been tagged, preparation for transport and/or field treatment can begin.

3.4. Field Treatment Following a Disaster

A. The Goals of Field Medical Care:

- Maintenance of an open airway.
- Bleeding and wound control.
  - Direct pressure will stop most external hemorrhages. If direct pressure fails, elevate the affected area and/or compress the nearest pressure point.
  - Cover all open wounds. The sight of blood after a disaster increases public anxiety. If dressing supplies are inadequate, clean bed sheets (or other material) can be cut and used.
  - Irrigate all wounds prior to closing. Use forceps to remove all obvious foreign material in the wound.
- Immobilization of suspected fractures:
  - Use cardboard, magazines, or padded pieces of wood.
  - Another option is to splint two body parts together ("buddy splint"). Make sure that there is adequate padding between the extremities.
  - Once the extremity is splinted, elevate it, and apply a cold pack (if available).
  - Always check distal nerve and circulatory function before and after splinting. If distal circulation is compromised after splinting, remove the splint and reapply. If distal circulation is compromised prior to splinting, a closed reduction is indicated.
  - Recognize and treat shock.
  - Pain relief.
  - Recognition of crush injury: Crush syndrome occurs secondary to prolonged (>4 h) continuous compression of the extremities and can lead to rhabdomyolysis, life-threatening myoglobinuria, renal failure, hyperkalemia, and disseminated intravascular coagulation. Prehospital care begins before the victim is removed from the disaster scene. After extrication, the hemodynamic status of the injured victim may rapidly deteriorate and the victim may develop due to the severe hypovolemia (extremity edema develops and upon release of the extremity, redistribution of body fluids). Treatment prior to removal of the trapped extremity includes:
    - Bolus intravenous hydration using normal saline.
    - Albuterol via hand-held nebulizer will help mitigate hyperkalemia.
    - Consider calcium chloride 1 g intravenous IV push.
    - Sodium bicarbonate (1 mEq/kg added to a liter of normal saline).

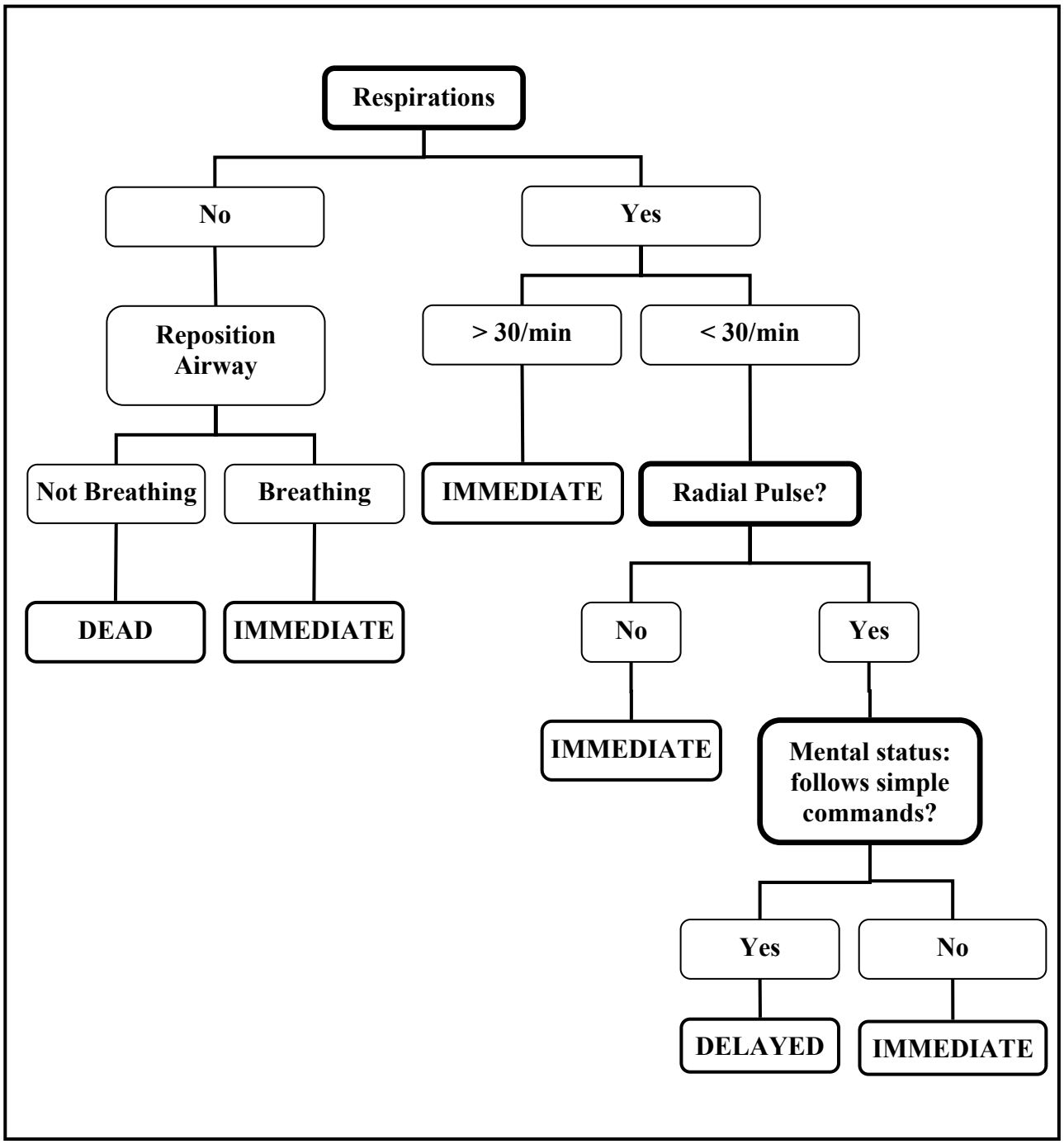
3.5. Disaster Management within the Hospital

- After a major disaster, a hospital must assess the damage, care for the injured, assess the ability of the hospital to function, and prepare for the potential influx of victims.
- Some hospitals have an organized system to determine their operational status. The following is an example of the system used:
  - "Green": the hospital is able to carry out both emergency and inpatient services in a normal manner.

- "Amber": some reduction in patient care services, but overall, the hospital is able to continue providing emergency and inpatient services.
- "Red": significant reductions in patient care services. Only emergency services being provided.
- "Black": hospital is severely impacted and unable to provide emergency or inpatient services.

Communication is important during a disaster. Documentation, organization, and situational reporting to higher levels (the regional or national disaster managing center) allows for continuity of care.

Figure 1.1: S.T.A.R.T. Divisionary Algorithm



Chapter 2: Triage

GUIDELINE OBJECTIVES:

- Define triage.
- Recognize objectives of patient triage.
- Understand triage guidelines.
- Identify triage categories.
- Demonstrate competency in triage response.
- Perform triage documentation.

1. INTRODUCTION

Triage is the process used to sort patients in order of acuity or the severity of their illness. Triage is designed to get the right patient, to the right place, at the right time, with the right care provider. Rapid access to health care provider assessment increases patient satisfaction, reduces client anxiety, and improves public relations. The goals of triage are to:

- Rapidly identify patients with urgent or life threatening conditions.
- Determine the most appropriate treatment area for patients presenting to the emergency department (ED).
- Decrease congestion in emergency treatment areas.
- Provide information to patients and families regarding expected care and waiting times.

1.1. Triage Doctor/Nurse Duties

- Should have rapid access to, or be in view of the registration and waiting areas at all times.
- Greeting patients and families in warm, empathetic manner.
- Performing brief visual assessment.
- Documenting the assessment.
- Triage patients into priority groups, using appropriate guidelines.
- Transporting patients to treatment areas, when necessary.
- Reporting to the treatment nurse or ED doctor.
- Measuring relevant vital signs for determination of triage category.
- Reassessment of patients that were directed to the waiting room.
- Notifying patients and families of delays.
- Instructing waiting patients and families to notify triage staff of changes in their condition.

1.2. Triage Guidelines

- All patients should be assessed (at least visually) within five minutes of arrival. The triage assessment should take no longer than two-to-five minutes.
- Routinely, full patient assessment should not occur in the triage area.
- Record all information obtained.

- Perform rapid assessments when two or more patients are waiting to be triaged.
- A patient's priority category may change after a complete assessment or as the patient's signs and symptoms evolve.

Triage is a dynamic process

A patient's condition may improve or deteriorate during the wait for entry into the treatment area.

1.3. Triage Assessment

The purpose of the triage assessment is to determine priority of care, not to establish a final medical diagnosis.

A. First Impression:

"Quick look"initial impression by triage personnel.

B. Chief Complaint.

C. Analysis of Chief Complaint.

D. Vital Signs.

2. CLASSIFICATION

- Based on initial assessment, patients should be placed into one of three categories (A, B, or C).
- Category A represents patients in immediate need (ie. life-threatening)
- Category B indicates moderate need
- Category C indicates minor need.

3. CLINICAL FEATURES

3.1. Category A

Refer to table 2.2.

A. Description of Category:

- Conditions that are life-threatening (or imminent risk of deterioration) and require immediate aggressive intervention.
- Critical treatment, which must be applied within a limited time window.
- Very severe pain.

B. Clinical Descriptors (indicative only):

- Cardiac arrest.
- Respiratory arrest.
- Immediate risk to airway- impending arrest (severe strider or drooling with distress):

  - Respiratory rate < 8/min.
  - Severe respiratory distress.
  - Systolic blood pressure (SBP) <80 (adult) or severely shocked child.
- Circulatory compromise:
  - Clammy or mottled skin, poor perfusion.
  - Heart rate (HR) <50 or >150 (adult).
  - Hypotension with hemodynamic effects.
  - Severe blood loss.
- Ongoing/prolonged seizure.
- Intravenous (IV) overdose.
- Glasgow coma scale (GCS) ≤ 13 (Drowsy, decreased response to stimuli or responds to pain only).
- Chest pain of cardiac nature.
- Very severe pain (any cause).
- Blood sugar level (BSL) <3 mmol/l.
- Acute hemiparesis or dysphasia.
- Fever with signs of lethargy (any age).
- Suspected meningococcemia.
- Major multi trauma.
- Severe localized trauma (major fracture).
- Amputation.
- Behavioral/psychiatric with immediate risk to harm to self or others.

3.2. Category B

A. Description of Category:

- Potentially life threatening.
- Potentially serious.
- Situational urgency.
- Significant complexity or severity.

B. Clinical Descriptors (indicative only):

- Severe hypertension.
- Moderate blood loss.
- Moderate shortness of breath.
- Sat.O_2: 90-95%
- Blood sugar level (BSL) > 16 mmol/l.
- Seizure (post ectal).
- Persistent vomiting.
- Dehydration.
- Head injury with short loss of consciousness (LOC).

- Non-cardiac chest pain.
- Stable neonate.
- Moderate limb injury.
- Trauma (high risk history with no other high risk features).
- Patient age > 65 years.
- Abdominal pain without high risk features.
- Mild hemorrhage.
- Foreign body (F.B.) aspiration, no respiratory distress.
- Difficulty swallowing, no respiratory distress.
- Minor head injury, no loss of consciousness.
- Moderate pain.
- Eye inflammation or F.B.
- Risk of fracture.
- Tight cast.
- Minor limb trauma (sprained ankle, uncomplicated laceration).

3.3. Category C

A. Description of Category:

- The patient's condition is chronic or minor. Clinical outcomes will not be significantly affected if assessment and treatment are delayed.

B. Clinical Descriptors (indicative only):

- Minimal pain.
- Minor symptoms of existing stable illnesses.
- Minor wounds, small abrasions, minor lacerations (not requiring sutures).
- Scheduled visit.
- Immunization only.

4. MANAGEMENT

4.1. Category A

Place patients in a resuscitation area and *immediately perform simultaneous full assessments and treatments.*

4.2. Category B

When triaging patient, perform and document the patient's full vital signs, indicating that it is safe for them to wait. Full assessment and treatment should occur within 30 minutes of patient's arrival.

4.3. Category C

During triage, vital signs can be delayed until re-evaluation in the waiting or treatment areas. *Full assessment and treatment should occur within 60 minutes of patient's arrival.*

5. MONITORING

5.1. Re-triage

To ensure the patient status is not deteriorating after initial triages. Re-triage should occur at the time intervals recommended in table 2.1.

Table 2.1: Re-triage Times

Category	A	B	C
Time Interval	Continuous Care	15 Minutes	>30 Minutes

5.2. Documentation Standards

The documentation of the triage assessment should minimally include the following essential details:

- Date & time of assessment.
- Name of the triage officer.
- Chief presenting problem(s).
- Limited relevant history.
- Relevant assessment findings.
- Initial triage category allocated
- Re-triage category with reasoning.
- Assessment and treatment area allocated.
- Any diagnostic, first aid, or treatment measures initiated.

Table 2.2: Examples of Patient Triage Categories

	Category A	Category B	Category C
Abdominal pain	• Protracted vomiting • Active Gl bleeding • Pallor and diaphoresis with abnormal vital signs • Syncope with abnormal vital signs • Suspected acute abdomen • Hypotension • Vaginal bleeding with abnormal vital signs • Pain radiating to shoulder or chest • 60 years or older with pain constant for > (1h) • Urinary retention with painfully full bladder	• Evidence of coffee ground emesis • Syncope with normal vital signs • Elderly age group • Abdominal distension • Vaginal bleeding with normal vital signs • Dark or black stools	• Constipation • Chronic or mild abdominal pain • Chronic diarrhea with no signs of dehydration • Recent vomiting or diarrhea without signs of dehydration

	Category A	Category B	Category C
	• 60 yrs or older with symptoms of UTI & fever	• Foreign body in rectum or vagina • Flank pain and dysuria without fever	
Allergic Reaction	• Oral swelling • Hypotension • Respiratory compromise or wheezing • Signs of impending anaphylaxis	• Increasing edema • Facial swelling • Increasing rash or urticaria	• Uncomplicated rash • Urticaria
Back Pain	• Severe disabling pain • Associated numbness, tingling, weakness, or decreased temperature of extremity • Bowel or bladder dysfunction • Inability to walk • Pain radiating to back from another area • Flank pain with fever and vomiting	• Pain radiating to legs • Inability to sit • Back or flank pain with fever or chills	• Minor back pain that is localized • Chronic back pain • Back pain associated with influenza
Chest	• 40 yrs or older with the following pain symptoms: tightness, crushing, squeezing, substernal, or radiating (may include jaw pain) • Chest pain with diaphoresis • Chest pain with cyanosis or pallor • Chest pain with nausea • Chest pain with palpitations • Chest pain with weakness • Chest pain with haemoptysis • Chest pain with cardiac history • History of chest trauma with respiratory distress	• Under 40 years of age • Increasing pain on inspiration • Cough with fever, history of lung/heart disease, or elderly	• Allergy or hayfever symptoms • Cough without fever or respiratory compromise • Mild upper respiratory infection

	Category A	Category B	Category C
Eye	• Chemical burns • Penetrating trauma • Sudden loss of vision • Hyphema • Sudden severe pain	• Decreasing visual field • Periorbital swelling with fever • Blurred or double vision after trauma • Lacerations of eyelid • Foreign body	• Eye infection • Subconjunctival hemorrhage • Eye(s) drainage • Gradual change in vision aquity • Ptosis without trauma • Nonpenetrating FB without pain, i.e. dust
Head	• Seizure • Ataxia • Incapacitating pain • Weakness or paralysis • Possible bacterial meningitis	• History of migraine headache • Headache with vision changes	• Headache • Sinus pain • Toothache or temporo-mandibular joint pain
Neck	• Uncontrolled bleeding • Inhaled foreign body • Possible meningitis or epiglottitis	• Wound to neck with controlled bleeding, no involvement of deep structures	• Chronic neck pain • Neck stiffness without signs of infection
Neurological	• Head injury with respiratory distress/seizure • Decreased level of consciousness or confusion • Active seizure or new onset of seizures • Confusion with fever or seizures • Paraplegic/quadriplegic with any of the following symptoms suggesting autonomic dysreflexia: ↑BP, diaphoresis, facial flushing, bounding pulse	• Focal or acute changes in seizure activity with history of confusion.	• History of seizures without current seizure activity • Depleted seizure medications • Chronic confused condition without acute changes
Nose	• Uncontrolled hemorrhage • Epistaxis with hypertension	• Partially controlled epistaxis	• Nasal discharge • Epistaxis without active bleeding

	Category A	Category B	Category C
		• Foreign body with possible F.B. enlargement	• Nasal trauma without bleeding • Possible fractured nose
Obstetrics	• Active labor, crowning • Delivery prior to arrival • Severe abdominal pain with history of pregnancy • Triage directly to obstetric unit, if in active labor and over five months gestation (20 weeks), having pregnancy related problems, or having non-acute medical problems (while pregnant)	• Vaginal spotting with history of pregnancy	
Orthopedics	• Amputations • Dislocations • Compartment syndrome • Uncontrolled bleeding	• Suspected fractures • Hand injuries • Joint pain with fever • Calf pain with dyspnea and/or cough	• Sprains / strains • Minor injury without deformity • Cast check or removal
Pediatrics	• Respiratory distress with any of the following: stridor, retractions, wheezing, flaring nares, or tachypnea • Pallor or cyanosis • Lethargy • Unexplained irritability • Bulging fontanelle • Petechial rash • Dehydration • Infant < 3 months with fever > 39º F • Any infant < 1 year with history of premature birth and experiencing respiratory distress • Febrile seizure	• Vomiting & diarrhea without signs of dehydration	• Cold symptoms without respiratory distress • Earache

	Category A	Category B	Category C
	• Diarrhea with signs of dehydration		
Respiratory	• Acute distress or dyspnea • Diaphoretic, pale, or cyanotic • Drooling due to the inability to swallow • Wheezing or stridor • Foreign bodies • Diabetics with rapid respiratory rate • (SOB) or COPD with pulse > 120/min or RR > 30/M • 60 yrs or older with new onset of SOB or respiratory rate > 24/min	• Pain on inspiration without trauma	• Sore throat without airway obstruction • Cough without respiratory compromise
Wounds	• Loss of pulses distal to the wound • Uncontrolled bleeding • Sever (>20%) deep partial thickness burns • Full thickness (3º degree) burns or possible inhalation injury • Snakebite from possible venomous snake • Chemical burns or contamination • Deep partial or full thickness burns of face, hands, feet, perineum, or skin overlying joints	• Wounds requiring suturing • Grossly infected/contaminated wounds • Partial thickness burns 10 - 20% BSA • Human or animal bite over joint space	• Abrasions • Suture removal • Superficial sunburn • Partial thickness burns < 10% BSA • Contusions • Non-suturable lacerations
GI= Gastrointestinal, **1H**= One hour **UTI=** Urinary tract infection **BP** = Blood pressure **F.B.**= Foreign body, **SOB**= Shortness of breath **COPD** = Chronic obstructive pulmonary disease, **BSA**= Body surface area **RR**= Respiratory rate			

Chapter 3: Airway, Ventilation and Oxygenation

GUIDELINE OBJECTIVES

- Identify the airway compromise clinical situations.
- Recognize the signs and symptoms of acute airway obstruction.
- Understand how to establish and maintain a patent airway using different airway adjuncts.
- Outline the steps necessary for maintaining oxygenation, before, during, and after establishing a definitive airway.

1. INTRODUCTION

Hypoxia is a state of oxygen deficiency which is sufficient to cause an impairment of function. Hypoxia is caused by the reduction in partial pressure of oxygen, inadequate oxygen transport, or the inability of the tissues to use oxygen. *The inadequate delivery of oxygenated blood to the brain and other vital structures is the quickest killer*. Prevention of hypoxemia requires:

- Unobstructed airway.
- Protected airway.
- Adequate ventilation.
- Begin with simple airway management and proceeds to more advanced management.

2. CLINICAL FEATURES

2.1. Physical Examination

A. Look:

Patient agitation suggest hypoxia, obtunded behavior suggest hypercarbia or cyanotic hypoxia.

B. Listen for Abnormal Sound:

Noisy breathing = obstructed breathing.

Snoring, striding = associated with partial occlusion in the pharynx and larynx.

C. Feel for Tracheal Location:

Airway compromise can be sudden and complete, partial, progressive and/or recurrent. Therefore, assessment is critical. During initial assessment of the airway, the talking patient provides reassurance that the airway is patent, ventilation is intact, and that brain perfusion is adequate (at least for the moment).

3. MANAGEMENT

The objective of respiratory support is to ensure a patent airway, provide supplemental oxygen, and institute positive-pressure ventilation, when spontaneous breathing is inadequate or absent. Special devices can help control the airway, ventilate the patient, and provide oxygenation.

3.1. Airway Control: Head and Jaw Position

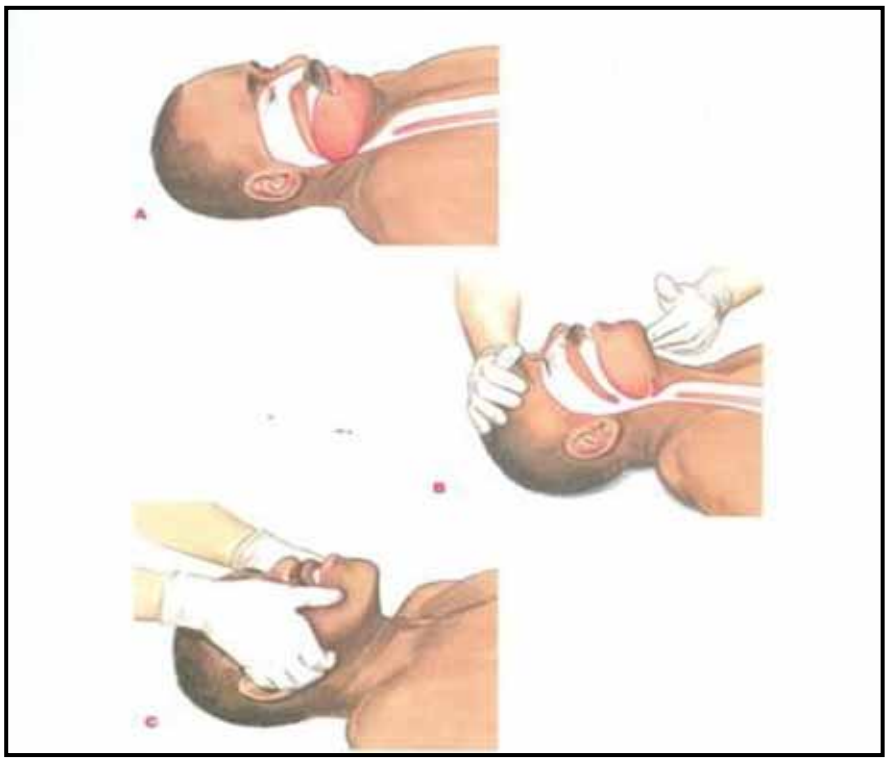
The most common cause of upper airway obstruction in the unresponsive victim, is tongue airway occlusion.

- Basic opening techniques.
- Head tilt-chin lift: The basic opening technique is head tilt with anterior displacement of the mandible (chin lift and if necessary jaw thrust).
- Jaw thrust maneuver: In the trauma victim with suspected neck injury.

3.2. Airway Adjuncts

Assume as a first step that the airway obstruction is produced by either the tongue or relaxed throat muscles. Use the head tilt-chin lift maneuver (see figure 3.1). If this seems insufficient, insert an oropharyngeal or nasopharyngeal airway (see figure 3.2), and (figure 3.3).

Figure 3.1: Respiratory Arrest



Reference: Terry L. Vanden Hoek, MD Chair. Robert O'Connor, MD immediate past chair,2004-2005. John E. Bellli, MD. Henry R. Halperin, MD. Todd J. Crovvo, MD. Steven K ronick, Md. Mark S. Link, MD Laurie Morrison, MD. Robert Neumar, MD. Mary Ann Peberdy, MD. Roger D. White, MD. Charles Otto, Md, PhD. *Advanced Cardiovascular Life* ***Support Provider Manual.The ACLS Cases: Respiratory Arrest.*** *Figure1. Page 25.*

A. Oropharyngeal Airways:

- *Oropharyngeal airways are most helpful for the unconscious spontaneously breathing patients*, who do not have a gag reflex, and is at risk of occluding the airway via tongue and pharyngeal relaxation.
- Oropharyngeal airways keep the airway open during bag-mask ventilation when rescuers tend to unknowingly push down on the chin, blocking the airway.

- These devices help suction the mouth and throat and prevent the patient from biting and occluding a tracheal tube.

Hazards:

- A long oropharyngeal airway may press the epiglottis against the entrance of the larynx, producing complete airway obstruction.
- If the airway is not inserted properly, it may push the tongue posteriorly, aggravating upper airway obstruction.
- The airway should be used only in the unconscious patient.

B. Nasopharyngeal Airways:

- They are frequently used for the intoxicated or semiconscious patient who cannot tolerate an oropharyngeal airway.
- A nasopharyngeal airway is indicated when insertion of an oropharyngeal airway is technically difficult or impossible (because of strong gag reflex, trismus or massive trauma around the mouth).

Hazards:

- A long nasopharyngeal airway may enter the esophagus.
- Although a nasopharyngeal airway is better tolerated by semiconscious patients, its use may also precipitate laryngospasm and vomiting.

C. Tracheal Intubation:

Tracheal intubation provides definitive airway management and should be performed by properly trained personnel as soon as possible during any resuscitative effort.

- Keeps the airway patent.
- Ensures delivery of a high concentration of oxygen.
- Ensures delivery of a selected tidal volume (6-7 mL/kg) to maintain adequate lung inflation.
- Isolates and protects the airway from aspiration of stomach contents or other substances in the mouth, throat, or upper airway.
- Permits effective suctioning of the trachea.
- Provides a route for administration of several medications.

For clinical reasons intubation should be restricted to medical/health care personnel who meet the following criteria:

- Personnel are well trained.
- Personnel perform intubation frequently.

Indications:

- Cardiac arrest with ongoing chest compressions.
- Inability of a conscious patient in respiratory compromise to breathe adequately.
- Inability of the patient to protect the airway (coma, areflexia or cardiac arrest).
- Inability of the rescuer to ventilate the unresponsive patient with conventional methods.

Before endotracheal intubation (ET):

- Check equipment and O_2 source.
- Have equipment ready including:

- Suction device.
- Laryngoscope.
- Endotracheal intubation (ET) tubes.
- Magill forceps.
- Medications.
- Monitors (pulse oximeter, electrocardiogram (ECG) tracing, blood pressure (BP) monitor).

- Consider sedation and paralysis, especially in head injury patients to avoid increase in intracranial pressure.
- The oral route is the preferred route for endotracheal intubation (ET):
  - Pre-oxygenate with a bag-valve-mask with oxygen reservoir to avoid hypoxia during intubation. Inspiratory time during bagging should be slow (1 second) to avoid gastric distension with air. During the use of the bag-valve-mask, observe a good air seal between mask and face with movement of the chest wall.
  - Cricoid pressure by an assistant may be needed to guard against aspiration during ET intubation.

Correct Placement of Tracheal Tube:

- Confirm tube placement immediately, assessing the first breath delivered by the bag-mask unit, observing chest movements and by auscultation.
- Ventilate with a tidal volume of 6 mL/kg.
- Ventilate at a rate of 1 breath every 6-7 seconds.
- Ventilate 1 second for each bag ventilation.
- Ventilate with 100% oxygen.
- Insert an oropharyngeal airway.
- Insert a bite protector.
- Secure the tracheal tube to prevent dislodgment.

Complications:

- Insertion of tube into esophagus
- Tracheal intubation can cause significant trauma to the patient, such as:
  - Lacerated tongue.
  - Chipped teeth.
  - Lacerated pharynx or trachea.
  - Injury to the vocal cords and pharyngeal-esophageal perforation.
- Insertion of tracheal tube into one lung.

D. The Laryngeal Mask Airway (LMA)

The LMA provides an airway adjunct with a cuffed (inflatable), masklike projection at the distal end. When the cuff is inflated, the mask is pushed up against the tracheal opening, providing an effective seal and a clear airway into the trachea. The LMA has several distinguishing features that account for its enthusiastic acceptance as part of the advanced cardiac life support (ACLS) airway management:

- Blind insertion means that the operator does not have to learn to visualize the tracheal opening.
- The need for proper positioning and alignment of the pharyngeal, oral, and tracheal axes is eliminated because the vocal cords are not visualized.

The LMA has significant advantages over the tracheal tube in the following circumstances:

- When a patient has possible unstable neck injuries, access to the patient is limited, or appropriate positioning of the patient for tracheal intubation is difficult or impossible.
- The possibility of fatal errors with the LMA is much lower than that associated with tracheal tubes. The LMA does not invade the trachea, and consequently there is less risk for the fatal.
- The LMA provides less airway protection from regurgitation than the tracheal tube. The device shows great promise for use by healthcare providers who cannot be trained to perform tracheal intubation and for in-hospital and out-of-hospital sites without early responses from advanced-level personnel.
- The LMA may also prove superior to the tracheal tube for the difficult airway.

E. The Combitube:

The esophageal tracheal combitube (ETC) is a plastic twin-lumen tube with a proximal low-pressure cuff that seals the pharyngeal area and a distal cuff that seals the esophagus, allowing ventilation between the cuffs. The proximal seal also removes the need for a facemask and minimizes dental damage to the cuff. The distal cuff is similar to an ET and serves to seal either the esophagus or the trachea when inflated. If the distal tube enters the esophagus, perforations in the esophageal lumen serve to ventilate the patient. If the trachea is intubated, the patient is ventilated directly, as with the cuffed ET.

Studies show that by comparison to the simple oral airway, the ETCs are superior in preventing regurgitation and thus aspiration. Compared with the ET, ventilation and oxygenation studies reveal varying results, but suggest that they are adequate during cardiac arrest. However, the method of choice for airway management for both hospital and prehospital therapy remains direct oral endotracheal intubation.

Advantages:

- Rapidly and easily inserted.
- Avoids need for laryngoscopy.
- Protects against aspiration.
- Can be used if inflation pressures are high.

Disadvantages:

- Available in 2 sizes only.
- Potential for ventilation via wrong lumen.
- Damage to cuffs on insertion.
- Trauma on insertion.
- Single use.

3.3. Airway Control in Trauma Patients

Excessive movement of the head and neck in patients with an unstable cervical spinal column can cause a disastrous injury to the spinal cord or make a minor cord injury much worse.

- Avoid unnecessary movement of the spine in trauma patients.
- Assume that any patient with multiple trauma, head injury, or facial trauma has a cervical spine injury.
- Maintain a high index of suspicion for spinal column or spinal cord injury.

Steps to follow, in known or suspected cervical spine trauma:

- With a suspected neck injury, perform the chin lift or jaw thrust without head tilt.
- Direct a trained rescuer to stabilize the head in a neutral position during all airway manipulation.
- In a patient with facial fractures and fractures at the base of the skull, attempt direct orotracheal intubation while a second rescuer provides spinal immobilization.
- Suction the upper airway as needed.
- If tracheal intubation cannot be performed, consider circothyrotomy.
- Use of laryngeal mask or combitube by an experienced operator can be considered.
- Blind nasal intubation is generally used in breathing patients. Immobilize the spine continuously during intubation attempts because the stimulus may result in spontaneous neck movement. As an advanced technique, blind nasal intubation should be performed only by someone with experience in this technique.
- Avoid manipulating the patient's head and neck during intubation.
- Use paralytic drugs in patients who cannot be intubated with the techniques described above.

3.4. Surgical Airway Creation

A Cricothyrotomy:

Cricothyrotomy allows rapid entrance into the airway for temporary ventilation and oxygenation of patients for whom airway control is not possible by other methods.

Indications for surgical airway (cricothyroidotomy):

- Inability to orotracheally or nasotracheally intubate when airway control is required.
- Upper airway obstruction (above level of vocal cords).

Methods:

- Needle cricothyroidostomy.
- Surgical cricothyroidostomy.

In this technique the cricothyroid membrane is opened with a scalpel and a tube inserted. Percutaneous dilational cricothyrotomy is an emergency variation, in which a small vertical incision is made and a cricothyrotomy tube is advanced over a guidewire and dilator.

B. Tracheostomy:

Surgical opening of the trachea and insertion of a tracheostomy tube should be performed under controlled conditions in the operating room by a skilled person. Tracheostomies should be performed after the airway has first been secured by a tracheal tube, a translaryngeal catheter, or cricothyrotomy. *Tracheostomies are not an appropriate procedure for urgent situations such as airway obstruction or cardiac arrest.*

3.5. Ventilation

Indications:

- Apnea.
- Inadequate ventilation in spite of O_2 therapy.
- Severe respiratory distress.
- Cyanosis or poor arterial blood gases (ABGs).
- As adjunct to treatment of increased intra cranial pressure.

A Mouth-to-Mouth and Mouth-to Nose Ventilation:

It should only be performed in out- of hospital circumstances. If the circumstances require a healthcare provider to provide mouth-to-mouth rescue breathing, it is because a major mistake has been made. Expired-air ventilation can provide adequate volumes of air to the victim. The only limitation is the rescuer's vital capacity and the reduced concentration of oxygen in exhaled air (approximately 17%).

B. Bag-Valve Masks:

An oropharyngeal airway should be inserted as soon as possible to help maintain the airway. The following are some calculations:

- The recommended tidal volume for most adults is 6 mL/kg.

  A man weighing 80 kg would need 500 to 700 mL of oxygen delivered with each squeeze of the bag.
- Most commercially available adult-sized bag-mask units have a 1600-mL bag. Using a 1-handed squeeze, most rescuers can empty no more than 50% of the bag (800 mL or less).

Many rescuers cannot provide a leakproof seal between the mask and face using one hand. The hand holding the mask must perform two tasks simultaneously: form a mask-to-face seal, which requires pushing down firmly on the mask, and tilt the head back, which requires a lifting action.

For this reason many experts and clinicians recommend that two well-trained, experienced rescuers work together during bag-mask ventilation. One rescuer should hold the mask with two hands in a leak proof seal against the mouth, while the other squeezes the bag slowly and gently over one to two seconds.

Complications:

- The most frequent problem with this type of device is the inability to provide adequate ventilatory volumes to a patient who is not intubated.
- Gastric dilatation which may cause aspiration pneumonia especially if aggressive ventilation is used.

Suction Devices:

The rigid pharyngeal catheter (Yankauer) is used to clear secretions, blood clots, and other foreign material from the mouth and pharynx.

The tracheobronchial suction catheter is used to clear secretions through the endotracheal tube or the nasopharynx.

High suction pressure is needed for pharyngeal suction (higher than -120 mm Hg). The tracheobronchial suction catheter should have a design that will:

- Produce minimal trauma to the mucosa with molded ends and side holes.
- Be long enough to pass through the tip of the endotracheal tube.
- Have minimal frictional resistance during insertion through the endotracheal tube.
- Be sterile and disposable.

3.6. Oxygen Therapy

Most acute cardiac patients (e.g. acute myocardial ischemia, congested heart failure, cases of shock) *need low flow 100% oxygen* (see table 3.1).

Nasal Cannula:

- Every liter oxygen/ minute will raise the fraction inspired of O_2 (FIO_2) by 0.04 (maximum up to 6 liter /min).
- It is a low flow system.
- For every liter/min of flow, oxygen inspired will increase by 4%.
- Oxygen flow between 1-6 L/min gives oxygen concentration between 24-44%.

Oxygen Mask:

- Well tolerated by adult patients.
- Oxygen flow should be higher than 5 L/min.
- Oxygen concentration between 40-50%.
- Minimum flow of 5-6 liters should be used to raise FIO2 to 0.4.

Face Mask with Reservoir:

- It provides oxygen concentration more than 60%.
- Six liters per minute will provide 60% oxygen concentration.
- Every L/min will increase oxygen concentration by 10%.
- 10 L/min provides 100% oxygen concentration.

Table 3.1: Ventilation Methods and Oxygen Concentration

Nasal Cannula		**O_2 Mask**	
100% O_2 flow (l/min.)		**100% O_2 flow (l/min.)**	
Liters	**Oxygen Concentration**	**Liters**	**Oxygen Concentration**
1	0.24	5-6	0.4
2	0.28	6-7	0.5
3	0.32	7-8	0.6
4	0.36		
5	0.40		
6	0.44		

Venturi Mask:

- It provides a high gas flow with a more fixed oxygen concentration.
- Oxygen concentration is adjusted by changing the size of the orifice and oxygen flow.
- Oxygen concentration can be adjusted to 24%, 28%, 35%, 40% and 50%.
- The patient is observed for respiratory depression and partial pressure of oxygen in arterial blood (PaO_2) is evaluated.
- The oxygen concentration is then titrated to the preferred level of PaO_2.

Potential Complications:

- Patients with severe chronic obstructive pulmonary disease (COPD) develop progressive hypoxemia and hypercapnia.
- Despite these alterations, arterial and cerebrospinal fluid pH are normal because of increased bicarbonate levels.
- Occasionally, patients become "desensitized" to the respiratory stimulant effects of carbon dioxide and presumably maintain ventilation by means of a reflex ventilatory response to a decrease in (PaO_2) originating in the carotid and aortic bodies.
- If this reflex is ablated by oxygen administration, so that the (PaO_2) exceeds 55 to 60 mm Hg, ventilation is reduced significantly, carbon dioxide retention is exacerbated, and (CO_2) narcosis can result.

Potential ventilatory depression should never contraindicate oxygen therapy in severe hypoxemia. If hypoventilation is a major problem, other support measures, including mechanical ventilation, can be employed.

There is substantial evidence that hyperoxia for prolonged periods can produce deleterious effects on the lungs.

It is unlikely that pulmonary oxygen toxicity develops in humans at a FIO_2 of less than 0.5 at 1 (ATA), even with prolonged exposure.

4. MONITORING

4.1. Pulse Oximetry

The pulse oximeter is designed to measure oxygen saturation and pulse rate in periphral circulation.

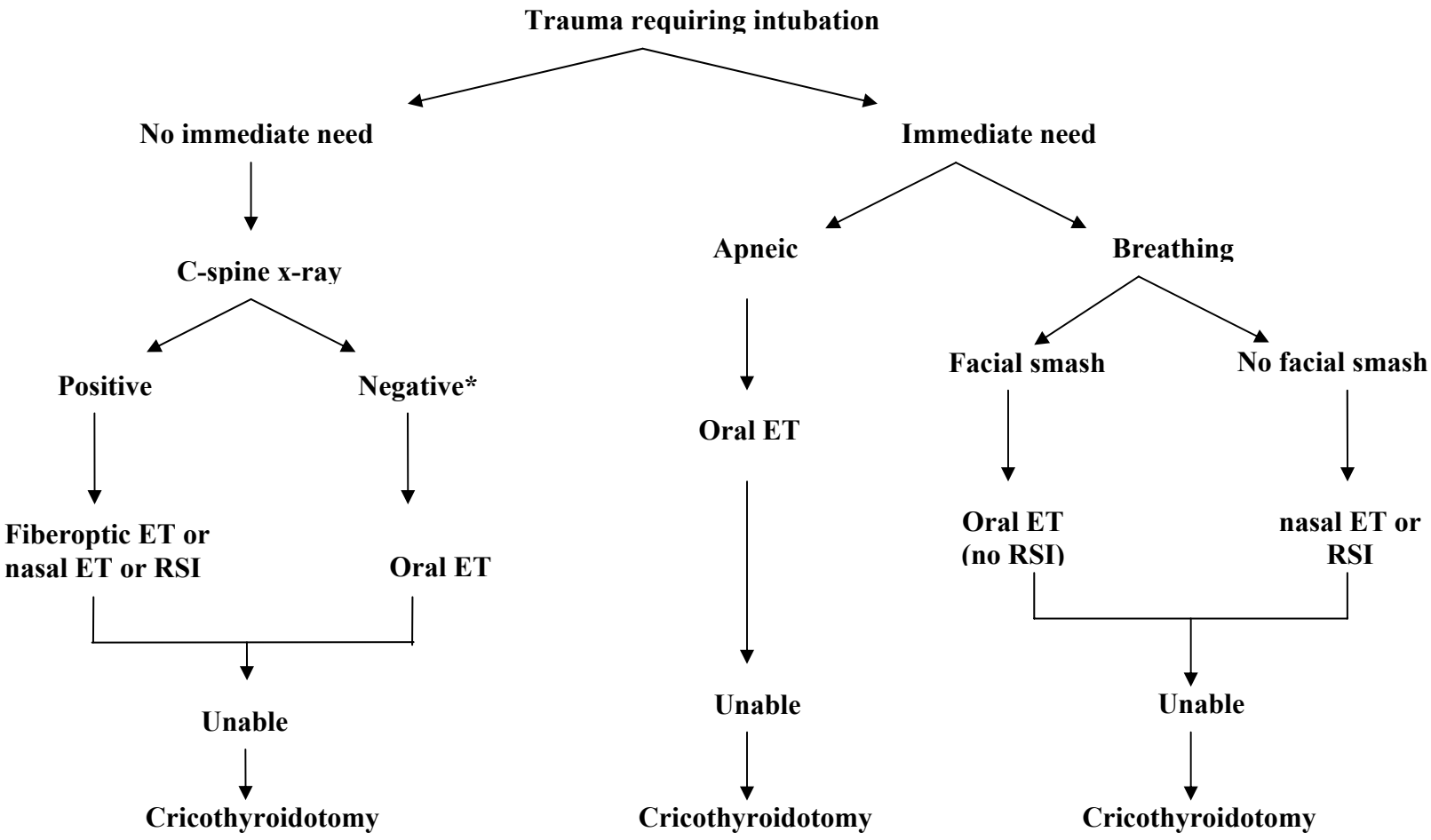
The accuracy of the pulse oximeter is unreliable when there is poor peripheral perfusion. This may be due to:

- Vasoconstriction.
- Hypotension.
- Blood pressure cuff that is inflated above the sensor.
- Hypothermia.
- Severe anemia.
- Excessive patient movement.
- Use of nail polish.
- Intense ambient light.
- Hemoglobinopathies.

When analyzing pulse oximeter results, evaluate the initial readings. Does the pulse rate correspond to the electrocardiogram monitor? Is the Pulse oximeter wave of good quality?

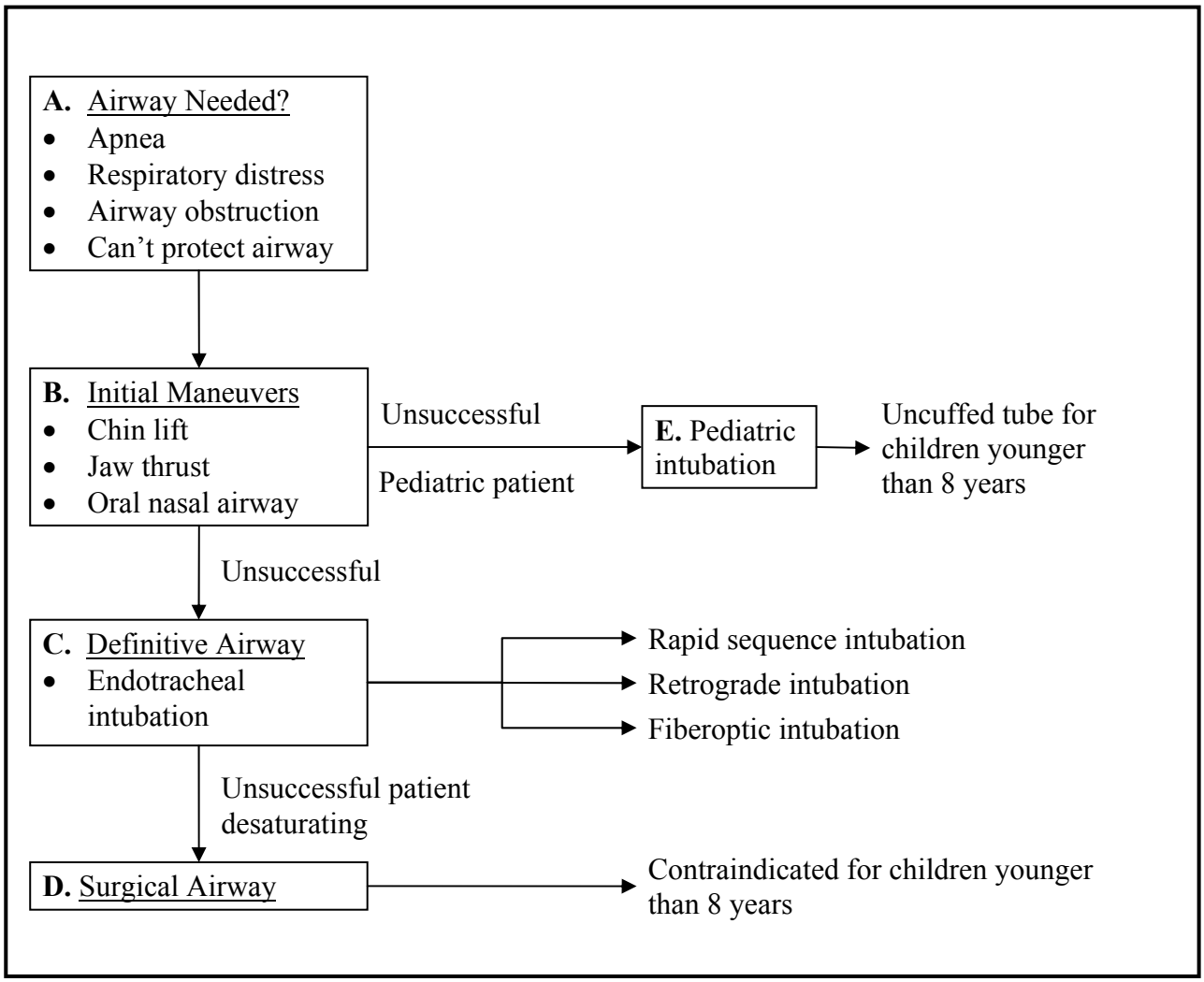
The relationship between partial pressure of oxygen in arterial blood (PaO_2 and SaO_2) is demonstrated by O_2 dissociation curve. The sigmoid shape of this curve indicates that this relationship is nonlinear. This is particularly important in the middle range, where small changes in PaO2 will cause large changes in saturation.

Figure 3.2: Intubation Algorithm



* Note: clearing the C-spine also requires clinical assessment (cannot rely on x-ray alone)
ET= Endotracheal intubation **RSI**= Rapid sequence intubation

Figure 3.3: Management of a Difficult Airway



Chapter 4: Choking and Foreign Body Airway Obstruction (FBAO)

GUIDELINE OBJECTIVES:

Diagnose and manage the following:

- Partial airway obstruction in a conscious adult.
- Complete airway obstruction in an un-conscious adult.
- Partial airway obstruction in a conscious child.
- Complete airway obstruction in an un-conscious child.
- Partial airway obstruction in a conscious infant.
- Complete airway obstruction in an un-conscious infant.

1. INTRODUCTION

Choking is the physiological response to a sudden airway obstruction. Obstruction may be partial or complete. Foreign body airway obstruction (FBAO) causes asphyxia. Asphyxia is an acutely occurring terrifying condition, with the victim often unable to explain what is happening to them. The victim will have rapid loss of consciousness/death if first aid is not undertaken quickly and successfully. Immediate recognition and response is important.

2. CLASSIFICATION

For adults and children chocking can be categorized into:

A. Partial Airway Occlusion:

Victim can still move some air to and from the lungs. The victim may make wheezing sounds or cough forcefully.

B. Complete Airway Occlusion while Conscious:

The victim cannot cough, speak, or breathe.

C. Complete Airway Occlusion with Loss of Consciousness:

No movement of air to and from the lungs with loss of oxygenation to the brain resulting in loss of consciousness.

3. CLINICAL FEATURES

3.1. History

Choking due to inhalation of a foreign body usually occurs while eating. Inhalation of a foreign object can happen while having a formal meal, eating a snack, or chewing gum. Beware of the so-called 'cafe coronary'.

3.2. Diagnosis

Consider the diagnosis when a victim collapses during or shortly after a meal or snack. In a chocking situation, events unfold almost instantaneously:

- The victim may clutch his neck, appear extremely distressed, and may become cyanosed.
- The victim may struggle to cough or breathe.
- Ask the victim directly, "Are you choking?" Speech is difficult or impossible, and so, the victim may utter, "Yes" in a muffled voice or simply nod his/her head.
- If the event has not been witnessed, the rescuer may simply find the victim unconscious without obvious cause.

3.3. Examination

Assess the severity of the situation:

- Is the victim able to speak, cough or breathe?
- The victim's breath may sound wheezy.
- There may be silent attempts at coughing.
- There may be loss of consciousness.

3.4. Differential Diagnosis

Rapid evaluation is key. Swiftly consider other conditions that may cause sudden respiratory distress, cyanosis, or loss of consciousness such as:

- Anaphylaxis.
- Syncope.
- Myocardial infarct.
- Seizure.

4. MANAGEMENT

4.1. Adults

Action depends upon the severity of the situation and the adequacy of the cough (refer to figure 4.1).

A. Partial Airway Occlusion:

Encourage the victim to continue coughing, more dramatic action is unnecessary.

B. Complete Airway Occlusion while Conscious:

Abdominal thrusts should be applied in rapid sequence until the obstruction is relieved (Heimlich Maneuver) refer to the box below. If this is not effective, chest thrusts can also be used. Chest thrusts can also be used in obese victims or victims in late pregnancy. Abdominal thrusts should not be used in infants under one year of age.

C. Complete Airway Occlusion with Loss of Consciousness:

If a victim becomes unresponsive he should be lowered to the ground, and initiate cardiopulmonary resuscitation (CPR). When the airway is opened during CPR, the rescuer should look into the victim's mouth for an object causing obstruction, and remove it, if it is evident.

The Heimlich Maneuver

This is an emergency technique for dislodging obstructing bodies in a choking victim's airway.

In a conscious adult/child over 1 yeas:

- Stand behind the victim; wrap your arms around the victim's waist, mid-way between the umbilicus and xiphoid process.
- Use one hand to make a fist, put the thumb of the fisted hand against the abdomen. The other hand grasps over the fist.
- The hands press up and into the abdomen in a sharp movement. Avoid squeezing the ribcage.
- Repeat up to five times.

With an unconscious adult:

- Lay the victim on his back.
- Begin CPR chest thrusts.

When the airway is opened during CPR, the rescuer should look into the mouth for the object causing obstruction, and remove it if it is evident.

4.2. Children (>1 year to puberty)

Airway obstruction is suggested by the sudden onset of respiratory distress with coughing, gagging, or stridor. Similar signs and symptoms will occur with other causes of airway obstruction, such as, laryngitis or epiglottitis. Suspect a foreign body if (refer to figure 4.2):

- There is very sudden onset.
- There are no other features of illness.
- The child may have been eating or playing with small items.

A. Partial Airway Occlusion:

With partial airway occlusions there is cough, crying, or reply to questions. The child is able to take deep breaths before making a loud cough and is fully responsive. A spontaneous cough is likely to be more effective and safer than any maneuver a rescuer might perform. With an inadequate cough response:

- The child cannot speak.
- The cough is quiet.
- The child may be cyanosed.
- There is a decreasing level of consciousness.
- If the situation is deteriorating, shout for urgent help.

B. Complete Airway Occlusion while Conscious:

- The Heimlich maneuver may be used in children greater than one year.
- The rescuer may need to stand or kneel depending on the height of the child.

C. Complete Airway Occlusion with Loss of Consciousness:

If the child victim becomes unconscious, place the child on a flat, firm surface. Send someone for help and begin CPR for pediatric basic life support.

4.3. Infants (<1 year)

A. Partial Airway Occlusion:

Support the child in trying to cough or dislodge the item.

B. Complete Airway Occlusion while Conscious:

- In a seated position, support the infant in a head-down, prone position.
- Support the head by placing the thumb of one hand at the angle of the lower jaw, and one or two fingers from the same hand at the same point on the other side of the jaw. Do not compress the soft tissues under the jaw, as this will aggravate the airway obstruction.
- After each blow assess to see if the foreign body has been dislodged, if not, repeat the maneuver up to five times.
- After five unsuccessful back blows, perform chest thrusts (do not use the Heimlich maneuver): turn the infant into a head downwards supine position by placing your free arm along the infant's back and encircling the occiput with your hand. Support the infant down your arm, which is placed down (or across) your thigh. Identify the landmark for chest compression, which is just below the nipple line. Deliver five chest thrusts. These are similar to chest compressions for CPR, but sharper in nature and delivered at a slower rate.

C. Complete Airway Occlusion with Loss of Consciousness:

- Place the infant in a neutral position and institute CPR.
- Compress at a rate of 100 compressions per minute utilizing two-to-three fingers on the center of the chest just below the nipple line. Compressing at a depth of 2-to-3 cm.
- Continue to visualize for the foreign aspirate. Lift the jaw and grasp the tongue and lower jaw between your thumb and fingers.
- If foreign object is seen, the rescuer should hook/sweep the object using their smallest finger.

5. MONITORING

If the victim is breathing and has a pulse but continues to be unconscious, place the victim in a recovery position, while angling the head towards the ground. The recovery position allows the victim to maintain an open airway for breathing.

5.1. Complications

- Bronchiectasis (lung abscess).
- Atelectasis.
- Gastric or splenic rupture.
- Hypoxic brain injury or death.

Figure 4.1: Adult Choking Management

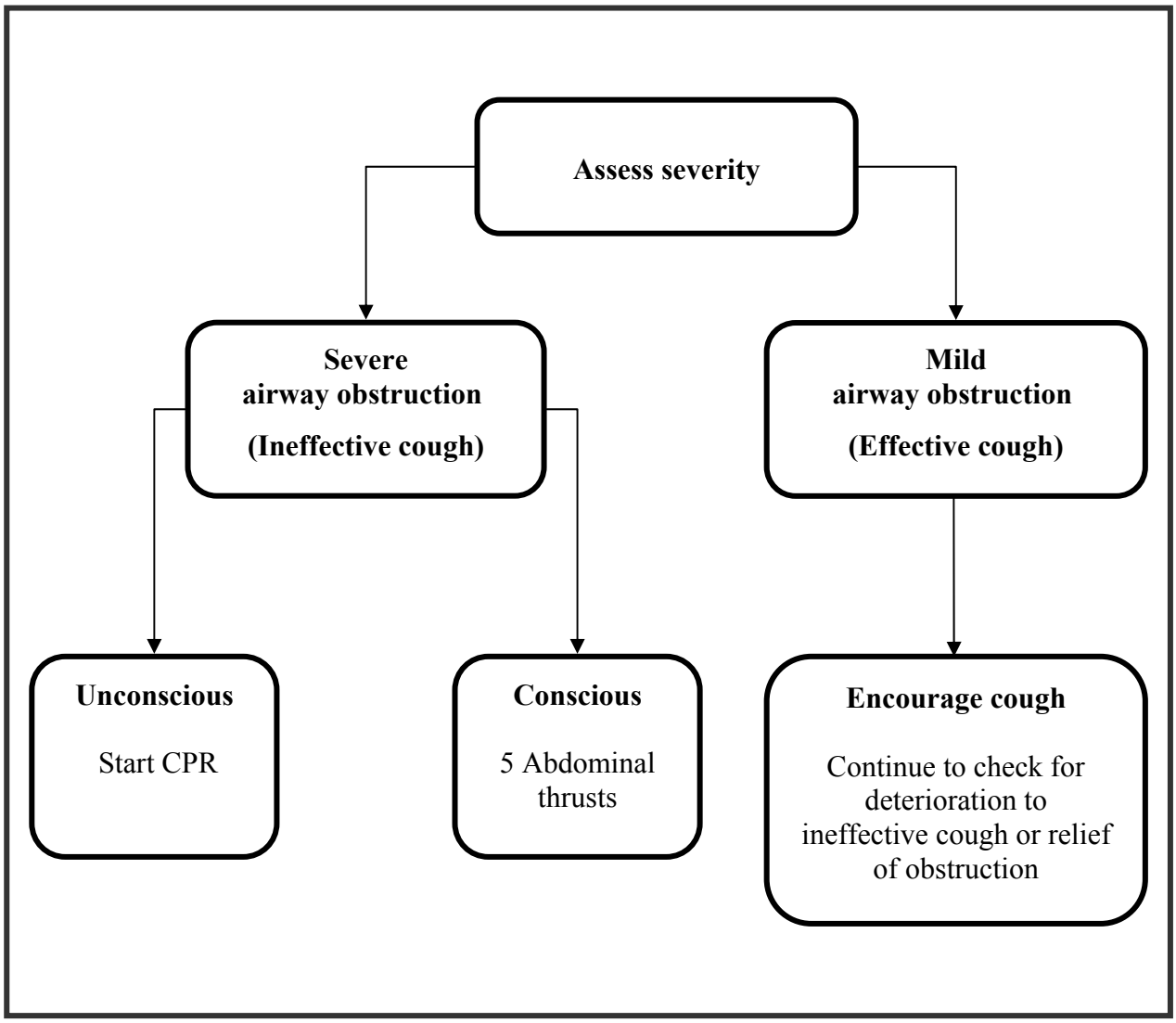
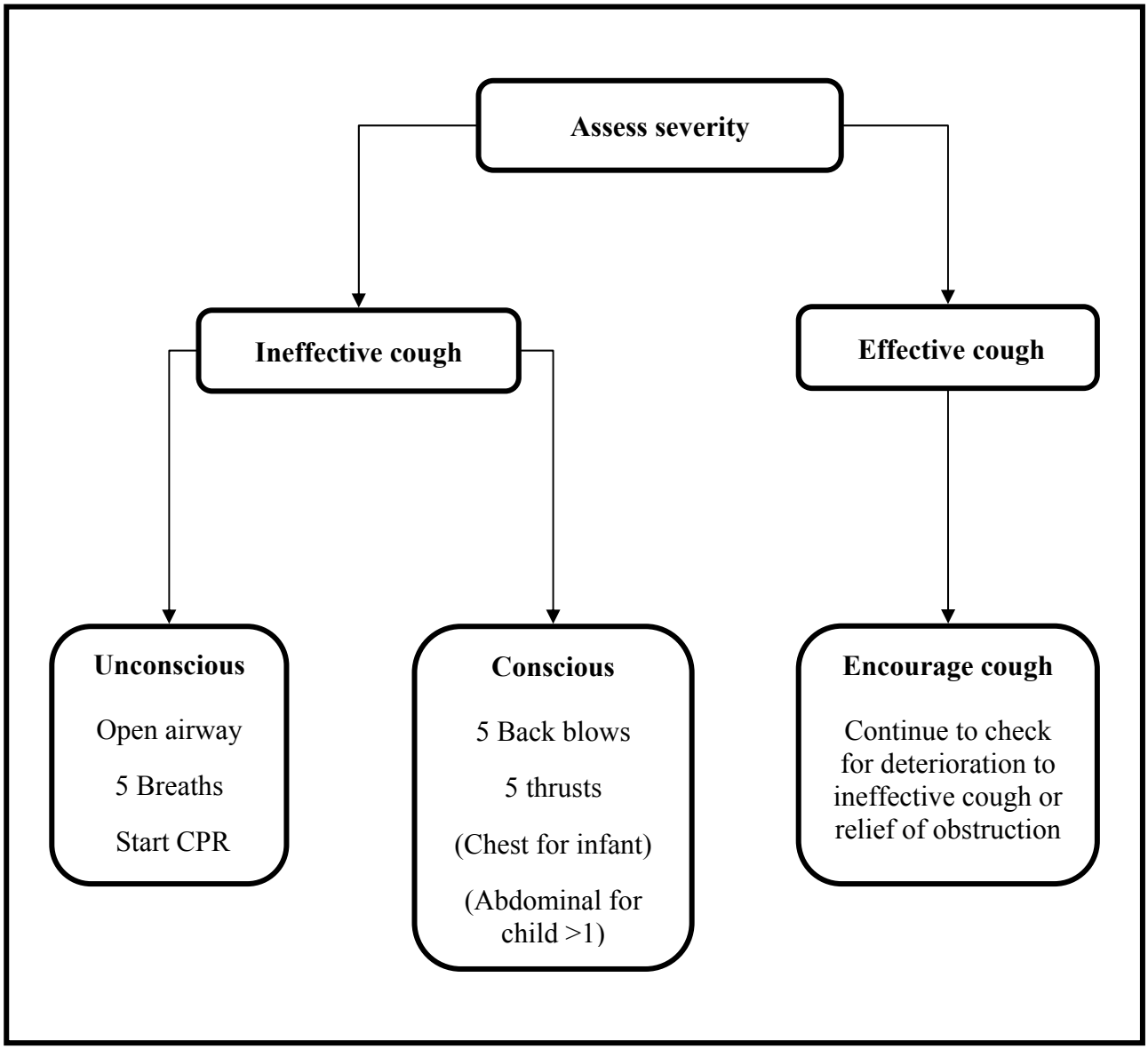


Figure 4.2: Pediatric / Infant Choking Treatment



Chapter 5: Patient Transportation

GUIDELINE OBJECTIVES:

- Define patient transfer.
- Recognize different modalities of patient transfer.
- Understand stabilization of the patient before, during, and after transport.
- Understand communication as key to the successful transfer of a patient.
- Appreciate the importance of the interdisciplinary approach in patient transfer.

1. INTRODUCTION

Transfer is inherently dangerous for the patient. There are well-recognized physiological responses to transfer, and these should be anticipated. Understand that all patients will require thorough assessment, resuscitation, and monitoring to ensure their airway is protected, respiratory function is supported, and that their cardiovascular status is stable. Because of the adverse transport environmental conditions, all interventions must be completed prior to departure. If interventions are necessary during transport, the safest option is to stop the ambulance before carrying out necessary interventions. Checklists for equipment and physiological status prior to transfer should be followed. The ultimate aim is to move a stable patient in a timely, but unhurried fashion, to the appropriate referral center.

1.1. Before Patient Transfer

- Indications, procedures, and communications are established.
- Obtain acceptance of the receiving hospital.
- Ambulances should be equipped with resuscitation devices.
- Follow checklists for equipment and physiological status prior to transfer.
- The airway is clear and secure.
- The spine is secure.
- Breathing is normal or ventilation is symmetrical.
- Two intravenous lines are infusing adequately.
- Assess the level of consciousness.
- All monitors are functioning.
- Drugs and equipment are accessible.

1.2. During Patient Transfer

- Ensure stabilization before and during transfer.
- The appropriate level of staff is accompanying the patient.
- Medical records are provided to the receiving hospital.
- Patient’s position is adequately observed.
- Perform ongoing resuscitation procedures as needed.
- Notified the receiving department before transport.

- Properly transfer responsibility for patient management to receiving department.

A. Packaging and Cannulation:

Two principles underpin transportation. First, do no further harm. Secondly, anticipate disaster with every transfer.

- All movement subjects the injured patient to energy changes, which are inherently harmful. From start to finish the journey should be as smooth as possible.
- Most patients are transported supine, usually on a long spine board or a Vacumat mattress. This allows patients to be tipped or turned if they start to vomit. The risk of vomiting can be reduced by inserting a nasogastric tube, but this is not a substitute for having a suction device.
- Pregnant patients should be transported with the uterus slightly displaced to the left. This prevents supine hypotension syndrome.
- Confused patients must be adequately restrained once cerebral hypoxia has been eliminated. If cerebral hypoxia cannot be ruled-out, anesthesia with paralysis (muscle relaxant) may be indicated.
- The airway is most effectively protected with a well secured, cuffed tracheal tube. Consider inserting a tracheal tube in an unconscious patient who is tolerating an oropharyngeal airway or has inhalation burns; particularly if transport will take a prolonged period of time. When there is the possibility of an unstable cervical spine, nasal intubation or surgical airway creation may be utilized.
- A high inspired oxygen concentration may need to be maintained. If a portable mechanical ventilator is used, remember that many machines use the same oxygen source to drive the machine and supply the patient with oxygen.
- The patient at risk of hypovolemic shock requires two secured functioning large bore intravenous cannulaes. If access has been inserted in the antecubital fossa, the use of an arm board ensures patency. Central venous access should not be attempted, except under controlled conditions, and by trained personnel.

B. Essential Information Given En Route to the Receiving Hospital:

- The number of patients.
- Age and sex of the patients.
- Vital signs at the scene.
- Initial findings of assessment.
- Procedures at the scene.
- Response to treatment given.
- Estimated time of arrival.

1.3. Concluding Transfer

A. Communication and Handover:

- Staff in the receiving hospital must be informed of the patient's impending arrival and condition. The ambulance staff or the accompanying medical attendant may report to the

receiving unit. Information received alerts the emergency department to prepare the resuscitation room and call the resuscitation team.

- On arrival, details about the accident, the patient's initial condition, treatment given, and response, must be communicated to the appropriate staff members.
- It is particularly important to communicate problems that occurred during transportation, highlighting improvements or deteriorations in vital signs during resuscitation.
- Pre-hospital care personnel must wait to ensure that the team leader has all the necessary information.

2. CLASSIFICATION OF TRANSPORT

2.1. Transportation to a Hospital

- Timing and type of transportation are governed by the patient's condition, and response to resuscitation.
- Although speed of transfer is important, take the time to secure the patient’s airway, institute effective ventilation, control external bleeding, gain intravenous access and splint the spine and limbs.
- If it is impossible to stabilize the patient at the scene without complex interventions, the patient must be transported to the closest appropriate hospital, utilizing the fastest transport, with continuous resuscitation measures.
- The most common cause of instability is uncontrollable hemorrhage.

2.2. Transfer between Hospitals

During the course of treatment, patients may have to be transferred to another hospital.

- Arrangements must be made between doctors initiating and receiving the transfer.
- Indications, procedures, and communication must be established for transfer.
- Ensure stabilization before and during transfer.
- A decision about the minimal level and specialty of accompanying personnel must be made before transfer. Unstable, ventilated patients require high levels of medical and anesthetic support. The patient must be fully stabilized.
- Make provisions for all requirements during transfer. This includes adequate amounts of intravenous fluid, blood, supplementary drugs, kits for re-intubation, and replacement of lines.
- Humidification should be provided on long journeys to prevent secretions blocking ventilation tubes.
- The nasogastric or orogastric tube should be suctioned regularly.
- The urinary catheter monitored for output.
- Take the patient's records, and the results of all investigations.
- Whenever possible, maintain communication with the receiving hospital.
- On arrival, the patient should be presented to the receiving doctor with their history, treatment, and investigation results. Problems encountered en route and actions taken are also important. All documentation should be given to receiving staff and transport equipment retrieved before leaving.

2.3. Transportation within the Hospital

- The patient must be reasonably stabilized. Intervention should not be delayed, trying to reach normal vital signs in patient requiring advanced intervention due to their condition (i.e. severe ongoing bleeding).
- Suspected spinal injury patient's position should be intensely monitored during patient transportation. An adequate number of personnel help is required.
- Ongoing resuscitation procedures must be maintained during transportation.

3. MONITORING

- Ideally the patient should be positioned with his/her head next to the attendant's seat.
- There must be room to gain access to the airway (to allow tracheal intubation).
- The attendant should be able to identify and relieve a tension pneumothorax in a ventilated patient.
- Additional intravenous lines may be established.
- In cases of cardiac arrest, cardiopulmonary resuscitation is difficult for a single operator and is also difficult when done by two attendants in a speeding vehicle. Under such circumstances, it is advisable to stop the ambulance and use the help of a trained driver.
- During transportation, one of the most important tasks of the medical attendant is the continued monitoring and assessment of the patient. Continuously record the heart rate, respiratory rate, blood pressure, oximetry and level of consciousness.
- The patient must be accompanied by an appropriate level of staff, according to his/her condition.
- Patient's management must be properly delivered to the receiving department.

Chapter 6: Resuscitation Room, Trauma Team and Crash Cart

GUIDELINE OBJECTIVES:

- Ensure proper organization of the crash cart contents and timely replacement of crash cart consumable items.
- Operate and maintain all crash cart equipment, including implementation of proper preventive maintenance procedures and operation of all crash cart life-saving equipment.
- Ensure proper organization of the resuscitation room equipment and supplies. Ensure timely replacement of resuscitation room consumable items.
- Operate and maintain all resuscitation room equipment properly, including preventive maintenance procedures and operation of all resuscitation room life-saving equipment.
- Describe a trauma team and define roles of each member.

1. INTRODUCTION

The crash cart is a mobile equipment, supply, and medication unit used in cardiopulmonary resuscitation (CPR) and emergency situations. Emergency department doctors and nurses must be knowledgeable of the crash cart's contents, organization, and maintenance. Emergency department (ED) team members must be trained in CPR and the proper use of life saving equipment. The charge nurse must review the crash cart contents, ensure all equipment is in place, and indicate crash cart readiness by signing the shift check list.

2. CRASH CART CONTENTS

The crash cart is divided into three levels and two sides (see figure 6.1).

2.1. The Upper Level (Top Surface)

The top surface holds large sized equipment (defibrillator, electrocardiogram (ECG) monitor, and suction devices) and Ambu bag.

2.2. Drawers

A. The First Drawer:

Contains all drugs used in cardiopulmonary resuscitation or emergency situations (see table 6.1).

B. The Second Drawer:

Contains intravenous (IV) items (i.e., infusion sets, syringes, bandages, adhesive tapes, IV cannulae), ECG monitor leads, and tongue depressors.

C. The Third Drawer:

Contains airway management items and endotracheal tube intubation (ET) equipment, including:

- Oral and nasopharyngeal airways (different sizes).
- Endotracheal tubes (different sizes).

- Laryngoscopes with different blade sizes (functioning batteries and bulbs).
- Magill forceps.
- KY jelly (lubricant gel).

D. The Fourth Drawer:

Contains emergency instrument set packs. All instrument set packs must be sterile:

- Central venous pressure (CVP) pack.
- Venous cut down pack.
- Intubation pack.

E. The Fifth Drawer:

Contains all emergency tubes and catheters (different sizes must be available):

- Suction catheters.
- Nasogastric tubes.
- Urinary catheters.
- Chest tubes.

2.3. The Lower Level

Contains emergency IV solutions, different sized sphygmomanometer cuffs, and procedure trays.

2.4. Sides

A cardiopulmonary resuscitation board and a portable oxygen cylinder are attached to opposite sides of the crash cart. The crash cart wheels must be in good working condition.

3. RESUSCITATION ROOM

The resuscitation room is designed to perform resuscitation procedures for all life threatening illnesses or injuries. The physical structure (including adequate space, good illumination, efficient ventilation, high number of electric sockets, and a basin facility) must allow for large numbers of staff and equipment to easily circulate during emergency situations.

3.1. Furniture and Equipment

Furniture must allow for resuscitation functions without obstructing patient or staff circulation and performance of resuscitation functions.

Life saving equipment must be available in immediate functioning condition. This includes different emergency procedure instrument packs, such as:

- Intubation set.
- Venous cutdown pack.
- Chest tube insertion pack.
- Central venous pressure (CVP) insertion pack.
- Stitching pack.
- Pericardio-centhesis pack.

3.2. Medications

The resuscitation room must have all life-saving medications, in roper amounts, and within valid dates, that would be needed during a resuscitation event. During a resuscitation event, the nurse should not exit the resuscitation room to retrieve medications. All medications in the resuscitation room should be routinely checked for expiration .

Equipment and medications must be checked every shift to ensure that the resuscitation room is ready to receive a critical patient at any time. Requirements for mass accidents must be considered.

4. TRAUMA TEAM

The advanced trauma life support (ATLS) is designed so that single doctor can safely look after a multiply injured patient. Tasks are performed in sequence. This 'vertical organization' ATLS design) is the least efficient method of proceeding. When more than one staff member is available, a team approach is usually employed. This 'horizontal organization', or team approach, has been shown to lead to significant reductions in resuscitation times and improved outcomes .

The trauma team is comprised of doctors, nurses, operating department assistants, radiographers, and other support personnel. In large facilities trauma teams have no other commitments, their daily task is to receive and treat trauma patients. Obviously, this is a very expensive arrangement, and most hospitals cannot afford this level of care.

Every hospital has to form a trauma team within the capacity of its available resources. The trauma team should closely resemble the below description.

The core trauma team is a group of professionals that receives and treats trauma patients. This includes a team leader, as needed ancillary staff, and varieties of imaging and surgical specialties.

Trauma members needed for one trauma patient include:

- Team leader.
- Anaesthetist.
- Anaesthetic assistant.
- General surgeon.
- Orthopaedic surgeon.
- Emergency room physician.
- Two nurses (three if no anaesthetic assistant).
- Radiographer.
- Scribe (nurse or doctor).
- Additional staff mobilized to provide ancillary services.
- Porters - to run samples to the lab, collect blood etc.
- Haematologist and biochemist to receive and process samples.
- Blood bank.
- Other staff, while not involved in every trauma call, they may need to be available to the trauma team immediately:

- Neurosurgeon.
- Thoracic Surgeon.
- Plastic surgeon.
- Radiologist.
- Certain areas that need early notification of the trauma victim.
- Computerized tomography (CT) scanner.
- Intensive care.
- Surgical theatres.

The core trauma team comprises ten people working around a single patient. It is vital that everyone knows their place and tasks, and that every member has the skills, equipment and support to accomplish them. The trauma room should be quiet, so that the voice of the team leader can be heard, and assessments from team members can be relayed back to him/her. Vital signs should be reported out loud every five minutes. With practice and exposure the trauma team becomes an efficient machine.

4.1. Trauma Team Tasks.

A. The Team Leader:

The team leader has limited physical contact with the patient; his/her main role is to orchestrate the trauma patient's care.

Responsibilities of the team leader:

- Obtain history.
- Direct team member actions.
- Establish investigation and management priorities.
- Order or authorize investigations and procedures.
- Receive and interpret all investigation results.
- Order fluid or blood administration.
- Supervise spinal maneuvers.
- Consult with specialties.
- Decide on patient discharge location (admission, home, or other facility).
- Talk with relatives.
- Document in patient records.
- Record audit information.
- Dismiss and debrief team members.
- Educate trauma team.

The trauma team leader should be the most experienced team member present before the patient arrives to the hospital. The leader's role should not be superseded by late arriving trauma members, or by passing senior staff. This avoids team confusion.

B. Anesthetist:

The anesthetist has a central role in the trauma team. Responsibilities include :

- Airway control.
- Ventilation.
- Vital sign monitoring.
- Monitoring of fluid and drug administration.
- Analgesia.
- Administering anesthesia for surgical procedures.

C. General Surgeon:

The general surgeon focuses on assessment of the thorax, abdomen, and head (if no neurosurgeon is on the core trauma team). Responsibilities include:

- Primary survey.
- Assessment of thorax and abdomen, head, and facial injuries, with a log roll.
- Thoracostomy or thoracotomy.
- Diagnostic peritoneal lavage.
- Urinary catheter.

D. Orthopedic Surgeon:

The orthopedic surgeon should focus on the care of large/small bone and limb injuries. Responsibilities include:

- Intravenous access.
- Assessment of spine and pelvis.
- Application of external fixator.
- Assessment of limb injury.
- Dressing of wounds and stabilization of fractures.
- Urinary catheter.

E. Emergency Department Physician:

The ED physician is a general specialist that focuses on the overall care of the trauma patient. Responsibilities include :

- Intravenous access.
- Venous and arterial blood samples.
- Thoracostomy.
- Urinary catheter.
- Assist with diagnostic peritoneal lavage.

Some overlap is necessary between the general surgeon, orthopedic surgeon, and emergency department physician. This ensures that tasks continue simultaneously, that no time is lost, and no hands are wasted .

F. Nursing Staff:

If the trauma team lacks an anesthetic assistant, one nurse should be solely dedicated to the anesthetist. Otherwise, one nurse should assist each hands-on surgeon or ED physician. Nurses should not leave the resuscitation room to fetch equipment or deliver lab samples.

Ancillary staff should be outside the main resuscitation area to deliver equipment and labs.

G. Radiographer:

Immediately the radiographer should begin taking a trauma series of X-rays, starting with the cervical spine, chest, and pelvis, unless directed otherwise by the team leader. Once processed, other views may be required indicated by the patient's injuries. The radiographer is the CT scanning department liaison.

H. Scribe:

The scribe is responsible for recording all events and interventions during the trauma call. A separate individual, either doctor or nurse, should fulfill this roll. They should be situated near the team leader so that all information passing through the leader is then passed and recorded by the scribe. Records must include :

- Time of arrival.
- Mechanism of injury.
- Personnel present during the call.
- Physical findings.
- Vital signs.
- Urine output.
- Glasgow coma scale.
- X-rays and investigation results.
- Fluids administered.
- Drugs administered.
- Previous medical history.
- Summary of injuries.
- Disposal of patient.

The trauma team's goal is to provide a safe and efficient evaluation of the patient. This includes identifying and initial definitive management of all trauma injuries. The golden hour begins at the time of injury. Therefore, trauma teams have approximately 30 minutes to stabilize the patient. Every team member should work towards the goal of quick and appropriate stabilization of the trauma patient.

Figure 6.1: Crash Cart Diagram

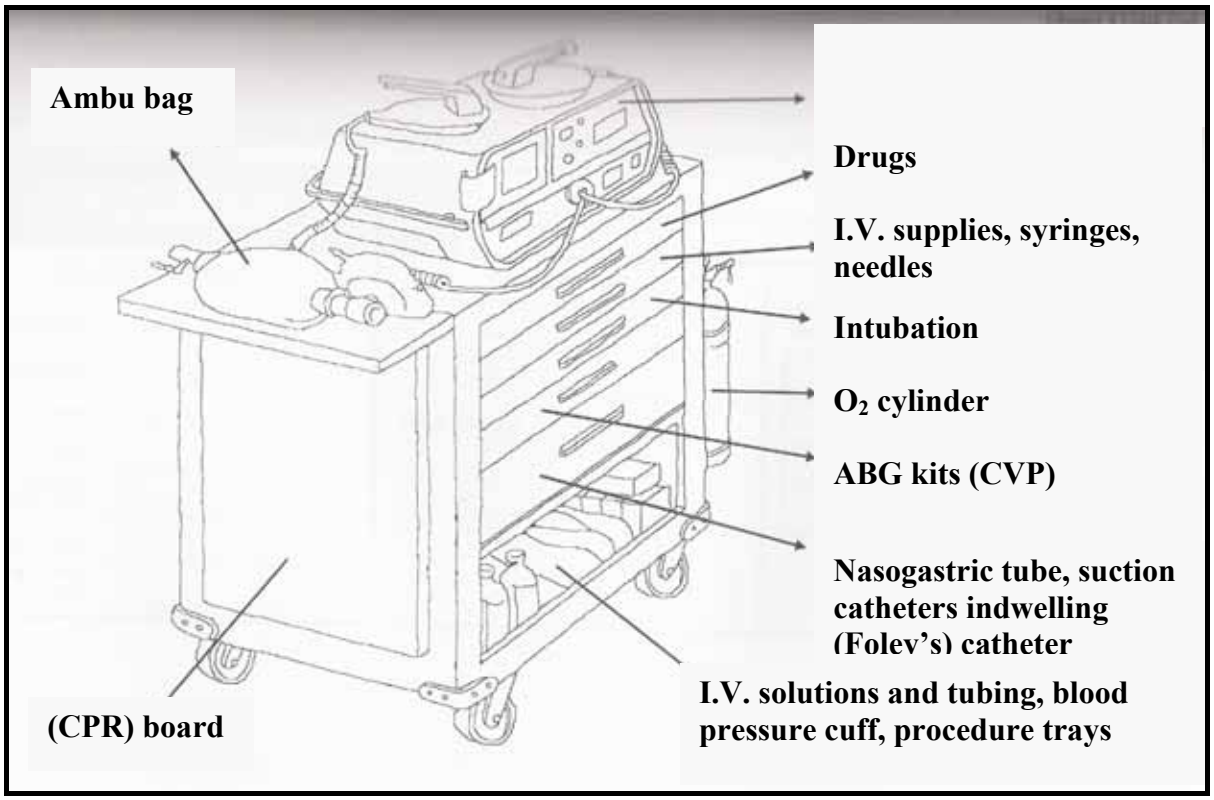


Table 6.1: Crash Cart Stock Drug Items

No.	Drug	Amount
1	Adrenaline	20 Ampoules
2	Atropine	20 Ampoules
3	Sodium Bicarbonate	40 Ampoules
4	Xylocaine	10 vials
5	Calcium Chloride	10 Ampoules
6	Dopamine	3 Ampoules
7	Dobutamine	3 Ampoules
8	Nitroglycerine	3 Ampoules
9	Lanoxin	5 Ampoules
10	Lasix	20 Ampoules
11	Aminophyline	20 Ampoules
12	Dextrose 50%	10 bottles
13	Magnesium Sulfate	20 Ampoules

Chapter 7: Infection Control

GUIDELINE OBJECTIVES:

- Understand the importance of utilizing protective tools such as gloves, gowns, and/or masks.
- Practice good hand washing techniques.
- Institute precautions during patient transfer.
- Follow disposal policy for contaminated and uncontaminated materials.
- Adhere to cleaning policy of the emergency department (ED).

1. INTRODUCTION

The purpose of infection control is to protect ED personnel, other employees, patients, and visitors from hospital acquired infections. Hospital acquired (nosocomial) infections are defined as infections which are a result of treatment in a hospital or a healthcare service unit, but not secondary to the patient's original condition. Infections are considered nosocomial if they first appear 48 hours or more after hospital admission or within 30 days after discharge.

2. MANAGEMENT

Interventions to minimize the risk of hospital acquired infections to patients and personnel while in the ED.

2.1. Hygiene

- Practice good hand washing techniques.
- Wash hands thoroughly before and after touching wound dressings.
- Utilize surgical aseptic techniques when performing all procedures.
- Clean environment daily, and as warranted, utilizing hospital approved chlorine base disinfectant.
- Wipe up blood spills, bodily fluids, or secretions promptly, using chlorine base disinfectant.
- Clean examination tables between patients and remove disposable sheets. Place clean sheets on examination tables between patients.

2.2. Education

- Recognize the signs and symptoms of communicable diseases.
- Prevent multiple exposures of infectious agents to patients and personnel, designated examination rooms should be used for suspected infectious cases.
- Educate patients about prevention and control measures.
- Notify infection control team of all communicable diseases.

2.3. Standard Precautions

- Institute individual case-based standard precautions by wearing protective equipment such as gloves, gowns, and/or masks.

- When transporting patients with infectious diseases:
  - Wear gloves, masks, gown, and protective glasses as necessary.
  - Have patient wear masks for infectious agents spread by droplet contact.
  - Cover draining/open wounds.
- Discharge patients from the department, either to home or other facilities, where appropriate isolation techniques can be instituted.

2.4. Disposal

Use correctly colored bags for waste disposal:

A. Blue/Black Plastic Bags:

Any disposable material, which is not contaminated with blood and/or bodily fluids.

Examples:

- Intravenous (IV) tubing/bag without visible blood.
- Syringes not used for parentral therapy.
- Ventilation tubing without visually present blood/bodily fluids.
- Empty plastic medication bottles and bags.
- Plates and cups.
- Diapers and underpads (not with feces).

B. Yellow/Red Plastic Bags:

Any disposable materials contaminated with blood and/or bodily fluids.

Examples:

- All bloody (IV) sets.
- Worn protective equipment (masks/gloves/gowns).
- Urinary catheters and bags.
- Used drainage bags.
- All soiled dressings.
- Disposable suction and endotracheal tubes.

C. Central Sterile Sanitizing Department (CSSD):

- Soiled equipment should be placed into special transparent central sterile sanitizing department (CSSD) bag.
- Ensure all invasive equipments are processed by CSSD.

D. Linen:

Soiled linen should be deposited into a laundry bag.

E. Sharps Materials:

Dispose used syringes with needles, broken glass, glass medication vials, and IV needles into puncture resistant containers.

Part 2: Surgical Emergencies

Chapter 8: Multiple Trauma

GUIDELINE OBJECTIVES:

- Perform initial assessment and resuscitation of multiple trauma patients utilizing the primary survey to identify life-threatening conditions.
- Perform a secondary survey to evaluate other injuries and patient status.
- Monitor for signs of patient deterioration and manage the trauma patient.
- Prioritize the management of the multiple trauma patient.
- Recognize and manage special categories of trauma patients (pregnant, pediatric, and geriatric).

1. INTRODUCTION

Effective management of multiple trauma patients requires a team approach. Many of the steps that appear in this text are written sequentially, but in fact they are performed simultaneously. For a team to function in harmony, predetermined plans and roles must be outlined. It is advisable that all emergency department (ED) staff become familiar with the different roles, as roles are frequently interchangeable (see figure 8.1).

2. MANAGEMENT PHASES

- Pre-hospital preparation.
- Triage.
- Primary survey.
- Secondary survey.
- Reassessment.

2.1. Preparation

- Pre-hospital Information:
  - Number and ages of victims.
  - Extent and mechanism of injuries.
  - Vital signs.
  - Estimated time of arrival to ED.
- Preparation of the ED for major trauma:
  - Alert ED and ancillary personnel.
  - Clear beds for the expected number of victims.
  - Prepare equipments and supplies.
- Safety and precautions of trauma personnel:
  - Wearing protective clothing is important. Minimum is latex glove, plastic aprons, and eye protection.

2.2. Triage

- Triage is needed in:
  - Multiple causalities.
  - Mass causalities.
- During the initial contact with the patient, the clinician can gain an overall clinical picture of the patient and his/her needs (critical immediate life support vs. monitoring and minor interventions).

2.3. Primary Survey

The purpose of the primary survey is to identify and treat life threatening conditions in order of priority. The primary survey should focus on:

(ABCDE):

A. Airway

B. Breathing

C. Circulations

D. Disability

E. Exposure

See figure 8.2.

A. Airway:

- Open the airway with the chin lift & jaw thrust maneuver, while maintaining the cervical spine in a neutral position.
- Clear the airway from blood, vomitus, or foreign bodies using suction and/or magill forceps. Use the finger sweep technique for deeply comatose patients to remove a foreign body, if it is visible. If there is vomiting while cervical spine status is not assured, lower the head and use suction to clear the airway, rather than turning the patient's face to the side.
- Maintain a patent airway:
  - Unconscious patients with no gag reflex require an oral airway of suitable size.
  - Unconscious patients with a gag reflex will not tolerate an oral airway.
  - Unconscious patients with a partial gag reflex can tolerate nasopharyngeal airways (contraindicated in fracture basal skull fracture).
- Oral endotracheal intubation (ET), with in line stabilization of neck, should be performed by an experienced staff member for patients who require active airway management.
- Surgical airway creation should be performed if ET is impossible.
- Deliver 100% oxygen once the airway is cleared and secured.
- Rapidly examine the neck for air crepitus, tracheal position, and venous congestion.
- Cervical spine control is maintained by a secured semi-rigid collar, sand bags, and tape (or other similar method). Except in restless patients who can cause more damage to their cervical spine if it is fixed. In such instances, semi-rigid collars may be utilized.

- Airway obstruction = noisy labored breathing
  or no breathing.
- Assume cervical spine is injured until proved otherwise, keep the spine in a neutral position.

B. Breathing:

During the assessment of the trauma patient, it is vital that the clinician look, listen, and feel for breath and oxygen exchange. Consider the following:

- Is the patient breathing?
- Is the patient breathing adequately?
- Is immediate intervention like needle or tube thoracostomy urgently needed? Which side?

Rapid killers in chest trauma:

1. Airway obstruction (immediate and late).
2. Tension pneumothorax.
3. Cardiac tamponade.
4. Open chest wound.
5. Massive haemothorax.
6. Flail chest.

Ominous signs in chest trauma:

1. Cyanosis.
2. Labored breathing.
3. Respiratory rate (RR) < 6 or > 30 per/minute.
4. Trachea not central.
5. Congested neck veins.
6. Unequal chest movements.
7. Absent air entry unilateral.
8. Paradoxical breathing.

Management:

- Insert a large bore needle in the intercostal space at the midclavicular line, if tube thoracotomy cannot wait.
- Large >28 french chest tube is inserted if diagnosis of tension pneumothorax or massive haemothorax is made. The chest tube is inserted in the 5th intercostal space, in the anterior axillary line, for the lung that is not moving and has no air entry.
- Paradoxical breathing may not be evident initially, because of intercostals muscle spasm.

- Paradoxical breathing is evident when the patient becomes exhausted with the work of breathing.
- Oxygen administration should be greater than 8 liters/minute.
- Oxygen saturation is monitored by pulse oximeter.
- If spontaneous breathing is absent, or ineffective, start bag-valve-mask.
- Bag valve mask may increase an undiagnosed pneumothorax.

C. Circulation and Bleeding Control:

> **All patients with multiple traumas, who are hypotensive, are considered to be hypovolaemic until prove otherwise.**

Assessment of circulation:

- Level of consciousness: Agitation is common due to hypovolaemia, but other causes include hypoxia, head injury, drugs, and alcohol.
- Pulse: Hypovolaemia is manifested by a rapid, thready pulse, but associated severe head injury, spinal cord injuries, B-blockers, cardiac injury, hypoxia, or severe coldness can result in a slowed pulse.
- Capillary refill time at fingers tips and skin color are indicators of adequacy of the peripheral circulation.

Management:

- Two large IV cannulae (gage 14 or 16) (wide, short cannula flow better than long cannula).
- 10-20 ml of blood is drawn for blood cross-matching and laboratory work.
- Look and control any source of bleeding, the use of haemostats is time consuming.
- Two liters of ringer lactate (RL) or normal saline (NS) infused at the fastest rate possible.

D. Disability:

Patients with severe head injury who survive to reach the hospital may die from depressed level of consciousness with loss of airway control , aspiration, and respiratory arrest. An injured brain requires higher oxygen delivery.

Assessment:

- Decreased level of consciousness can be due to head injury or decreased cerebral oxygenation resulting from hypoxia or hypovolaemia.
- Level of consciousness is graded by the (AVUP) system in primary survey and by the glasgow coma scale (GCS) in secondary survey.

  (AVUP):

  A Alert
  V Reacts to voice
  U Unresponsive
  P Reacts to pain

Key Points:

- Blood preussure may remain normal in the initial stage of hypovolaemia, until volume loss exceeds 30 %.
- Quickly infuse 2-3 liters of lactated Rivger's solution.
- Oxygen administration is critical, start oxygen flow immediately.
- Guard against hypothermia by warming the intravenous fluid (IVF).
- Vasopressors, steroids, and bicarbonates have no role in the initial treatment of hypovolaemic shock.

E. Exposure:

Two sites (at least) must be exposed during the primary survey; the chest and the site of external bleeding. Avoid hypothermia by covering the patient with blankets or use an external warmer.

2.4. Primary Survey Interventions

Monitoring:

Electrocardiogram (ECG) monitor, automatic blood pressure (BP) recorder, and pulse oximeter are started.

Foley's catheter:

- Insert a urinary catheter unless contraindicated (contraindicated if urethral injury is suspected).
- Observe urine color and monitor hourly urine output. Urine should be clear and greater than 30 ml/h.

Gastric tube:

A nasogastric tube is inserted. If a fracture in the skull base is suspected, or severe faciomaxillary injury is present, insert orogastric tube. This gastric tube decompresses the stomach, *but care should be taken, because the patient may vomit during insertion trial.*

X-ray:

If patient is stable, chest anterior posterior (AP and lateral), and cervical x-rays are essential. AP x-rays of the pelvis are critical, especially when blood loss is suspected.

Focused Abdominal Ultrasound for Trauma (FAST & Doppler (DPL)):

- FAST is a focused abdominal ultrasound (U/S). It is an important diagnostic tool to be utilized for any severely injured trauma patient, as an adjunct to the assessment of the patient's circulatory system status. The FAST can identify the presence or absence of free intra-abdominal, thoracic, or pelvic hemorrhage, pneumothorax, pericardial effusion, or cardiac tamponade.
- Doppler (DPL) is rarely used due to the wide availability of U/S.
- One member of the medical team should take the responsibility to talk to family and friends about the patient.
- Consider the need for patient transfer, if his injuries require advanced care at another facility.

2.5. Secondary Survey

The secondary survey should be utilized for history taking and detailed evaluation/management of non life-threatening injuries (see figures 8.3 and 8.4).

A. History:

The pneumonic (AMPLE), is helpful in gaining full history from the patient, patient's relatives, or ambulance crew. This history allows for a general overall picture of the patient and his/her injuries (AMPLE):

A. Allergies
M. Medications
P. Past history
P. Last meal
E. Events of accident mechanism.

B. Physical Examination:

The secondary examination should be very detailed. The trauma team must perform a complete head to toe assessment, evaluating both the front and back, and all orifices of the patient.

Keep in mind that drugs, alcohol, and hypoglycemia can cause decrease altered level of consciousness.

C. Laboratory & Radiological Assessment:

During the secondary assessment, the patient is somewhat stabilized, allowing for further investigation into his/her injuries. The trauma team should utilize this time to perform further laboratory and radiological inquiries.

D. Management:

Once the patient is stable and the secondary survey is performed, the overall picture of the patient injuries evolves. Management plans should be formulated and the trauma team should decide if the patient should be transferred to another department or to a more advanced facility.

E. Monitoring:

The team leader must constantly re-evaluate the response to resuscitation:

- Is the patient improving, deteriorating, or unchanged since resuscitation started?
  - If the patient is not improving then airway, breathing, and circulation, and disability must be reassessed.
  - The patient's condition can change rapidly, repeated examination and constant monitoring are essential.
- What is the extent of the injuries and what are the priorities for treatment?
- Has an injury been missed? (e.g. the area between two injured regions in blunt trauma).
- Has analgesia been given? Victims of major trauma require pain relief.
- What is the patient's tetanus status and are antibiotics required?

- Are any further radiological investigations required? Is it safe to complete an x-ray examination?

X-rays should not delay resuscitation, they should only be performed if the patient is stable.

F. Documentation:

It is the responsibility of the trauma team to document all events, injuries, and management when time allows.

G. Definite Care and Transfer:

The trauma team's responsibility continues until the patient is transferred, and the patient's management is properly taken over by the definitive care team (transfer may be either inside or outside the initial receiving facility).

Head Trauma

GUIDELINE OBJECTIVES:

- Evaluate patients with head and brain injuries.
- Perform neurological examinations.
- Explain the importance of resuscitation in limiting secondary brain injuries.
- Determine the need for admission consultation or discharge.

1. INTRODUCTION

The aim of patient management is:

- To provide the best recovery conditions for already sustained brain damage.
- To prevent and treat any complication that can cause secondary brain damage (e.g. hypoxia and increased intracranial tension).

Raised intracranial pressure occurs in 70% of comatose severe head injury patients. It causes a drop in brain perfusion leading to ischemia.

The role of the emergency department team:

- Resuscitation, diagnosis, and recording.
- Detection and exclusion of other injuries.
- Request and interpret radiography and initial investigations.
- Decide admission or transfer.
- Contact neurosurgeons for serious cases.
- Ensure adequate arrangement for observation and transfer.
- Short duration observation of patients with minor injuries.

2. CLASSIFICATION

- Minor head injury.
- Moderate head injury.
- Severe head injury.

See table 8.1.

3. CLINICAL FEATURES

3.1. Minor Head Injury

- Glasgow coma scale (GCS) 13-14 (80% of head trauma), or GCS of 15 with any one of the following:
  - Suspected open or depressed skull fracture.
  - Sign of basal skull fracture.
  - Vomiting more than two times.
  - Age greater than 65 years.

3.2. Moderate Head Injury

Glasgow coma scale (GCS) 8-12 (10% of head trauma).

3.3. Severe Head Injury

Glasgow coma scale (GCS) 3-7 (accepted definition of coma).

4. MANAGMENT

4.1. Minor Head Injury

- History & examination:
  - General examination to exclude systemic injury.
  - Limited neurological examination.
  - Screen for drugs.
- Radiological investigation:
    - Skull X-ray A+P, lateral.
    - Computerized tomography (CT) scan – indications:
      - Witnessed loss of consciousness.
      - Definite amnesia.
      - Witnessed disorientation in a patient with (GCS) score of 13-15.
- Observe or admit to the hospital:
  - No (CT) available.
  - Abnormal CT scan.
  - All penetrating head injuries.
  - Prolonged loss of consciousness.
  - Sign of basal skull fracture.
  - No reliable companion at home.
  - Glasgow coma scale (GCS) < 15.

4.2. Moderate Head Injury

Resembles the management of minor head injury.

- Computerized tomography (CT) scan must be obtained.
- Admission for observation.
- Obtain neurosurgical consultation.

4.3. Severe Head Injury

The patient is unable to follow even simple commands because of impaired consciousness.

- Neurosurgical consultation.
- Primary survey and resuscitation.
- Secondary survey and AMPLE history.
- Neurological re-evaluation and GCS.

- A therapeutic agent (after consultation with neurosurgeon).
- Mannitol 20% solution 0.25 to 1g/kg.
- Moderate hyperventilation PCO_2 = 32-35 mmhg).
- Anticonvulsant drugs if needed.
- Computerized tomography (CT) scan.
- Transfer to definitive neurosurgical care.

Indications for hospital admission:

- Confusion or any other depression of consciousness at the time of examination.
- Skull fractures.
- Neurological symptoms and/or signs.
- Difficulty of assessment due to:
  - Drug or alcohol intake.
  - Epilepsy.
  - Other medical conditions that cloud the consciousness.
- For children:
  - The absence of responsible adult to observe the patient at home.
  - Mild cases with a short duration of amnesia, followed by a full recovery can be sent home under the care of a responsible adult with a "Head injury warning Card"; outlining danger signs and warnings to follow up with the hospital if they occur. **These signs include:**
    - Drowsiness or excessive sleeping.
    - Confusion or disorientation.
    - Severe headache, vomiting, or fever.
    - Weakness of any limb or double vision.
    - Convulsions, seizures, or passing out.
    - Discharge of blood or fluid from the ears or nose.

4.4. Medications

A. Sedation and Analgesia:

- Opiates and respiratory depressant sedatives should be used very cautiously with close monitoring (usually requires transfer to the ICU).
- Paracetamol 500 mg every 6 hours can be given safely.
- Combined paracetamol and codeine can also be given every 6 hours.
- Metoclopromide 10 mg every 12 hours can be given to control nausea and vomiting.

B. Antibiotics:

- Prophylactic antibiotics are of value for compound fractures and in CSF leakage. In other situations the use of antibiotics is controversial.
- For treatment of established infection.

C. Mannitol:

- It is a powerful osmotic diuretic used to reduce intracranial tension. *It can be life saving, specially during preparation for transfer to another center.*
- Dose is 0.5- 1 g/kg as a bolus over 10-30 minutes (about 250-400 ml of 20% solution in an adult).

D. Steroids:

Recent studies *dispute any benefit from the use of corticosteroids* in the management of head injury (crash study).

E. Symptom Management:

- Seizures:
  - Seizures carry a high risk of brain hypoxia.
  - Phenytoin 10-15 mg/kg bolus is given intravenous (IV) over 20 minutes with electrocardiogram (ECG) monitoring. Followed by IV infusion of 250-500 mg over 4 hours. Then 100 mg every 8 hours (IV or orally).
  - Diazepam 5-10 mg IV in persistent seizures.
  - *Watch for respiratory depression and ventilate the patient in the ICU.*
- Restlessness:
  - Is a warning sign
  - Sedation: Selective sedation should not be performed, unless the causes below are excluded or properly treated.
- Full bladder (needs catheterization).
- Hypoxia: Check arterial blood gas tension, respiratory rate, chest x-rays and oxygen therapy.
- Ischemias: Check pulse, blood pressure, ECG, and complete blood panel. Exclude intra abdominal hemorrhage, metabolic changes, dehydration, blood urea, serum electrolytes and bicarbonates, and blood glucose level.
- Missed intracranial hematoma (repeat CT scanning).
- Seizures: See management above. Consult the neurosurgeon.

Thoracic Trauma

GUIDELINE OBJECTIVES:

- Identify and initiate treatment for the following injuries during primary survey:
  - Airway obstruction.
  - Tension pneumothorax.
  - Open pneumothorax.
  - Massive haemothorax.
  - Cardiac tamponade.
  - Sever flail chest.
  - Thoracic aorta injury.
- Identify and initiate treatment of the following injuries during the secondary survey:
  - Simple pneumothorax.
  - Haemothorax.
  - Lung contusion.
  - Cardiac contusion.
  - Diaphragmatic rupture.

1. INTRODUCTION

- Chest injuries are estimated to be responsible for 25% of trauma deaths.
- The majority of patients who survive to reach the hospital require simple, yet vital interventions, within the capabilities of the ED trauma team. These, patients if properly recognized, can be treated or their conditions temporally stabilized, by early management in ED.
- Less than 15% of patients with chest trauma require thoracotomy.
- The aim of initial resuscitation is to restore adequate oxygen delivery to tissues by supporting oxygenation and circulation.
- Some major thoracic injuries may occur without chest wall damage. Diagnosis depends on prediction and exclusion rather than direct manifestations.

2. CLASSIFICATION

Thoracic trauma is categorized by its level of severity and time of recognition (during the primary or secondary survey).

3. CLINICAL FEATURES

3.1. Primary Survey

During the primary survey, the usual resuscitation steps of airway, breathing, and circulation are followed with immediate corrective interventions (as problems are identified).

A. Tension Pneumothorax:

- Tension pneumothorax is a clinical diagnosis which requires immediate decompression without awaiting further investigations.
- It develops when air in the pleural space is trapped under tension.
- This may be aggravated by mechanical ventilation attempts.
- The clinical picture shows:
  - Respiratory distress.
  - Tracheal deviation away from affected site.
  - Unilateral absence of breath sounds.
  - Distended neck veins and shock.
- Results in the complete collapse of one lung with mediastinal shift. Impairment of venous return will follow and cause low cardiac output and shock. The opposite lung may also be compromised.

Management:

- Intravenous access must be established as soon as possible.
- Decompression is initially done by inserting a needle (large cannula) *in the second intercostals space at the midclavicular line, on the side with no air entry.*
- Chest tube insertion *in the fifth intercostal space at the anterior axillary line.*

B. Open Pneumothorax:

Sucking chest wounds reduce respiratory efficiency.

Management:

- Sucking chest wounds should be covered initially with a sterile occlusive dressing, secured on 3 sides to act as a flutter valve. Do not secure the fourth side, this will cause respiratory collapse.
- As soon as practically possible, a chest tube should be inserted away from the wound, changing an open pneumothorax into a simple peumothorax.
- Surgical closure of the wound is done later.

C. Massive Hemothorax:

Is a collection of more than 1500 ml of blood in the chest.

Management:

- Insertion of a large chest tube > 32F in the fifth intercostal space.
- Simultaneous fluid resuscitation via two large bore IV cannulae.
- Penetrating wounds medial to the nipple or scapula, indicate possible injury to the heart, great vessels, or hilar structures.
- Initial drainage of > 1500 ml of blood from chest tube or > 200 ml/h for 3-4 hours, is an indication for thoracotomy.

D. Flail Chest:

- Crush injuries, causing multiple rib and sternal fractures, may cause a segment of the chest wall to become loose, with no continuity to the bony thoracic cage. Resulting in inefficient paradoxical respiration movements.
- The paradoxical movements may not be apparent until respiratory fatigue occurs, except when the flail segment is large.
- Indicates underlying pulmonary contusion.
- Pain leads to decreased respiratory movements, atelectasis, and decreased cough; resulting in chest infections.

Management:

- The degree of respiratory distress and hypoxia indicates the need for mechanical ventilation. Functional, not anatomical integrity is the aim of treatment.

E. Cardiac Tamponade:

- An acute collection of a small amount of blood in the pericardium may cause tamponade.
- This may not be clearly evident in chest x-ray (CXR), therefore a FAST should be performed for positive diagnosis.
- Beck's triad (increase central venous pressure, hypotension, and decrease heart sounds) may not be present. Neck veins may be empty due to severe hypovolaemia.

Management:

- If cardiac tamponade is suspected, pericardiocentesis is indicated. Removal of as little as 20 ml can improve the condition. Serious injury to the heart during pericardiocentesis is rare in trained hands. However, the posterior descending branch of right coronary artery may be injured.
- *Pericardiocentesis requires a cardio thoracic surgeon consult.*

3.2. Secondary Survey

Many serious chest injuries are missed on clinical examination. Serious chest injuries should be ruled out using base line investigations, (adding special investigations when needed). The history of trauma, CXR, arterial blood gases (ABGs), and ECG are base line investigations. *The erect CXR is the most important chest trauma investigation*. The erect position may not be feasible for hypovolaemic (or other injured) patients.

A. Pulmonary Contusion:

- Is a potentially lethal condition due to the insidious development of respiratory distress syndrome.
- There is associated atelectasis, shunting of blood, decreased lung compliance, and increased airway resistance.
- Some patients can be managed without intubation and mechanical ventilation.

Management:

- Early intubation (within the first hour) must be considered when:
  - There is hypoxia or worsening respiratory status.
  - The level of consciousness is impaired.

- The patient is being transferred to another hospital.

Lung contusions may not be present in the initial chest x-ray (CXR). Clinical respiratory distress mandates close observation of the patient and a follow up CXR.

B. Myocardial Contusion:

- Contusions of the heart are the most common undiagnosed fatal injury.
- It is often associated with sternal fractures; in such cases the right ventricle is more commonly damaged.
- Electrocardiogram (ECG) changes and creatine kinase (CK-MB) and Triponin may mimic acute myocardial infarction (AMI).

Management:

- An area of contused myocardium behaves as an area of infarction would, and the patient should be treated accordingly.
- Complications mimic myocardial infarction; and are treated the same (with no anticoagulants or fibrinolytics).
- Conduction defects may require a pacemaker.

C. Diaphragmatic Rupture:

- Penetrating chest injuries cause minor small diaphragmatic perforations (limited significance). It should be suspected in any stab wound below the nipple line.
- By contrast blunt trauma produces large radial tears of the diaphragm and herniation of the abdominal viscera. Rupture can result from a sudden severe increase in intra- abdominal pressure in an event as common as a minor fall.
- Left sided ruptures are more common, and more easily diagnosed because of the appearance of the gut in the chest.
- The chest radiograph can be misinterpreted as showing a raised hemidiaphragm, acute gastric dilatation, or a loculated pneumothorax. Contrast radiography or locating the abnormal position of the stomach on plain radiography with a nasogastric tube, confirms the diagnosis.

Management: Surgical repair (can be performed laparoscopically).

D. Major Airway Injury:

- It is suspected if there is extensive surgical emphysema in the neck, mediastinum, and chest wall. *Haemoptysis may be present.*
- Large persistent air leaks from the chest tube, which may result in incomplete expansion of the lung and characteristic whistling sounds on auscultation, is suggestive of bronchopleural fistula.
- The condition is temporarily relieved in the emergency department by inserting a chest tube. Early referral to a thoracic surgeon is indicated for further management.
- Indications for thoracotomy after initial resuscitation:
  - Cardiac tamponade.

- Massive air leak suggestive of major airway injury.
- Initial chest drainage > 2000 ml, or three consecutive hours of > 200 ml/h blood collection.
- Chest wall defects.
- Diaphragmatic lacerations.

- Other indications for chest tube insertion:
  - Selected patients with suspected severe lung injury, especially those being transferred by air or ground vehicle.
  - Individuals undergoing general anesthesia for treatment of other injuries (e.g. cranial or extremity), who have suspected significant lung injury.
  - Individuals requiring positive pressure ventilation who are suspected of having substantial chest injury.

Pitfalls in the management of chest trauma:

- A simple pneumothorax in a trauma patient should not be ignored or overlooked. It can progress to a tension pneumothorax.
- A simple hemothorax (not fully evacuated) can result in a retained, clotted hemothorax, or if infected can develop into an empyema.
- Diaphragm injuries are notorious for being overlooked in the initial trauma evaluation. Undiagnosed diaphragm injury can result in pulmonary compromise due to the entrapment and strangulation of peritoneal contents.
- Delayed or extensive evaluation of the wide mediastinum without cardiothoracic surgery capabilities can result in an contained hematoma rupture and rapid death from exsanguinations. All patients with injury and simple chest x-ray findings suggestive of aortic disruption should be transferred to a facility capable of rapid definitive diagnosis and treatment of this injury.
- Underestimating severe pathophysiology of the rib fractures, particularly in the elderly patient, has dire consequences. Aggressive pain control without respiratory depression is the key management principle.
- Avoid underestimating blunt pulmonary injury severity. Pulmonary contusions can present in a wide spectrum of clinical signs, often not well correlated with chest x-ray findings. Careful monitoring of ventilation, oxygenation, and fluid status is required, often for several days. Mechanical ventilation is required frequently.

Abdomen

GUIDELINE OBJECTIVES:

- Identify patients with abdominal injuries who require immediate resuscitation and urgent management.
- Recognize pitfalls in management of abdominal injuries.

1. INTRODUCTION

The initial assessment and resuscitation are essentially the same in blunt and in penetrating trauma. Intra-abdominal injuries are associated with high incidence of morbidity and mortality because they are often overlooked. A high index of suspicion is needed to avoid missing abdominal injuries especially in patients with multiple trauma.

2. CLASSIFICATION

According to mechanism of injury.

A. Blunt Trauma:

- Compression or crushing injuries.
- Deceleration injuries.

B. Penetrating Trauma:

- Stabs and low velocity projectiles: cause damage by laceration or penetration only.
- High velocity projectiles: have the added effects of cavitation, tumbling, and secondary missiles with more tissue damage.

3. CLINICAL FEATURES

The airway, breathing, and circulation, (ABCs) of primary survey, should be done as for all trauma patients. Resuscitation of the patient has the first priority.

3.1. History

- From the patient, relatives, or friends.
- From the police and ambulance crew.

3.2. Assessment

A. Look:

- Completely expose the patient.
- Anterior views: Inspect the chest, abdomen, urethral meatus (in men) and flanks.
- Posterior views: Inspect the back, buttocks, and perineum.
- Search for bruises, lacerations, entry and exit wounds, impressions of the seat belt or tires, spinal deformities, or paravertebral hematoma. Record all findings.

B. Listen:

- Listen for bowel sounds and their quality if present.
- *The presence of bowel sounds does not exclude major peritoneal injury.*

C. Feel:

Superficial and Deep Palpation:

- Muscle guarding results from intra-peritoneal injury. However, injury to abdominal wall muscles can cause guarding too.
- Rigidity usually indicates peritoneal irritation due to bleeding or rupture of a hollow viscus.
- Injuries to the lower chest can include the abdomen.
- Tenderness on cough is another indicator of peritoneal irritation.
- Check for stability of the pelvic ring by pressing against the anterior superior iliac spines in opposite directions.
- Palpate the symphysis pubis and superior pubic rami.
- Re-evaluate repeatedly for new signs.

D. Rectal Examination:

- Detect if there is loss of rectal wall integrity or bleeding from the rectal wall indicating damage to the rectal or large bowl wall.
- Check for high riding prostate which indicates urethral disruption.

E. Vaginal Examination:

- Detect vaginal wall integrity.
- Detect symphysis pubis disruption or fractures of superior or inferior pubic rami.

F. Examination of Urethral Meatus:

If bleeding from the external meatus is *found, do not pass a catheter.* Call for urological consultation.

G. Examination of the Gluteal Region:

Penetrating injuries to the gluteal folds are associated with 50% incidence of significant intra abdominal injuries (e.g. rectal injuries).

4. MANAGEMENT

4.1. Baseline Investigations

- Withdraw blood samples for complete blood paneling including a complete blood count (CBC) and blood group with cross matching. Reserve blood for later use.
- Urea, electrolyte concentrations, serum amylase activity, and blood gas tensions should be determined.

4.2. Pass a Nasogastric Tube

Pass the tube *orally* if there is fracture of the cribriform plate. It helps to empty the stomach and may detect injury if blood is aspirated from the stomach.

4.3. Pass a Urethral Catheter

It is essential to monitor the urinary output and the efficiency of fluid replacement. The supra-pubic route should be used if there is evidence of urethral injury.

4.4. Abdominal Ultrasonography

After stabilization of the patient and if there is no indication for immediate laparotomy, ultrasonography can help detect fluid collection, subcapsular splenic hematoma, and renal injuries.

(If ultra sound is not available, diagnostic peritoneal lavage (DPL) is done).

4.5. Plain X-Rays

- An erect chest film may show free air under the diaphragm or rib fractures.
- Abdominal x-ray films may show lower rib fractures, which may suggest injuries to the liver or spleen. Fractures of transverse processes may suggest ureteric injuries.
- Radio opaque foreign bodies, bullets or shrapnel may be detected.
- The lateral cervical spine and pelvis should be radiographed.

4.6. Computerized Tomography (CT) Scan

If available, and the patient is stable, or there is an indication for CT scanning of the brain.
Abdominal CT scanning helps to diagnose pancreatic and retroperitoneal injuries.

4.7. Diagnosis Laporscopy

4.8. Indications for Emergency Surgical Intervention

- Unexplained shock (deterionatry vital signs).
- Rigid silent abdomen.
- Evisceration.
- Radiological evidence of free intraperitoneal gas.
- Radiological evidence of ruptured diaphragm.
- All gunshot penetrating wounds.
- Positive FAST.

Urinary Tract

GUIDELINE OBJECTIVES:

- Implement the initial assessment of the lower and upper urinary tracts.
- Institute the initial management of lower and upper urinary tract injures.

1. INTRODUCTION

The upper urinary tract includes the kidneys and ureters, the lower urinary tract involves the bladder and urethra.

2. CLASSIFICATION

- Blunt abdominal trauma.
- Penetrating wounds.

3. CLINICAL FEATURES

The ABCs of the primary survey should be done as usual. Resuscitation of the patient has the first priority.

3.1. History and Symptoms

- Groin pain.
- Gross hematuria.
- Colicky pain after hematuria.

3.2. Clinical Examination

A. Look:

- Bruising or lacerations of the groin, upper abdomen, or lower chest.
- Flattening of the groin contour.
- Presence of stab wounds, entrance, or exit wound in the groin.
- Gross hematuria. It may be absent in severe renal vascular injury.
- Scoliosis with concavity on the affected side.

B. Feel:

- Renal angle tenderness.
- Rigidity of upper abdomen.
- Palpable renal angle mass.

4. MANAGMENT

4.1. Basic Laboratory Tests

- Withdraw blood sample for a complete blood panel, including complete blood count (CBC), blood group, and cross matching. Reserve blood for later use.
- Urea, electrolyte concentrations, serum amylase activity, and blood gas tensions should be determined.
- Urine analysis for microscopic hematuria.

4.2. Ultrasonography

In clinically stable patients, ultrasonography gives further information on the state of the injured kidney. It may show perirenal collection, intrarenal or subcapsular hematoma, or the presence of parenchymal disruption.

4.3. Intravenous Urography

Indicated for all patients with gross hematuria and for patients with microscopic hematuria and blood pressure < 90 mm Hg.

4.4. Computerized Tomography (CT) Scan

It does not give reliable in information in renal emergency trauma patients.

5. LOWER URINARY TRACT

5.1. Consider Lower Urinary Tract Injuries

- When a patient presents with a fractured pelvis. Fractures of the symphysis pubis should raise the suspicion of bladder or urethral injury.
- Inability to pass urine or high riding prostate in rectal examination is a sign of urethral injuries.
- Intraperitoneal rupture of the bladder causes lower abdominal peritonism. If urethral injury is suspected, or if a perineum or scrotal hematoma is present, insert a suprapubic cystofix.
- If intraperitoneal rupture of the bladder is suspected, order an ascending cystogram because, intraperitoneal rupture of the bladder dictates laparotomy.

5.2. Management

- Serial clinical observations should include vital signs and abdominal palpation.
- Serial CBC and urine analysis test.
- Consult the specialist if there is micro and/or macroscopic hematuria, surgical intervention is warranted.

Blood on the external urinary meatus indicates damage to the bladder or urethra. Call for urologic consultation.

Do not pass a catheter.

Limb Injuries

GUIDELINE OBJECTIVES:

- Identify patients with limb injuries who require immediate resuscitation.
- Identify limb threatening injuries.
- Identify limb trauma with associated neuro-vascular injuries.
- Recognize and manage the compartment syndrome.
- Recognize pitfalls in the management of limb injuries.

1. INTRODUCTION

In polytrauma patients, 70% have limb injuries with fractures or dislocations. Severe limb injuries should not distract the attention from the ABCs of resuscitation, unless causing exsanguinating bleeding.

2. CLASSIFICATION

- Life threatening limb injuries include:
  - Traumatic amputation.
  - Major vascular injury.
  - Pelvic fracture disruption.
  - Hemorrhage from an open fracture.
  - Multiple long bone fractures.
  - Severe crush injury.
- Limb threatening injuries include:
  - Vascular injuries.
  - Major joint dislocation.
  - Crush injury.
  - Open fracture.
  - Compartment syndrome.
  - Nerve injury.

3. CLINICAL FEATURES

3.1. History

- Ask about the injury mechanism, environment, time, and the immediate care given.
- A falling patient that lands on his/her heels can cause compression fractures of the calcaneum, ankle, tibial plateau, and vertebrae.
- Trauma to the knee in the sitting position (e.g. car accident) may cause a fractured patella, femur, acetabulum, or hip dislocation.

- Estimated blood loss caused by bone fractures:
  - Pelvis…… 1.0-4.0 liters.
  - Femur…… 1.0-2.5 liters.
  - Humerus…… 0.5-1.5 liters.
  - Tibia…….. 0.5-1.5 liters.

3.2. Physical Examination

A. Look:

- Look for swellings, contusions, hematomas, or open wounds along the course of major vessels, indicating vascular injuries.
- Look for limb shortening or abnormal rotation, which indicates fractures or dislocations.
- Pallor or blue-grey color of the skin indicates major vascular injury.

B. Feel:

- Carefully palpate along the axes of bones and bony prominences for tenderness, angulations, crepitus, and abnormal movements.
- Gently examine the joints for restricted movements or sub-luxations. But not in the presence of fractures or ischemia.
- Distal pulses should be felt and recorded on both sides.
- Check for differences in temperature between both limbs (indication of ischemia). Test for capillary filling.
- Peripheral nerves suffer early, total loss of sensation in a hand or foot is an indication of ischemia.
- Test for sensation and motor function along the nerve distribution to detect and document nerve injury for further management.

4. MANAGEMENT

4.1. Airway, Breathing, and Circulation (ABCs)

- Immediate fluid replacement with available crystalloids or colloids, till blood is available, is life saving.
- Bleeding from wounds can be reduced with compressive bandages or digital pressure over a sterile pad. *No tourniquet* should be used, except in life threatening bleeding after traumatic amputation.
- Immobilization of fractures by splinting before transport is mandatory. Reduction of a major deformity should be done. After reduction check for distal pulses in the affected limb.

4.2. Investigations

A. Blood:

- Complete blood count (CBC).
- Blood grouping and cross matching (order all available units in major bone injuries).

B. Radiography:

- Plain X-rays.
  - X-rays for limbs and joints are done in two perpendicular projections. After resuscitation, perform the three standard X-rays (i.e. chest, lateral cervical spine and pelvis). To detect fractures or dislocations and help plan the definitive patient management.
  - Images should include the joint above and below the fracture.
- Doppler study: Doppler ultrasonography is used to assess limb perfusion.

4.3. Wound Management

- The wound should be covered with sterile compressive dressings.
- Antibiotic administration.
- Tetanus immunization according to the immunization state of the patient.
- Surgical toilet in the operating theatre.

4.4. Fracture Management

- Splinting affords considerable relief of pain, avoids further soft tissue damage, and facilitates transfer of the patient.
- Immobilize the joint above and below the fracture, and the bones on either side of a dislocated joint.

Examples:

- The arm is supported by a sling and bandaged to the body.
- The forearm and wrist are immobilized in padded splints.
- The hand is splinted gripping a bandage roll in a functional position.
- Lower limb fractures are immobilized using a Thomas splint.

- Definitive management of fractures occur in the orthopedic department.

A. Pelvic Injuries:

- Pelvic fractures should be actively investigated to avoid catastrophic consequences.
- Assessment and examination of the pelvis should occur from outside moving inwards.
- Pelvic fractures are usually associated with serious soft tissue and visceral injuries (e.g. urinary tract, vascular, and abdominal).
- Life threatening bleeding is common in pelvic fractures.
- External fixation of the pelvic ring is helped by:
  - Reducing the rate of bleeding and the retroperitoneal hematoma.
  - Reducing the magnitude of soft tissue damage.
- External fixation does not interfere with x-rays, CT scanning, diagnostic peritoneal lavage, or laparotomy (when needed).
- External fixation can be done in the emergency room within 10 minutes.
- Call the orthopedic surgeon early.

B. Compartment Syndrome:

- Increased swelling inside a fascial compartment due to contusion of the tissues will cause a marked drop in local tissue perfusion. This can lead to infarction of the muscles in that compartment.
- High degree of suspicion is needed to diagnose.
- Pulse is palpable in most cases.
- Clinical picture includes:
  - Increasing pain despite immobilization.
  - Altered sensation in the dermatome of the nerve passing through the compartment.
  - Palpable raised tension and tenderness in the muscle compartment.
  - Pain on passively stretching the muscles within the compartment.
  - Removal of all constricting bands, dressings, and casts should be done promptly. If no relief, fasciotomy is to be done immediately (to be done by specialist).

C. Traumatic Amputations:

- It is a life threatening catastrophe.
- Bleeding should be controlled as a priority.
- The amputated part should be cleaned, wrapped in sterile cloth, soaked in saline, and put in a plastic bag which is immersed in a container of crushed ice and water. It should not freeze.
- The patient after resuscitation can be transferred, with the amputated part, to a center with replantation facilities.
- If replantation is not possible, the amputated part can help the patient by serving as a source of skin, nerve, vessels, and bone autografts.

The Spine and Spinal Cord

GUIDELINE OBJECTIVES:

- Understand the probability of cervical spine injury in unconscious trauma patients.
- Recognize spinal shock in thoracic injuries.
- Utilize spinal immobilization during transport of suspected spinal fracture patients.
- Understand the role of steroid medication use in spinal cord injury management.

1. INTRODUCTION

- Any patient with trauma who is not fully conscious should be assumed to have an injury of the cervical spine, until prove otherwise.
- Collars and neck supports should not be removed, unless a cervical spine injury has been conclusively excluded. Sand bags and a forehead strap may be needed as a lateral support with a neck collar.
- Collars alone are inadequate; collars do not provide 100% cervical protection. When cervical injury is clinically suspected, collars must be supplemented by manual stabilization or lateral support (sandbags and forehead tape).
- In the unconscious patient, the supine position facilitates clinical examination, cardio-pulmonary resuscitation, respiratory movements, and control of the neck; however, aspiration is a risk that can be avoided with early endotracheal intubation.
- In patients with spinal cord injury, the early use of high dose corticosteroids (e.g. methylprednisolone) over the first 48 hours has been shown to improve clinical outcomes.
- Injury above the clavicle should prompt a cervical spine injury search.
- The neck must be aligned in a neutral position without longitudinal compression or distraction, during transport and manipulations.
- About 55% of spinal injuries occur in the cervical region and about 15% in the lumbosacral area.

2. CLINICAL FEATURES

2.1. Primary Survey

A. Airway Care with Protection of Cervical Spine:

- Vigorous suction, manipulation of oral airway, or intubation in a patient with cervical spine injury may cause unopposed vagal stimulation and cardiac arrest.
- Whenever possible, tracheal intubation should be done by an experienced anesthetist and assistant to minimize neck movement.

B. Circulation:

- Cervical or high thoracic spine injuries may cause neurogenic or spinal shock.
- Hypovolemia should be corrected, but pulmonary edema should be avoided.
- If the cardiac rate is <50 beats/min, atropine should be given (0.5 mg/ IV push).

- If systolic blood pressure is < 80 mmHg, inotropic support is mandatory.

2.2. Secondary Survey

A. Neurological Examination:

- The conscious patient:
  - A full neurological examination must be performed, including testing of cranial nerves, sensation to fine touch and pin prick, power, tone, coordination, and reflexes.
  - The neurological level is the most caudal segment of the spinal cord with normal sensory and motor function on both sides of the body. The level is detected by charting sensation in different dermotomes and different reflexes.
  - The bony level of injury is the vertebral at which the bones are damaged, causing injury to the spinal cord.
  - Injuries of the first 8 segments of the spinal cord cause quadriplegia, while lesions below the T1 level result in paraplegia.
  - Complete cord lesion: Loss of sensation and paralysis below the neurological level.
  - Partial cord lesion: Some neurological functions are preserved below the neurological level.
- The unconscious patient:
  - Features of spinal cord injury include flaccid paralysis, diaphragmatic breathing, bradycardia with hypotension, and upward movement of the umbilicus when tensing the abdomen.
  - The patient is log rolled to one side by four persons to stabilize the spine.
  - Glasgow coma scale scoring.
  - Fundoscopy, assessment of muscle tone, and nervous reflexes.
  - Abdominal, anal, and bulbocavernosus reflexes should be assessed.
  - Flaccidity and areflexia in an arm may be caused by a brachial plexus injury and/or spinal cord injury.

3. MANAGEMENT

A. Airway, breathing, and circulation (ABCs).

B. Call for Specialists.

C. Adequate Immobilization of Cervical and Thoracolumbar Spine:

- Respiratory insufficiency can occur early in patients with spinal cord injuries.
- Narcotic analgesics can cause further depression of respiration.
- Cardiac arrest can occur due to respiratory failure.
- Blood gases tension must be checked.
- Pulse oximetry is important.
- Abdominal trauma is difficult to diagnose in quadriplegic patients.

- A Foley's catheter should be placed under strict aseptic conditions to empty the bladder and measure the urine output. Urine output is used to monitor the fluid therapy and the renal function. A suprapubic catheter can be used if urethral injury is present.

3.1. Radiology

- Good quality radiographs in the radiology department (supervised by a doctor ensuring patient spinal stabilization) are necessary to reach an accurate diagnosis.
- In patients with multiple trauma, x-rays of the cervical spine, chest, and pelvis are mandatory. Skull x-rays and CT scans (if available) are needed for patients with depressed sensorium.

A. Assessing Spinal Radiographs:

- For al spinal radiographs, it is important to check:
  - Vertebral borders (anterior and posterior).
  - Posterior facet margins.
  - Anterior and posterior borders of the spinous processes.
  - Integrity of vertebral bodies, laminae, pedicles, and arches.
  - Prevertebral space.
  - Interspinous gap.
  - For rotation deformity.

B. Cervical Spine & Thoracolumber Spine:

- A standard lateral radiograph of the cervical spine showing all the seven vertebrae and (C7-T1) junction will show almost all the detectable abnormalities. This view may necessitate applying traction to both arms. A swimmer's view may be needed to show the (C7-T1) junction.
- A normal x-ray of the spine dose not rule out cord injuries in the presence of abnormal physical signs. A CT of the spine may be needed.
- Look for fractures, subluxations, and dislocations. Look for a prevertebral hematoma that causes widening of the retropharyngeal space.
- The open mouth odontoid view and the anteroposterior projection will highlight injuries of the odontoid, atlantoaxial joint, and the transverse ligament of the atlas.
- Cervical spine x-rays in patients with a short neck can be inadequate. In such cases, a CT of the cervical spine may be essential for diagnosis.
- Anteroposterior and lateral radiographs are needed for thoracolumber vertebral injuries.
- Thoracic spine fractures associated with rib or sternal fractures are unstable. X-rays of ribs and sternum are important in this context.

Spinal injuries with paraplegia or quadriplegia:

- A high dose of corticosteroids is given as early as possible (methylprednisolone 30 mg/kg intravenously over 15 minutes and then 5.4 mg/kg/h for 48 hours).
- Routine administration of manitol and antibiotics has been proven to be nonbeneficial.
- Early referral to spinal centers with intensive care units and magnetic resonance imaging is recommended after resuscitation and management of life threatening conditions.

Trauma during Pregnancy

GUIDELINE OBJECTIVES:

- Understand the physiological difference for the pregnant trauma patient.
- Recognize the clinical impaction of pregnancy on trauma management.
- Understand the trauma assessment difficulties in pregnant patients.
- Recognize fetal hypoperfusion in hypovolemic events of the mother.
- Understand pregnancy-related supine hypotension syndrome.

1. INTRODUCTION

- Pregnancy alters the clinical assessment of the trauma patient:
  - Plasma volume expands by 50% by the sixth month.
  - Cardiac output increases by one third.
  - Consequently, normal pregnancy may mimic hypovolemia with tachycardia and hypotension.
- Another important change is the physiologic anemia of pregnancy.

These changes make it difficult to rely on vital signs to assess hemodynamic status.

As the uterus enlarges, abdominal contents are displaced upward and diaphragmatic excursion is limited. Apart from gastrointestinal problems, this anatomic change results in a characteristic "hyperventilation of pregnancy" as the respiratory rate increases to compensate for the decrease in tidal volume. Consequently, PCO_2 levels are typically low, and the kidney excretes bicarbonate to maintain physiologic pH. The average level of PCO_2 is 30 mmHg, and bicarbonate (HCO_3) averages 19-22 mEq/L. Both tachypnea and low bicarbonate levels may be misconstrued as signs of decompensated shock. In addition, displaced abdominal contents and a higher diaphragm are more susceptible to penetrating injury in the upper abdomen and lower chest. One of the immediate responses to maternal blood loss is that blood flow to the abdominal viscera and uterus will decrease; in order to preserve circulation to the mother's essential organs. Consequently, the patient may appear relatively stable, but the placenta and fetus may be significantly underperfused. In order to prevent unrecognized fetal hypoperfusion, all pregnant trauma patients should be transported with high flow oxygen and a fluid challenge test through a large-bore infusing IV.

The supine position should be avoided, and the patient should be transported with the backboard tilted 15^o to the left.

2. CLASSIFICATION

- Abruptio placentae.
- Uterine rupture.
- Amniotic fluid embolus.
- Fetomaternal hemorrhage.
- Premature labor.
- Premature rupture of membranes.

3. CLINICAL FEATURES

3.1. Physical Examination

A. The Initial Resuscitation of the Pregnant Trauma Victim:

- Although the patient should be assessed in a slightly left lateral position, the primary survey for life-threatening injuries should be conducted as usual.
- Once life-threatening injuries have been addressed, a complete head-to-toe examination should be performed, including assessment of the fetus.
- Fetal heart tones can be obtained with a stethoscope by 12 weeks gestation, and these should be recorded.
- If the patient is able to provide history, the date of the last menstrual period (LMP) and estimated date of confinement (EDC) should be determined, as well as, any complications during the current and previous pregnancies, presence of contractions, and fetal movements.
- Physical examination of the pregnant abdomen is difficult as abdominal organs are displaced by the growing uterus.
- Pelvic examination is essential; it may reveal vaginal lacerations, rupture of membranes, vaginal bleeding, cervical dilation, presenting fetal parts, and pelvic or sacral fractures that may make vaginal delivery impossible. *The exam must be done under sterile conditions due to the possibility of ruptured membranes.* This examination should be performed by an obstetrician to avoid repeat examinations.

3.2. Ancillary Methods in Evaluating the Fetus

- Bedside ultrasound evaluation of the abdomen is a noninvasive method to detect organ injury and hemoperitoneum, and it is particularly useful in the pregnant patient.
- Transabdominal ultrasound can detect the fetal heart rate by 7-8 weeks gestation.

3.3. Injuries That Occur Specifically During Pregnancy

A. Abruptio Placenta (revealed and concealed):

- Dissection of blood between the layers of the placenta, and uteru 5 disrupts the supply of oxygen and nutrients to the fetus.
- After approximately 50% of the placental surface is disrupted, the fetus cannot survive.
- Lesser degrees of abruption present as abdominal pain, uterine irritability, contractions, and a variable amount of bright red vaginal bleeding.

B. Uterine Rupture:

- The uterus can be palpated as a contracted mass and fetal parts can be palpated subcutaneously.

C. Amniotic Fluid Embolus:

- Following any trauma of the pregnant uterus, there is a risk of introducing a bolus of amniotic fluid into the maternal circulation.
- This causes a devastating condition with a high patient mortality.

- The diagnosis is suggested by the sudden onset of dyspnea, hypoxemia, and tachypnea, which may be followed by adult respiratory distress syndrome (ARDS) and disseminated intravascular coagulation (DIC).

D. Fetomaternal Hemorrhage:

- Introduction of only a few milliliters of fetal blood into the maternal circulation will cause isosensitization of the patient if there is an Rh mismatch.
- If the woman is Rh^- and the fetus Rh^+, the woman will form antibodies to the Rh factor. These antibodies will attack subsequent Rh positive fetuses resulting in erythroblastosis fetalis.
- Because no tests are sensitive enough to detect such a small amount of fetal blood, it is routine to administer "mini anti-d also called (RhoGAM)" (50 μg) in the first trimester and regular dose RhoGAM (300 μg) subsequently.
- This dosage will block the formation of maternal antibody against the Rh factor.

E. Premature Labor:

- Organized uterine contractions are common after trauma to the abdomen.
- Abruptio palcentae is the most common cause of premature labor following trauma.

F. Premature Rupture of Membrane:

- Occasionally, the placental membranes will rupture as a result of increased intrauterine pressure or from direct trauma.
- The principle risk of premature rupture of the membranes is ascending infection, amnionitis, and fetal loss.

4. MANAGEMENT

- Airway, breathing, and circulation, (ABCs).
- Call for a specialist.
- If the patient has injuries requiring surgery, surgery should never be deferred or delayed because of pregnancy.
- If the fetus is previable and in severe distress, there are no specific interventions to correct the distress, other than optimizing the woman's condition.

Resuscitation of the mother takes first priority.

Trauma in Pediatrics

GUIDELINE OBJECTIVES:

- Understand the pediatric physiological differences.
- Utilization of the ABCs of trauma management for pediatric patients.

1. INTRODUCTION

Infants and children under ten years of age have important physiological differences that influence trauma response and management.

****Children are not just little adults****

Key Points:

- Infants and small children have less physiological reserves than adults, and minor deviations from normal levels require early attention.
- Infants and children are at special risk of becoming dehydrated and hypoglycemic.
- Monitor fluid status, electrolytes, and hemoglobin diligently and correct any abnormalities promptly.

2. PREPARATION

- Upon notification of a pediatric admission:
  - Notify the appropriate consultants based on the pre-hospital report.
  - If the age or weight of the child is known, assign someone to calculate fluid bolus, endotracheal tube size, and doses of first-line drugs (see table 8.2).
  - Attempt to warm the stretcher as much as possible utilizing warming lights or other aids. It is much easier to keep a patient warm than to rewarm a hypothermic patient. Have warm blankets and IV fluids.
  - Assign someone to obtain the child's medical history from the parent (who may be in another trauma victim):
    - Normal pregnancy and delivery?
    - Met all developmental milestones?
    - Any medical problems?
    - Normal weight for height?
    - Is the tetanus immunization up to date?
    - Allergies to foods or medications?

3. PHYSIOLOGIC CONSIDERATIONS

3.1. Compensatory Mechanisms for Shock

Children compensate for shock differently than adults, mainly by increasing their heart rate. A rapid heart rate in a child may be a sign of impending circulatory collapse. Do not ignore a decreased blood pressure. *A slow heart rate in a child is hypoxia until proven otherwise.*

A. Blood Volume:

- Children have smaller blood volumes:
  - Even small amounts of blood loss can be life threatening.
  - Intravenous fluid replacement is needed when blood loss exceeds 10% of the total blood volume.
- Blood volume (ml/kg body weight):
  - Neonates 85–90 m1/kg.
  - Children 80 m1/kg.
  - Adults 70 m1/kg.

4. PEDIATRIC RESUSCITATION

While care of the pediatric trauma patient can be technically challenging and emotionally stressful, remember to follow the basic primary and secondary surveys and treatment goals associated with the care of the adult trauma patient.

4.1. Airway

- The airway should be briefly suctioned if secretions are present.
- The relatively large pediatric tongue may make ventilation and maintenance of an oral airway difficult. Measure oral airways for the unconscious child, from the corner of the mouth to the bottom of the ear lobe.
- Endotracheal intubation is never the first step of airway control in the pediatric patient and should only be performed by individuals who are already proficient with this procedure.
- Refer to standard tables to determine endotracheal tube and laryngoscope blade sizes.
- Children must be well oxygenated prior to intubation because of their smaller functional residual capacity (FRC) and higher metabolic rates.
- Atropine and succinylcholine must be available for rapid sequence intubations.
- The pediatric trachea is short; do not advance the endotracheal tube too far. The black line on the tube should be positioned at the vocal cords.
- After intubation, confirm tube placement by observation of bilateral chest movement and auscultation over the lungs and stomach (be careful, as a right main stem intubation may still allow breath sounds to be heard over the left anterior chest). Confirm tube placement each time the patient is turned or moved.
- After intubation, decompress the stomach with an appropriate sized nasogastric or orogastric tube.

4.2. Breathing

Until prove otherwise, bradycardia = hypoxia!

- Children can be given oxygen via nasal cannula, facemask, bag valve mask (only if the mask is held firmly against the face) or "blow by" tubing (remember that oxygen is heavier than air).
- Neonates and pediatric patients may be ventilated using a pediatric (even for neonates) bag and appropriately sized facemasks. Remember to turn on the oxygen flow meter and use just enough ventilation to cause chest expansion.
- The respiratory (hence ventillatory) rate is greater in children. If positive pressure ventilation is required, ventilate at 20-40 breaths/minute.

4.3. Vascular Access

- Peripheral IV line (s) should be placed.
- Intraosseous infusion may be used in children six years of age and younger to provide a rapid means of fluid administration. Additionally, any drug may be given by the intraosseous route.
- Central line placement in the pediatric patient can be technically challenging and should not be attempted by inexperienced providers.

4.4. Fluid Resuscitation

- All fluids and blood products administered to children should be warmed to prevent hypothermia.
- Initial fluid resuscitation in the hypovolemic child should consist of 20 ml/kg of warmed ringer's Lactate solution. This may be repeated once or twice.
- At this time, consider administering 10 ml/kg of warmed type-specific or O negative packed red cells.

See table 8.4.

4.5. Drug Therapy

Drugs should be administered on a "per kilogram" basis (see table 8.3).

4.6. Temperature Regulation

- Infants and children have a higher metabolic rate than adults. This is reflected in their normal vital signs. Children are especially prone to hypothermia, prevention of hypothermia is essential.
- Avoid hypothermia, which is common because of the child's greater body surface area/weight ratio:
  - Turning off any air conditioning (aim for a room temperature of >28°C).
  - Use warmed intravenous fluids and respiratory gases.
  - Overhead heating lights and forced air heating devices are all important.
  - To decrease heat loss, the extremities can be covered with plastic.

Trauma in Elderly

GUIDELINE OBJECTIVES:

- Recognize physiological differences of the elderly.
- Utilize the ABCs of trauma management in the elderly.
- Know and utilize drugs and medications in traumatized elderly patients.

1. INTRODUCTION

Mortality rates for elderly trauma patients (56 years and above) are higher, for any injury severity score (ISS) elderly patients are more likely to die. The elderly have reduced adaptation capacity in illness and injury, and increasing co-morbidities. Early aggressive management of the elderly trauma patient is appropriate, and required to achieve good outcomes. There are wide individual variations for the effects of aging.

2. MANAGMENT

The resuscitation time window is reduced in elderly patient; therefore, early aggressive interventions, assessments, and monitoring are justified.

2.1. Airway

- There are aspects of airway management in elderly trauma patients that need to be considered:
  - Upper airway obstruction is more likely.
  - Dentures.
  - Air tight seal for mask ventilation is more difficult (due to facial fat loss).
  - A brittle trachea.
  - Cervical spine diseases result in difficult airway management.

2.2. Breathing

Restrictive and obstructive respiratory diseases plus spinal diseases lead to difficult breathing control.

2.3. Circulation

- Consider age specific cardiovascular diseases and cardiac medications.
- Careful attention to volume status is fundamentally important.

2.4. Disability and Drugs

- There is a decreased requirement for sedation/anaesthesia.
- Pain threshold remains the same; but pain tolerance may be increased.
- Polypharmacy results in increased drug sensitivity.
- High drug levels and prolonged half-lives require decreased dosing and prolonged drug intervals.
- Consider pre-morbid neurological status.

2.5. Exposure

- Hypothermia is common in the elderly trauma patient, even in warm environments.
- Careful attention to thermoregulation must be ensured.
- Don't forget: pressure sores, adrenal insufficiency, and hypothyroidism.

Elderly trauma patients deserve the same aggressive resuscitation and management as younger trauma patients. Optimal outcomes are more likely with careful monitoring and appropriate timely interventions. To achieve this, admission to HDU and ICU is appropriate at a lower threshold (than with younger trauma patients).

Table 8.1: Glasgow Coma Scale (EVM)

Sign	Response	Score
Eyes Open	Spontaneously	4
	To speech	3
	To pain	2
	None	1
Best Verbal Response	Orientated	5
	Confused	4
	Inappropriate	3
	Incomprehensible	2
	None	1
Best Motor Response	Obey commands	6
	Localize pain	5
	Flexion to pain	4
	Abnormal flexion	3
	Extension to pain	2
	None	1

Table 8.2: Pediatric Vital Signs and Airway Equipment Size

	WEIGHT	RANGE	PULSE	BP	RESP	ET TUBE	BLADE
Premature	2.0		120/170	60/40	40-60	2.5–3	0 Miller
Term Newborn	3.3	2.4-4	100-150	65/40	40-60	3–3.5	1 Miller
6 Months	7.5	6-9	90-120	85-60	40-60	3.5–4.5	1-1/2 Wis-Hipple
1 Year	10	8-12	80-120	90/60	30-40	4–4.5	1-1/2 Wis-Hipple
2 Years	12	10-14	70-110	96-65	20-40	4.5	1-1/2 Wis-Hipple
3 Years	14	12-17	70-110	95/65	20-40	4.5	2 Miller
4 Years	17	14-20	65-110	100/65	20-35	5	2 Miller
5 Years	19	15-23	65-110	100/65	20-35	5	2 Miller
6 Years	21	17-26	65-110	100/55	15-30	5.5	2 Miller
7 Years	23	18-31	60-95	100/55	15-30	5.5	2 Miller
8 Years	25	20-35	60-95	105/55	15-30	6 cuffed	2 Miller
9 Years	29	22-41	60-95	105-55	15-30	6.5 cuffed	2 Miller
10 Years	33	24-46	60-95	105-55	15-30	6.5 cuffed	2 Miller
11 Years	37	27-53	60-95	105/55	15-30	6.5 cuffed	2 Miller
12 Years	41	31-60	60-95	110/60	15-25	7 cuffed	2 Miller
13 Years	46	34-67	55-85	115/60	15-25	7 cuffed	3 Miller, 3 Mac
14 Years	51	38-73	55-85	115/60	15-25	7.5 cuffed	2 Miller, 3 Mac

Table 8.3: Pediatric Drug Dosages

• ANY drug or fluid may be given by the intraosseous route • ENDOTRACHEAL drug dose = 2 – 2,5 x (IV) dose, except Epinephrine	
Adenosine	0.1 mg/kg (IV) (SLAM); (Direct, very rapid IV shot may repeat 0.2 mg/kg x 2)
Albuterol	2.5-5.0 mg QS 3 ml with NSS; Inhalation
Amiodarone	5 mg/kg RAPID IV Bolus AFTER PEDIATRIC CONSULTATION
Atropine	0.02 mg/kg IV; may repeat x 1; ET possible MIN DOSE = 0.10 mg; MAX SINGLE DOSE = 0.5 mg Child, 1.0 mg Adolescent
Bicarbonate	1 mEq/kg SLOW (IV); assure adequate ventilation (ABGs) available: mEq = kg x 0.3 x (BD); give 1/2 calculated
Calcium Chloride	20 mg/kg SLOW IV
50% Dextrose1 ml/kg	(SLOW) (IV) into running IV; follow with infusion
Diazepam	0.04-0.10 mg/kg (SLOW) (IV); TITRATE
Diphenhydramine	1 mg/kg
Electricity	Defib: 2 J/kg, 2-4 J/kg, 4 J/kg; Cardioversion 0.5-1.0 J/kg
Epinephrine	
• Bradycardia	(IV/IO) 0.01 mg/kg (0.1 ml/kg 1:10,000) (ET) 0.10 mg/kg (0.1 ml/kg 1:1,000)
• Pulseless Arrest	<u>First Dose</u> (IV/IO) 0.01 mg/kg (0.1 ml/kg 1:10,000) (ET) 0.10 mg/kg (0.1 ml/kg 1:1,000) <u>Second & subsequent</u> Repeat initial (IV)/IO dose or may increase up to 10 times (0.1 mg/kg, 0.1 ml/kg 1:1,000) Q3-5 minutes
• Bronchospasm	0.01 mg/kg subcutaneous (0.01 ml/kg 1:1,1000); MAX 0.30 mg
Fentanyl	1-2 μg/kg SLOW (IV); TITRATE
Furosemide	1 mg/kg (IV)
Lidocaine	1 mg/kg (IV; ET) possible; may repeat x 2 20-50 μg/kg/minute infusion
Meperidine	1-1.5 mg/kg SLOW (IV); TITRATE
Midazolam	0.03-0.1 mg/kg SLOW (IV); TITRATE
Morphine	0.0-0.15 mg/kg SLOW (IV); TITRATE
Naloxone	0.01 mg/kg IV or IM; ET possible; may repeat up to 0.1 mg/kg, especially neonatal
Succinylcholine	1 mg/kg (IV) (2 mg/kg under 1 year); PRETREAT with atropine
Vecuronium	0.1-0.15 mg/kg initially; subsequent titration
IV= Intravenous **IO**= Intrasseous **ET**= Endotracheal **ABGs**= Arterial blood gases	

Table 8.4: Pediatric Drug Infusion

	Usual Dose (μg/kg/min)	**mg diluted to total 100 ml**	**IV Infusion Rate**
Dopamine μg/kg/min	2-20	6.0 x wt in kg	1 ml/hr = 1.0
Dobutamine ***μg/kg/min***	***5-20***	***6.0 x wt in kg***	***1 ml/hr = 1.0***
Epinephrine μg/kg/min	0.1-1.0	0.6 x wt in kg	1 ml/hr = 0.1
Isoproterenol μg/kg/min	0.1-1.0	0.6 x wt in kg	1 ml/hr = 0.1
Lidocaine	20-50	120 mg	1 ml/kg/hr = 20 μg/kg/min
General Formula	X mg added to Y ml IV fluid = 1000X/Y μg/ml D μg/kg/min x K kg = DK μg/min DK μg/min x 60 min/hr = 60DK μg/hr $\frac{60DKY}{1000X}$ = ml/hr needed to deliver D μg/kg/min		

Figure 8.1: Management of Multiple Trauma Algorithm

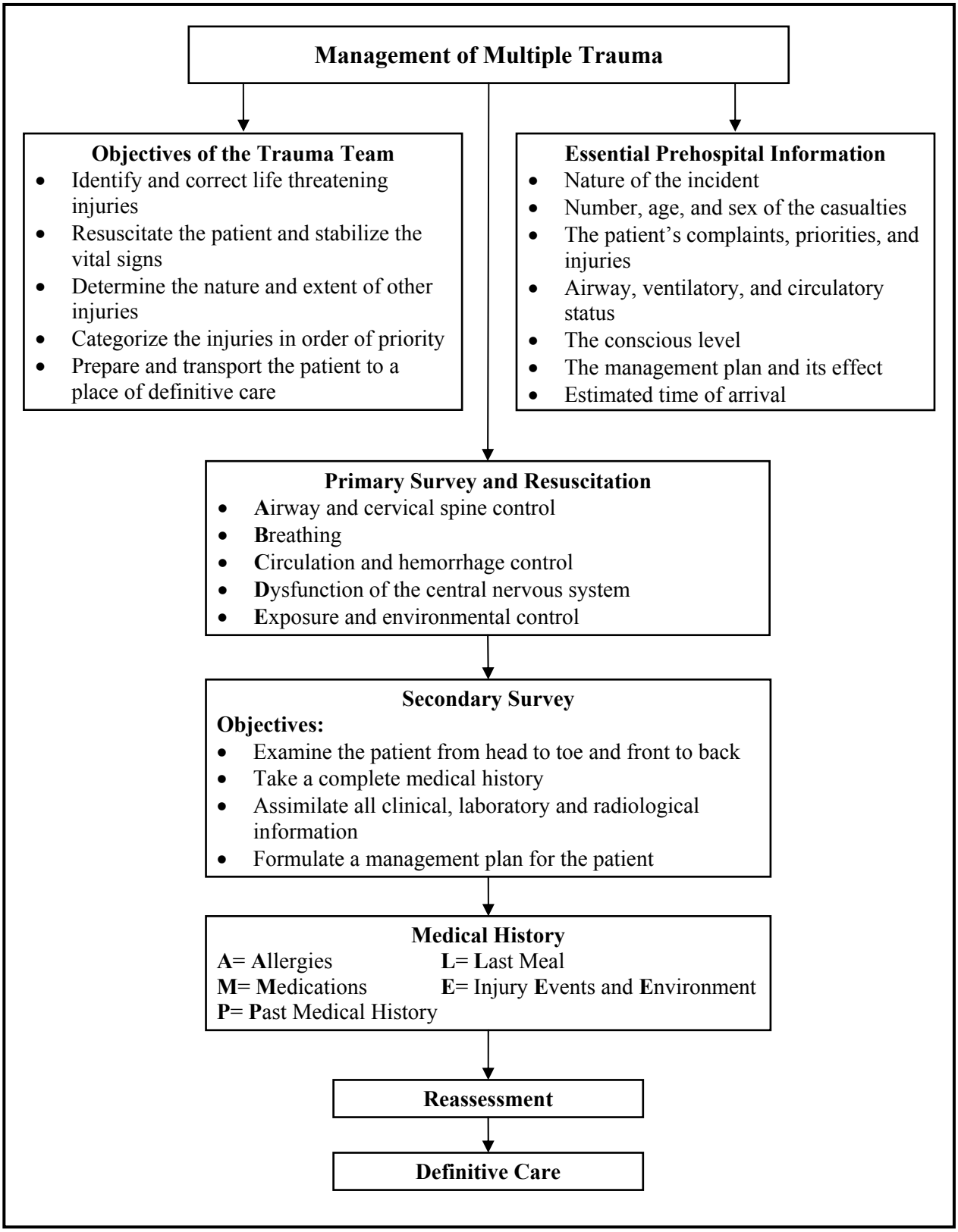


Figure 8.2: Primary Survey and Resuscitation Algorithm

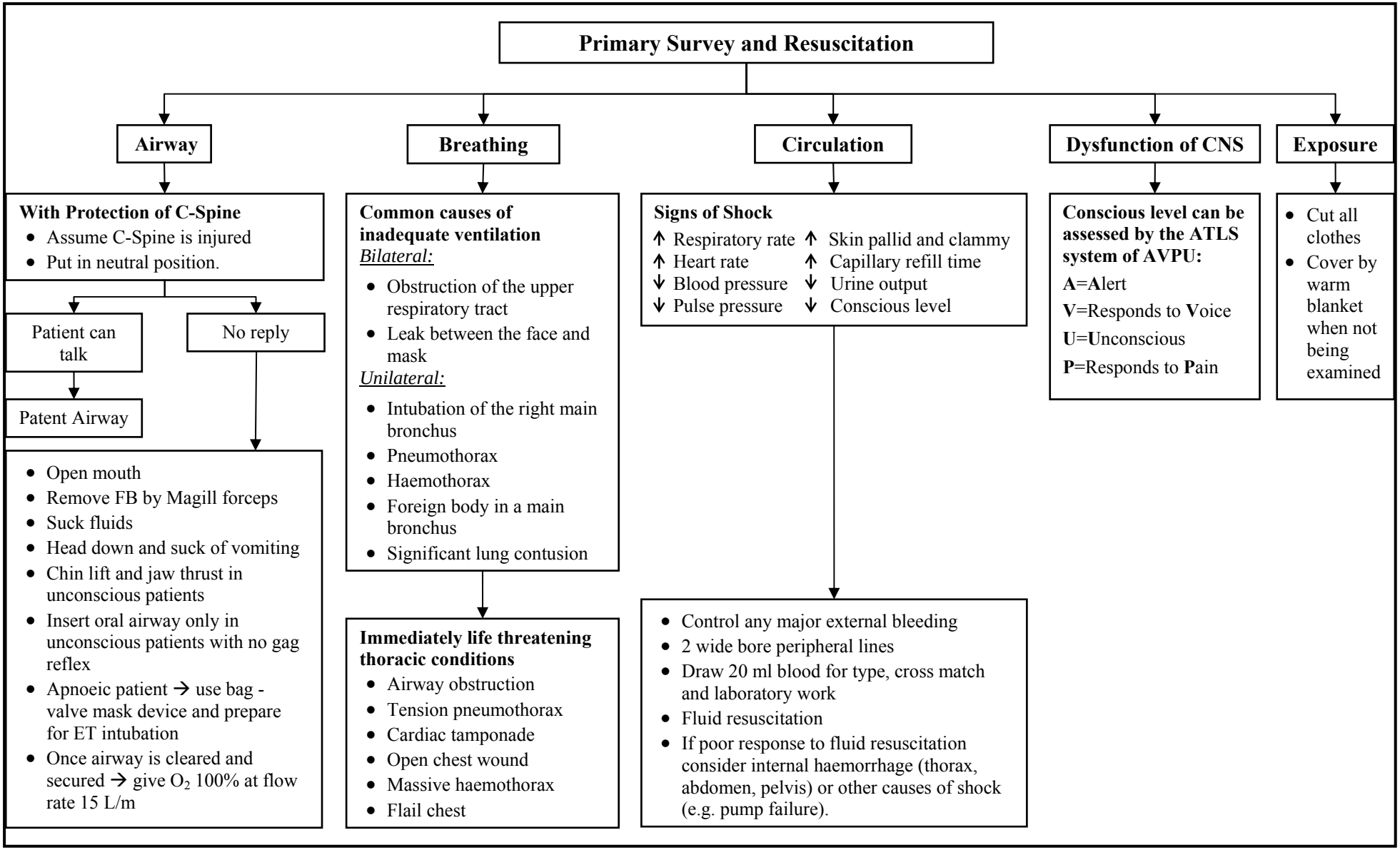


Figure 8.3: Secondary Survey Algorithm

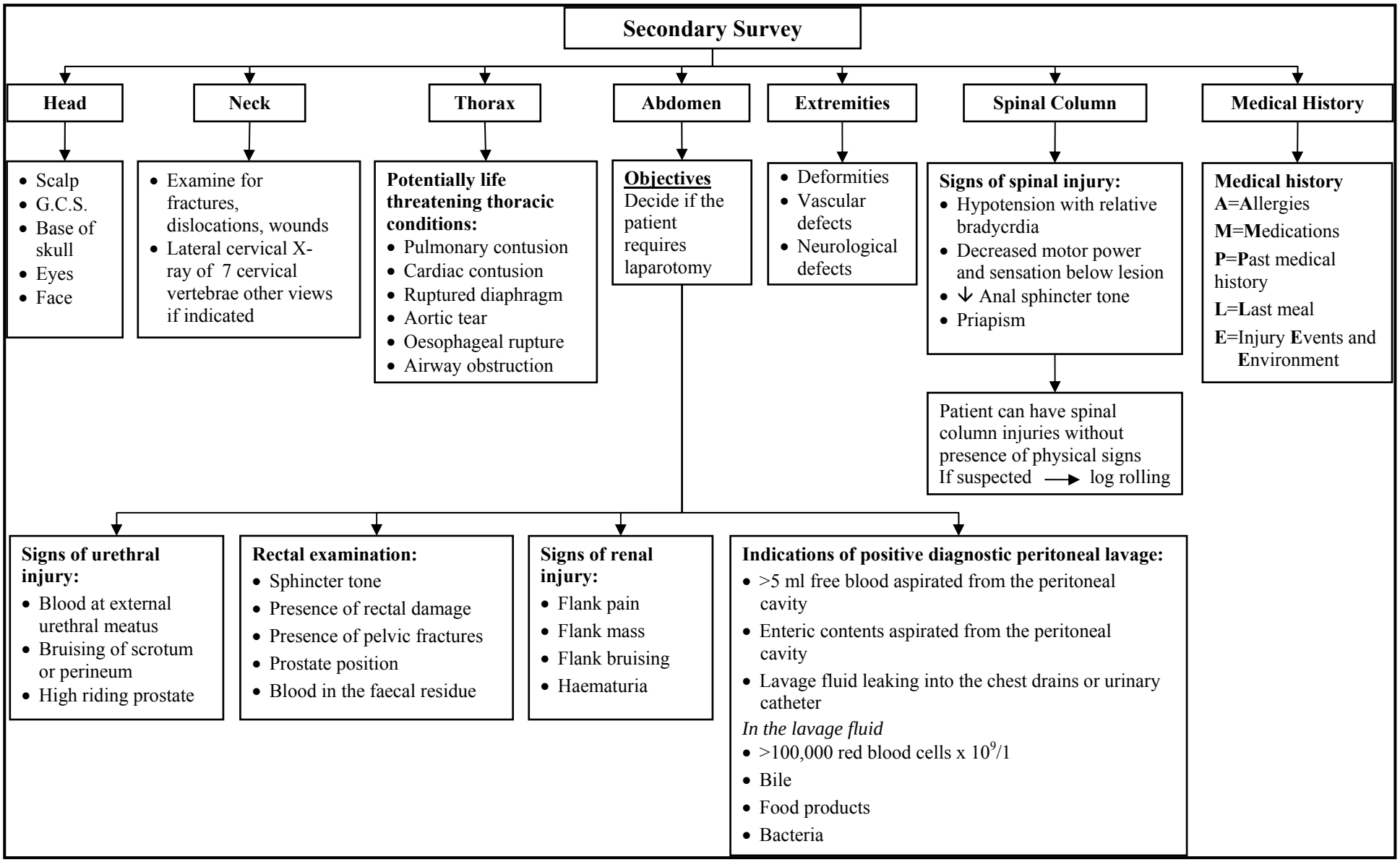
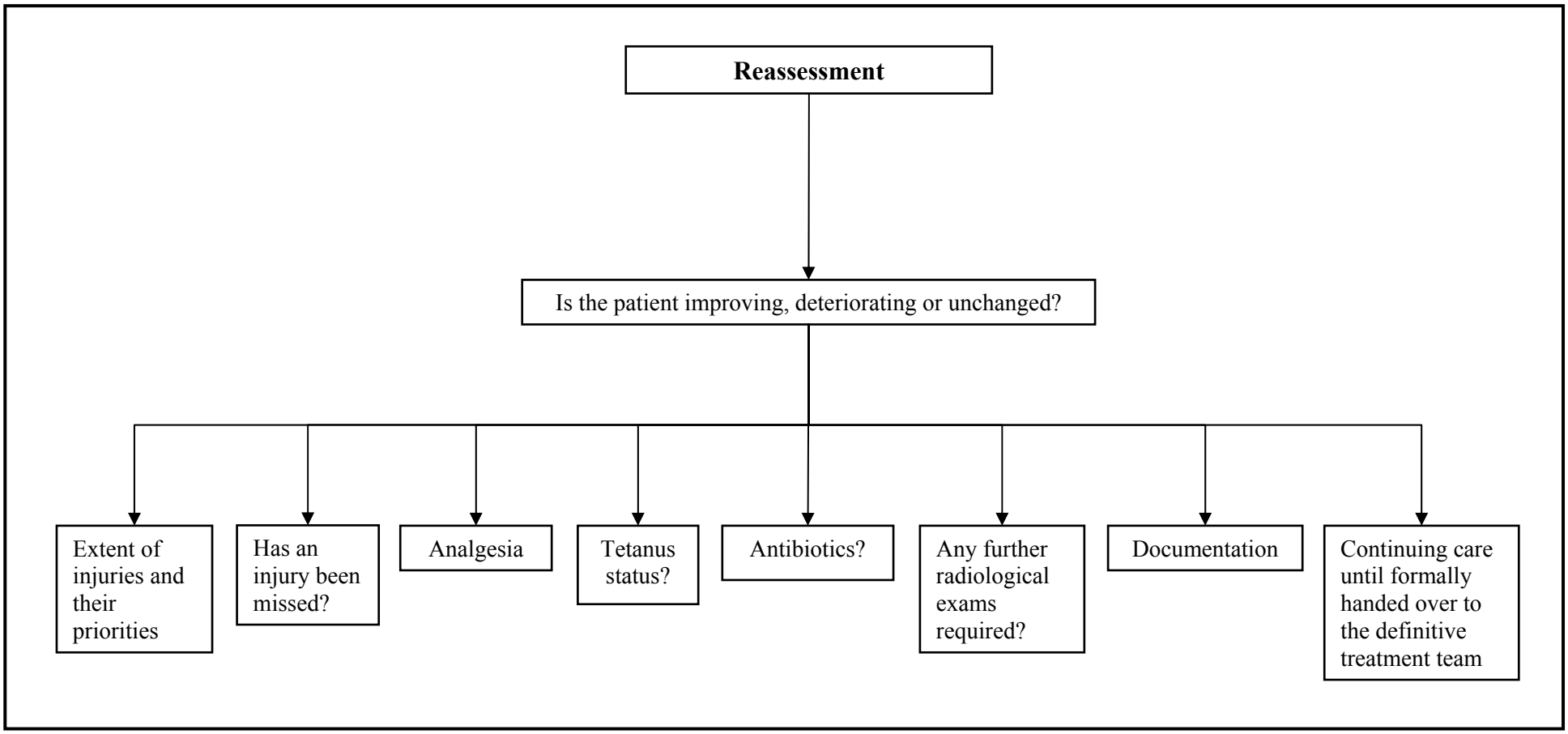


Figure 8.4: Reassessment Algorithm



Chapter 9: Acute Abdomen

GUIDELINE OBJECTIVES:

- Identify patients who require urgent resuscitation and management.
- Understand the importance of consulting senior staff in a timely manner.
- Diagnose abdominal pain using clinical judgment, laboratory investigations, and radiological tools.
- Rule out the most serious causes of abdominal pain before the most common causes.

1. DEFENITION

The "acute abdomen" is a term used to encompass a spectrum of surgical, medical, and gynecological conditions. Ranging from the trivial to the life-threatening, which require hospital admission, investigation, and treatment. The primary symptom of the condition is abdominal pain.

2. CLASSIFICATION

The acute abdomen is classified by the location, symptomatology, and intensity of pain.

Abdominal pain types include:

- Visceral.
- Parietal.
- Referred.
- Extra-abdominal.
- Metabolic and blood disease pain.
- Non-specific abdominal pain.

3. CLINICAL FEATURES

3.1. History

- History of:
  - A. Allergy to drugs or asthma.
  - M. Medication or history of drugs taken.
  - P. Past medical and surgical history including blood transfusion.
  - L. Last meal and last menses (female patients).
  - E. Event that could have led to the problem (in patient's opinion).
- Family history.
- Social history: alcohol abuse and smoking.
- History of present illness.

A. Abdominal Pain is the Presenting Complaint:

- Patient history (see table 9.1).

Characterization of the pain:

Any change in the character of the pain over time should be noted because change may highlight regarding the organ involved. For example, appendicitis often begins as a poorly localized cramping pain that becomes sharp and localizes in the right lower quadrant.

- Location (see table 9.2):
  - Epigastric pain is associated with pancreatitis, peptic ulcer diseases, myocardial infraction (MI), aortic aneurysms, and gastritis.
  - Right upper quadrant pain is consistent with hepatitis and cholecystitis (see figure 9.3).
  - Right lower quadrant pain may be seen in patient with appendicitis, Crohn's disease, diverticulitis, or gynecologic disorders (see figure 9.2).
  - Left lower quadrant pain is associated with diverticulitits and gynecologic disorders (see figure 9.1).
- Quality:
  - Cramping pain suggests obstruction of a hallow viscus, such as occurs in cholecystitis, small bowel obstruction, or renal colic.
  - A burning pain is characteristic of gastroesophageal reflux and peptic ulcer disease.
  - Sharp, localized pain suggests peritoneal irritation.
- Radiation:
  - Left shoulder: Pain may radiate to the left shoulder in patients with a perforated peptic ulcer, subphrenic abscess, splenic rupture, or mononucleosis.
  - Chest: Pain may radiate to the chest in patients with gastroeosphageal reflux disease (GERD), peptic ulcer disease, or hiatal hernia.
  - Back: Radiation of pain to the back is seen primarily in association with pancreatitis, abdominal aneurysms, and acute aortic dissection.
- Timing:
  - An abrupt onset of pain occurs with the perforation of hollow viscus.
  - A waxing and waning pain is inductive of obstruction (e.g. small bowel obstruction, (cholecystitis).
- Provocative and palliative factors:
  - Movement is painful for patients with peritonitis.
  - Patients with peritonitis often lean forward to improve the pain.
  - Food may exacerbate the pain (as in pancreaitis) or alleviate the pain (as in peptic ulcer disease).
  - Antacid medications usually relieve peptic ulcer disease pain.

B. Associated Symptoms:

Gastrointestinal symptoms:

- Anorexia and nausea are important to note, because the diagnosis of appendicitis is excluded if anorexia is not present.

- Nausea & vomiting:
  - Bilious vomiting in small bowel obstruction.
  - Strangulation.
- Vomiting:
  - Bilious or feculent vomitous suggests a bowel obstruction.
  - "Coffee grounds" or frank blood in the vomitus suggests peptic ulcer disease, a Mallory-Weiss tear, or bleeding esophageal varices.
  - Non-bilious vomiting for obstructions proximal to ampulla of vater.
  - Feculent vomiting in distal small gut obstruction, large bowel obstruction, and bowel.

  Vomiting is very prominent in:
  - Acute gastritis.
  - Acute pancreatitis.
  - High intestinal obstruction.
  - Biliary colic and acute cholecystitis.
  - Ureteric colic.
  - Acute appendicitis (anorexia with pain is usually seen or infrequent vomiting).
- Diarrhea:
  - Bloody diarrhea suggests inflammatory bowel disease or diverticultitis.
  - Melena is consistent with an upper gastrointestinal bleeding.

  Diarrhea with pain is mainly medical. The following are some exceptions:
  - Obstructed richter's hernia.
  - Superior mesenteric vascular occlusion.
  - Pelvic abscess.
  - Pelvic appendicitis.
- Constipation:
  - Constipation or obstication suggests obstruction.
  - Progressive intestinal obstruction from a neoplasm or inflammatory bowel disease.
  - Paralytic Ileus.
  - Post operative.
  - Obstructed groin hernia.

Fever, sweats, and chills:

Are noted in infectious processes. Weight loss may be seen with cancer, inflammatory bowel disease, or ischemic bowel syndromes.

Gynecological and urological symptoms:

Should be noted because these symptoms may rule out a gastrointestinal process.

3.2. Physical Examination

In cases where the diagnosis is not clear, repeated physical examinations at frequent intervals will help clarify the diagnosis. *Determine the vital signs, including blood pressure, pulse, temperature, respiratory rate and oxygen saturation.*

The physical exam progresses with inspection first, followed by auscultation, and then palpation. The patient's general appearance indicates disease severity, duration, and often the cause of the underlying condition.

A. Vital Signs:

A full evaluation of vital signs can highlight patient compensation, de-compensation, and sometimes help pinpoint the cause of the abdominal pain.

Examples:

- Rapid respiration may indicate pneumonia.
- Tachycardia and hypotension indicate patient de-compensation.
- Irregular pulse with atrial fibrillation (AF) may indicate mesenteric ischemia.
- Temperature is elevated in gastrointestinal perforation and normal in gastrointestinal obstruction.

Look, Listen, and then Feel

B. Inspection:

On inspection, visualize abdominal asymmetry, peristaltic intestinal movement, and general condition.

Look for abdominal distension:

- Scaphoid or flat in peptic ulcer.
- Distended in ascitis or intestinal obstruction.
- Visible peristalsis in a thin or malnourished patient (with obstruction).
- Erythema or discoloration:
  - Peri-umbilical - Cullen sign.
  - Inguinal – Fox sign.
  - Flanks - Grey Turner sign. Seen in Hemorrhagic pancreatitis or any other cause of haemoperitoneum.

C. Auscultation:

Auscultation should always be performed before palpation so that abdominal sounds may be evaluated before they are altered by palpation, and for the general compliance of the patient.

The presence of abdominal sounds does not rule out serious abdominal pathology.

Listen for bowel sounds:

- Absence may be a sign of peritonitis or ileus.
- High pitched tinkling may indicate obstruction.

Cardiopulmonary examination, check for:

- Possible myocardial infarction (MI).
- Basal pneumonia.
- Pleural effusion.

D. Palpation:

Generally, gentle percussion should precede palpation. Ask the patient to point to the site of maximal pain, palpating this area last.

- Percuss to differentiate gas from liquid.
- Palpate the abdomen.
- Start away from the site of tenderness.
- Check for masses or tumors.
- Determine the site of maximum tenderness.
- Check for abdominal rigidity.
- Guarding- *involuntary spasm of muscles during palpation.*
- Rigidity- *when abdominal muscles are tense and board-like.*
- Local Right Iliac Fossa tenderness, consider:
  - Acute appendicitis, positive rovsing's sign in acute appendicitis, positive obturator sign in pelvic appendicitis, and positive psoas sign in retrocaecal appendicitis.
  - Acute salpingitis in females.
  - Amoebiasis of caecum.
  - Low grade, poorly localized tenderness, consider intestinal obstruction.
- Tenderness out of proportion to examination, consider:
  - Mesenteric ischemia.
  - Acute pancreatitis.
- Flank tenderness: Consider principle abscess.
- Positive Murphy's sign in acute cholecystitis.
- Thumping tenderness over lower ribs with inflamed diaphragm, liver, or spleen.
- Pulsatile abdominal mass with hypotension (leaking abdominal aortic aneurysm AAA).
- Cutaneous hyperesthesia indicates involvement of parietal peritoneum.

E. Genitourinary and Rectal Examinations:

- Groin for incarcerated hernia.
- Rectum for signs of trauma, abscess, obstruction.
- Vagina for pelvic abscess, ectopic pregnancy, distended pouch of Douglas.

Abdominal examination is not complete unless a back, rectal, and hernial orifices examination occurs.

- Per male genital examination:
  - Tenderness mass.
  - Pulsations.
  - Testicular torsion is an emergency.
- Per rectal examination:
  - Tenderness.
  - Indurations mass.
  - Frank blood.
- Per vaginal examination:
  - Bleeding, discharge.
  - Cervical motion tenderness.
  - Adnexial masses or tenderness.
  - Uterine size or contour.

See figure 9.4.

3.3. Specific Findings on Physical Examination:

A. Peritoneal Irritation:

The location of peritoneal irritation (somatic pain) depends on the anatomical position of the diseased organ. Peritoneal irritation can be localized or generalized. Findings that are important indications for surgery are:

- Abdominal tenderness, suggesting inflammation of an underlying organ.
- Rebound abdominal tenderness elicited by percussion, which confirms peritoneal irritation.
- Involuntary contraction of the abdominal wall, a sign of peritoneal irritation, which presents as local guarding or generalized rigidity.

B. Non-Specific Abdominal Pain:

It is the most common cause of abdominal pain in late childhood and early adolescence. It is a colicky pain with some localization that becomes worse after meals. Bowel sounds may be increased and a palpable mass of feces may be present in the right or left iliac fossa. Commonly, the cause of pain is constipation, irritable bowel, and chronic spasm.

C. Referred Abdominal Pain:

- The location of referred abdominal pain is based on the embryological origin of the affected organ.
- Fore gut pain (stomach, duodenum, gall bladder) is referred to the upper abdomen.
- Mid gut pain (small intestine, appendix, right colon) is referred to the mid abdomen.
- Hind gut pain (mid transverse, descending, sigmoid colon and rectum) occurs in the lower abdomen.
- Diseased retroperitoneal organs (kidney, pancreas) may present with back pain.
- Ureteric pain radiates to the testicle or labia.
- Diaphragmatic irritation presents as shoulder tip pain.

D. Extra-Abdominal Causes:

- Otitis media and upper respiratory tract infections especially in children.
- Pneumonia especially in children.

E. Metabolic and Blood Diseases:

- Diabetic keto-acidosis.
- Hypercalcemia.
- Sickle cell.

3.4. Investigations

A. Laboratory.

- Complete blood count with differential.
- Blood glucose.
- Electrolytes, blood urea, and creatinine.
- Urine dipstick.
- Amylase and lipase.
- Liver function test.

B. Radiology.

- Chest x-ray A.P. lateral for:
  - Basal pneumonia.
  - Ruptured esophagus.
  - Elevated hemi- diaphragm.
  - Free gas under diaphragm.
- Abdominal x-ray film; supine and standing:
  - Air-fluid levels.
  - Stones.
  - Ascites.
  - Eggshell calcification in AAA.
  - Air in the biliary tree.
  - Obliteration of psoas shadow in retro- peritoneal disease.
  - Right lower quadrant sentinel loop in acute appendicitis.
- Ultrasonography.
- Computerized tomography (CT) abdomen for AAA, pancreatic disease, or ureteric colic (non- contrast).
- Intravenous urogram (IVU).
- Magnetic resonance image (MRI).
- Selective mesenteric angiography for ischemia or hemorrhage.
- Upper gastrointestinal (GI) series.
- Barium enema.

C. Various Forms of Endoscopy (proctoscopy upper and lower).

D. Diagnostic Laparoscopy.

4. MANAGEMENT

- The treatment of patients with abdominal pain depends on the severity of the pain, its rapidity of onset, and the nature of the causal condition:
  - Severe pain with an abrupt onset often reflects a GI disorder that will require surgical intervention; immediate consultation with a surgeon is indicated.
  - Less severe pain, the pain that doesn't completely incapacitate the patient, should not be treated aggressively with drugs until a diagnosis is established.
- As a general rule, analgesic drugs may be prescribed to patients with persistent pain; but opiates should be avoided, if possible. Opiates can aggravate the underlying medical condition causing the pain.
- General use of antispasmodics is a mainstay of abdominal pain treatment.

Key points

- Abdominal pain is one of the most common presentations in emergency department.

  The most important concern is to decide if the condition requires surgical intervention or can be managed medically.
- Although abdominal pain is common and often trivial, acute and severe pain accompanies intra-abdominal disease.
- Pain out of proportion to physical signs may suggest conditions such as mesenteric vascular occlusion – pancreatitis.
- Consider gynecological causes in women of child bearing age.
- Amylase may be normal in acute pancreatitis and it can be raised in other pathologies.
- Steroids can mask the classic inflammatory signs.
- B-blockers can block the tachycardic response to inflammation and hypovolemia.
- White blood cells (WBCs) may be normal in the face of acute inflammation.
- Analgesics are not contraindicated after the diagnosis is made.

Table 9.1: History of Patients with Acute Abdominal Pain

Question	Potential Responses and Indications
Where is the pain?	• Depends on location.
What is the pain like?	• Acute waves of sharp constricting pain that "take the breath away" (renal or biliary colic). • Waves of dull pain with vomiting (intestinal obstruction). • Colicky pain that becomes steady (appendicitis, strangulating intestinal obstruction, mesenteric ischemia). • Sharp, constant pain, worsened by movement (peritonitis). • Tearing pain (dissecting aneurysm). • Dull ache (appendicitis, diverticulitis, pyelonephritis).
Have you had it before?	• Yes suggests recurrent problems such as ulcer disease, gallstone colic, diverticulitis, or mittelschmerz.
Was the onset sudden?	• Sudden: "like a light switching on" (perforated ulcer, renal stone, ruptured ectopic pregnancy, torsion of ovary or testis, some ruptured aneurysms). • Less sudden: most other causes.
How severe is the pain?	• Severe pain (perforated viscera, kidney stone, peritonitis, pancreatitis). • Pain out of proportion to physical findings (mesenteric ischemia).
Does the pain travel to any other part of the body?	• Right scapula (gallbladder pain). • Left shoulder region (ruptured spleen, pancreatitis). • Pubis or vagina (renal pain). • Back (ruptured aortic aneurysm).
What relieves the pain?	• Antacids (peptic ulcer disease). • Lying as quietly as possible (peritonitis).
What other symptoms occur with the pain?	• Vomiting precedes pain and is followed by diarrhoea (gastroenteritis). • Delayed vomiting, absent bowel movement, and flatus (acute intestinal obstruction; the delay increases with a lower site of obstruction). • Severe vomiting precedes intense epigastric, left chest, or shoulder pain (emetic perforation of the intra-abdominal oesophagus).

Table 9.2: Selected Differential Diagnosis of Abdominal Pain

Pain Location	Possible Diagnosis
Right Upper Quadrant	**Biliary:** cholecystitis**,** cholellthasis, cholangitls.
	Colonic: colitis, diverticulitis.
	Hepatic: abscess, hepatitis, mass.
	Pulmonary: pneumonia, embolus.
	Renal: nephrolithlasis, pyelonephritis.
Epigastric	**Biliary:** cholecystitis, cholelithlasis, cholangitls.
	Cardiac: myocardial infarction, pericarditis.
	Gastric: esophagitis, gastritis, peptic ulcer.
	Pancreatic: mass, pancreatitis.
	Vascular: aortic dissection, mesenteric ischemia.
Left Upper Quadrant	**Cardiac**: angina, myocardial infarction, pericarditis.
	Gastric: esophagitis, gastritis, peptic ulcer.
	Pancreatic : mass, pancreatitis**.**
	Renal: nephrolithiasis, pyelonephritis.
	Vascular: aortic dissection, mesenteric ischemia.
Periumbilical	**Colonic:** early appendicitis.
	Gastric: esophagitis, gastritis, peptic ulcer, small- bowel mass or obstruction.
	Vascular: aortic dissection, mesenteric ischemia.
Right Lower Quadrant	**Colonic:** appendicitis, colitis, diverticulitis, (IBD, IBS).
	Gynecologic: ectopic pregnancy, fibroids, ovarian mass, torsion, (PID).
	Renal: nephrolithiasis, pyelonephritis.
Suprapublic	**Colonic:** appendicitis, colitis, diverticulitis, (IBD, IBS).
	Gynecologic: ectopic pregnancy, fibroids, ovarian mass, torsion, (PID).
	Renal: cystitis, nephrolithiasis, pyelonephritis
Left Lower Quadrant	**Colonic:** colitis, diverticulitis, (IBD, IBS).
	Gynecologic: ectopic pregnancy, fibrolds, ovarian, mass, torsion, (PID).
	Renal: nephrolithlasis, pyelonephritis.
Any Location	**Abdominal wall:** herpes zoster, musde strain, hernia.
	Other: bowel obstruction, mesenteric ischemia, peritonitis, narcotic withdrawal, sickle cell crises, porphyria, (IBD), heavy metal poisoning.

IBD= Inflammatory bowel disease **IBS**= Irritable bowel syndrome
PID= Pelvic inflammatory disease

Figure 9.1: Algorithm for Evaluation of Left Lower Quadrant Abdominal Pain

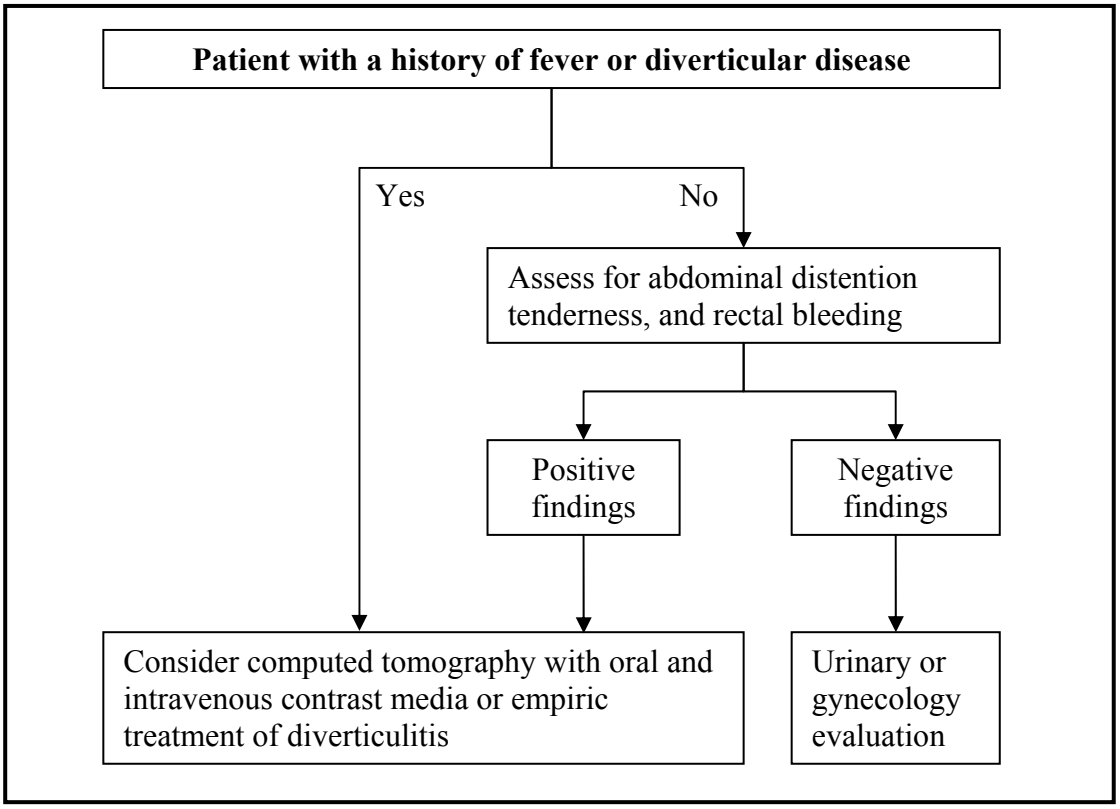


Figure 9.2: Algorithm for Evaluation of Right Lower Quadrant Abdominal Pain

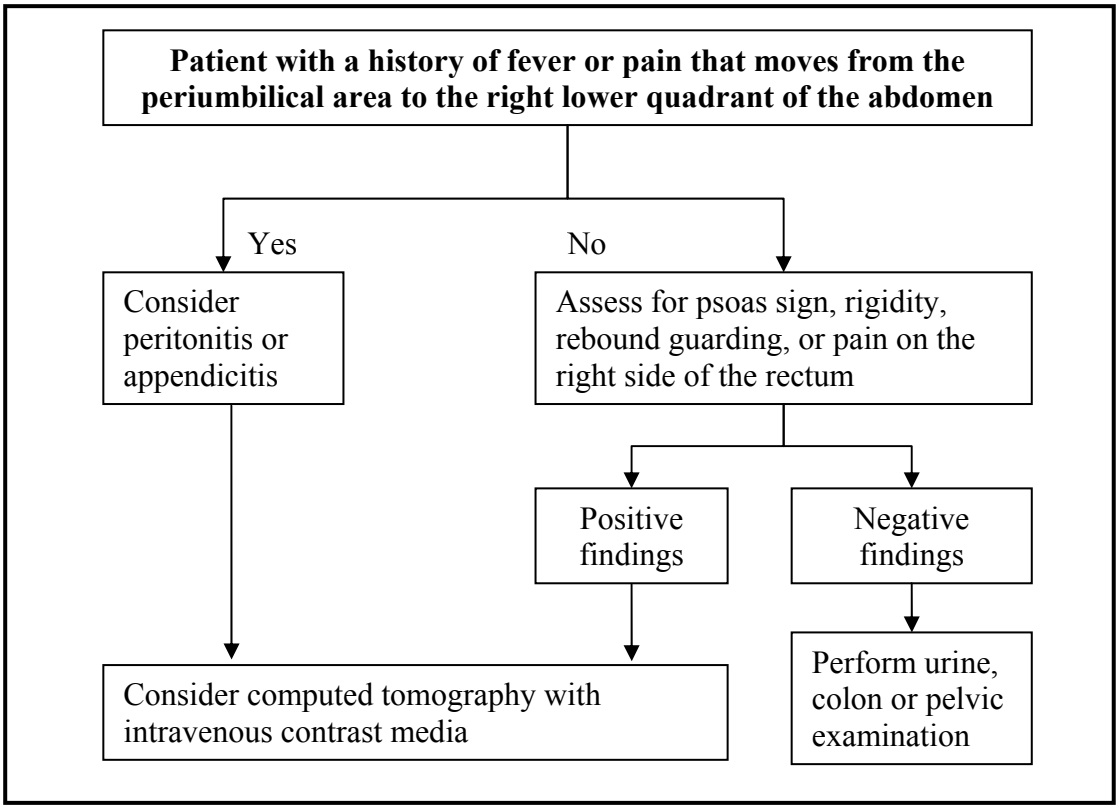


Figure 9.3: Algorithm for Evaluation of Right Upper Quadrant Abdominal Pain

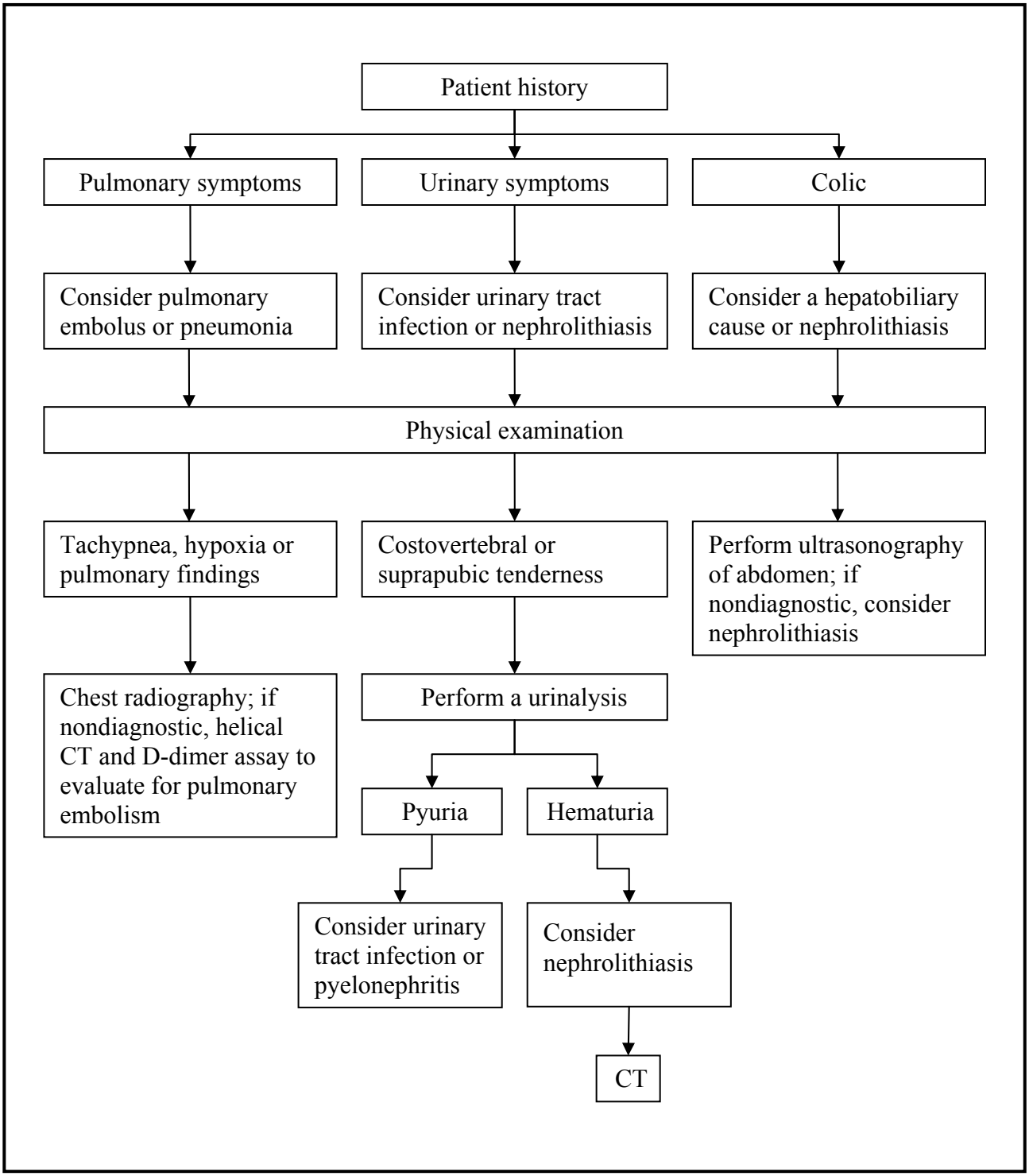
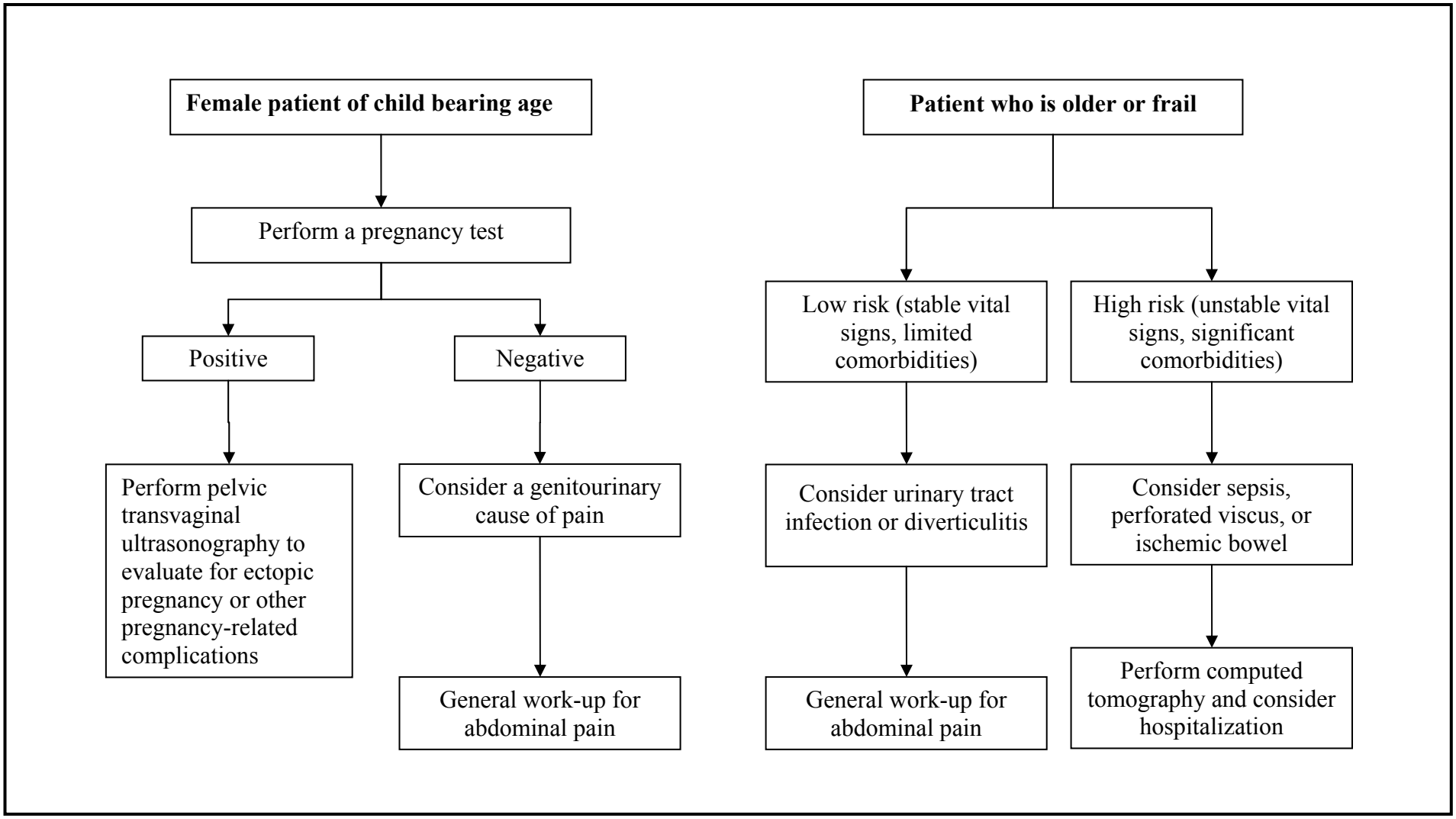


Figure 9.4: Algorithm for Evaluation of Abdominal Pain in Special Populations



Chapter 10: Shock

GUIDELINE OBJECTIVES:

- Define shock.
- Recognize types of shock.
- Recognize presence of shock with variations in clinical presentation.
- Apply principles of management of shock.
- Monitor response to treatment.

1. INTRODUCTION

Shock is a state of inadequate tissue perfusion. During shock, the body's ability to maintain normal cellular function and structure is impaired. Shock may involve decreased circulating volume, decreased cardiac output, and vasodilatation, sometimes with shutting of blood to bypass capillary exchange beds. However, *shock does not equal hypotension*. Whether shock results from hemorrhage, sepsis, or cardiac failure, mortality rates exceed 20%. Diagnosed traumatic, cardiogenic, or septic shock constitutes about 1-3% of all emergency department visits.

2. CLASSIFICATION

Shock can be classified into:

A. Hypovolemic Shock:

- Hemorrhagic
- Non- haemorrhagic

B. Neurogenic Shock.

C. Cardiogenic Shock.

D. Septic Shock.

E. Anaphylactic Shock.

F. Obstructive Shock:

Due to pericardial tamponade, tension pneumothorax, massive pulmonary embolism.

3. CLINICAL FEATURES

Patients frequently present to the emergency department (ED) in shock with no obvious cause. Rapid recognition of shock requires the integration of information from immediate history and physical examination, and it can be strongly supported by the presence of a worsening base deficit or lactic acidosis (see figure 10.1). The diagnosis of shock depends on the clinical presentation and interpretation of the clinical findings.

3.1. Hypovolemic Shock

A. Types:

Hemorrhagic:

Caused by decreased circulating volume from loss of one or combination of the following: red cell mass, plasma, and extracellular fluid. Hemorrhagic shock is the most common cause of shock in injured patients, and usually results from acute blood loss.

> **Initially, haemoglobin and heamatocrite levels do not reflect the degree of bleeding, hemoconcentraition causes these readings to be falsely normal.**

A healthy adult can usually maintain his systolic blood pressure until blood loss exceeds 30% of his blood volume.

Classes of Hemorrhagic Shock:

- Class 1 hemorrhage: 0 to 15% total blood volume (TBV) lost:
  - Slightly tachycardic.
  - Usually no significant change in blood pressure (BP), pulse pressure, or respiratory rate.
- Class 2 hemorrhage: 15 to 30% of TBV lost:
  - Heart rate (HR) >100 beats per minute, tachypnea, and decreased pulse pressure.
- Class 3 hemorrhage: 30 to 40% of TBV lost:
  - Marked tachycardia, tachypnea, decreased systolic BP, and oliguria.
- Class 4 hemorrhage: > 40% of TBV lost:
  - Marked tachycardia, and severe or immeasurable BP.
  - Narrowed pulse pressure, markedly decreased (or no) urinary output.
  - Immediately life threatening.

Non –Hemorrhagic:

Caused by loss of plasma (burns) and loss of fluid and electrolytes (emesis, diarrhea, or bowel obstruction).

B. Management:

Treatment of hypovolemia focuses on simultaneous cessation of ongoing hemorrhage and restoration of circulating blood volume. Treatment for hypovolemia is usually instituted before a cause is identified *(Call the surgeon as early as possible).*

- Airway:
  - Open and secure the airway.
- Breathing:
  - Ensure adequate ventilation/oxygenation.
- Circulation:
  - Peripheral venous access established with two 18 gauge or larger cannulae.
  - Stop external hemorrhage with direct pressure.
  - Start intravenous (IV) fluid resuscitation.

- Fluid resuscitation should be instituted with a balanced crystalloid solution until blood is available (if needed). In an adult, start with 2 L lactated Ringer's solution. In a child, 20 ml/kg of lactated Ringer's solution should be infused as bolus, and may be repeated once. Continue with type specific blood or O negative blood (if available). If blood is not available, repeat another 2 L of crystalloid solution.
- Monitor indices of resuscitation.
- Assess for 'obstructive shock':

  a. Tension pneumothorax: needle thoracostomy.

  b. Cardiac tamponade: pericardocentesis.

- Class 1 and 2 hemorrhage can usually be treated by crystalloids and/or colloids with no blood transfusion.
- Class 3 usually requires blood transfusion.
- Class 4 hemorrhage, blood transfusion is life saving.

3.2. Neurogenic Shock

Neurogenic shock is usually due to injuries to the spinal cord from fractures of the cervical or high thoracic vertebrae that disrupt sympathetic regulation of peripheral vascular tone. This will result in loss of vasomotor tone to peripheral arterial beds, resulting in increased vascular capacitance, decreased venous return, and decreased cardiac output.

A. Clinical Features:

- Hypotension and bradycardia, with warm, perfused extremities. Presence of a sensory or motor deficit consistent with cord injury.
- Tachycardia can be present.
- The diagnosis is often made once hypovolemia has been excluded, and a vertebral fracture is identified.

B. Management:

- Airway:
  - Open and secure the airway with attention to cervical spine control.
- Breathing:
  - Oxygenate.
  - Ventilate.
- Circulation:
  - Start IV fluid resuscitation, restoration of intravascular volume may be sufficient to restore blood pressure and perfusion.
  - If hypotension persists, initiate vasopressor support.

3.3. Cardiogenic Shock

Cardiogenic shock refers to a failure of the circulatory pump leading to diminished forward flow and subsequent tissue hypoxia. Cardiogenic shock can be caused by either:

- Significant cardiac injury (myocardial contusion).

- Intrinsic cardiac disease (myocardial infarction, cardiac arrhythmia).

A. Clinical Features:

The diagnosis of shock due to cardiac pump failure requires exclusion of other causes, and demonstration of diminished cardiac function (decreased cardiac output/echocardiographic evidence of cardiac dysfunction).

B. Management:

Call for specialist as soon as possible.

- Airway:
  - Open and secure the airway.
- Breathing:
  - Oxygenate.
  - Ventilate.
- Circulation:
  - Fluid resuscitation should be instituted judiciously in patients with known cardiac dysfunction.
  - Invasive hemodynamic monitoring guides therapy and assesses the success of treatment
  - Begin vasopressor or inotropic support; norepinephine (0.5 μg/min) and dobutamine (5 μg/kg/min) are common empirical agents.

3.4. Septic Shock

The mortality rate from septic shock is 50%, and increases when patients have co morbid problems such as immunosuppression, diabetes, or cardiovascular compromise. Septic shock is defined as the presence of sepsis with refractory hypotension. Septic shock is caused by an infectious agent.

A Clinical Features:

Septic shock causes three major effects that must be addressed during resuscitation:

- Relative hypovolemia.
- Cardiovascular depression.
- Induction of systemic inflammation.

B. Management:

- Call for specialists as soon as possible.
- Ensure adequate oxygenation; remove the patient's work of breathing.
- Administer 20 mL/kg of crystalloid or 5 mL/kg of colloid solution, and titrate the infusion to adequate central venous pressure and urine output.
- Begin antimicrobial therapy; attempt surgical drainage or debridement.
- *Early use of steroids in severe sepsis is now recommended.*

3.5. Anaphylactic Shock

Anaphylaxis is a severe hypersensitivity reaction with multi system involvement that commonly includes airway compromise and hypotension. The main three causes of anaphylaxis include: medications, foods, and insects.

A Clinical Features:

- Shortness of breath.
- Hypotension.
- Altered mental status.
- Cardiac arrest (in some cases).
- Peripheral skin flushing, hives, and uticaria.

B. Management:

- Airway, breathing, and circulation, (ABCs): oxygen, IV fluids, and epinephrine.
- Decontamination for allergic reactions: antihistamines (e.g., diphenhydrageneralmine and ranitidine), corticosteroids, and asthma medications (e.g., albuterol).

4. LABORATORY INVESTIGATIONS

Laboratory and diagnostic testing that should be obtained for all shock emergencies:

- Complete blood count (CBC).
- Blood grouping and cross matching.
- Serum chemistry (glucose, blood urea nitrogen, ,and creatinine).
- Liver function test.
- Coagulation profile.
- Chest x-ray.

If cardiogenic shock suspected:

- Cardiac profile should be evaluated.
- Electrocardiogram (ECG).

In hypo-perfused patients.

- Serum lactate level.
- Arterial blood gas values.
- Base deficit levels.

Specific radiographic testing should be ordered based on the cause of shock.

5. MONITORING

Circulation must be monitored by:

- Continuous ECG.
- Continuous pulse oximetry.
- Serial blood pressures.
- Urine output (expected rate of 1ml/kg/hr).

Response to therapy judged by:

- Improvement in patient's mental status and speech.
- Pulse, blood pressure, and respirations return to baseline.
- Adequate urine output (should be at least 30 cc/hr in adults).
- Return of capillary refill/ skin perfusion.
- Normal parameter central venous pressure (CVP).
- Lab data (less important than clinical parameters).

Monitoring CVP in shock patients:

- Central venous pressure (CVP) measures the ability of the heart (right side) to accept a fluid load.
- Central venous pressure (CVP) line measurements may be helpful for patients with:
  - Preexistent cardiac dysfunction chronic heart failure (e.g. CHF).
  - Neurogenic shock.
  - Myocardial contusion.
  - Suspected cardiac tamponade.
- A central venous line is not needed for most trauma patients.

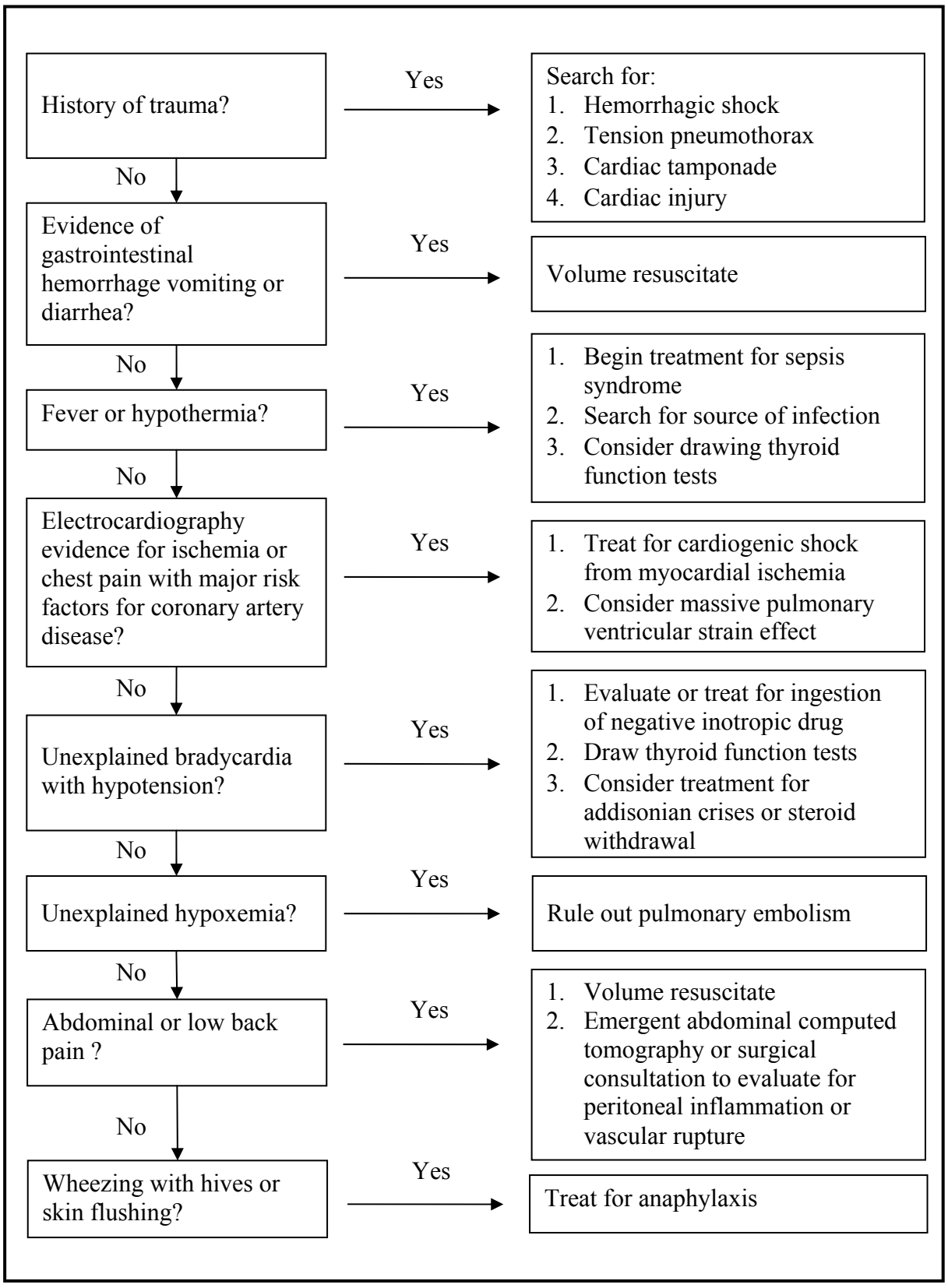
Considerations if the patient fails to respond to shock treatment:

- Unrecognized fluid loss.
- Ventilation problems.
- Acute gastric distention: Treat by naso-gastric (NG) tube/ suction.
- Cardiac tamponade.
- Acute myocardial infarction.
- Diabetic ketoacidosis.
- Neurogenic shock.
- Hypothermia.

Key Points:

- Circulatory shock can occur with normal arterial BP readings, and not all patients with arterial hypotension have circulatory shock.
- A base deficit less than 4mEq/L or a serum lactate > 4.0 mmol/L indicates the presence of widespread circulatory insufficiency in suspected shock.
- Urine output is a reliable index of vital organ perfusion in patients with suspected shock.
- Ill patients with tachycardia, a worsening base deficit, and low urine output should be diagnosed with circulatory shock.
- A downward trend of the serum lactate concentration or upward trend of the base deficit, with improving vital signs and urine output, is a reliable gauge of the adequacy of resuscitation for any cause of shock.
- Resuscitation should continue until the lactate concentration drops below 2mM/L.

Figure 10.1: Clinical Algorithm for Undifferentiated Shock



Chapter 11: Burn Management

GUIDELINE OBJECTIVES:

- Identify causes of burns.
- Recognize burn classification according to surface area and depth.
- Understand and be able to perform primary and secondary burn surveys.
- Recognize indications for hospital admission.
- Recognize indications for transfer to a burn unit.
- Recognize and be able to recognize high risk burns.

1. INTRODUCTION

Burns are one of the most devastating conditions encountered in medicine. The injury represents an assault on all aspects of the patient, from the physical to the psychological. Burns affect all ages, from infants to the elderly, and are a problem in both the developed and developing world. Most burns are due to flame injuries, followed by burns due to scalds. The most infrequent burns are those caused by electrocution and chemical injuries.

2. CLASSIFICATION

Burn injuries can be classified according to the depth or extent of the injury.

2.1. Depth

Classified as partial or full thickness, and divided into four degrees. Burn depths are routinely underestimated during the initial examination. Devitalized tissue may appear viable for some time after the injury, and often, some degree of progressive microvascular thrombosis is observed on the wound periphery. Consequently, the wound appearance changes over the days following injury. *Serial examination of burn wounds is recommended*.

A. First-Degree Burns:

- Affects epidermis, usually red, dry, and painful (i.e. sun burn).
- Burns initially termed first-degree are often actually superficial second-degree burns, with sloughing occurring the next day.

B. Second-Degree Burns:

- Affects dermal layer.
- Red, wet, and very painful.
- Their depth, ability to heal, and propensity to form hypertrophic scars vary enormously.

C. Third-Degree Burns:

- Affects deep dermal layer & subcutaneous tissues. Third degree burns appear leathery in consistency, dry, insensate, and waxy.
- Heals by contraction and limited epithelial migration, with resulting hypertrophic and unstable cover.

- Burn blisters can overlie both second- and third-degree burns.

D. Fourth-Degree Burns:

- Involve underlying subcutaneous tissue, tendon, or bone.

2.2. Extent

The extent is expressed as the percentage of the total body surface area that was burned. Erythematous skin areas should not be included when calculating burn areas. Erythema will fade within a few hours.

A. Palmar Surface:

The surface area of a patient's palm (including fingers) is roughly 1% of total body surface area. Palmar surface can be used to estimate relatively small burns (< 15% of total surface area) or very large burns (> 85%, when unburnt skin is counted). This method is inaccurate for medium-sized burns.

B. Wallace Rule of Nines:

For adults, the rule of nines is a good, quick way of estimating medium to large burns. The body is divided into areas of 9%, and the total burn area can be calculated. It is not accurate in children. See figure 11.1.

C. Lund and Browder Chart:

This chart, if used correctly, is the most accurate burn estimation method. It compensates for the variation in body shape with age. Therefore, it can give an accurate assessment of burn areas in children. See figure 11.2.

3. CLINICAL FEATURES

3.1. Burn Causative Agent

A. Thermal Injuries:

- Scalds:

  Approximately 70% of burns in children are caused by scalds, although they frequently occur in the elderly. The common mechanisms are spilling hot drinks, liquids, or being exposed to hot bathing water. *Scalds tend to cause superficial to superficial dermal burns* (see later for burn depth).
- Flame:

  Flame burns comprise 50% of adult burns. They are often associated with inhalational injury and other concomitant trauma. *Flame burns tend to be deep dermal or full thickness.*
- Contact:

  To be burnt from direct contact, the object touched must either have been extremely hot or the contact was abnormally long. *The latter* is more common, and these types of burns are commonly seen in people with epilepsy or those who misuse alcohol or drugs. They are also seen in elderly people after a loss of consciousness; such a presentation requires a full investigation as to the cause of the blackout.

Burns from brief contact with very hot substances are usually due to industrial accidents. *Contact burns tend to be deep dermal or full thickness.*

B. Electrical Injuries:

An electric current will travel through the body from one point to another, *creating "entry" and"exit" points*. The tissue between these two points can be damaged by the current. The voltage is the main determinant of the degree of tissue damage. Electrocution injuries are divided into the following:

- Domestic electricity:

  Low voltages tend to cause small, deep contact burns at the exit and entry sites. The alternating nature of domestic current can interfere with the cardiac cycle, giving rise to arrhythmias.
- "True" high tension injuries:

  Occur when the voltage is 1000 V or greater.
  - There is extensive tissue damage and often limb loss.
  - There is usually a large amount of soft and bony tissue necrosis.
  - Muscle damage leads to rhabdomyolysis, and renal failure may occur.
  - In comparison to other types of burns, high tension injuries require more aggressive resuscitation and debridement.

  Contact with voltage greater than 70 000 V is invariably fatal.
- "Flash" injury:

  This burn occurs when there has been an arc of current from a high tension voltage source.
  - The heat from this arc can cause superficial flash burns to exposed body parts, typically the face and hands. However, clothing can also be ignited, resulting in deeper burns. No current actually passes through the victim's body.

- A particular concern after an electrical injury is the need for cardiac monitoring.
- If the patient's electrocardiogram on admission is normal, and there is no history of loss of consciousness, then cardiac monitoring is not required.
- If there are electrocardiographic abnormalities or a loss of consciousness, admit and monitor the patient for 24 hours.

C. Chemical Injuries:

Chemical injuries generally occur in industrial accidents, but may occur with household chemical products. These burns tend to be deep, as the corrosive agent continues to cause coagulative necrosis until completely removed. *Alkalis tend to penetrate deeper and cause worse burns than acids.* Cement is a common cause of alkali burns.

Certain industrial agents may require specific treatments in addition to standard first aid. Hydrofluoric acid, widely used for glass etching, and in the manufacture of circuit boards, is one of the more common culprits. It causes a continuing, penetrating injury that must be neutralized with calcium gluconate, either applied topically in a gel form, or injected into the affected tissues.

4. MANAGEMENT

Management depends on severity

Severity of Burns

A. Critical Burns – Adults:

- Full - thickness of hands, feet, face, genitalia or joints.
- Burns associated with respiratory injury or smoke inhalation.
- Full - thickness of more than 10% of body surface.
- Partial thickness of more than 25% of body surface.
- Burns coexisting with a painful, swollen, or deformed extremity.
- Moderate burns in patients under 5 or over 55.

B. Moderate Burns – Adults:

- Full - thickness burns of 2% to 10% of the body surface area, excluding critical areas.
- Partial - thickness burns of 15% to 25% of the body surface area.
- Superficial burns of greater than 50% of the body surface area.

C. Minor Burns – Adults:

- Full - thickness burns of less than 2% of the body surface area.
- Partial - thickness burns of less than 15% of the body surface area.

Major Burns

4.1. Initial Evaluation and Resuscitation

Before the management of the patient's burn wound, the patient should be properly and completely evaluated. During assessment, *the environment should be kept warm*, and small segments of skin should be exposed sequentially to reduce heat loss. Evaluation of the burn patient is organized into primary and secondary surveys (see figure 11.3).

A. Primary Survey:

- Airway with cervical spine control:

  This assessment determines if the airway is compromised or at risk of compromise. Unless cervical injury has been ruled out, the cervical spine should be protected. Inhalation of hot gases will result in a burn above the vocal cords. This type of burn will become edematous within hours after the initial trauma, especially after beginning fluid resuscitation. Signs of inhalational injury include:
  - History of flame burns or burns in an enclosed space.
  - Full thickness or deep dermal burns to face, neck, or upper torso.
  - Singed nasal hair.
  - Carbonaceous sputum or carbon particles in oropharynx.

- Breathing:

  On presentation to the ED, *all burn patients should receive 100% oxygen* through a humidified non-rebreathing mask. Deep dermal or full thickness circumferential burns of the chest can limit chest excursion and prevent adequate ventilation. This may require escharotomies. In smoke inhalation injuries, the products of combustion lead to bronchospasm, inflammation, and bronchorrhoea. The inflammatory exudates created are not cleared, and atelectasis or pneumonia follows. Management can be attempted, with:

  - Adequate oxygenation
  - Nebulizers
  - Intubation if indicated by (all intubated patients must be transferred):
    - Erythema or swelling of oropharynx on direct visualization.
    - Change in voice, with hoarseness or harsh cough.
    - Stridor, tachypnoea, or dyspnoea.

- Circulation:

  Intravenous access should be established with two large bore cannulas, preferably placed through unburnt tissue. When establishing access, withdraw blood for lab evaluation. Initial laboratory tests should include: full blood count, urea and electrolytes, blood group, and clotting screen.

  - Peripheral circulation

    Peripheral circulation must be checked. Any deep or full thickness circumferential extremity burns can act as a tourniquet. Reduced blood flow can occur several hours after injury due to edema caused by fluid resuscitation. Serial reassessment is necessary to reduce the chance of decrease perfusion to an extremity. If there is any suspicion of decreased perfusion in an extremity with a circumferential burn, the tissue must be released with escharotomies.

    Profound hypovolaemia is not a normal initial response to a burn; if a patient is hypotensive, consider delayed presentation, cardiogenic dysfunction, or an occult source of blood loss (chest, abdomen, or pelvis).

- Neurological disability:

  All patients should be assessed for responsiveness with the Glasgow coma scale; they may be confused because of hypoxia or hypovolaemia.

- Exposure with environment control:

  The entire patient should be examined (including the back) to get an accurate estimate of the burn area, and to check for any concomitant injuries. Burn patients, especially children, easily become hypothermic, leading to hypoperfusion and deepening of burn wounds. Patients should be covered and warmed as soon as possible.

- Fluid resuscitation:

  Immediately, the resuscitation regimen should be determined and instituted. This is based on the estimation of the burn surface area. Fluid needs include:

  - Warmed crystalloid solution (lactated Ringer) 4 ml /kg/% burned BSA within the initial 24 hours (½ in first 8 hours from the onset of injury, and the second ½ in the remaining 16 hours).

- Monitor heart rate and urinary output:

- *A urinary catheter is mandatory to monitor urinary output in all adult patients with injuries covering > 20% of their total body surface area.* Children's urine output can be monitored with external catchment devices, or by weighing nappies, provided the injury is < 20% of total body area. In children the interosseous route can be used for fluid administration if intravenous access cannot be obtained, but should be replaced by intravenous lines as soon as possible.
- Analgesia:

  Superficial burns can be extremely painful. All patients with large burns should receive intravenous morphine at a dose appropriate to body weight. This can be titrated against pain and respiratory depression. Reassessment for pain should be performed 30 minutes after administering pain medication.

B. Secondary Survey:

At the end of the primary survey and the start of emergency management, a secondary survey should be performed. This is a full head-to-toe examination, looking for any concomitant injuries. Any concomitant trauma will have their own investigations.

4.2. Investigations for Major Burns

A. General:

- Perform serum full blood count, packed cell volume, urea and electrolyte concentration, clotting screen, blood type and crossmatch.

B. Electrical Injuries:

- 12 lead electrocardiography (attention to arrhythmia).
- Cardiac enzymes (for high tension injuries).

C. Inhalational Injuries:

- Chest x-ray.
- Arterial blood gas analysis.

  Arterial blood gases are useful in any burn case, because the base excess is predictive of the amount of fluid resuscitation required. ABG's are also helpful in determining successful fluid resuscitation, and essential in inhalational injuries or exposure to carbon monoxide.

D. Carboxyhaemoglobin (COHB):

Signs of carboxyhaemoglobinaemia (see table 11.1).

Table 11.1: Signs of Carboxyhaemoglobinaemia

COHB levels	Symptoms
0-10% Minimal	Normal level in heavy smokers
10-20%	Nausea, headache
20-30%	Drowsiness, lethargy
30-40%	Confusion, agitation
40-50%	Coma, respiratory depression
> 50%	Death

- Pulse oximetry cannot differentiate between oxyhaemoglobin and carboxyhaemoglobin. Therefore, oximetry will give false normal results.
- Treat patients with 100% oxygen. Patients with carboxyhaemoglobin levels greater than 25-30% should be ventilated.

Key points:
- Perform a systematic assessment, similar to other trauma patients (don't get distracted by the burn).
- Beware of airway compromise.
- Provide adequate analgesia.
- Exclude any concomitant injuries.
- Coordinate with a burn unit early in patient treatment.
- If in doubt, reassess.

Hospital admission criteria:
- Adults with:
  - partial thickness burns > 15%
  - full thickness burns > 5%
- Children with:
  - partial thickness burns > 10%
  - full thickness burns > 3%
- Burns to the face, hands, perineum, or feet.
- Circumferential burns.
- Burns over major joints.
- Chemical burns.
- Inhalation injury or trauma.
- Electrical injuries.

Minor Burns

Minor burns suitable for outpatient management:

- Partial thickness burns covering < 10% of total body surface area in adults.
- Partial thickness burns covering < 5% of body surface area in children.
- Full thickness burns covering < 1% of body surface.
- No comorbidity.

First Aid

- Stop the burning process.
- Remove the heat source.

  Clothing can retain heat, even in a scald burn, and should be removed as soon as possible. Adherent material, such as nylon clothing, should be left on. Tar burns should be cooled with water, but the tar itself should not be removed. In the case of electrical burns, the victim should be disconnected from the source of electricity before first aid is attempted.
- Cooling the burn:

  Cooling the burn is effective if performed within 20 minutes of the injury. Immersion or irrigation with running tepid water (15°C) should be continued for up to 20 minutes. This removes the noxious agents, reduces pain, and may reduce edema by stabilizing mast cells and histamine release. Iced water should not be used, as intense vasoconstriction can cause burn progression. Cooling large areas of skin can lead to hypothermia, especially in children. Liquid chemical burns should be irrigated with copious amounts of water.
- Analgesia:

  Exposed nerve endings resulting from a burn, will cause pain. Cooling and covering the exposed burn will reduce the pain. Initially, opioids may be required to control pain, but once first aid measures have been effective, oral non-steroidal anti-inflammatory drugs, such as ibuprofen or co-dydramol will suffice.
- Dressing Changes for burns
  - Utilize aseptic technique.
  - First dressing change is performed after 48 hours, and then every 3-5 days.
  - Criteria for early dressing change:
    - Excessive “strike through” of fluid from wound.
    - Smelly wound.
    - Contaminated or soiled dressings.
    - Slipped dressings.
    - Signs of infection (such as fever).

Key points:

- Initial first aid can influence final cosmetic outcome.
- Cooling with tepid tap water is one of the most important first aid measures.
- Routine use of antibiotics is discouraged.
- Simple dressings suffice.
- Utilize aseptic technique for dressing changes.

Chemical Burns

All chemical burns are managed the same irrespective of the agent.

- All contaminated clothing must be removed, and the area thoroughly irrigated. This has been shown to limit the depth of the burn.
- Dry chemical burns, react with water, which worsen the burn. Therefore, utilize the "brush - then flush" method. Litmus paper can be used to confirm removal of alkali or acid. Eye injuries should be irrigated copiously and referred to an ophthalmologist.

5. MONITORING

5.1. Indications for Referral to a Burn Unit

All complex injuries should be referred. A burn injury is likely to be complex if associated with:

A. Extremes of Age:

- Under 5 or over 60 years.

B. Site of Injury:

- Face, hands, or perineum.
- Feet (dermal or full thickness loss).
- Any flexure, particularly the neck or axilla.
- Circumferential dermal or full thickness burns of limbs, torso, or neck.

C. Inhalational Injury:

- Any substantial injury, excluding pure carbon monoxide poisoning.

D. Mechanism of Injury:

- Chemical injury > 5% of total body surface area.
- Exposure to ionizing radiation.
- High pressure steam injury.
- High tension electrical injury.
- Hydrofluoric acid burn >1% of total body surface area.
- Suspicion of non-accidental injury.

E. Large Size (dermal or full thickness loss):

- Dermal or full thickness loss >5% of total body surface area in pediatric patient (<16 years of age).
- Dermal or full thickness loss >10% of total body surface area in adult patient (+16 years of age).

F. Coexisting Conditions:

- Any serious medical conditions (cardiac dysfunction, immunosuppression, pregnancy).
- Any associated injuries (fractures, head injuries, crush injuries).

Figure 11.1: Wallace Rule of Nines

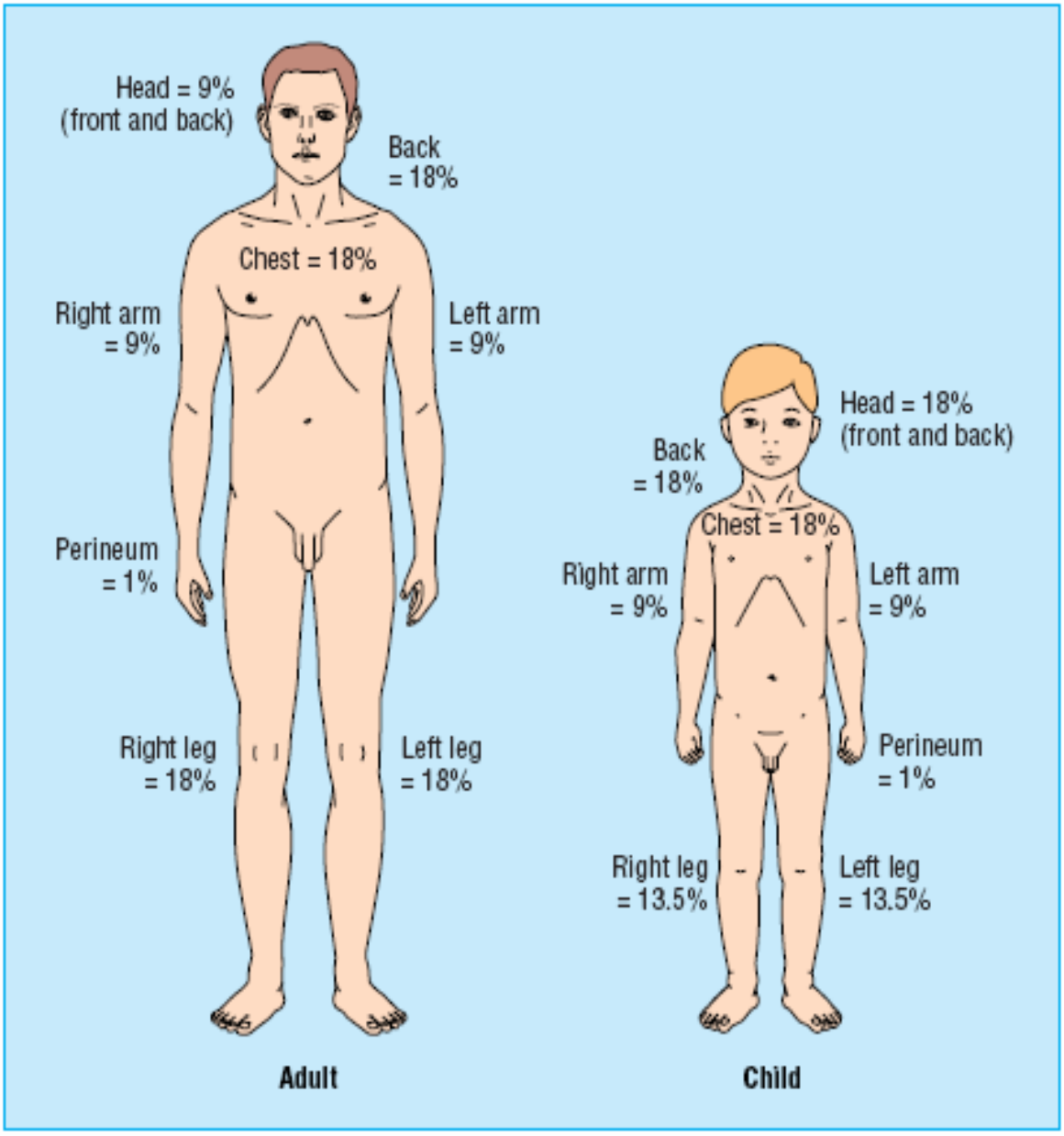
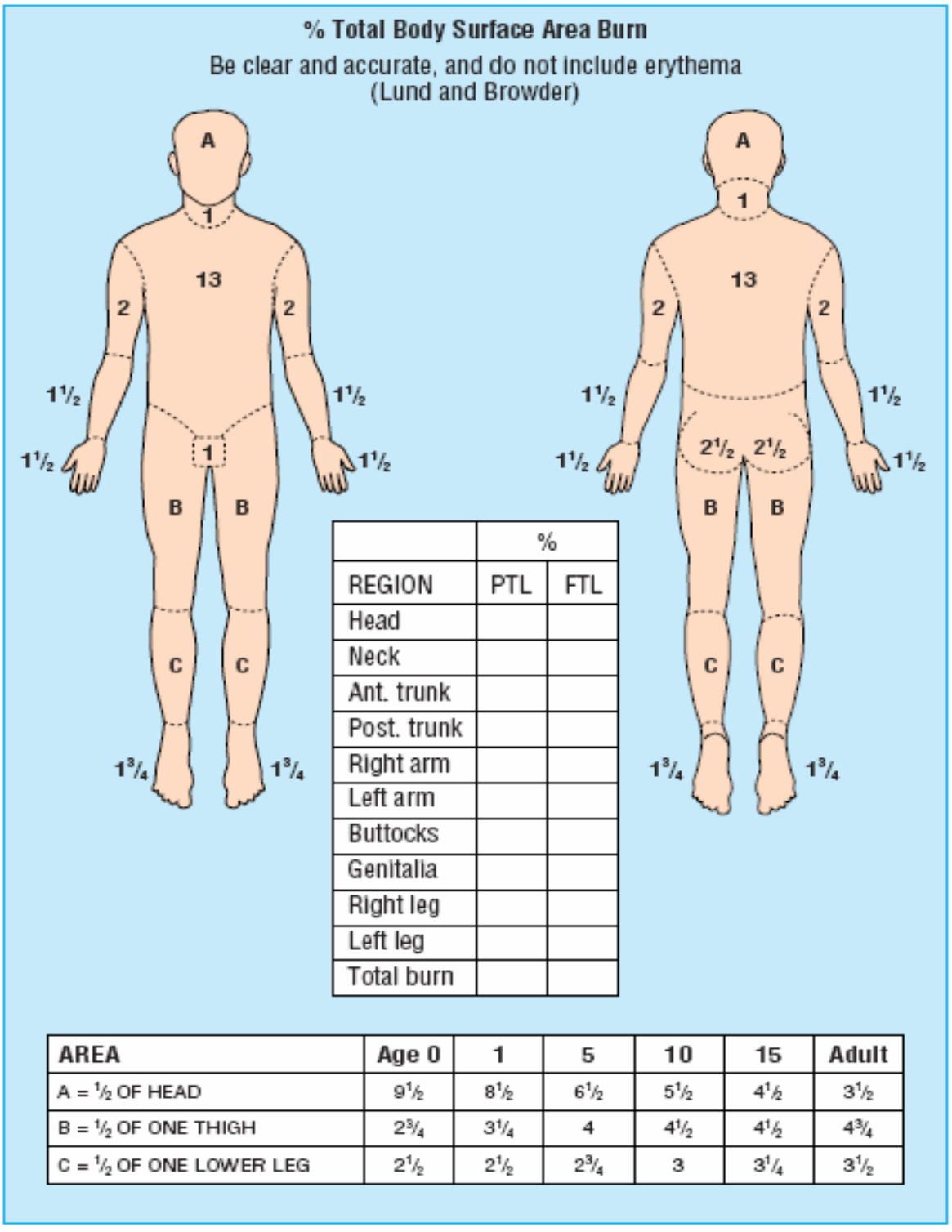


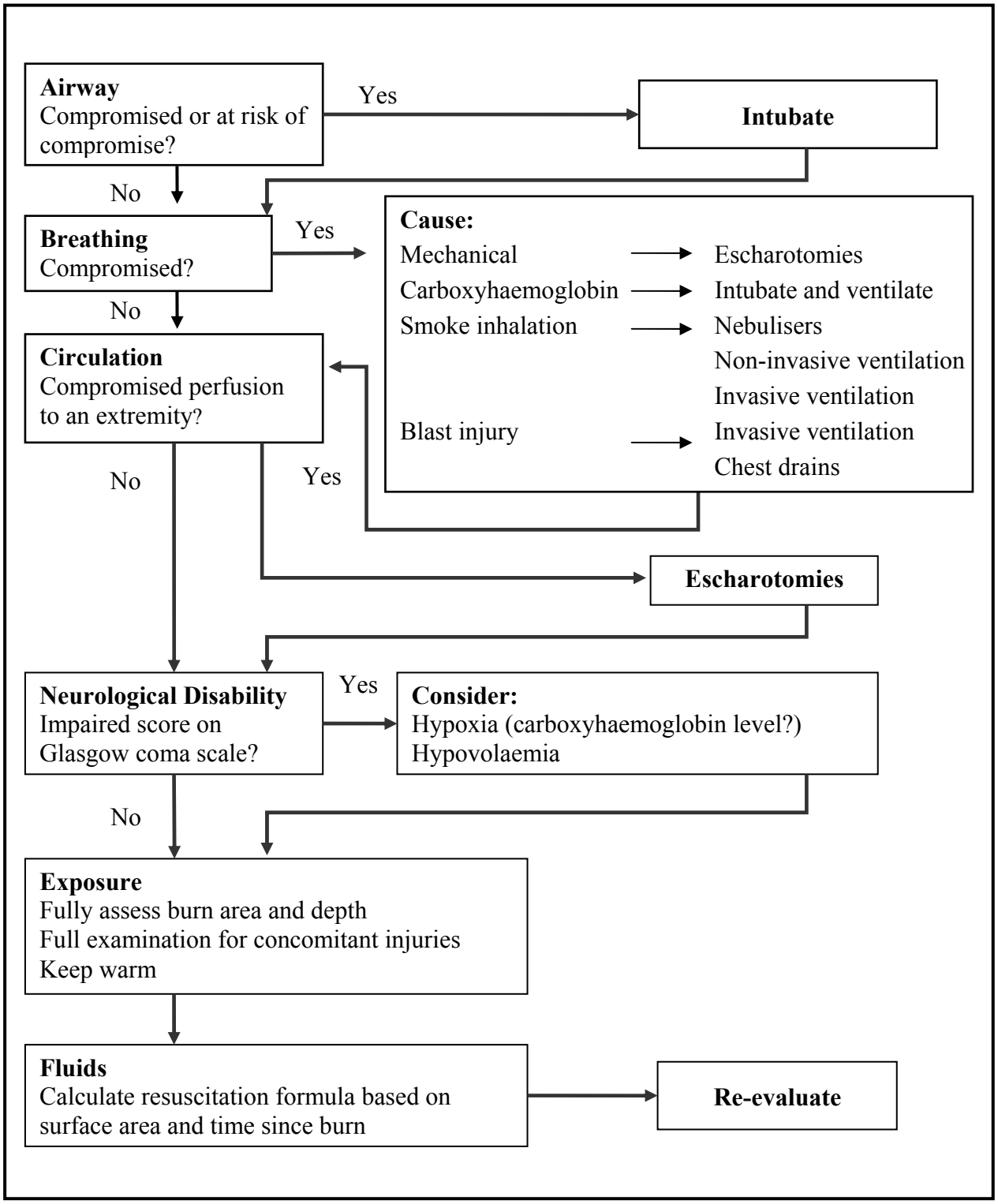
Figure 11.2: Lund and Browder Chart



	%	
REGION	PTL	FTL
Head		
Neck		
Ant. trunk		
Post. trunk		
Right arm		
Left arm		
Buttocks		
Genitalia		
Right leg		
Left leg		
Total burn		

AREA	Age 0	1	5	10	15	Adult
A = ½ OF HEAD	9½	8½	6½	5½	4½	3½
B = ½ OF ONE THIGH	2¾	3¼	4	4½	4½	4¾
C = ½ OF ONE LOWER LEG	2½	2½	2¾	3	3¼	3½

Figure 11.3: Algorithm for Primary Survey of a Major Burn Injury



Part 3: Medical Emergencies

Chapter 12: Sudden Cardiac Arrest

GUIDELINE OBJECTIVES:

- Understand common causes of sudden cardiac arrest.
- Perform basic life support for adults and children.
- Understand indications for initiating and discontinuing cardiopulmonary resuscitation (CPR).
- Utilize guidelines for advanced cardiac life support.

1. INTRODUCTION

The "chain of survival", are the tasks performed during a resuscitation event. They are performed in sequence to revive a victim in the quickest and most successful way. These steps include:

- Early access to care.
- Early basic life support (BLS).
- Early defibrillation.
- Early advanced cardiac life support (ACLS).

1.1. Cardiopulmonary Resuscitation (CPR)

Understanding critical concepts in providing CPR ensures delivery of quality CPR that improves the victim survival. During CPR, push hard and fast. Allow for full chest recoil with each compression without removing your hands from the chest and minimizing compression interruption. Also, avoid hyperventilation of the victim and remember quality chest compressions are critical.

A. Who Needs (CPR):

- Unresponsive.
- Not breathing.
- No pulse.
- Gasping is not adequate breathing, a victim who is gasping needs CPR.
- Brain injury does not usually lead to cardiac arrest unless if respiratory arrest occurs.

B. Cardiac Arrest:

Sudden cardiac arrest occurs when the heart stops beating abruptly and unexpectedly. Sudden cardiac arrest may occur:

- During the first 4 hours after the onset of symptoms of heart attack (most common).
- As the initial and only symptoms of coronary heart disease (CHD).
- Other causes of sudden cardiac arrest include:
  - Primary respiratory arrest.
  - Direct injuries to the heart.
  - Heart arrhythmia.
  - Use of drugs.

2. CLASSIFICATION

Many avenues can be employed to revive the sudden arrested victim. These include basic life support (BLS), advanced life support (ALS), and automatic external defibrillator (AED). BLS and AED use occurs outside the hospital, however, ACLS and defibrillation occurs within the hospital. BLS, ACLS, and defibrillation can occur for both the adult and pediatric populations.

3. FEATURES

3.1. Adult Basic Life Support (BLS)

- Step 1: Check the victim for response:
  - Action:
    - Tap the victim shoulders and shout: “Are u alright”.
- Step 2: If no response:
  - Action:
    - Shout for help, activate the emergency response system and get (AED).
- Step 3: Assess breathing:
  - Action:
    - Open victim’s airway with a head tilt-chain - lift maneuver.
    - Place your ear near the victim.
    - Look for the chest to rise and fall.
    - Listen for air escaping during exhalation.
    - Feel for the flow of air against your cheek.
- Step 4: If the victim is not breathing adequately:
  - Action:
    - Use a barrier to give two rescue breaths and watch for the chest to rise.
- Step 5: Check pulse:
  - Action:
    - Maintain a head tilt with one hand.
    - Locate the trachea, using two or three fingers of your free hand.
    - Slide the fingers into the groove between the trachea and the muscles at the side of the neck.
    - Palpate the carotid artery for 5 to 10 seconds.
- Step 6: If the pulse is nonexistent, begin chest compressions:
  - Action:
    - *Use a ventilation ratio of 30 compressions to 2 breaths at the rate of 100 compressions per minute.*
- Step 7: If no response:
  - Use (AED) (if available).

3.2. Defibrillation

- Defibrillation that occurs within 3-5 minutes of collapse can provide survival rates as high as 49 – 75%.
- The rescuer must make every effort to minimize any interruption in chest compressions.
- Causes for interruption of cardiac compressions may include:
    - Prolonged pulse check.
    - Prolonged rescue breaths.
    - Moving the victim.
    - Using the AED.

Remember: Assess…..... then perform appropriate action

3.3. The Advanced Cardiac Life Support (ACLS) Secondary Survey

Advanced cardiac life support (ACLS) is conducted and started after the BLS.

A. Advanced Airway:

- Endotracheal intubation.
- Laryngeal mask.
- Combitube.

B. Advanced Circulation:

- Use of drugs after securing an intravenous (IV) / intraosseous (IO) access to control heart rhythm and blood pressure (BP).

C. Rhythm Recognition:

- An important component of ACLS is the recognition of cardiac arrest rhythm.
- Shockable:
    - Ventricular fibrillation (VF).
    - Pulseless ventricular tachycardia (VT).
- Non shockable:
    - Asystole
    - Pulseless electrical activity (PEA).
- Don't forget to search for, and treat reversible causes of cardiac arrest.

D. Drugs:

- Epinephrine.
- Vasopressin.
- Amiodarone.
- Lidocain.
- Magnesium Sulphate.

E. Performing Resuscitation for Shockable Rhythms:

See figure 12.1.

- Give 1 shock:
  - 120 – 200 J – Biphasic.
  - 360 J in – Monophasic.
  - Resume CPR (chest compression) immediately, and continue for 5 cycles with 30 compressions and 2 ventilations.
- Check rhythm, if shockable:
  - Give 1 shock.
  - Resume CPR immediately and continue for 5 cycles (2 minute).
  - Give Epinephrine 1 mg IV push (repeated every 3-5 minute) or vasopressin 40 unite (U) IV push.
- IF still shockable:
  - Give 1 shock.
  - Resume CPR immediately.
  - 5 cycles (2 minute) with 30 compression and 2 ventilations.
  - Give amiodarone 300 mg IV push.
- Still shockable:
  - Shock plus five cycles of CPR.
  - Epinephrine 1 mg IV push.
- IF still shockable:
  - Shock plus five cycles of CPR.
  - Amiodarone 150 mg IV push.
- Still shockable:
  - Shock plus five cycles of CPR.
  - Epinephrine1 mg IV push.
- Still shockable:
  - Shock plus five cycles of CPR.
  - Lidocaine 1.5 mg/kg IV push.
- Still shockable:
  - Shock plus five cycles of CPR.
  - Epinephrine 1 mg IV push.
- Still shockable:
  - Shock plus five cycles of CPR plus Lidocane 0.75mg/kg IV push.
  - Magnesium sulphate for torsades depoint: 1-2 g Iv diluted in 10 ml D5w
  - Pulseless arrest (VF, VT) treatment sequence is →rhythm →checks→ shock →drugs.

Remember:

- Follow each drug with a 20ml bolus of IV fluid.
- Elevate the extremity for about to 20 seconds.
- Administer drugs during CPR, do not stop CPR to administer drugs.

F. Non-Shockable Rhythm Pulseless Resuscitation or Pulseless Electrical Activity (PEA).

- See table 12.1 and figure 12.2.
- Continue high-quality CPR for 5 cycles.
- Epinephrine 1mg IV push, repeated every 3-5 minutes or vasopressin 40 units (U) IV push may replace the first or second dose of Epinephrine.
- Consider giving atropine especially if PEA rate is slow (Atropine 1mg IV push, may repeat every 3-5 minute for a total of three doses).
- In pulseless electrical activity (PEA) look for the reversible causes.

If the rhythm check reveals a non shockable rhythm and there is no pulse, then proceed along the asystole / PEA pathway as shown on the right side of ACLS pulseless arrest algorithm.

3.4. Pediatric Basic Life Support (BLS)

A. Definitions:

- Neonate: Infants in the first 28 days (1 month) of life.
- Newborn: The first minutes to hours from birth.
- Infant: Includes the neonatal period and extends to one year of age (12 calendar months).
- Child: from 1 year of age to the onset of puberty.

B. Causes of Cardiac Arrest:

- Respiratory failure is the common cause of cardiac arrest during infancy and childhood.
- Bradycardia associated with a rapid fall in cardiac output, leads to rapid deterioration in systemic perfusion.
- Asphyxia and choking.
- Sudden infant death syndrome.
- Drowning.
- Trauma.
- Pneumonia and severe asthma.
- Poisoning.

C. Basic Life Support (BLS) Step-Wise Management:

Causes of cardiac arrest affect the priorities of resuscitation. If cardiac arrest is due to respiratory failure (as most pediatric cases), immediately institute CPR (by opening the airway and performing rescue breathing) before activating of the emergency response system.

- Step 1:
  - Unresponsive.
  - Supine position.
- Step 2:

A - Open airway:

  - The airway is opened by the head tilt, chin lift method.
  - An alternative method is to use a modified jaw-thrust.
- Step 3:

B- Breathing:

  - Look for chest movement.
  - Listen for air from the mouth and/or nose.
  - Feel for air movement.
  - Do this for 5-10 seconds.
  - If no adequate breathing, *give 2 breaths, each breath should make the chest rise.*
  - Check for breathing and movement.
  - If no response, call for help.
- Step 4:

C- Circulation:

  - Check pulse within 10 seconds.
  - Use the brachial pulse in infants and carotid pulse in children.
  - If the pulse is less than 60 beats per minute or nonexistent, begin chest compressions.
  - Compression hand position:
    - Infant- 1 finger breadth below the nipple line.
    - Child- heal of hand on the center of the chest.
    - Begin chest compression- 30 compressions and 2 breaths for one rescuer, and 15 compressions and 2 breaths for two rescuers.
    - Compress at rate 100 compressions per minute (push hard and fast, release completely, and minimize interruptions in compressions).

3.5. Discontinuing Basic Life support (BLS)

- Restoration of effective spontaneous circulation and ventilation.
- Transfer of care to emergency medical responders or other trained personnel (who continue BLS or initiate advanced life support).
- Inability to continue resuscitation because of exhaustion, environmental hazards that endanger the rescuer, or if continued resuscitation would jeopardize the lives of others.

Figure 12.1: Algorithm for Shock Disbursement and Drug Delivery

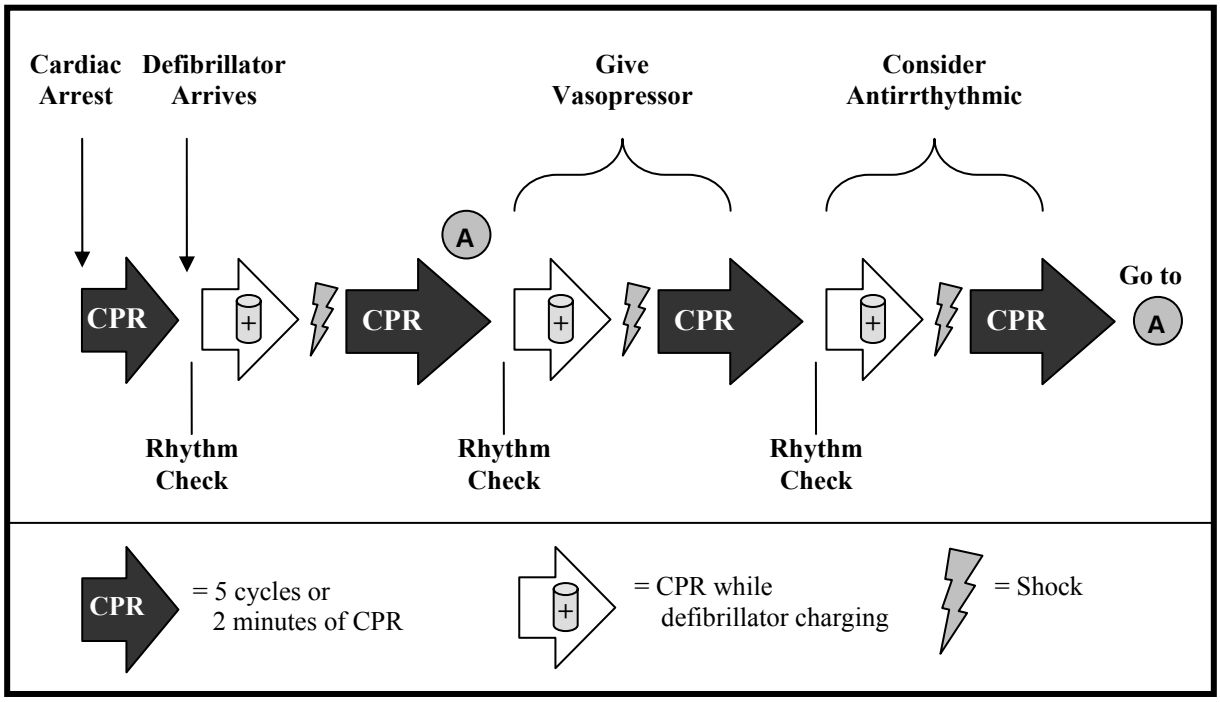
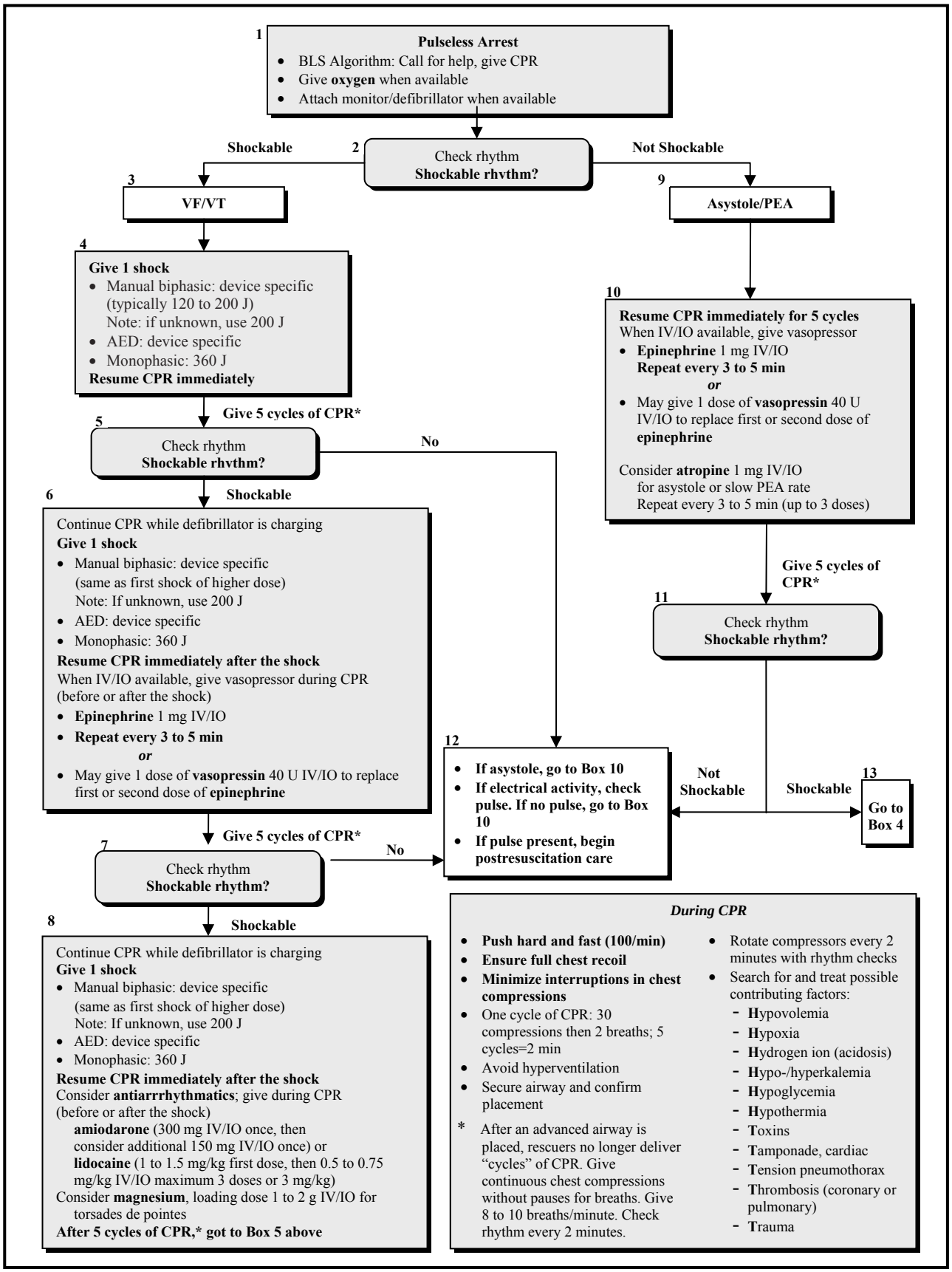


Table 12.1: Causes for Pulseless Electrical Activity (PEA)

Hs	**Ts**
Hypovolemia	Toxin
Hypoxia	Tamponade (cardiac)
Hydrogen ion (Acidosis)	Tensionpneumothorax
Hyper/ Hypokalemia	Thrombosis (coronary – pulmonary)
Hypoglycemia	Trauma
Hypothermia	

Figure 12.2: Pulseless Arrest Algorithm



Chapter 13: Chest Pain

GUIDELINE OBJECTIVES:

- Understand causes and assessment of chest pain in emergency department (ED).
- Identify life threatening causes of chest pain.
- Recognize initial management and approach towards the different causes of chest pain.
- Define and manage different varieties of acute coronary syndromes in the ED.
- Follow an algorithm approach to manage acute coronary syndrome.

1. INTRODUCTION

Chest pain is one of the most common complaints that will bring a patient to the emergency department. Seeking immediate care may be lifesaving. While the patient may be worried about a heart attack, there are many other causes of pain in the chest that the healthcare provider will need to consider. Some diagnoses are life threatening, while others are less dangerous.

1.1. Causes of Cardiovascular Chest Pain

- Coronary artery disease (CAD):
  - Stable angina pectoris.
  - Unstable angina.
  - Myocardial infarction.
- Coronary vasomotor disease:
  - Variant angina.
  - Microvascular angina.
  - Pericarditis.
  - Myocarditis.
  - Valvular heart disease.
- Aortic stenosis:
  - Mitral stenosis.
  - Hypertrophic cardiomyopathy.
  - Aortic dissection.
  - Post-pericardiotomy.

2. CLASSIFICTION

Life-threatening causes of chest pain include:

- Acute coronary syndrome.
- Aortic dissection.
- Pulmonary embolism.
- Tension pneumothorax.

3. MANAGEMENT

Myocardial infarction (MI), pulmonary embolism (PE), aortic dissection, & tension pneumothorax may result in sudden death. Any patient with rapid onset of chest pain, who may be potentially unstable (history, appearance, vital signs), should be transported immediately to the resuscitation room.

Features that predict a cardiologic cause to chest pain:

- Location in chest.
- Character or quality of discomfort.
- Response to Nitroglycerin.
- Relationship to exertion.

3.1. Immediate Goals

- Resuscitation and stabilization: Airway, breathing, and circulation (ABC's).
- Oxygen administration.
- Intravenous (IV) line (s).
- Monotoring: ECG, pulse oximeter, and blood pressure.
- Rule out life-threatening causes.

Acute Coronary Syndromes (ACS)

1. INTRODUCTION

Etiology:

- Ischemia and imbalance between oxygen demand and oxygen supply.
- Fixed atherosclerotic lesion (progressive narrowing).
- Plaque disruption with platelet/thrombi aggregation (partial or complete blocks).
- Spasm.

2. CLASSIFICATION

Acute coronary syndrome (ACS) includes a spectrum of clinical presentations ranging from unstable angina (UA), non –ST segment elevation myocardial infraction (NSTEMI), to ST segment elevation myocardial infraction (STEMI).

3. CLINICAL FEATURES

- How to diagnose an acute coronary syndrome (ACS)?
  - History.
  - Physical examination.
  - Electrocardiogram (ECG).
  - Cardiac enzymes.
- Analysis of pain:
  - Quality.
  - Location.
  - Radiation.
  - Provocation.
  - Onset.
  - Duration.
  - Palliation.
  - Severity.
- Associated symptoms.
- Risk factors.

3.1. Pain

A. Quality of the Pain:

- Typical: squeezing, tightness, pressure, constriction, strangling, burning, heart burn, fullness in the chest, a band-like sensation, knot in the center of the chest, lump in the throat, ache, heavy weight on chest, like a bra too tight, and jaw or toothache.
- Sometimes indescribable, patient places the palm in the center of the chest.

B. Location of Pain:

Diffuse, retrosternal, but may be difficult to localize.

C. Radiation of the Pain:

- Neck, throat, mandible, teeth, upper extremity, or shoulder.
- Wide extension increases the probability of MI.

D. Onset of Pain:

- Onset of ischemic pain is often gradual or stuttering with an increasing intensity over time.
- Early morning onset may indicate an acute MI.

E. Duration of Pain:

The pain from myocardial ischemia generally lasts for a few minutes; it may be more prolonged in the setting of a myocardial infarction.

F. Provocation of the Pain:

- Exertion: Classic of angina, also esophageal.
- Cold, emotional stress, and coitus.

3.2. Associated Symptoms

- Vomiting may occur in MI (transmural).
- Sweating is more frequently associated with MI than esophageal disease (forehead, diaphoresis with pallor).
- Dyspnea + chest pain: myocardial ischemia, lung pathology (airways, parenchyma, vessels), or panic disorder.
- Syncope + chest pain: severe ischemia, aortic dissection, massive PE, ruptured AAA, or critical aortic stenosis (AS).

3.3. Risk Factors

- Non-changeable risk factors:
  - Heredity.
  - Sex.
  - Race.
  - Age.
- Changeable & controllable risk factors :
  - Smoking.
  - Hyperlipidemia.
  - Diabetes.
  - High blood pressure.

- Contributing risk factors:
  - Stress.
  - Obesity.
  - Lack of Exercise.

3.4. The Past History

Coronary artery disease CAD and risk factors.

4. ANGINA

4.1. Introduction

A transient discomfort (which may or may not be perceived as pain) caused by an inadequate blood flow and oxygen delivery to the heart muscle. Frequently located in the center of the chest (called precordial or substernal) but may be more diffuse throughout the front of the chest. The most frequent cause of angina is Coronary Atherosclerosis.

Angina is often precipitated by any factor that increases the heart rate (increase of myocardial oxygen demand):

- Exercise.
- Unusual exertion.
- Strong emotions.
- Extreme temperatures.

4.2. Classification

- Types of angina:
  - Stable angina.
  - Prinzmetal angina.
  - Unstable angina.

4.3. Clinical Features

See table 13.1.

A. Symptoms:

- Typical:
  - Commonly lasts from 2 to 15 minutes.
  - Usually described as uncomfortable pressure, fullness, squeezing, or pain in the center of the chest.
  - May spread to one (more often the left) or both shoulders, arms, neck, jaw, back, or upper mid portion of the abdomen (epigastrium).
  - As the severity of the coronary narrowing increases, the amount of exertion needed to bring on angina decreases.

- Atypical:
  - Women, the elderly, and persons with diabetes often present with angina that is more diffuse in location and vague in description. Descriptions may include:
    - Shortness of breath.
    - Syncope.
    - Lightheadedness.
    - Weakness.
    - Nausea or vomiting.
    - Diffuse pain.

B. Angina Types:

- Stable angina pectoris:
  - Chest pain occurs intermittently over a long period of time with the same pattern of onset, duration, and intensity of symptoms.
  - Can be controlled with medications on an outpatient basis.
  - Pain usually lasts 3 to 5 minutes.
  - Subsides when the precipitating factor is relieved.
  - Pain at rest is unusual.
- Prinzmetal angina:
  - Occurs at rest, usually due to spasms of major coronary artery.
  - Spasm may occur in the absence of CAD.
- Unstable angina:
  - New in onset.
  - Occurs at rest.
  - Has a worsening pattern.
  - Unpredictable.
  - Considered to be an acute coronary syndrome.
  - Associated with deterioration of a once stable atherosclerotic plaque.

5. ACUTE MYOCARDIAL INFARCTION

5.1. Introduction

Severe narrowing or complete blockage of a diseased coronary artery, which leads to injury of the myocardium, then death of the heart muscle.

5.2. Clinical Features

- Pain is severe and may occur at rest or during sleeping.
- Usually lasts for more than 15 minutes.
- May be associated with nausea and sweating.
- Pain may be atypical and not relieved by rest or nitroglycerine.
- Heart attack can occur under a wide variety of circumstances.

- Most episodes of acute coronary syndromes occur at rest or with modest daily activity.
- Heavy physical exertion is a precipitating event in a minority of patients, perhaps 10% to 15%.
- An MI event may show signs of complications (hypotension, bradycardia, arrhythmia, heart failure).
- Sudden cardiac death (cardiac arrest) is due to ventricular fibrillation (80% of patients), asystole (15% of patients), and pulseless electrical activity PEA (5% of patients).

6. ACUTE CORONARY SYNDROMES (ACS) MANAGEMENT

See figure 13.2.

6.1. Diagnostic Studies

- Electrocardiogram (ECG) (see figure 13.1 and table 13.2).
- Cardiac markers (CK, MB Troponin).

6.2. Stabilization

- Supplemental oxygen.
- Intravenous (IV) access.
- Cardiac monitoring.
- Electrocardiogram (ECG).
- Blood sample for cardiac enzymes (See table 13.3).

6.3. Medication Management

A. Myocardial infarction (MI):

- Aspirin (160-325 mg p.o.): Decreased platelet aggregation.
- Nitroglycerin: Sublingually in 0.3-0.5 mg tablets every 5 minutes up to three doses, or if the systolic blood pressure (SBP) decreases below 90 mm Hg, whichever occurs first.
  - Not given unless SBP is at least 90 mm Hg.
  - Especially helpful treatment in acute myocardial infarction (AMI) with hypertension or chronic heart failure (CHF).
  - Not to be used in right ventricular infarction (cautious in inferior wall (IWMI)).
  - If nitroglycerin fails to relieve chest pain, give morphine 2 to 5 mg IV every 5 to 30 minutes (cautious in IWMI):
    - Monitor respiratory status and hemodynamic response carefully after each morphine administration.

B. Angina:

- Unstable angina:
  - Above plus Heparin in a bolus of 80 U/Kg as an infusion at a rate of 15 U/Kg/hour.
  - Or, low molecular weight heparin (e.g. enoxaparin 1 mg/kg subcutaneous (SC).
  - Clopidogrel: Oral antiplatelet agent (75mg IVdaily).

C. ST Elevation Myocardial Infarction:

- Above plus Streptokinase 1.5 million over one hour (see table 13.4).
- Heparinization is not necessary.

Adjuncts:

- Beta blocker if indicated (if the patient is hypertensive or tachycardic).
- In antiarrhythmic, anti-ischemic, anti-hypertensive patients. This will decrease infarct size, cardiovascular complications and mortality.
- Consider percutaneous transluminal coronary angioplasty, fibrinolytics, and glycoprotein IIb/IIIa inhibitors (antiplatelet drug).

Aortic Dissection

1. DEFENITION

An aortic dissection is defined as an intimal tear with entry of blood into the media that "dissects" between the intima and adventitia.

2. CLASSIFICATION

Stanford classification is a gold standard for delineating the type of aortic dissection. Under these criteria aortic dissections are classified as either types A or B.

2.1. Classification Types

- Type A: 80% of dissections involve the ascending aorta (w/ or w/o descending), especially at the ligamentum arteriosum.
- Type B: Descending aorta only.

2.2. Increased Risk

- Type A: Patient older 50 years of age with hypertension.
- Type B: Younger patients, those with marfan's syndrome, and patients that are pregnant.

2.3. Mortality

A. Type A:

- Untreated: 75%.
- Surgically treated: 15-20%.

B. Type B:

32-36% with or without surgical intervention.

3. CLINICAL FEATURES

3.1. History:

- Pain:
  - Greater than 90 percent of patients will exhibit abrupt and severe pain in the chest or between the scapulae.
    - Type of pain:
      - "Tearing" or "ripping".
      - Can be dull or pressure-like.
    - Location of pain:
      - Anterior chest ~ ascending aorta.
      - Back ~ descending.
- Nausea, vomiting, and diaphoresis are common.

3.2. Associated Symptoms Based on Progression of Dissection

- Carotid arteries: Stroke.
- Spinal arteries: Paraplegia.
- Abdominal aorta/renal arteries/iliacs: Abdominal/flank pain.
- Coronary arteries: Aortic insufficiency, pericardial effusion, or tamponade.
- Laryngeal nerve compression: Hoarseness.
- Tracheal compression: Dyspnea/stridor/wheezing.
- Esophageal compression: Dysphagia.

3.3. Physical Examination

- Symptoms/signs as above.
- Most commonly: Normal heart and lungs.
- Aortic insufficiency murmur in 16-20% of patients.
- Unequal, decreased, peripheral pulses and blood pressure (50% of patients).

4. MANAGEMENT

4.1. Investigations

- Chest x-ray (CXR):
  - Double density appearance of the aorta.
  - 85% with some abnormality.
  - Most frequent presentation is widened mediastinum.
  - Left pleural effusion, indistinct aortic knob, displaced trachea, calcified intima > 6mm from outer aortic wall.
  - Computerized tomography (CT), or transesophageal echocardiogram (TEE) aortogram may be helpful.

4.2. Management of Aortic Dissection

- Airway, breathing, and circulation (ABCs).
- Two large bore intravenous (IV)'s and monitor electrocardiogram (ECG).
- Lower arterial blood pressure to decrease the shear force on the intimae to minimize progression.
- Decrease left ventricular (LV) contractility.
- Goal: Systolic blood pressure (SBP) 100-110 mm Hg, and heart rate (HR) between bpm 60-80.

4.3. Management Options

- Nipride + esmolol.
- Labetalol.
- Early computerized tomography CT surgical involvement.

Pulmonary Embolism

1. DEFENITION

Pulmonary embolism occurs when a venous thrombus (or occasionally, another substance such as a fat or amniotic fluid) embolizes to the lung causing occlusion of a pulmonary artery.

- The source is lower extremity deep vein thrombosis (DVT) (80-90% of cases) and upper extremity DVT (10-15%).
- Others origin sources include: pelvic vein thrombosis, fat emboli, septic emboli, right heart thrombosis and only account for a small percentage of embolisms.

2. CLINICAL FEATURES

2.1. Predisposing Conditions and Risk Factors

A. Virchow's Triad:

- Venous stasis:
  - Prolonged travel and bed rest etc.
- Hypercoagulability:
  - Pregnancy malignancy, estrogen therapy, deficiencies of protein C, protein S, and AT III.
- Endothelial damage:
  - Recent surgery, trauma.
  - Heart diseases (e.g., acute myocardial infraction, CHF, arrhythmia).
  - Obesity.
  - Deep venous thrombosis.

2.2. Symptoms

The presence of any one of these should make the clinician consider pulmonary embolism (PE). Patients with a large pulmonary embolism may present with syncope or in cardiopulmonary arrest.

A. "Classic Triad":

Pleuritic chest pain, dyspnea, and hemoptysis is present in less than 25% of patients.

B. Notable Findings:

- Pleuritic chest pain in 74%.
- Dyspnea in 84%.
- Respiratory rate > 16 in 92%.
- Heart rate > 100 in only 44%.

2.3. Physical Examination Findings

- Tachypnea is seen in approximately 70% of patients.
- Tachycardia.

- An accentuated second heart sound.
- S3 or S4 gallop may also be observed.
- Pulseless electrical activity (PEA) is often seen in patients with pulmonary embolism and cardiopulmonary arrest.
- Lower extremity edema may be observed.

2.4. Diagnosis

- Clinical suspicion.
- Electrocardiogram (ECG):
  - Often normal.
  - Greater than 40% of patients will present with nonspecific ST and T wave abnormalities.
  - Sinus tachycardia is the most common rhythm disturbance.
- Chest x-ray (CXR):
  - Normal in ~30% of patients.
  - Atelectasis in ~50% of patients.
  - Elevated hemi diaphragm in 40% of patients.
  - Greatest role of the CXR in PE is to rule out other causes of patients symptoms (pneumothorax/pneumonia).
- Laboratory Investigations:
  - D-Dimer: An elevated plasma concentration indicates the presence of a clot from somewhere in the body within 72 hours.

3. MANAGEMENT

- Intravenous (IV), oxygen as needed, monitor, and continuous pulse oximetry.
- High pretest probability:
  - Anticoagulate 1st, then order your study:
    - Heparin 80 U/kg i.v. bolus followed by a 18 U/kg/hr i.v. drip.
- Low (+/- intermediate) pretest probability:
  - Study 1st, then anticoagulate if appropriate.
  - Contraindications to thrombolytic therapy (See table 13.5).
- Pulmonary angiography testing is the gold standard for the diagnosis of PE.

Tension Pneumothorax

1. DEFENITION

A tension peneumoithroax develops when a "one – way valve" air leak occurs from the lung or through the chest wall. Air is forced into the thoracic cavity without any means of escape, completely collapsing the effected lung.

2. CLASSIFICATION

- Simple pneumothorax – 85 % are often recurrent.
- Spontaneous pneumothorax.
- Traumatic pneumothorax.
- Tension pneumothorax.

3. CLINICAL FEATURES

Tension pneumothorax is a clinical diagnosis of air under pressure in the pleural space. Treatment should not be delayed to wait for radiological confirmation.

3.1. Signs and Symptoms

A. Early:

- Chest pain.
- Respiratory distress.
- Tachycardia.
- Hyperresonant sounds on lung percussion.
- Unilatral absence of breathe sounds.

B. Late:

- Tracheal deviation.
- Neck vein distension.
- Hypotension.
- Cyanosis.

4. MANAGMENT

A. Initial Management:

Immediate decompression by rapidly inserting a large calber needle into the second intercostals space at the midclavicular line.

B. Definitive Management:

Insertion of chest tube into the fifth intercostals space anterior to the midaxillary line.

C. Chest Tube Use Indications:

- Tension pneumothorax.
- Pneumothorax, (if it involves more than 20%).
- Hemothorax.
- Pleural effusion.
- Pyothorax.
- Post thoracotomy.

Table 13.1: Clinical Characteristics of Classic Anginal Chest Discomfort

Characteristic	More Likely to Be Angina	Less Likely to Be Angina
Type of pain	Dull, pressure	Sharp, stabbing
Duration	2-5 min, always <15-20 min	Seconds or hours
Onset	Gradual	Rapid
Location	Substernal	Lateral chest wall, back
Reproducible	With exertion	With inspiration
Associated Symptoms	Present	Absent
Palpation of Chest Wall	Not painful	Painful, exactly reproduces pain complaint

Table 13.2: Location of Cardiac Ischemia or Infarction as Suggested by ECG Lead Findings

Location of Ischemia or infarct	Leads in which abnormalities are seen
Inerior wall	II, III, aVF
Lateral wall	I, aVL, V5, V6
Anterolateral region	V1-V6,I, aVL
Anterior wall	V1,V2
Anteroseptal region	V1-V4
Right ventricle	V3R-V6R
Posterior wall	V1, V2, V7-V5

Table 13.3: Cardiac Biomarkers for Acute Myocardial Infarction

Cardiac Biomarkers	Initial Elevation after Acute Myocardial Infarction	Mean Peak Time	Time to Return to Baseline
Myoglobin	1-4 hours	6-7 hours	18-24 hours
Troponin I (cardiac specific)	3-12 hours	10-24 hours	3-7 days
Troponin T (cardiac specific)	3-12 hours	12-48 hours	10-14 days
Creatine kinase-MB (CK-MB)	4-12 hours	10-24 hours	48-72 hours

Table 13.5: Contraindications to Thrombolytic Therapy

Absolute	Relative
• Suspected aortic dissection or pericarditis • Cerebrovascular accident • Cerebrovascular surgery within the previous 2 months • Cerebrovascular neoplasm or aneurysm • Active bleeding in the gastrointestinal tract or other noncompressible site; hemorrhagic diathesis • Major surgery within the previous 2 weeks • Pregnancy or 2 weeks postpartum status • Allergy to thrombolytic agent • Unstable angina	• Cerebrovascular accident • Conditions placing the patient at high risk of intracardiac thrombus (e.g., atrial fibrillation, mitral valve stenosis) • Major surgery within the previous 2 months • Puncture of, or recent injury to, a noncompressible vessel within the previous 2 weeks • Uncontrolled hypertension • Prolonged cardiopulmonary resuscitation • Metastatic cancer • History of gastrointestinal bleeding • Hemorrhagic retinopathy

Table 13.5: Evaluation and Treatment of Patient Suspected of Having Pulmonary Embolism Based on VQ Scanning Results and Clinical Suspicion

Clinical Suspicion for Pulmonary Embolism	V/Q Scanning Results			
	Normal	Low Probability	Intermediate Probability	High Probability
Low	No treatment	No treatment	NIS (+): treat	NIS (+): treat
			NIS (-): no treatment	NIS (-): PA
Medium	NIS (+): treat	NIS (+): treat	NIS (+): treat	NIS (+): treat
	NIS (-): no treatment	NIS (-): no treatment	NIS (-): PA, consider serial IPG	NIS (-): PA
High	NIS (+): treat	NIS (+): treat	NIS (+): treat	
	NIS (-): no treatment	NIS (-): PA, consider serial IPG	NIS (-): PA, consider serial IPG	
IPG = impedance plethysmography NIS = noninvasive study of the lower extremities PA = pulmonary angiography				

Figure 13.1: ECG MI Findings

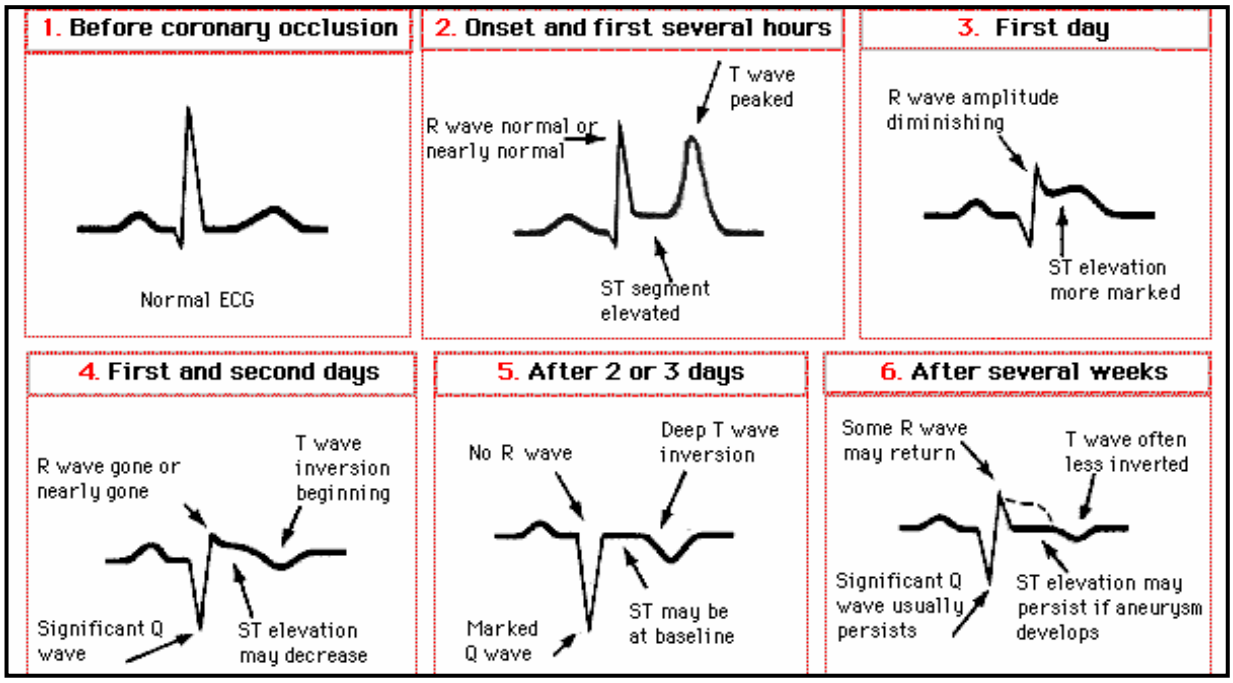
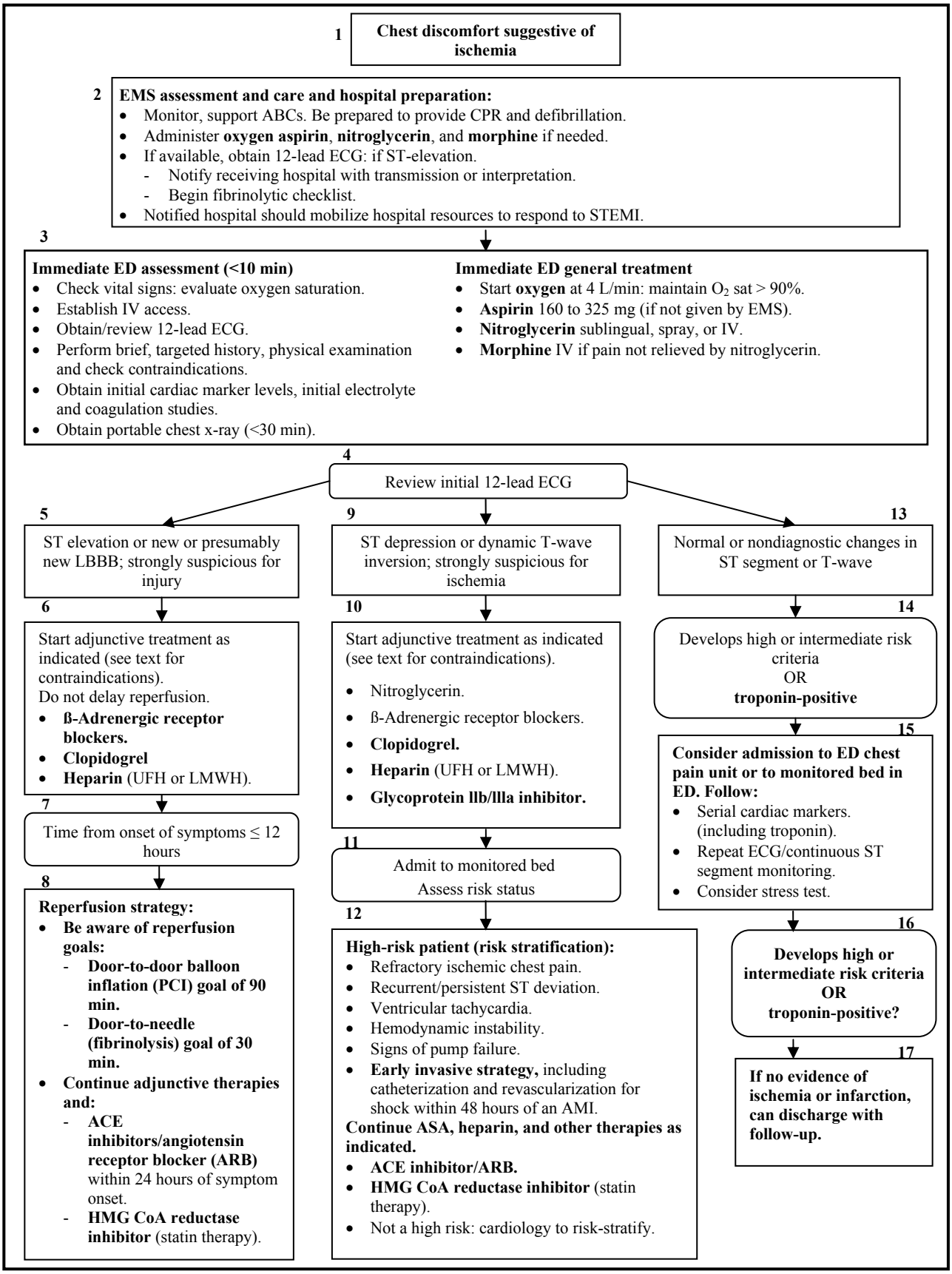


Figure 13.2: Acute Coronary Syndromes Algorithm



Chapter 14: Severe Bronchial Asthma

GUIDELINE OBJECTIVES:

- Diagnose severe bronchial asthma.
- Identify common precipitating factors.
- Understand bronchial asthma and status asthmaticus management.
- Recognize and manage bronchial asthma during pregnancy.

1. INTRODUCTION

Asthma is a chronic inflammatory disorder characterized by increased airway responsiveness to multiple stimuli. In susceptible individuals, the inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning. These episodes are usually associated with widespread, but variable, airflow obstruction, that is often reversible either spontaneously, or with treatment. The recognition that asthma is a chronic inflammatory disorder of the airways has significant implications for the diagnosis, management, and prevention of acute exacerbations.

2. CLASSIFICATION

Bronchial asthma has a wide range of disease severity (mild to severe) and two states (chronic and acute). Chronic asthma is insidious, and the patient lives with various levels of disability. The patient's condition can change rapidly for a variety of allergen and environmental triggers. Acute exacerbations require the patient to seek urgent care in the emergency department (ED) (including exercise and pregnancy). Chronic forms of asthma include mild intermittent, mild persistent, moderate persistent, and severe persistent. The acute refractory attack is referred to as status asthmaticus, and is life threatening for the patient.

3. CLINICAL FEATURES

3.1. History

The history should include prior medical history highlighting, intubations, hypoxic seizure, or ICU admissions.

3.2. Asthma Categories

See table 14.1.

A. Chronic:

- Mild intermittent asthma:
  - Symptoms < once/week.
  - Night symptoms < twice/month.
  - Normal pulmonary expiratory force (PEF) between exacerbations.

- Mild persistent:
  - Symptoms > once/week < once/day.
  - Night symptoms < twice/month.
  - Pulmonary expiratory force (PEF) > 80% of predicted.
- Moderate persistent:
  - Daily symptoms.
  - Exacerbations affect activity and sleep.
  - Night symptoms > once/week.
  - Pulmonary expiratory force (PEF) 60-80% of predicted.
- Severe persistent:
  - Continuous symptoms and frequent exacerbations.
  - Frequent nighttime symptoms.
  - Physical activity limited by symptoms.
  - Pulmonary expiratory force (PEF)< 60% of predicted.

B. Acute:

- Severe shortness of breath.
- Usual precipitating factors:
- Non-compliance with medications.
- Exposure to allergens or medications.
- Sinus infection.
- Stress.

3.3. Examination

- Cannot complete one sentence.
- Respiratory rate > 25/min.
- Heart rate > 110/min.
- On auscultation, bilateral wheezes with respiratory distress.
- Arterial blood gases indicate respiratory alkalosis (but acidosis in prolonged severe attacks).

3.4. Life-Threatening Asthma

- Silent chest.
- Feeble respiratory effort.
- Bradycardia, arrhythmias, and hypotension.
- Exhaustion, confusion, and coma.
- Saturation of oxygen (SaO_2) <92%.
- Hypercapnia requiring mechanical ventilation occurs in near fatal asthma.

4. MANAGEMENT

4.1. Medical Management

See figures 14.1 and 14.2.

A. Chronic Asthma:

- Mild intermittent:
  - Short-acting β-agonist (Ventolin) 2 puffs qid & prn. Oral steroids may be added for severe exacerbations.
- Mild persistent:
  - Long-acting β-agonist (Serevent and formeterol), steroid inhaler and (PRN) short-acting β-agonist (Ventolin).
- Moderate persistent:
  - Inhaled long-acting β-agonist (Serevent and formeterol) and inhaled corticosteroids (Fluticasone or Budesonide).
- Severe persistent:
  - Long-acting β-agonist (Serevent and formeterol) and (PRN) short-acting β-agonist (Ventolin) and inhaled steroid (Fluticasone or Budesonide) and/or oral steroid.

Key Points:
- In cardiac patients, aminophylline may cause tachycardia.
- In diabetic patients, replace glucose with normal saline, or add 10 units regular insulin to the glucose drip.
- In hypertensive diabetic patients, use glucose with insulin in the drip.

B. Acute Asthma Exacerbation:

- If respiratory arrest is imminent alert the cardio pulmonary resuscitation (CPR) team:
  - Give oxygen freely.
  - Start β-agonist (Ventolin) by nebulization 2.5mg every 15-20 minutes delivered with O_2.
- If a nebulizer is not available, then administer four to eight puffs of ventolin (equivalent to 2.5 mg nebulized ventolin):
  - Continuous nebulization is more effective than intermittent dosing.
  - In young patients with severe airflow limitation (life-threatening), consider administering subcutaneous epinephrine (0.2-0.5 ml of 1:1000 solution) with close monitoring.
  - Ipratropium bromide (Atrovent) 500μg may be added to ventolin nebulizer.
  - Those who fail to respond to initial β-agonist therapy should be given intravenous (IV) or oral corticosteroids. Start with methylprednisolone 125mg IV once, then prednisone 60 mg orally every twelve hours. The anti-inflammatory effect requires six hours to manifest; and it is used to prevent relapse, and decrease theβ-agonist requirements.
  - Consider Magnesium Sulphate 2 gm IV over 20 minutes in severe acute attacks with poor response to previously applied management.

- Due to the lack of demonstrated efficacy and adverse side effects, administration of IV aminophylline or oral theophylline is not recommended in acute severe asthma attacks.
- Patients previously on oral theophylline, may continue theophylline therapy after checking serum levels, and when the severe attack is controlled.

- If the patient fails to improve four hours after initiation of treatment, they should be admitted.
  - Patients with severe hypoxia, increasing (PCO_2), worsen mental status, or hypoxic seizure, should be intubated and mechanically ventilated.
  - Treat any documented exacerbating factor (antibiotics for sinusitis or chest infections, H2-blocker for gastro esophageal reflux disease (GERD).

C. Exercise Induced Asthma:

- Advise patient to use Salbutamol or Cromolyn before initiating exercise.
- Routine use of an inhaled steroid should be considered with more severe and frequent symptoms.

D. Asthma in Pregnancy:

- One third of asthma patients worsen during pregnancy.
- β-agonists have strong tocolytic effects.
- Avoid leukotriene modulators.
- Manage acute severe attacks like non-pregnant patients.
- The harmful effects of acute asthma to the mother and the fetus outweigh the potential drug adverse effects.

4.2. Laboratory Investigations

- Complete blood count (CBC).
- Random blood glucose (RBG).
- Plain x-ray.
- Electrocardiogram (ECG): To exclude cardiac asthma.
- Arterial blood gases (ABGs): if the patient does not respond to treatment, and the clinician is evaluating if the patient is suffering from respiratory failure.

Table 14.1: Classifying Asthma Exacerbation Severity

	Severe	Respiratory Arrest Imminent
Symptoms	• Breathlessness at rest. • Sits upright and erect. • Agitated speaking.	• Drowsy or confused.
Signs	• Respiratory rate usually greater than 30 breaths/minute. • Usually, loud wheezing heard throughout inhalation and exhalation. • Pulse >120 beats per minute.	• Use of accessory muscles: paradoxical thoracoabdominal movement. • Absence of wheeze (silent chest). • Bradycardia.
Functional Assessment	• <50% predicted or personal best pulmonary expiratory force (PEF).	

Figure 14.1: Emergency Department Management of Acute Asthma

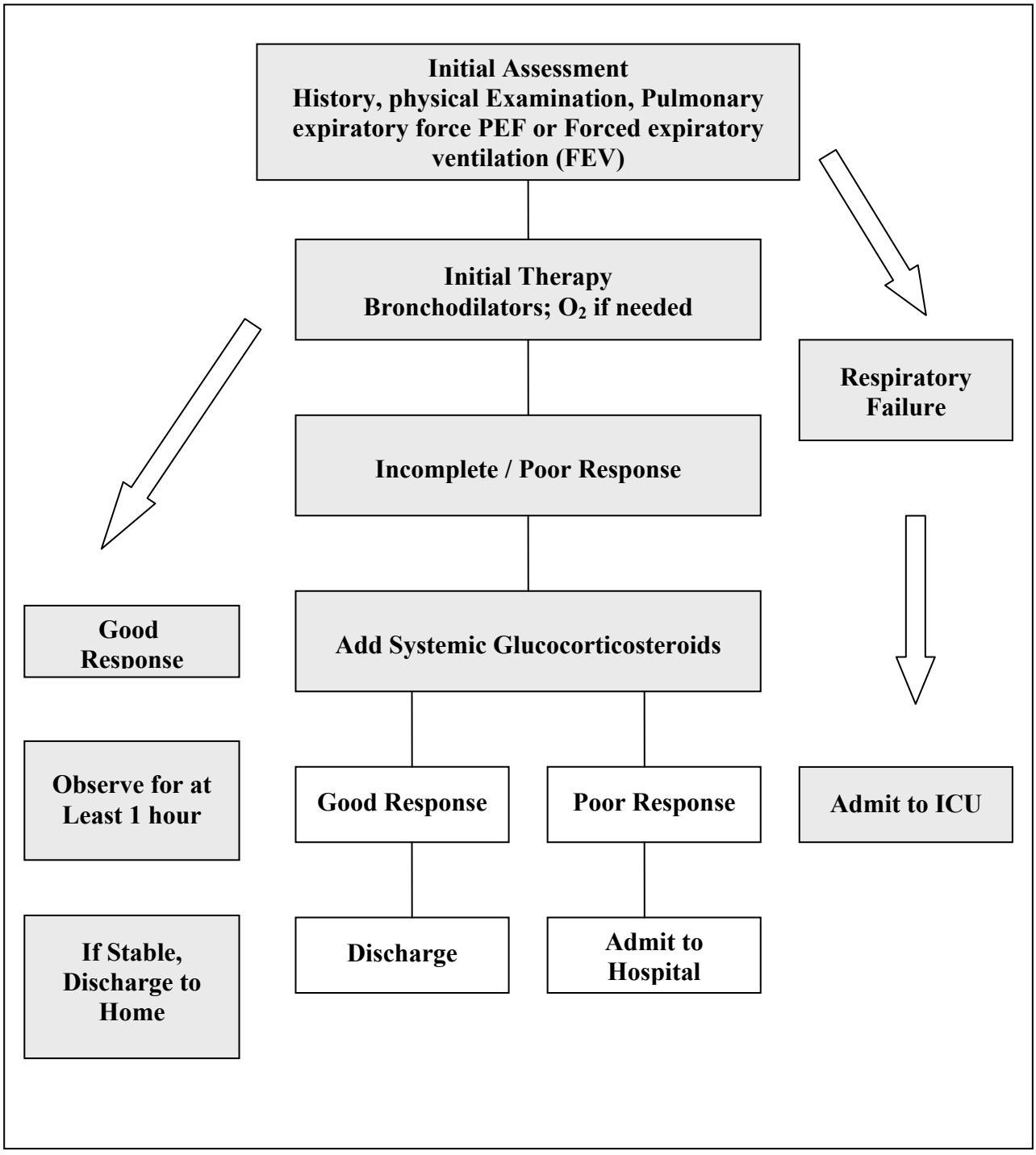
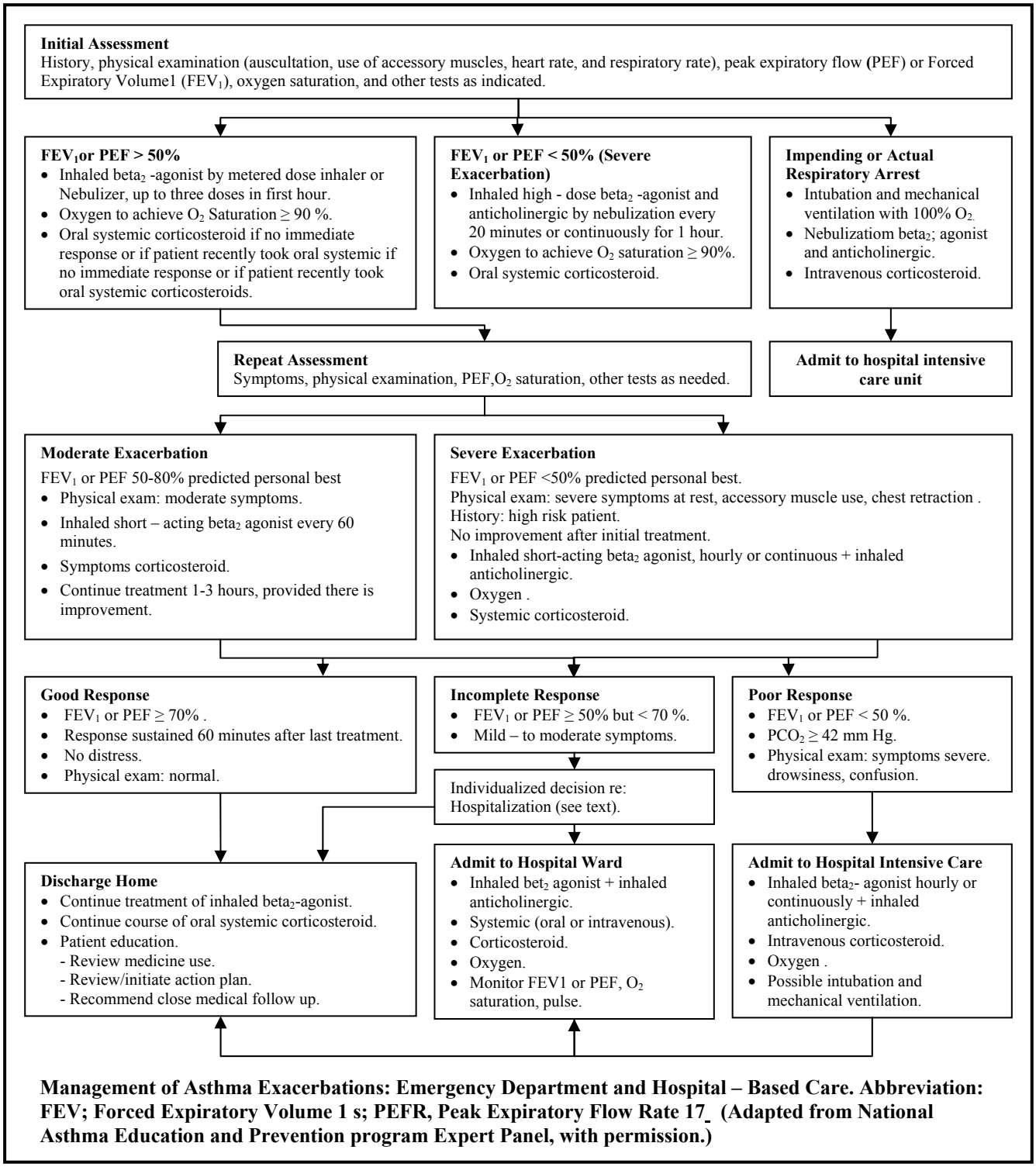


Figure 14.2: Management of Asthma Exacerbations: Emergency Department and Hospital-Based Care



Management of Asthma Exacerbations: Emergency Department and Hospital – Based Care. Abbreviation: FEV; Forced Expiratory Volume 1 s; PEFR, Peak Expiratory Flow Rate 17_ (Adapted from National Asthma Education and Prevention program Expert Panel, with permission.)

Chapter 15: Diabetic Emergencies

GUIDELINE OBJECTIVES:

- Understand the causes and treatment of hypoglycemic emergencies.
- Recognize diabetic keto-acidosis (DKA) and hyperosmolar hyperglycemic state (HHS) and treat the patient appropriately.

1. INTRODUCTION

Diabetes Mellitus is the most common endocrine disease. It comprises a heterogeneous group of hyperglycemic disorders characterized by high serum glucose and disturbances of carbohydrate and lipid metabolism. Acute complications include hypoglycemia, diabetic ketoacidosis (DKA), and hyperglycemic hyperosmolar nonketonic coma (HHNG).

According to the Jordanian National Center for Diabetes, Endocrinology and Genetics 2006. In Jordan, the age-standardized prevalence of diabetes and IFG was 17.1% and 7.8% respectively, with no significant differences between women and men. More than half (54%) of those previously diagnosed were found to be with unsatisfactory glycemic control.

2. DEFINITION

Biochemically, hypoglycemia is defined as a decrease blood glucose level below 50mg/dl (2.7mmol/dl). More specifically, it is defined as a decreased blood glucose level that results in demonstrable signs or symptoms. The glucose level at which an individual becomes symptomatic is highly variable. Insulin or oral hypoglycemic diabetic drug (Sulphonylurea, Repaglinide, Nateglinide) therapy accounts for the vast majority of cases of severe hypoglycemia encountered in ED due to:

- Delaying meals.
- Unusual physical exertion.
- Excessive doses of exogenous insulin.
- Unusual fluctuation in insulin absorption from varying injection sites.
- Impaired counter regulatory mechanisms due to autonomic neuropathy.
- Infection.
- Renal impairment.

Oral hypoglycemic drugs which don't cause hypoglycemia:

- Metformine.
- Thiazolidinediones: (Rosiglitazone & Pioglitazone).
- Glucosidase inhibitors (Acarbose & Miglitol).

Diabetic ketoacidosis DKA and hyperosmolar hyperglycemic state HHS, also known as non-ketotic hyperglycemia) are two of the most serious acute complications of diabetes. DKA and HHS differ clinically according to the presence of ketoacidosis and the degree of hyperglycemia.

3. CLASSIFICATION

- Hypoglycemia.
- Diabetic ketoacidosis (DKA).
- Hyperosmolar hyperglycemic state (HHS).

4. CLINICAL FEATURES

4.1. Hypoglycemia

Whipples triad is the basis of clinical diagnosis, it includes:

- Hypoglycemic symptoms.
- Associated low blood glucose levels.
- Symptomatic relief with glucose administration.

Typical symptoms of hypoglycemia include: sweating, shakiness, anxiety, nausea, headache, confusion, bizarre behavior, slurred speech, blurred vision and coma. Neurologic manifestations are cranial nerve palsy, hemiplegia, leading to seizure. Breathing may be normal or depressed.

4.2. Diabetic Ketoacidosis (DKA)

DKA is characterized by the triad of:

- Hyperglycemia.
- Anion gap metabolic acidosis.
- Ketonemia.

The serum glucose concentration is usually greater than 500 mg/dL (27.8 mmol/L) and less than 800 mg/dL (44.4 mmol/L). However, serum glucose concentrations may exceed 900 mg/dL (50 mmol/L) in patients with DKA who are comatose. In certain instances, such as DKA in the setting of starvation or pregnancy, or treatment with insulin prior to arrival in the emergency department, the glucose may be only mildly elevated.

A. Precipitating Factors:

- Concurrent medical illness (60%).
- Omission of treatment.
- Emotional factors and stress.
- New onset type 1 diabetes, in which DKA is a common presentation.

B. Features:

- Nausea, vomiting, and abdominal pain (mainly in children).
- Unexplained tachycardia.
- Dehydration.
- Kaussmul respiratory pattern.
- Pain, coma, and shock.

C. Laboratory Findings:

- Blood sugar 500-600 mg/dl.
- Blood glucose readings of less than 350 mg/dl (15% of patients),
- Anion gap is high.
- Metabolic acidosis.
- Na could be low.
- Low (pH).
- Serum creatinin is elevated.
- Positive ketones in serum and urine.

Elevated anion gap metabolic acidosis is due to the production and accumulation of beta-hydroxybutyrate and acetoacetate. Patients with DKA usually present with a serum anion gap (AG) greater than 20 meq/L. *The normal AG had been considered to range between 7 and 13 meq/L*.

Serum anion gap = Serum sodium – (serum chloride + bicarbonate)

4.3. Hyperosmolar hyperglycemic state (HHS)

Hyperosmolar hyperglycemic state HHS, also known as non-ketotic, hyperglycemia). HHS is diagnosed by random blood glucose (RBG)>500mg% + no acetone in urine.

- There is little or no ketoacid accumulation.
- The serum glucose concentration frequently exceeds 1000 mg/dL (56 mmol/L).
- The plasma osmolality may reach 380 mosmol/kg.
- Neurological abnormalities are frequently present (including coma in 25 to 50 percent of cases).
- Most patients with HHS have on admission pH >7.30, a serum bicarbonate >20 meq/L, a serum glucose >600 mg/dL (33.3 mmol/L), *and test negative for ketones in serum and urine;* although mild ketonemia may be present minimal ketonuria and ketonemia .
- If plasma osmolarity cannot be directly measured, it may be calculated with this formula: plasma osmolarity = 2 sodium+ potassium ($Na + K^+$) + BUN/3 + glucose/18.
- Urine osmolarity also is increased.

5. MANAGMENT

5.1. Hypoglycemia

- The initial approach should include the following:
- Airway breathing circulations (ABCs), intravenous (IV) access, oxygen, monitoring,
- and gluco-check.
- Administration of glucose as part of the initial evaluation of altered mental status often corrects hypoglycemia.
- *Intravenous (I.V) glucose: 50ml of 50% dextrose at 10ml/min for a comatose patient*. A continuous infusion of 10% dextrose may be required to maintain the blood sugar above 100mg/dl.

- If intravenous (IV) glucose is not available or there is difficulty gaining IV access. A subcutaneous or intramuscular injection of 0.5 to 1.0 mg of glucagon will usually lead to recovery of consciousness within 10 to 15 minutes, although it is often followed by marked nausea 60 to 90 minutes later.
- Oral feeding of fruit juice and long acting carbohydrates as soon as the patient regains consciousness.

A. Discharge:

Factors considered in determining disposition include: the patient's response to treatment, cause of hypoglycemia, co-morbid conditions, and social situation. Most diabetics with uncomplicated insulin reaction respond rapidly. They can be discharged with instructions *to continue oral intake of carbohydrates and closely monitor their finger stick glucose*.

- Admission criteria:
  - No obvious cause.
  - Oral hypoglycemic use.
  - Long-acting insulin.
  - Persistent neurological deficits.
  - If there is not a prompt response to glucose infusion.

5.2. Diabetic Ketoacidosis (DKA)

The most important treatment in DKAin the ED is to give (0.9 percent) saline as rapidly as possible to patients with signs of shock. IV fluids should be instituted before Insulin treatment.

A. Insulin:

- 0.1 unit/kg (IV) (10 UNITS) regular insulin.
- 0.1 unit/kg 1hour after.
- Then follow the insulin infusion protocol:
- Standard insulin concentration: 1 unit regular insulin/10ml normal saline (NS (25 units mixed with 250 ml NS).
- Capillary glucose measured hourly for the first 6-8 hours while receiving insulin infusion.
  - If blood glucose is stable, less frequent monitoring (every 2 hours) is acceptable.
- A lower dose of 0.05 unit/kg per hour may be used initially in younger children who may be more sensitive to insulin. *An insulin bolus should NOT be given:*
  - If the blood sugar doesn't decrease by 50-70mg/dl in the first hour, insulin doses can be doubled.
  - If blood sugar reduction is more than 100-150 mg/hour, the dose of infused insulin should be reduced.
- A common mistake is to allow blood glucose to drop to hypoglycemic levels.

B. Fluid Replacement:

Normal saline (isotonic saline 0.9%) should be infused as quickly as possible in patients who are in shock. In the absence of cardiac compromise, normal saline 0.9% is infused at a rate of 15 to 20 mL/kg/hour during the first few hour.

- One liter 0.9% (NS) infused quickly.
- Then, three liters over three hours.
- Then, 250-500 ml/hour of 0.45% NS. One-half isotonic saline is preferred since the addition of potassium to isotonic saline will result in a hypertonic solution that will delay correction of the hyperosmolality
- When plasma glucose reaches <250mg/dl change to 5%dextrose with 0.45 saline 150-250 ml/hour and reduce insulin to 0.05-0.1 unit per Kg.

C. Potassium Concentration:

The range of K^+ administration depends on serum K^+ level (see table 15.1).

Table 15.1: Range of Potassium Administration

Potassium Level	Potassium Administration Needed
5.3 mEq	no K^+ required
4.5-5.3	10mEq/hour
4-4-5mEq	20 mEq/hour
3-4mEq	30 mEq/hour

- Give 10-30 mEq of potassium in each liter of fluid to maintain serum potassium at 4-5 mEq/L. (assuming an adequate urine output >50 mL/h).
- If serum potassium is less than < 3.3 mEq/L, insulin treatment should be delayed till serum K^+ is restored by IV potassium infusion.

D. Bicarbonate ($HCO3^-$), Replacement:

HCO_3^- *therapy is not indicated in routine management of (DKA), and is not recommended.*

E. Diabetic Ketoacidosis (DKA) Resolution:

- Ketoacidosis is considered to be resolved when the following goals are reached:
  - Serum glucose below 200 mg/dL (11.1 mmol/L).
  - Serum anion gap <12 meq/L.
  - Serum bicarbonate ≥ 18 meq/L.
  - Venous pH >7.30.
  - The patient is able to eat.
  - Negative ketones in the serum.
  - Regardless of when rapid or short-acting subcutaneous insulin is started, intravenous insulin infusion should be continued for an overlap of one to two hours.
  - Patients with known diabetes who were previously treated with insulin may be given insulin at the dose they were receiving before the onset of DKA.
  - In insulin-naive patients, a multi-dose insulin regimen should be started at a dose of 0.5 to 0.8 U/kg per day.

F. Complications:

- Hypokalemia and cardiac dysrhythmia.
- Hyperglycemia.
- Pulmonary edema (non cardiogenic).
- Cerebral edema.

5.3. Hyperosmolar Hyperglycemic State (HHS)

A. Fluid Resuscitation:

- Normal saline should be infused as quickly as possible in patients who are in shock.
- Isotonic sodium chloride solution is the fluid of choice to begin treatment. Usually, two liters of 0.9% isotonic sodium chloride solution may be infused safely over the first hour of treatment.
- Then 250-500 ml one-half isotonic saline (0.45% NS) infused if the corrected serum sodium is normal or elevated; isotonic saline at a similar rate is appropriate if the corrected serum sodium is low.
- Serum Na^+ should be corrected for hyperglycemia (for each 100 mg/dl glucose >100 mg/dl, add 1.6 mEq to sodium value for corrected serum sodium value).

B. Correction of Hyperglycemia:

- Immediate treatment with insulin is contraindicated in the initial management of patients with HHS.
- After the kidneys show evidence perfusion, as evidenced by adequate urinary output, initiating insulin therapy is safe.
- Infuse insulin separately from other fluids, and do not interrupt or suspend the infusion of insulin once therapy is started. The following guideline for insulin infusion may be used:
  - Begin a continuous insulin infusion of 0.1 U/kg/h.
  - Hourly monitor blood glucose at the bedside. If stable for 3 hours, then decrease the frequency of testing to every 2 hours.
  - Set the target blood glucose level at 250-300 mg/dL. This may be adjusted downwards after the patient is stabilized.
  - For blood glucose concentrations of less than 250 mg/dL, decrease the insulin infusion rate by 0.5 U/h.
  - For a blood glucose concentration of 250-300 mg/dL, do not change the insulin infusion rate.
  - For a blood glucose concentration of 301-350 mg/dL, increase the insulin infusion rate by 0.5 U/h.
  - For a blood glucose concentration of 351 mg/dL or greater, increase the insulin infusion rate by 1 U/h.
  - Do not discontinue the insulin drip.
  - If a decrease of more than 100 mg/dL occurs between consecutive readings, wait do not increase the insulin infusion rate.

C. Goals of Therapy:

- Gradual drop of serum sodium and osmolality.
- Gradual drop in serum glucose.
- Rapid change in the above may cause brain insult termed disequilibrium syndrome.
- Keep serum glucose between 250-300 mg/dl until plasma osmolality <315mosmol/kg.
- Treat the underlying diseases.

D. Hyperosmolar Hyperglycemic State (HHS) Resolution:

- HHS is considered to be resolved when the following goals are reached:
  - Patients are mentally alert.
  - The plasma effective osmolality is below 315 mosmol/kg.
  - The patient is able to eat.

Chapter 16: Coma

GUIDELINE OBJECTIVES:

- Implement the initial assessment of a comatose patient.
- Institute the airway breathing circulations (ABCs) of resuscitation.
- Utilize the diagnostic approach with a comatose patient.
- Order appropriate investigations for a comatose patient.
- Consult appropriate specialties in a timely manner.

1. INTRODUCTION

Coma is a condition in which the patient appears to be asleep, but is incapable of responding either to external stimuli or inner needs. A patient in a coma is unable to interact appropriately with his environment. Coma can be produced by bilateral cerebral hemispheric dysfunction or by injury to the reticular activating system (RAS).

2. CLASSIFICATION

Based on the anatomic location of the lesion and the neurologic disease mechanism, the causes of coma can be classified into four major groups:

- Metabolic and diffuse cerebral disorders.
- Supratentorial lesions.
- Infratentorial lesions.
- Psychogenic coma.

Up to 75% of patients in a comatose state, without obvious cause, will likely have a diffuse systemic disorder. Structural lesions make up the remaining causes of coma; supratentorial lesions are more common than subtentorial lesions.

3. CLINICAL FEATURES

3.1. History

The history is vital and should include:

- Onset (sudden or gradual).
- The state of the patient's health prior to the onset of coma.
- The patient accessibility to drugs or poisons.
- Inquiry concerning condition that commonly cause coma (trauma, epilepsy, drug abuse, cerbrovascular (CV) diseases, pulmonary disease, cerebravascular disease, metabolic disorders, infections, neoplasm).
- History of psychiatric illness.
- Occupational or environmental exposures (e.g. CO). cyanide, organic solvents, lead exposure).
- Past medical history: Diabetes mellitus (DM), hypertension (HTN), epilepsy, liver, renal, respiratory failure, endocrine disorder.

3.2. Vital Signs

A. Temperature:

Fever may suggest infection, thyroid storm, heat stroke, or anticholinergic toxicity.

Hypothermia may suggest myxoedema, cold exposure, intoxication with ethanol, or barbiturate abuse.

B. Heart Rate:

Bradycardia, hypertension, and bradypnea may indicate increase intracranial pressure (ICP) (cushing's triad). Tachycardia (>100 beats per minute) in ectopic paroxysmal tachyarrhythmias.

C. Blood Pressure:

Hypotension may indicate ethanol or barbiturates intoxication, hemorrhage (Hge), shock, or myocardial infarction (MI). Hypertension may indicate hypertensive encephalopathy, cerebral or brain stem infraction, or subarachnoid hemorrhage.

D. Respiratory Rate:

- Bradypnea: ethanol, narcotic or barbiturates intoxication.
- Tachypnea: hypoxia or sepsis.
- Hyperpnea: metabolic acidosis.

3.3. Physical Examination

A. Respiratory Pattern:

- A normal breathing pattern suggests the absence of brain stem damage.
- Chyne-stokes respiration (periods of waxing and waning hyperpnea alternating with shorter periods of apnea) implies bilateral hemispheric dysfunction with the brain stem intact. It may occur in metabolic disorders and congestive heart failure.
- Kussmaul respiration: metabolic acidosis.
- Apneustic respiration (prolonged inspiration followed by an expiratory pause) signifies a pontine lesion.
- Ataxic (irregular) breathing signifies a medullary lesion.
- Central neurogenic hyperventilation (deep rapid breathing) indicates involvement of the brain stem between the midbrain and pons.

B. Odor of the Breath:

- A fruity odor suggests diabetic keto-acidosis.
- A uriniferous odor is found in uremia.
- Fetor hepaticus points to hepatic encephalopathy .
- The odor of alcohol is characteristic.
- A burnt almond odor is found with cyanide toxicity.
- A garlic scent maybe smelled in arsenic poisoning.

C. Skin:

- Jaundice, spider angiomata, and palmar erythema point to hepatic encephalopathy.
- Petechiae and ecchymoses suggest a coagulation abnormality or thrombocytopenia.
- A maculohemorrhagic rash suggests meningococcal infection or staphylococcal endocarditis.
- Cherry-red skin suggests carbon monoxide poisoning.
- Needle marks on extremities indicate possible drug abuse.
- Check skin turgor.

D. Body Orifices:

- Bleeding from the ears or nose suggests cranial trauma.
- Bleeding from other orifices suggests coma from a bleeding disorder or hemorrhage.

E. Central Nervous System:

Posture in bed:

- Decorticate rigidity is characterized by flexion of arms and elbows with hyperextension of the legs, signifies bilateral hemispheric dysfunction with the brain stem intact.
- In decerebrate rigidity, the arms and legs are in an extended position; it reflects damage to the midbrain and upper pons.

Meningeal signs:

- Resistance to passive flexion of the neck without resistance to other neck movements is evidence of meningitis or subarachnoid Hge.
- Restriction of movement of the neck in all directions may occur in generalized rigidity or disease of the cervical spine.
- Positive brudzinki's sign i.e. flexion of the hips on passive flexion of the neck.
- Positive Kernig's sign i.e. pain or resistance of the hamstrings when the knees are extended with the hips flexed at 90 degree.
- Eye movements.
- When eyelids are opened, if the eyes flutter upwards, exposing only the sclera, suspect psychogenic coma.
- In comatose patients without involvement of the neural pathways influencing ocular movements, the eyes usually directed straight ahead or display slow roving (spontaneous eye) movements.
- Sustained, involuntary conjugate deviation of the eyes toward the unaffected side suggests a hemispheric lesion; towards the paralyzed side, a pontine lesion.
- Absence of oculocephalic (doll's eye movement) and corneal reflexes indicates pontine dysfunction.
- Oculovestibular reflexes (cold calorics) may be lost in brain stem lesions.

Pupils:

- Equal, round, reactive pupils exclude midbrain damage as a cause and suggest metabolic abnormality.
- Reactive pupils in association with absent oculocephalic and corneal reflexes, generally signify a metabolic encephalopathy or drug overdose.

- Pinpoint pupils (<1mm) may indicate opiate, pilocarine or pontine lesion.
- A unilateral, fixed and dilated pupil suggests ipsilateral temporal lobe herniation.
- Bilateral, fixed and dilated pupils suggest anticholinergic poisoning, anoxia, severe midbrain lesion or brain death. Also in response to some drugs, such as atropine or glutethimide.

Motor system:

- Hemiplegia, hyperreflexia and an extensor plantar response indicate a structural lesion of the brain as the cause of coma.
- Hyporeflexia without paralysis and with preservation of normal planter response suggests a metabolic cause or drug ingestion.

Sensory system:

- Sensory loss may be suspected if the patient exhibits variations in responsiveness to noxious stimuli.

3.4. Coma Types

A. Metabolic Encephalopathy:

- Hypoventilation, abnormal respiratory pattern.
- Reactive pupils (a midbrain function) in the presence of impaired function of the lower brain stem (e.g. hypoventilation, loss of extra ocular movements).
- Symmetric neurologic findings.
- No focal hemispheric lesions (hemi paresis, hemi sensory loss, aphasia) before loss of consciousness.
- Random eye movements, but not persistent ocular deviations.
- Tremors, asterixis, multifocal myoclonic jerks and seizures.

B. Supratentorial Lesions:

- Premonitory symptoms as headache or seizure.
- Symptoms and signs of hemispherical dysfunction are usually present (sensory or motor disturbances, aphasia, visual field defects) before onset of coma.
- Signs of bilateral hemispheric dysfunction (e.g. decorticate posturing).
- When coma is progressive, the patient exhibits signs of transtentorial herniation.
- Uncal herniation (herniation of the medial portion of the temporal lobe i.e. the uncus across the cerebellar tentorium) produces midbrain signs.
- Ipsilateral (unilateral) pupil dilatation and oculomotor nerve paralysis.
- Progressive impairment of the level of consciousness.

C. Central Herniation:

- Loss of consciousness.
- Marked unilateral papillary dilatation, loss of light reactivity, and oculocephalogyric reflex.
- Decerebrate posturing.

D. Infratentorial Lesions:

- Coma (sudden onset).
- Conjugate gaze toward lesion i.e. the eyes are directed away from the side of the lesion and towards the hemisphere.
- Disconjugate eye movements with doll's eye or caloric testing, strongly suggest a subtentorial lesion.
- Pinpoint non reactive pupils, are often present in pontine or cerebellar Hge or infarction.

Cholinesterase inhibitors and opiates also produce pinpoint pupils.

E. Psychogenic Coma:

- Patient is unresponsive.
- Normal physical examination.
- Flaccid symmetric decreased muscle tone.
- Normal and symmetric reflexes.
- Normal Babinski (down response).
- The pupils are normal in size (2-3mm).
- Voluntary muscle tone of the eyelids during passive examination.
- Ice water caloric test.

4. MANAGEMENT

See figure 16.1.

- Priorities include airway, breathing, and circulation, (ABCs) monitoring, venous access, and high flow oxygen.
- Maintain cervical spine stabilization, if trauma cannot be ruled out.
- Examine pupils or reactivity to light, and evaluate movement of extremities for rapid determination of coma cause (i.e. diffuse versus structural).
- The "coma cocktail" is an empiric treatment for common conditions presenting as coma.

It consists of:

- Thiamine 100mg intravenous (IV).
- Glucose: 50ml- D 50% (in children 2ml / kg of D25W) IV.
- Naloxone: 0.4-2mg IV repeated doses, up to 5 to 10 mg may be required.

Flumazenil is not included in the coma cocktail because of its ability to induce seizures and cardiac arrhythmias. It should be used only if the patient's coma is definitively caused by benzodiazepine use. Do not give Flumazenil if patient has ingested a tricyclic antidepressant, other convulsant drug, or has a seizure disorder.

4.1. Investigations

- Complete blood count (CBC).
- Biochemistry: glucose, urea, creatinine, bilirubin, alkaline phophatase, transaminases, Calcium, Magnesium, (serum glucose can be rapidly checked with a glucometer).

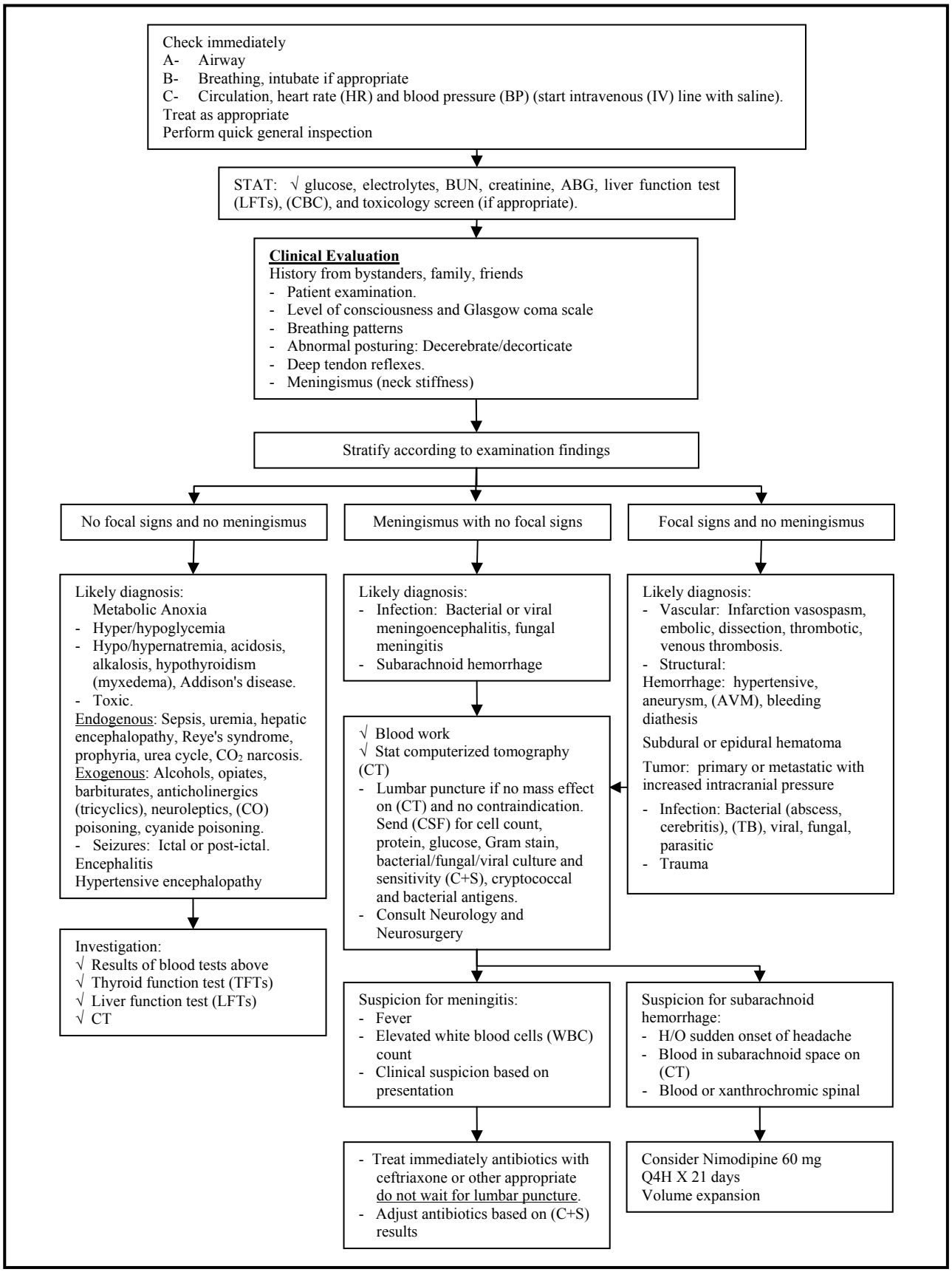
- Platelets and coagulation profile.
- Blood and urine culture if infection is suspected.
- Arterial blood gases (ABG).
- Toxicology screening.
- Chest films.
- Electrocardiogram (ECG).
- Computerized tomography (CT) scan of the head. Especially in the presence of focal neurologic signs, papilledema, or in the absence of any other etiology.

Key Points:

- Coma is commonly associated with ingestion of large doses of antihistamines, barbiturates, benzodiazepines, other sedative hypnotic drugs, γ-hydroxybutyrate(GHB), ethanol, opioids, phenothiazines, or antidepressants.
- The most common cause of death for the comatose patients is respiratory failure, which may occur abruptly.
- Aspiration of gastric contents may also occur, especially in victims who are deeply obtunded or convulsing.
- Hypoxia and hypoventilation may cause or aggravate hypotension, arrhythmias, and seizures.

Thus, protection of the airway and assisted ventilation are the most important treatment measures for any comatose patient.

Figure 16.1: Management of Coma Patient Algorithm



Chapter 17: Management of Seizures (Convulsion)

GUIDELINE OBJECTIVES:

- Define different types of convulsions.
- Implement the airway, breathing, and circulation, (ABC's) of resuscitation.
- Utilize medications to control convulsions.
- Identify initial investigations needed (laboratory and radiological) and interpret the results.
- Recognize indications for hospital admission.

1. INTRODUCTION

The most common convulsion cause is epilepsy. Epilepsy is the continuing tendency to have seizures even if attacks are separated by long intervals of time. A seizure is a convulsion, or transient abnormal event resulting from paroxysmal discharge of cerebral neurons. The most life-threatening form of seizure is status epileptics. Status epilepticus is defined by continuous seizures that occur without recovery of consciousness, or serial seizures with incomplete recovery of consciousness. Epilepsy is idiopathic in 75% of cases, and secondary in 25% of cases.

Other causes of convulsions include:

- Genetic and development abnormality.
- Traumatic hypoxia.
- Pyrexia.
- Brain mass.
- Alcohol – drug withdrawal.
- History of drug addiction.
- Metabolic abnormality like:
  - Hypoglycemia.
  - Hypocalcaemia.
  - Hyponatraemia.
  - Intoxication.
  - Uraemia.
  - Cholaemia.
  - Eclampsia.
  - Hypoxia.
  - Alkalosis.
  - Water retention.

1.1. Seizures Side Effects

- Increased brain activity, consumes more oxygen and glucose.
- Increased body muscle tone leads to respiratory insufficiency or aspiration, which results in brain anoxia.

- Sudden diaphragmatic contraction leads to vomiting and possible aspiration. *Aspiration is the main cause of death after an epileptic fit.* The second cause of death is trauma due to convulsive movements and falling down.
- Muscular pains, exhaustion, then sleep (post-ictal stage).

2. CLASSIFICATION

- Generalized: implies abnormal electrical activity that is belated in the brain, conscious is impaired.
- Partial seizures: Localized seizures
- Simple: Without loss of consciousness.
- Complex: With loss of consciousness.

3. CLINICAL FEATURES

3.1. History

Important avenues of inquiry include:

- Preceding aura.
- Onset (abrupt or gradual).
- Progression of motor activity.
- Loss of bowel or bladder control, seizure activity (local or generalized), symmetry.
- Any postictal confusion or lethargy.

The patient should be asked whether he or she has any recollection of the attack. Next, the history should be directed toward factors that may have precipitated epileptic activity. Including missed doses or recent alterations in antiepileptic medications, dosage changes, or the conversion from brand name to generic formulations. Other possible factors that might provoke a seizure include sleep deprivation, alcohol withdrawal, infection, and use/cessation of other drugs.

If there is no previous history of seizures, a more detailed history is needed. Symptoms that might suggest previous unwitnessed or unrecognized seizures include blank or staring spells in school, involuntary movements, unexplained injuries, nocturnal tongue biting, and enuresis. A history of recent or remote head injury; persistent, severe, or sudden headache should prompt a search for intracranial pathology. Concurrent pregnancy or recent delivery suggests the possibility of eclampsia. A history of metabolic derangements, electrolyte abnormalities, hypoxia, systemic illness (especially cancer), coagulopathy/anticoagulation, drug ingestion/withdrawal (licit and illicit), and alcohol use, may help identify factors that predispose the patient to seizures.

3.2. Physical Examination

The general physical examination should be directed toward discovering any injuries, especially for the head or spine. Tongue lacerations and aspiration are frequent sequelae. A search for any systemic illness that may have caused the attack should be undertaken. Temperature should be noted, and a bedside glucose determination should be obtained. A directed neurologic examination should be performed with follow-up serial exams. Level of consciousness and mentation should be followed closely. Profound obtundation that improves steadily is likely benign; however, progressive deterioration requires prompt intervention. Investigate for signs of increased

intracranial pressure. Any focal neurologic deficit should be noted. A transient focal deficit following a simple or complex focal seizure is referred to as Todd ´s paralysis, and should resolve within 48 hours.

3.3. Differential Diagnosis

Many episodic disturbances of neurologic function may be mistaken for seizures, this includes:

- Syncope.
- Psychogenic seizures or pseudoseizures.
- Hyperventilation syndrome.
- Movement disorders, such as dystonia, chorea, myoclonic jerks, tremors, or tics, may occur in a variety of neurologic conditions.
- Migraines, those proceeded by an aura, are similar in nature to partial seizures.

Clinical features that help to distinguish seizures include:

- Abrupt onset and termination. Although some focal seizures are preceded by auras that last 20 to 30 seconds (or more), most attacks begin abruptly. Attacks that develop over several minutes, or longer, should be regarded with suspicion. Most seizures last only one or two minutes, unless the patient is in status epilepticus.
- Lack of recall. Except for simple partial seizures, patients cannot recall the details of an attack.
- Movements or behavior during the attack are purposeless or inappropriate. Rare exceptions have been described.
- Most seizures, except for simple absence attacks (petit mal) or simple partial seizures are followed by a period of postictal confusion and lethargy.

Protect the patient form injury during resuscitation.

3.4. Laboratory Investigations

A. Blood:

- Complete blood count (CBC).
- Arterial blood gases (ABG).
- Liver function
- Electrolytes (especially calcium (Ca)).
- Blood sugar.
- Drug/toxic screen.

B. Imagine Studies:

- Electro encephalopathy (EEG).
- Brain computerized tomography (CT).
- Electrocardiogram (ECG) and rhythm trace monitoring.

4. MANAGEMENT

4.1. Airway Management

Open the patient's airway and institute aspiration precautions utilizing suction and the recovery position. (Left lateral decubitus position), any dentures should be removed a bite block should be placed to protect the tongue.

4.2. Breathing

Supply pure oxygen, guided by serial pulse oximeter readings, to *ensure oxygen saturation greater than 94%.* Generally, oxygen delivery occurs via face mask; however, if seizures persist, the patient may need intubation.

4.3. Circulation

Insert a large bore peripheral line and withdrawal appropriate laboratory samples for investigation.

Correct hypoglycemia if present with simultaneous administration of thiamine.

4.4. Immediate Management

See table 17.1.

Medications administered in a step-wise fashion.

- First – Line drug:
  - Lorazepam (Ativan): 2mg intravenous (IV) push, a maximum dose of 10 mg.
  - Diazepam (Valium):
    - Adult 5-10 mg/dose, may repeat every ten minutes, as needed for a total of three doses.
    - Pediatric: 0.3-0.5 mg/kg/dose, may repeat every five minutes, as needed for a total of two doses.
    - In pediatric patients, if there is no IV access, give valium rectally.
- Characteristics:
  - Rapid efficacy: second to minutes.
  - Short duration action.
  - Sedative effect.
  - Potential for hypotension and respiratory depress.

If first line medications fail to control the seizure, or if the seizure restarts use:

- Second – Line drug:
  - Pheyntoin sodium (Dilantin):
    - Adult: 15-20 mg/kg bolus over 20 minutes. If initial bolus does not control the convulsion, Phenytoin sodium may be repeated at a dose of 10mg/kg
    - Pediatric: Loading dose 10 mg/kg, may repeat bolus of 5mg/kg after 2 hours. After bolus, begin steady infusion of 1mg/kg/min.
  - Phenobarbital (Tuminal):
    - Adult: 15-20 mg/kg over 30 min. If convulsions persist, a second dose of 7mg/kg can be given.
    - Pediatric: 2-5 mg/kg over 15 min.

- Thiopentone sodium: 2-3 mg/kg/dose, maintenance 1 mg/kg as needed

- Characteristics:
  - Pheyntoin doesn't sedate the patient neither causes respiratory depression.
  - It may cause hypotention, bradydysrhythmias, and vascular injury (venous thrombosis)
  - Onset of action 10-30 minute, duration of action 24 hours
- Thiamin 100 mg, used for the alcoholic patient.
- Intravenous dextrose to correct hypoglycemia (if present).

Table 17.1: Drugs Used in the Abortive Treatment of Status Epilepticus in the Emergency Department

Generic Name	Brand Name	Adult Dose	Comments
Diazepam	Valium	5-10 mg IV every 10 minutes up to 30 mg per 8-hour period	May be given per rectum in pediatrics (0.3-0.5 mg/kg)
Lorazepam	Ativan	0.1 mg/kg IV (usually 4 mg in adult); May repeat in 10 minutes, then 0.01 – 0.1 mg/kg per hour infusion	Preferred benzodiazepine owing to its longer duration of action
Midazolam	Versed	0.2 mg/kg IV bolus, then 0.05-0.6 mg/kg per hour infusion	May be given intranasally (0.2 mg/kg)
Phenytoin	Dilantin	20 mg/kg IV at < 50 mg/minute	Cardiac and blood pressure monitoring during infusion; large-bore intravenous line
Fosphenytoin	Cerebyx	20 PE/kg IV at 150 mg PE/minute	Cardiac monitoring Less risk of infusion site reaction; may be given IM
Phenobarbital	Luminal	20 mg/kg IV, then 5-10 mg/kg every 20 minutes, up to 2 g	May be given as IM loading dose
Valproate	Depakote	20-40 mg/kg at ≤6 mg/kg per minute	Unlabeled use
Propofol	Diprivan	1-2 mg/kg IV bolus, then 5-10 mg/kg per hour infusion	Intubation required; monitor hemodynamics
Pentobarbital	Numbutal	10-20 mg/kg IV load over 1-2 hours, then 0.5-1 mg/kg per hour infusion	Intubation required; monitor hemodynamics
Isoflurane	Forane , Terrell	Via general endotracheal anesthesia	Monitor with EEG
IV = Intravenous		**IM** = Intra muscular	**EEG** = Electro- Encephalogram

4.5. Indications for Hospital Admission:

American College of Emergency Physicians (ACEP) guidelines for hospital admission of patients with new-onset seizure include:

- Persistent altered mental status.
- Central nervous system (CNS) infection.
- New intracranial lesion.
- Underlying correctable medical problem.
- Significant hypoxia.
- Hypoglycemia.
- Hyponatremia.
- Dysrhythmia.
- Significant alcohol withdrawal.
- Acute head trauma.
- Status epilepticus.
- Eclampsia.

Chapter 18: Poisoning

GUIDELINE OBJECTIVES:

After the completion of this guideline the reader should be able to clinically approach and manage:

- Unknown drug ingestion.
- Insecticide exposure.
- Snake bite.
- Scorpion sting.
- Animal bite.

1. INTRODUCTION

In practice, emergency departments receive a relatively high number or poisoning cases, as they function on a round-the-clock basis and are provided with trained personnel and basic equipment for decontamination and life-support measures.

2. CLASSIFICATION

Poisoning can occur through different routes; ingestion, inhalation, skin injection or contact. The most commonly encountered are: drug and insecticide ingestion, snake bites, scorpion stings, and animal bites.

3. CLINICAL FEATURES

3.1. Patient History

What?

- What drugs is the patient taking?
- What drugs or chemicals are available to the patient?
- What chemicals or toxins is the patient exposed to at work?
- What (e.g., pill bottles, chemicals containers, drug paraphernalia) was present at the scene?
- What events have occurred since the ingestion or exposure?

How much?

- How much of the drug or chemical did the patient consume/absorb?
- How much of the drug or chemical is remaining in the bottles or container?

When?

- When was the patient last observed at his or her baseline?
- When did the patient ingest or become exposed to the chemical or toxin?

3.2. Physical Examination

Vital signs:

- Neurological findings. Document level of consciousness and any focal neurological abnormalities. The neurological exam should include pupil size and reactivity.

- "Toxidromes" are physical manifestations of toxicology syndromes that may aid in the diagnosis.

Sympathomimetic "toxidrome":

- Hyperthermia.
- Tachycardia.
- Hypertension.
- Dilated pupils.
- Warm, moist skin.
- Altered mental status (e.g., agitation hallucination, combativeness) and seizures.

Anti-cholinergic "toxidrome":

- Hyperthermia ("hot as Hades").
- Tachycardia.
- Hypertension.
- Hot, flushed, dry skin ("red as Hades").
- Dilated pupils ("blind as a bat").
- Dry mucous membranes ("dry as a bone").
- Diminished bowel sounds.
- Urinary retention.
- Altered mental status (e.g., agitation, hallucination) and seizures ("mad as a hatter").

Cholinergic toxidrome:

- Profuse salivation.
- Bradycardia or tachycardia.
- Pinpoint pupils.
- Diaphoresis.
- Excessive bronchial secretions and bronchospasm.
- Hyperactive bowel sounds.
- Urinary or fecal incontinence (or both).
- Muscle fasciculation and weakness.
- Altered mental status and seizures.

Narcotic "toxidrome":

- Pinpoint pupils.
- Respiratory depression.
- Altered mental status (e.g., obtundation).

Other findings: Evidence of trauma (e.g. head trauma) or a medical disorder (e.g. hypo/ hyperglycemia, hypothyroidism).

4. MANAGEMENT

4.1. Initial Stabilization

As with any other patient in the emergency department (ED), initial stabilization of the poisoned patient involves assessment and stabilization of the patient's airway, breathing, and circulation (the ABCs).

A. Airway:

Check for the presence of gag reflex, intubation may be warranted in airway compromise. Causes of airway compromise include:

- Posterior displacement of the tongue.
- Oropharyngeal mucosal injury or edema.
- Angioedema.
- Trauma.

B. Breathing:

Asses the adequacy of oxygenation and ventilation with pulse oximetry and serial arterial blood gas (ABG) testing.

C. Circulation:

Assess the heart rate, heart rhythm, blood pressure, and the adequacy of perfusion.

D. Administration of the "Coma Cocktail":

The coma cocktail consists of a group of antidotes that maybe of value, both diagnostically and therapeutically, during the initial assessment and treatment of patients with altered mental status (see figures 18.1 and 18.2).

- Thiamine 100mg intravenously. Aids in the diagnosis for alcoholic patients, as well as, preventing wernicke-korsakoff syndrome.
- Dextrose, given intravenously, can rapidly treat and correct hypoglycemia.
- Naloxone 0.01 mg/kg intravenously, is administered as an initial dose.
- Flumazenil: A benzodiazepine antagonist is capable of reversing benzodiazenpine- induced central nervous system (CNS) depression. The dose is 0.5 mg (to a maximum dose of 5.0 mg) administered intravenous slowly. Flumazenil has a half life of 57 minutes, so reoccurring sedation after administration may occur within 1-2 hours, and repeat doses may be required.

See table 18.1.

E. Gastric Decontamination Procedures:

- Active charcoal, the initial dose of 1g/kg.
- Gastric lavage, in general, has no advantage over activated charcoal in terms of preventing the absorption of toxins.
- Whole bowel irrigation utilizing magnesium sulfate (adult -161g, child 250 mg/ kg).

F. Laboratory and Diagnostic Studies:

- Laboratory studies.
- Ferric chloride test.
- Urinary fluorescence.
- Urinalysis.
- Blood clotting.
- Toxicology screen.
- Quantitative serum drug levels.
- Electrocardiography.
- Radiography.
- Radio-opaque studies.
- Halogenated hydrocarbons.
- Iron-containing preparations.
- Potassium preparations.
- Iodinated compounds.
- Heroin or cocaine packets.

5. MONITORING

Level of consciousness and vital signs should be monitored regularly until complete recovery. After recovery, keep the patient under observation for at least 24 hours.

Snake Bite

1. INTRODUCTION

There are 2 main types of poisonous snakes:

- Crotalids or pit vipers (hemotoxic venom): Causing hemolysis, necrosis, and disseminated intravascular coagulation (e.g. rattlesnakes).
- Elapids (Neurotoxic venom): disrupt neuromuscular activity (e.g. coral snakes, cobras).

2. CLASSIFICATION

Snake bites are classified according to severity and seriousness of envenomation, ranging from no injury, to mild, moderate, and lastly severe.

3. CLINICAL FEATURES

3.1. Assessment

A. Severity and Envenomation:

- None:
  - Puncture with no to minimal pain/tenderness.
- Mild:
  - Local swelling, pain, perioral parasthesia, but no systemic symptoms.
- Moderate:
  - Local symptoms, systemic symptoms, and mild coagulopathy.
- Severe:
  - Severe symptoms of the entire extremity, severe systemic symptoms, and gross coagulopathy.

B. Snake Type:

- Crotalids:
  - Puncture marks (may have 1-4).
  - Local symptoms (pain, swelling, erythema, necrosis, edema, ecchymoses).
  - Systemic symptoms (weakness, nausea, vomiting, numbness, tingling, perioral parasthesia, bruising, tachycardia, shock, death).
- Coral snakes:
  - Multiple, painless, and small wounds.
  - Local symptoms of numbness & fasciculation.
  - Systemic symptoms: slurred speech, weakness, cranial never palsies dysphagia, ptosis, diplopia, diaphoresis, impaired consciousness, respiratory failure, and then death.

3.2. Libratory Investigations

Investigations may be abnormal in crotalid bites (but usually normal with neurotoxic venom).

- Complete blood count (CBC): Hemolysis
- PT/PTT, DIC screen: Coagulation abnormalities.
- Renal function: May reveal acute renal failure.
- Electrolytes and liver function test (LFTs): Maybe abnormal.

4. MANAGEMENT

Do not handle dead snakes as reflex biting may occur.

- If possible, identify the type of snake.
- Immobilize the bitten extremity/area, as if it were a fracture, and hold it below the level of the heart.
- Avoid incision, suction, and tourniquet/ice application.
- Monitor vital sign and perform resuscitation via advanced cardiac life support (ACLS) protocols.
- Establish intravenous (IV) access and withdraw appropriate blood samples for investigation/monitoring.
- Local wound care and tetanus immunization should be given. Limb circumference at several sites above and below the wound should be checked every 30 minutes and the border of advancing edema should be marked. Patients with no evidence of envenomation after 8-12 hours may be discharged.
- Any patient with progressive local swelling, systemic effects, or coagulopathy; should receive immediately antivenom therapy in accordance with the following principles:
  - Perform skin testing as follows:
    - 0.1 ml of antivenom injected subcutaneously, followed by 15 minutes of patient observation. If no reaction occurs, inject 0.25 ml of antivenom subcutaneously and observe the patient for another 15 minutes. Erythema and a wheal reaction constitutes an allergy, patients with a positive skin test can be given less allergenic goat antivenom. However, a negative reaction is no guarantee against anaphylaxis.
  - After the tolerated skin testing, the patient may be treated with antivenom. Dilute 50 ml (5 x 10 ml ampules) antivenom in 250 ml N saline. The antivenom infusion should proceed at a slow rate for the first 10 minutes; if the patient is stable, the infusion can be increased to the rate 250 ml/h.
  - More antivenom should be given if severe envenomation signs persist after 1-2 hours. Doses can be repeated every 4-6 hours until the arrest of progressive symptoms and coagulopathy.
  - Children must be given the same dose as adults.
  - Once initial control has been achieved, the protocol is completed by administering additional two vial doses every six hours over a total of 18 hours (3 more complete doses).

Key Points:

- Polyvalent crotalidae immune fab (CroFab), new sheep-derived antivenom has generally replaced antivenom polyvalent, an equine-derived product.
- Antivenom is the only specific antidote available at present time for the treatment of venomous snakebite.
- In case of anaphylaxis: see management of anaphylaxis.

5. MONITORING

Serum sickness reactions (late reactions) are common after antivenom use. Serum sickness usually occurs 5-10 days after antivenom, and is treated by prednisolone 45-60 mg daily with tapering doses over 1-2 weeks. An antihistamine (e.g. chlorpheniramine malleate), 2 mg every six hours for adults (0.25 mg/kg/day in two divided doses for children) may be added.

Scorpion Sting

1. INTRODUCTION

Androctonus Crassicaude (black scorpion) and Leiurus Quinquestriatus (yellow scorpion) are the two most dangerous and toxic scorpions in the world.

2. CLINICAL FEATURES

2.1 Assessment

The toxins cause local pain, tenderness, swelling, and rarely systemic manifestations like vomiting, dyspnea, hyperthermia, hypertension, convulsions, acidosis, and shock.

2.2. Libratory Investigations

- Complete blood count (CBC): Lecucocytosis
- Biochemistry: Elevated blood glucose, creatine phosphokinase (CPK), lactae deydrogenase (LDH), amylase.
- Electrolytes: Elevated sodium and calcium and decreased potassium.
- Arterial blood gases (ABG): Acidosis
- Electrocardiogram (ECG): Abnormalities

3. MANAGEMENT

- Give scorpion antivenom to all patients confirmed to have scorpion sting, after a skin test. (Perform skin testing by injecting 0.1 ml of antivenom intradermally).
- Administer polyvalent scorpion antivenom by diluting 5 x 1 ml ampules in 20-50 ml ½ (NaCl) to be given IV over 20 minutes. Infants and children (up to 6 years) should have the antivenom diluted in ¼ NaCl, but at the same dose.
- If systemic manifestations continue to exist, repeat drug administration every two hours, for a total of four doses.
- Children must be given the same dose of antivenom as adults.

Contraindicated drugs:

- Barbiturates.
- Morphine.
- Pethedine.
- Beta blockers.

4. MONITORING

- Keep under observation for at least 24 hours after recovery.
- Adjunctive therapy to support vital functions includes:
  - Severe local pain: 0.5 ml (maximum) of 1% xylocaine, infiltrated at the site of sting.
  - Vomiting: Chlorpromazine 0.5-1 mg/kg I.M, may repeat if necessary.

- Convulsion: Diazepam IV slowly.
- Pulmonary edema: Oxygen administration, furosemide, and fluid restriction.
- Hypertension: Hydralazine or Nifedipine.
- Shock : Central venous pressure (CVP) line with 0.5 N saline to keep CVP value at 8-12 cm (H_2O), and maintain blood pressure at a level to perfuse vital organs (systolic BP 60-70 mmHg in children).

Animal Bite

1. INTRODUCTION

Rabies is a fatal disease, due to infection of the human CNS by the rhabdo virus. The virus is transmitted by inoculation with infectious saliva, or by salivary contact with a break in the victim's skin or mucus membranes. Foxes, cats, dogs, horses, camels, and raccoons are more likely to be infected. Rats, mice, rabbits, and guinea pigs rarely transmit the virus to humans.

2. CLINICAL FEATURES

2.1. Assessment

Initial manifestations include fever, headache, sore throat, pain, and parasthesia at the wound site.

CNS symptoms develop 1-2 weeks after the prodrome.

The encephalitic form entails bulbar and peripheral muscle spasms, opisthotonus, agitation, and hydrophobia. Paralytic forms entail symmetric, ascending, and flaccid paralysis.

Coma, apnea, and death usually occur 4-7 days after the onset of CNS symptoms.

2.2. Differential Diagnosis:

Guillain-Barre syndrome, tetanus, meningitis, encephalitis, polio, and brain abscess.

3. MANAGEMENT

- Thorough cleaning, debridement, and repeated flushing of wounds with soap and water.
- Wounds caused by animal bites should not be sutured because this promotes rhabdo virus replication.
- Vaccination of non-immunized patients:
  - Passive immunization:
    - Human rabies immunoglobulin 20 IU/Kg, half of the dose infiltrated locally at the exposure site and the remaining injected IM distally from the wound.
    - If human rabies immunoglobulin is not available, equine rabies antiserum 40 I.U/Kg can be used after skin testing.
  - Active immunization:
    - Human diploid cell vaccine (HDCV), given as 5 injections of 0.5 ml IM in the deltoid muscle (anterolateral region of the thigh muscle in children). Do not inject in the gluteal region. Vaccine is given 0, 3, 7, 14, and 28 days after exposure.
- Vaccination of subjects already immunized:
  - Vaccination administered less than 5 years previously: give 2 injections (days 0 and 3).
  - Vaccination administered over 5 years previously or incomplete, give 5 injections on days 0, 3,4,14, and 28 with administration of immunoglobulin (if required).
  - Prevention or pre-exposure vaccination (HDCV).
  - Primary vaccination: three injections total with injections given on days 0, 7, 21 or 28.
  - Booster injection: one year later and then every five years thereafter.

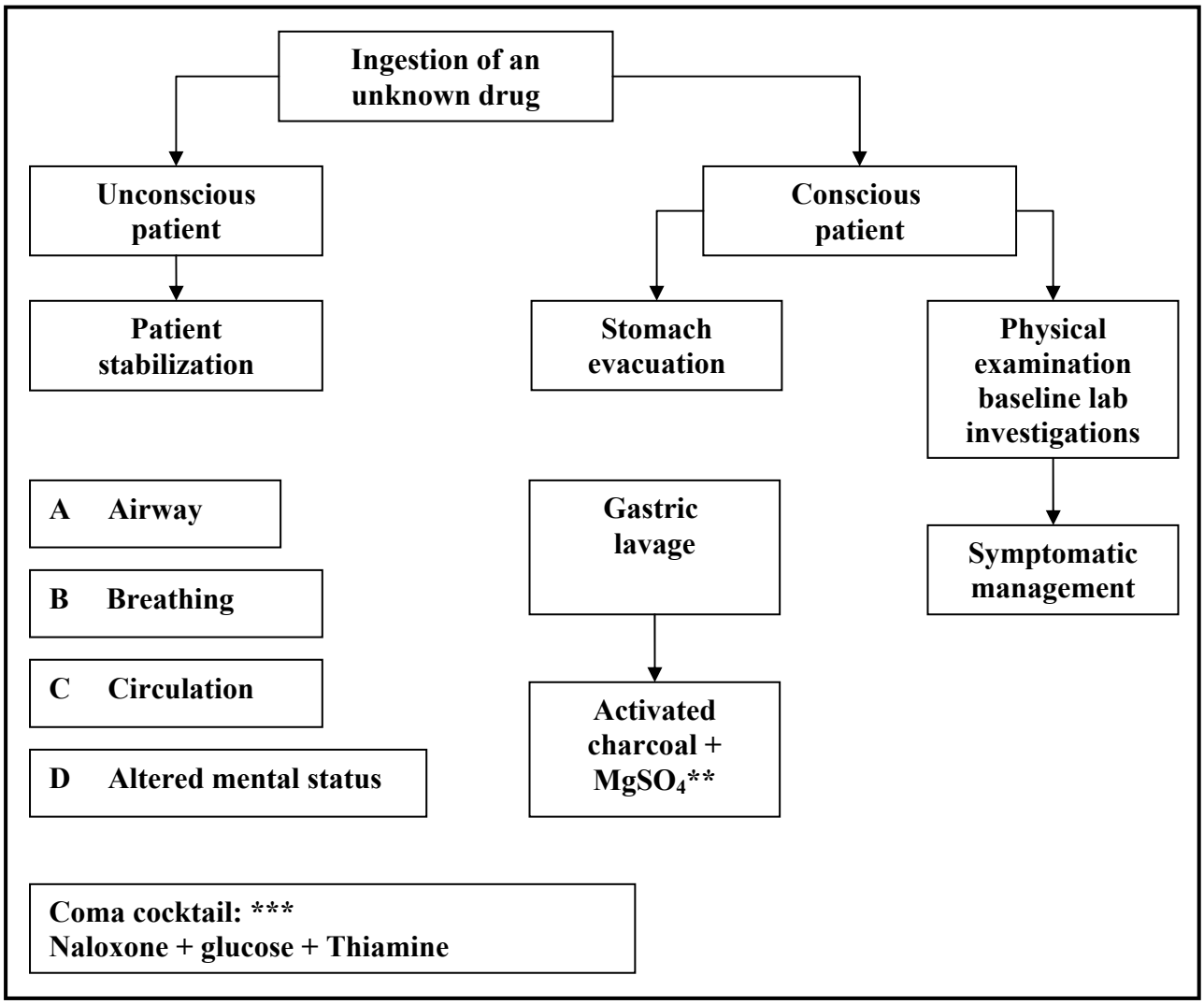
Key Points:

- Schedule of vaccination is the same for adults and for children.
- Rabies immunoglobulin and rabies vaccine should never be given in the same syringe or the same site.

Table 18.1: Antidotes Used in the Emergency Department

Toxic Used For	Antidote	Dose and Comments
Acetaminophen	N- Acetylcystein	140 mg/kg q4h for up to 17 does or 150 mg/kg IV load over 1 hr with 50 mg/kg over 40 hr followed by 100 mg/kg over 16
Arsenic, lead, and mercury	BAL	3-5 mg/kg IM only
Benzodiazepines	Flumazenil	0.2 mg, then 0.3 mg, then 05 mg, up to be used if patient has signs of TCA toxicity, not approved for use in children but probably safe
Beta- blockers	Glucagon	5-10mg in adults, then infusion of same dose per hour
Calcium channel blockers	Calcium	1g calcium chloride IV in adults 20-30 mg /kg dose in children, over a few minutes with continues monitoring. Repeat as needed
Cyanide	Hydroxyocobalamin	5 mg in 100 ml of NS over 15 min. Repeat as needed.
Digitals glycoside	Digoxin-specific Fab	10-20vials if patient in ventricular fibrillation; otherwise dose fragments based on serum digoxin concentration or a mount ingested
Iron	Deferoamine	15 mg/kg IV; higher dose reported to be safe
Isoniazid, hydrazine, and monomethylydrazine	Pyridoxine	5 g in adults, 1 g in children, if ingested dose unknown; antidote may cause neuropathy in very large dose.
Opioids	Nalmefene	2 mg; much longer half-half than naloxone
Organophosphates and carbamates	Atropine	Test dose, 1-2 mg IV adults, 0.03 mg/kg in children; titrate to drying of pulmonary secretions
Sulfonureas	Octreotide	50 mg SC q 12h, 5-10 mg/kg/24 hr IV
Tricyclic antidepressants	Bicarbonate	44-88 mEq in adults, 1-2 mEq/kg in children; best used by IV push and not by slow infusion
Valproic	Carnitine	100mg/kg IV or PO loading dose with 23 mg/kg q6h

Figure 18.1: Unknown Poison Ingestion Management



**** Activated charcoal**

- Adults: 60-100 mg
- Child: 1-2 mg/kg

$MgSo_4$:

- Adult: 16 mg
- Child: 250 mg/kg

***** Coma cocktail:**

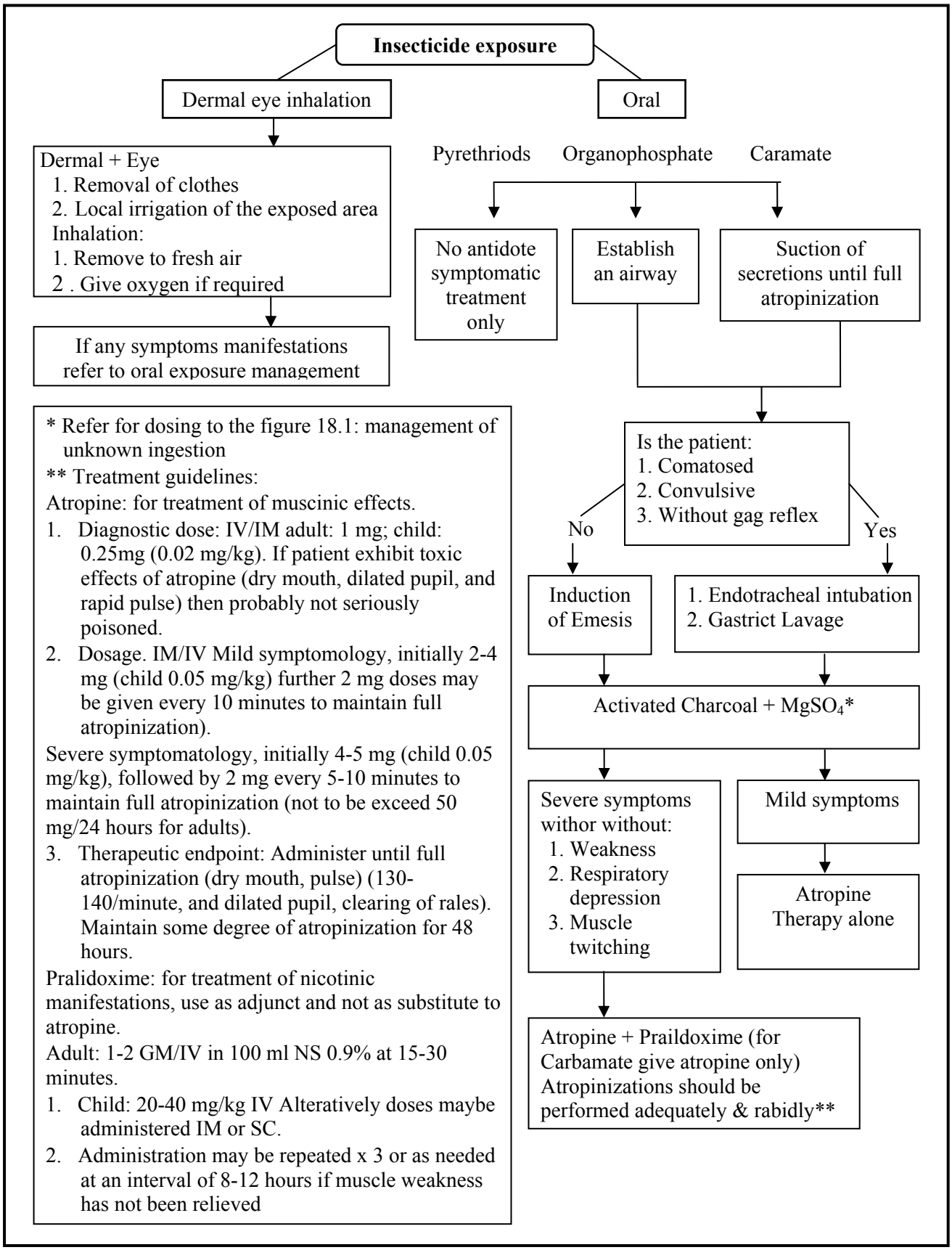
- Naloxone: opioid antagonist
  - Adult/Child: 0.2-2 mg IV; repeat at 2-3 minutes intervals until desired effect or a total dose of 10-15 mg.
  - Careful in opioids-dependent patient.
- Flumazenil: Benzodiazepine antagonist.
  - Adult: 0.2 mg IV over 30 seconds, then 0.3 mg, then 0.5 mg (every 30 seconds) up to 3 mg.
  - Child: 0.01 mg/kg, titrate up to 1 mg
  - Careful use in case of mixed ingestion, may precipitate seizures or arrhythmias

Glucose:

- Adults: 50-100 ml of 50%
- Child: 2-4 ml/kg of 25%

Thiamine 100 mg is indicated concomitantly for alcoholic or malnourished patients.

Figure 18.2: Management of Insecticide Exposure



Part 4: Obstetric Emergencies

Chapter 19: Obstetric Hemorrhage

GUIDELINE OBJECTIVES:

- Understand the mechanisms and causes of obstetric hemorrhage.
- Perform a physical examination for cases of obstetric hemorrhage.
- Order and interpret the investigations required.
- Diagnose obstetric hemorrhage.
- Provide first aid management and referral for obstetric hemorrhage.

1. INTRODUCTION

According to the JNMMS 1995/1996, obstetric hemorrhage is a major cause of maternal mortality; accounting for 22% of Jordanian maternal deaths. Hemorrhage related avoidable factors include substandard care and service provider harmful practices.

2. CLINICAL FEATURES

2.1. Causes of Obstetric Hemorrhage

A. Abortion:

The termination of pregnancy by any means before the fetus is sufficiently developed to survive (less than 24 weeks gestation based on the date of last menstrual period).

- Types of abortion:
  - Threatened abortion: Mild uterine bleeding occurring with or without uterine colic in the presence of a living fetus and closed cervix.
  - Inevitable abortion: Uterine bleeding occurring with continuous and progressive dilation of the cervix, without expulsion of the products of conception. Rupture of membranes before 24 weeks is considered an inevitable abortion.
  - Incomplete abortion: Expulsion of some but not all products of conception.
  - Complete abortion: Expulsion of all products of conception.
  - Missed abortion: Abortion in which the embryo or the fetus dies but is retained in utero.
  - Septic abortion: Any type of abortion with superadded infection where microorganisms and their products can be disseminated into the maternal systemic circulation.
  - Habitual abortion: Occurrence of three or more consecutive abortions.

B. Ectopic Pregnancy:

Pregnancy that occurs outside the normal uterine cavity (tubal, ovarian, abdominal, or cervical).

C. Gestational Trophoblastic Diseases (GTD):

Mainly vesicular mole and choriocarcinoma.

D. Antepartum Hemorrhage:

Antepartum hemorrhage is bleeding from or within the genital tract after 24 weeks of gestation.

- Placenta previa: Is the implantation of the placenta in the lower uterine segment with different grades of encroachment on the cervix. There is usually no pain and the uterus is soft. Bleeding is usually causeless, but may be precipitated by sexual intercourse. The principal danger in placenta previa is heavy blood loss when labor begins or during uterine contractions prior to the initiation of true labor.
- Abruptio placentae: Is the premature separation of a normally implanted placenta that may be precipitated by a sudden increase in blood pressure or trauma. The uterus may be painful and tense (as if in a tetanic contraction). Fetal parts are difficult to feel and fetal heart sounds may be absent. There are often signs of hypovolemia (tachycardia, hypotension, nausea, vomiting, oliguria and shock) in the absence of visible external hemorrhage. Coagulopathies occur in 30% of cases in which the abruption is severe enough to kill the fetus.

E. Postpartum Hemorrhage:

Postpartum hemorrhage is excessive blood loss after delivery of the fetus, sufficient to affect the general condition of the mother, demonstrated by tachycardia and/or hypotension. Postpartum hemorrhage can be caused by:

- Uterine atony: Poor or ineffective uterine contractions and retraction.
- Genital tract trauma (e.g., vaginal and cervical tears or uterine rupture).
- Third stage complications: acute inversion of the uterus, abnormal or incomplete placental separation.
- Coagulation disorders.

F. Local Causes:

Obstetric hemorrhage from a disease process or external source (e.g. polyps, cervicitis, foreign body).

2.2. History

Patient history highlights the causes of and incidents to obstetric hemorrhage.

- Obstetric history: Previous ectopic pregnancy, previous abortions, previous operative deliveries, previous postpartum hemorrhage.
- Past history: Tubal surgery, pelvic/abdominal surgery, history of infertility, use of drugs for ovulation induction and/or assisted reproductive techniques, and previous blood transfusion.
- Contraceptive history: Last contraceptive method used, its duration and cause of discontinuation.
- History of the present pregnancy:
  - Vaginal bleeding and its amount.
  - Lower abdominal pain, colic, or menstrual-like cramps.
  - Fainting episodes or dizziness.
  - Exaggerated symptoms of normal pregnancy e.g., nausea or vomiting.
  - Cessation or absent fetal movements.

2.3. Physical Examination

A. General Examination:

Signs of shock such as pallor, sweating, cold and clammy skin, hypotension, tachycardia, oliguria, and fainting may be seen in severe hemorrhage.

B. Abdominal Examination:

- Tenderness or rigidity.
- The size of the uterus.
- Any palpable masses or rebound tenderness.

C. Local Examination:

- Before 24 weeks gestation: To diagnose different types of abortion as well as ectopic pregnancy (tender cervix with or without a palpable adnexal mass).
- *After 24 weeks gestation: Never perform vaginal examination.*
- After delivery: To diagnose atonic and traumatic post partum hemorrhage.

D. Perform A Gentle Bimanual Examination Documenting When Suspecting Ectopic Pregnancy:

- Open or closed cervix.
- Size and consistency of the uterus.
- Cervical motion tenderness.
- Adnexal masses and/or tenderness.

Alarming symptoms and signs of severe hypovolemia:

- History of severe blood loss with sweating, fainting, and pallor.
- Tachycardia, tachypnea, hypotension and hypothermia.
- Reduction of the urine output (less than 30 ml/hour).

2.4. Harmful Effects of Blood Loss (hypovolemia)

- Mild to moderate loss is associated with anemia.
- Severe loss is associated with hypovolemic shock:
  - Reversible stage (reversible organ ischemia).
  - Irreversible stage (irreversible organ failure).

2.5. Investigations

A. Pelvic Ultrasound:

- To save time, emergency health care providers may arrange for ultrasound examination while preparing the woman for transfer/referral.
- In case of hypovolemia, emergency health care providers should not wait for an ultrasound, or deduct valuable time from resuscitation and referral (to obstetric department).

B. Laboratory Investigations:

- Complete blood count (CBC), (ABO) and (Rh) typing.
- Urine for albumin and glucose.
- Other specific relevant investigations.

3. MANAGEMENT

3.1. First Aid Management

First aid management is performed to save the life of the patient. Once stable, the patient can be referred to the obstetric department.

- Resuscitation:
  - Insert two wide bore cannulae.
  - Take 10 cc blood for CBC, cross-matching, coagulation profile and kidney function tests.
  - Replace the loss by fluids (crystalloids and/or colloids) until blood becomes available.
  - Insert a Foley's catheter.

3.2. Referral

After providing first aid management and resuscitation, women with obstetric hemorrhage should be referred to the obstetric department.

- Cases with severe bleeding before 24 weeks:
  - Perform resuscitation.
  - Ask for the obstetrician's help.
  - Give ecbolics (oxytocin, ergometrine).
  - Refer to the operating theatre (obstetric department).
- Cases with mild to moderate bleeding before 24 weeks:
  - Fix a cannula.
  - Take a blood sample for CBC and cross-matching.
  - Ask for an ultrasound.
  - Refer to the ward to be reviewed by a specialist (obstetric department).
- Cases with severe bleeding after 24 weeks:
  - Perform resuscitation.
  - Ask for the obstetrician's help.
  - Refer to the operating theatre (obstetric department).
- Cases with mild to moderate bleeding after 24 weeks:
  - Fix a cannula.
  - Take a blood sample for CBC and cross-matching.
  - Refer to the ward to be reviewed by a specialist (obstetric department).

- Cases with severe bleeding after delivery:
  - Perform resuscitation.
  - Ask for the obstetrician's help.
  - Give ecbolics.
  - Refer to the (obstetric department).
- Cases with mild to moderate bleeding after delivery:
  - Fix a cannula.
  - Take a blood sample for CBC and cross-matching.
  - Refer to the ward to be reviewed by a specialist (obstetric department).

Chapter 20: Pregnancy Induced Hypertension (PIH)

GUIDELINE OBJECTIVES:

- Understand and diagnose pregnancy induced hypertension (PIH).
- List PIH risk factors and common PIH-related complaints.
- Categorize pre-eclampsia severity using established criteria.
- Order and interpret relavent laboratory investigations.
- Perform physical examination for cases of (PIH).
- Provide PIH first aid management and referral.

1. INTRODUCTION

The (*JNMMS) 1995/1996)* revealed that hypertensive disease during pregnancy is a major direct cause of maternal mortality in Jordan. The most important avoidable factors were:

- Delay in referral (90%).
- Substandard care by obstetricians (80%).
- Delay in seeking care (40%).
- Transportation problems (28.6%).

Deaths attributed to pregnancy-related hypertensive diseases demonstrated irregular or late antenatal care (40% of cases).

2. DEFINITION

It is a hypertension which usually appears after 20 weeks gestation and is characterized by:

- Hypertension (≥ 140/90 mmHg).
- Proteinuria (≥ +).
- Edema.

3. CLINICAL FEATURES

3.1. Risk Factors

- Age: < 20 & > 35 years
- Parity: primigravidas have approximately double the incidence of the disease.
- Socioeconomic status: pre-eclampsia and eclampsia are more common in lower socio-economic groups.
- Genetic predisposition: evidence of genetic inheritance is most likely due to a recessive trait
- History of essential hypertension.
- Family history of hypertension.
- Previous antihypertensive medical treatment.
- Previous renal impairment.
- Systemic diseases (diabetes, SLE).

3.2. History Taking

Emergency health care providers should follow the history taking and keep in mind the following important data:

A. Complaints and History of Present Pregnancy:

- Headache: Frontal headache is more significant to pre-eclampsia, however all types of headaches should be given full attention.
- Gastrointestinal manifestations: nausea, vomiting, epigastric / or right hypochondria pain.
- Visual symptoms: blurring of vision, scotomata, diplopia, and flashes of light, usually in severe cases.

Document and report to the obstetrician indicators of severity of pre-eclampsia:

- Presence of symptoms, specifically headache, visual disturbances and/or upper abdominal pain (epigastric or right hypochondrial).
- Diastolic blood pressure (BP) ≥ 110 mmHg.
- Proteinuria of 2+ or more by urine dipstick or a total protein level of ≥5 gm/liter in a 24-hour urine sampling.
- Eclamptic convulsion.

3.3. Physical Exam

Emergency health care providers should perform a general and a abdominal examination, note the following important data:

A. General Examination:

- Hypertension: Defined as a sustained (for at least 6 hours) BP reading of ≥ 140/90 mmHg, or a single measurement of diastolic BP≥110 mmHg. The blood pressure reading of a pregnant woman is sensitive to body position, the sitting position is recommended for blood pressure measurement.
- Edema: Although neither sensitive nor specific, edema is observed in a large percentage of women with pre-eclampsia.

B. Abdominal Examination:

Looking for blood pressure related complication signs:

- Signs of complications, e.g. (intrauterine fetal growth retardation (IUGR), and accidental hemorrhage.
- Associated clinical conditions, e.g. multiple pregnancy, and polyhydramnios.

3.4. Harmful Effects of Pregnancy Induced Hypertension

Pre-eclampsia is associated with generalized tissue and organ ischemia. It might end with:

- Organ failure.
- Hemorrhage.

- Disseminated intravascular coagulation (DIC).
- Eclampsia.

3.5. Investigations

A. Laboratory Investigations:

- Urine analysis for proteinuria.
- Complete blood count.
- Serum glulamic oxaloacetic thrausaminase , Serum glutamate pyruvate
- Kidney function tests: creatinine clearance, serum creatinine, uric acid, total protein in urine, and blood urea: all may be impaired in severe cases.

B. Ultrasound:

Help to diagnose complications as IUGR, oligohydramnios, and abruptio placentae.

4. MANAGEMENT

4.1. First Aid Management

First aid is utilized to save the life of the patient, and facilitate a timely transferred to the obstetric department. Pre-eclampsia and eclampsia can be fatal, strict adherence to management guidelines is essential to reduce maternal and neonatal morbidity/mortality from these diseases.

- Mild pre-eclampsia:
  - Cases with mild pre-eclampsia should be referred to obstetric department to continue the antenatal care under strict follow up from the obstetric team.
- Severe pre-eclampsia/eclampsia:
  - Cases with severe pre-eclampsia and eclampsia are considered emergencies and should be referred *immediately* to obstetric department for active management and termination of pregnancy.

Chapter 21: Puerperal Sepsis

GUIDELINE OBJECTIVES:

- Understand the magnitude of the problem.
- Define puerperal sepsis.
- List risk factors of puerperal sepsis.
- Perform physical examination for cases of puerperal sepsis.
- Order and interpret required investigations.
- Diagnose puerperal sepsis.
- Provide first aid management for puerperal sepsis.

1. INTRODUCTION

The *JNMMS 1995/1996* reported that sepsis was associated with 5 maternal deaths, contributing to 13.9% of direct obstetric deaths and 9.8% of all maternal deaths. The MMR for sepsis was 4/100,000. The most important avoidable factors were:

- Substandard care by obstetricians in the form of inappropriate intrapartum and postpartum management. (80 %)
- Delay in seeking care (60%)
- Delay in referral (40%)

Among the deaths due to sepsis, lack of or poor quality antenatal care was an avoidable factor in 60% of cases.

2. DEFINITION

Puerperal sepsis is defined as a bacterial infection of the genital tract following delivery or abortion. In addition to fever, there may be pelvic pain and tenderness, an abnormal malodorous vaginal discharge and delayed involution of the uterus.

3. CLINICAL FEATURES

3.1. Risk factors

- Management of labor or delivery under unhygienic conditions.
- Multiple vaginal examinations.
- Prolonged rupture of membranes.
- Operative delivery.
- Anemia, uremia, and uncontrolled hyperglycemia.
- Manual removal of the placenta.
- Immunosuppressant and/or immunocompromising drug treatment.
- Vaginal tears.
- Genital infection prior to the onset of labor.
- Thrombophlebitis.

3.2. History Taking

- Emergency health care providers should obtain and full history, including health state before, during, and after pregnancy, specifically highlighting delivery and immediate post-delivery care.
- History of risk factors.
- History of present illness:
  - Discharge: Foul odor vaginal discharge following delivery or abortion.
  - Abdominal pain: This may be severe in complicated cases.
  - Fever and systemic manifestations of sepsis.

3.3. Physical Exam

Emergency health care providers should perform a general and abdominal examination, evaluating for the following important data:

A. General Examination:

Fever: Fever is defined as an oral temperature of > 38° C measured on two occasions, excluding the first 24 hours postpartum, or a temperature greater than or equal to 38.5° C anytime.

B. Abdominal Examination:

- Assess the size of the uterus.
- Presence of any tenderness (both abdominal and bimanual examination).
- Evaluate for signs of peritonitis, e.g. generalized tenderness and rigidity.
- Look for signs of peritonitis, e.g. generalized tenderness and rigidity.

C. Pelvic Examination:

- Inspect external genitalia and perineum to detect any tears or episiotomy.
- Investigate the amount, smell and color of discharge.

3.4. Harmful Effects of Puerperal Sepsis

Puerperal sepsis is a serious illness which might end with:

- Organ failure.
- Septicemia and septic shock.
- Disseminated intravascular coagulation (DIC).

3.5. Laboratory Investigations

- Complete blood count (CBC) with a differential leucocytic count.
- Urinalysis (plus culture if white blood cells or bacteria are seen in the urinalysis).
- Blood cultures for more severely ill patients.
- Uterine and cervical cultures for exogenous pathogens.
- Obtain other laboratory examinations and diagnostic tests as indicated by clinical examination.
- Ultrasound: To diagnose pelvic collections or retained product of conception.

4. MANAGEMENT

4.1. First Aid Management

- First aid management for women with puerperal sepsis includes an immediate administration of an antibiotic combination to cover both gram positive and negative aerobes and anaerobes. Start with the most relevant broad-spectrum antibiotics according to the current, locally available list of effective antibiotics. Recommended antibiotics initial treatment includes (prior to referral):
  - Ampicilin 2 gm intravenous (IV).
  - Gentamicin 1.5 mg/kg IV.
  - Metronidazole500 mg IV.
- In severely septic cases:
  - Ensure patent airways.
  - Insert wide bore IV cannulae and start infusion of crystalloids.
  - Provide oxygen via mask.
  - Insert Foley's catheter.
  - Monitor urine output while referring the case to the hospital.

4.2. Referral

After providing first aid management and resuscitation, women with severe sepsis should be referred to a higher level acuity health acre facility.

Evidence Based Medicine for Emergency Health Care

Chapter 22: Evidence Based Medicine

INTRODUCTION

Until recently, medical decision making was largely driven by anecdote, expert opinions, intuition, and a limited understanding of pathophysiology. Although, the scientific gaps remain wide, the recent explosion of high quality medical research and clinical trials justifies an evidence based approach. This allows the physician to reliably identify the best care available for each given patient when selecting diagnostic tests, choosing treatment strategies, and determining prognosis.

1. CARDIOVASCULAR

Recommendations outlined in the American College of Cardiology (ACC) and the American Heart Association (AHA).

1.2. Author's Conclusions

A. Classification:

- Class I: Evidence/general agreement that the procedure or treatment is useful and effective
- Class II: Conflicting evidence/divergence of opinion regarding usefulness and efficacy of the procedure or treatment:
  - Class IIa: Weight of evidence in favor of procedure or treatment.
  - Class IIb: Usefulness/efficacy not well established.
- Class III: Evidence/general agreement that the procedure or treatment is not useful or effective and may be harmful.

B. level of Evidence:

- Level A (highest): The data were derived from multiple randomized clinical trials that involved large numbers of patients.
- Level B (intermediate): The data were derived from a limited number of randomized trials that involved small numbers of patients or, from careful analysis of nonrandomized studies or, observational registries.
- Level C (low): A lower rank was given when expert consensus was the primary basis for the recommendation.

C. Adult Basic Life Support (BLS) Sequence:

- Airway:
  - A healthcare provider should use the head tilt– chin lift maneuver to open the airway in a victim without evidence of head or neck trauma.
  - If a healthcare provider suspects a cervical spine injury, open the airway using a jaw thrust without head extension (Class IIb).
  - Maintaining a patent airway and providing adequate ventilation is a priority in cardiopulmonary resuscitation (CPR) (Class I), use a head tilt– chin lift maneuver if the jaw thrust does not open the airway.

- Use manual spinal motion restriction rather than immobilization devices for victims with suspected spinal injury (Class IIb). Manual spinal motion restriction is safer, and immobilization devices may interfere with a patent airway. Cervical collars may complicate airway management during CPR and they can cause increased intracranial pressure in a victim with a head injury (Class IIb). Spine immobilization devices, however, are necessary during transport.

- Breathing:
  - Occasional gasps are not effective breaths. Treat the victim who has occasional gasps as if he or she is not breathing (Class I) and give rescue breaths.
  - Cardiopulmonary resuscitation CPR training should emphasize how to recognize occasional gasps and should instruct rescuers to give rescue breaths and proceed with the steps of CPR (Class IIa).
  - Deliver each rescue breath over one second (Class IIa).
  - Give a sufficient tidal volume (by mouth-to-mouth/mask or bag mask with or without supplementary oxygen) to produce visible chest rise (Class IIa).
  - Avoid rapid or forceful breaths.
  - When an advanced airway (i.e., endotracheal tube, combitube, or laryngeal mask (LMA) is in place during 2-person CPR, ventilate at a rate of 8 to 10 breaths per minute without attempting to synchronize breaths between compressions.
  - There should be no pause in chest compressions for delivery of ventilations (Class IIa).
  - When giving rescue breaths, give sufficient volume to cause visible chest rise (Class IIa).
- Chest Compressions:
  - "Effective" chest compressions are essential for providing blood flow during CPR (Class I). To give "effective" chest compressions, "push hard and push fast." Compress the adult chest at a rate of about 100 compressions per minute (Class IIa), with a compression depth of 1.5 to 2 inches (approximately 4 to 5 cm). Allow the chest to recoil completely after each compression, and allow approximately equal compression and relaxation times.
  - Minimize interruptions in chest compressions.
  - Compression-ventilation ratio:
    - Adults: A compression-ventilation ratio of 30:2 is recommended (Class IIa).
    - In infants and children: 2 rescuers should use a ratio of 15:2 (Class IIb).
- Defibrillation:
  - Immediate defibrillation is the treatment of choice for short duration ventricular fibrillation (VF), such as witnessed (SCA) (Class I).
- Drugs used in advanced cardiac life support (ACLS):
  - Epinephrine:

    It is appropriate to administer a 1-mg dose of epinephrine IV/IO every 3 to 5 minutes during adult cardiac arrest (Class IIb).

- Vasopressin:

  Vasopressin 40 U intravenous/ intrasseous (IV/IO) may replace either the first or second dose of epinephrine in the treatment of pulseless arrest (Level of Evidence: B).
- Atropine:

  The recommended dose of atropine for cardiac arrest is 1 mg IV, which can be repeated every 3 to 5 minutes (maximum total of 3 doses or 3 mg) if asystole persists (Level of Evidence: B).
- Amiodarone (Class IIb):

  Rapid infusion of 300 mg in 20-30 ml NS IV push (cardiac arrest dose). If (VF) / pulseless ventricular tachycardia (VT) recurs, rapidly administer 150 mg IV by, followed by 1 mg/min for 6 hours and then 0.5 mg/min (maximum daily dose of 2 g).
- Lignocaine (Level of Evidence: B)

  Initial bolus of 1.0-1.5 mg/kg. Additional bolus of 0.5-0.75 mg/kg with a maximum daily total of 3 mg/kg. Maintenance infusion of 1-4 mg/min.
- Magnesium sulphate

  Infuse 1-2 g diluted in 100 ml D5 over 1-2 minutes. Class IIb in torsades de pointes or suspected hypomagnesaemia or severe refractory VF.

D. Unstable Angina (UA) or Non–(ST)-Segment Elevation (MI):

- The use of antiplatelet and antithrombotic therapy for the treatment of unstable angina UAor non–(ST)-segment elevation myocardial infarction MI (non-Q–wave MI. Current (Class I) recommendations include the following:
  - Antiplatelet therapy should be initiated promptly. Aspirin (ASA) should be administered as soon as possible after presentation and continued indefinitely (level of evidence: A).
  - Clopidogrel should be administered to hospitalized patients who are unable to take (ASA) due to hypersensitivity or major GI intolerance (Level of Evidence: A).
  - In hospitalized patients for whom an early non-interventional approach is planned, clopidogrel should be added to (ASA) as soon as possible on admission. Clopidogrel should be administered for at least 1 month (level of evidence: A) and continued up to 9 months (Level of Evidence: B).
  - In patients for whom percutaneous coronary intervention (PCI) is planned, clopidogrel should be started and continued for at least 1 month (level of evidence: A) and up to 9 months in patients who are not at high risk for bleeding (Level of Evidence: B).
- Additional Class I recommendations include the following:
  - In patients taking clopidogrel in whom elective coronary artery bypass graft (CABG) is planned, the drug should be withheld for 5 to 7 days (Level of Evidence: B).
  - Anticoagulation with subcutaneous low–molecular-weight heparin (LMWH) or intravenous unfractionated heparin (UFH) should be added to antiplatelet therapy with aspirin (ASA) and/or clopidogrel (Level of Evidence: A).
  - A platelet (GP) IIb/IIIa antagonist should be administered, in addition to (ASA) (and/or clopidogrel) and heparin, to patients in whom catheterization and percutaneous coronary intervention PCI are planned. The (GP) IIb/IIIa antagonist may also be administered just prior to PCI (Level of Evidence: A).

F. Anaphylactic Shock:

The current grades of evidence for the treatment of anaphylaxis and anaphylactic shock are insufficient:

- Antihistamines—Level of Evidence: C. However, there is Level A evidence that the combination of (H1) and (H2) antihistamines is more effective in the resolution of allergic cutaneous syndromes than H1 antagonists alone.
- Epinephrine—Level of Evidence: C.
- Corticoids—Level of Evidence: D.
- Fluids—Level of Evidence: D.

2. TRAUMA

2.1. Antifibrinolytic Drugs For Acute Traumatic Injury

A. Authors:

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B. Authors' Conclusions:

- Implications for practice:

  To date, there is no evidence to support the use of antifibrinolytic agents in trauma.
- Implications for research:

  Further randomized controlled trials of antifibrinolytic agents in trauma are required.

2.2. Aminosteroids For Acute Traumatic Brain Injury

A. Authors:

- Ian G Roberts1.
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B. Authors' Conclusions:

- Implications for practice:

  There is no evidence to support the routine use of aminosteroids in the management of traumatic head injury.
- Implications for research:

  On the basis of existing evidence from randomised trials of aminosteroids in head injury, it is not possible to refute the possibility of moderate but potentially clinically important benefits or harms. A further randomized controlled trial of tirilazad mesylate with 1156 participants has been completed, the results of which should become available in the near future.

2.2. Emergency Intubation for Acutely Ill and Injured Patients

A. Authors:

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B. Authors' Conclusions:

- Implications for practice:

  Clinicians need to establish a safe airway and adequate ventilation for patients in emergency situations bearing in mind that the efficacy of emergency endotracheal intubation, as currently practiced, has not been rigorously studied. The skill of the operator may be a key determinant of efficacy in all patient groups. Success rates are not reported in most studies and in paramedic studies quoted rates are often less than desirable (< 95% after three attempts), which may reflect skill retention and operating conditions. In non-traumatic cardiac arrest it is unlikely that intubation carries the same life saving benefit as early defibrillation and bystander CPR. In pediatric and trauma patients the current evidence base provides no imperative to extend the practice of pre-hospital intubation in urban and short transit time systems.
- Implications for research:

  Given the review of findings and the large investment in paramedic intubation training, it would seem ethical to initiate a large, high quality randomized trial comparing endotracheal intubation (ETI) efficacy (involving competent practitioners) to basic maneuvers bag-valve-mask (BVM) in urban out-of-hospital adult cardiac arrest. The findings of this trial would then determine the nature of future studies in trauma patients and other groups.

2.3. Hyperventilation Therapy for Acute Traumatic Brain Injury

A. Authors:

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B. Authors' Conclusions:

- Implications for practice:

  The data available are insufficient to assess any potential benefit or harm that might result from hyperventilation in severe head injury.
- Implications for research:

  Further randomized controlled trials to asses the effectiveness of hyperventilation therapy following severe head injury are needed. The ($PaCO_2$) that should be targeted in hyperventilation, the timing, duration, and whether or not the intervention has a net benefit for certain groups of patients, such as those with raised Intracranial pressure (ICP), needs to be investigated.

2.4. Colloid Solutions For Fluid Resuscitation

A. Authors:

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B. Background:

Colloids are widely used for fluid volume replacement, however doubts remain as to which colloid is best. Colloids vary in their molecular weight and therefore in the length of time they remain in the circulatory system. Because of this and other characteristics, they may differ in their safety and efficacy.

C. Objectives:

To compare the effects of different colloid solutions in patients thought to need volume replacement.

D. Selection Criteria:

Randomized and quasi-randomized trials comparing colloid solutions in critically ill and surgical patients thought to need volume replacement were compared. The outcomes measured were death, amount of whole blood transfused, and incidence of adverse reactions.

E. Authors' Conclusions:

From this review, there is no evidence that one colloid solution is more effective or safe than any other, although the confidence intervals are wide and does not exclude clinically significant differences between colloids. Larger trials of fluid therapy are needed if clinically significant differences in mortality are to be detected or excluded.

- Implications for practice:

  Previous reviews have failed to show any benefit of colloids over crystalloids for volume replacement. This review does not provide any evidence that one colloid is safer than another, but does not rule out clinically significant differences.
- Implications for research:

  Trials of fluid therapy need to be larger in order to exclude clinically significant differences between colloids in patient relevant outcomes. However, trials should probably first address the question of whether colloids are any more effective than crystalloid solutions. Use of surrogate outcomes, such as physiological measurements should be discouraged.

2.5. Hypertonic Versus Near Isotonic Crystalloid for Fluid Resuscitation in Critically Ill Patients

A. Authors:

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3University of Cambridge School of Clinical Medicine, Department of Paediatrics, Cambridge, UK

B. Authors' Conclusions:

- Implications for practice

  This review does not provide any evidence that hypertonic crystalloid is better than isotonic crystalloid, but it does not rule-out clinically important differences.

- Implications for research:

  Further trials are needed comparing hypertonic to isotonic crystalloid. These trials should be multi-centre prospective randomized controlled trials, which are large enough to detect a clinically important difference. Clinically relevant outcomes such as mortality should be used and trials should specify the type and amount of fluid used.

2.6. Mannitol for Acute Traumatic Brain Injury

A. Authors:

Abel Wakai1, Ian G Roberts2, Gillian Schierhout3

1Division of Emergency Medicine, Sunnybrook Health Sciences Centre, Toronto, Canada. 2Cochrane Injuries Group, London School of Hygiene & Tropical Medicine, London, UK. 3c/o Cochrane Injuries Group, London School of Hygiene & Tropical Medicine, London, UK

B. Background:

Mannitol is sometimes effective in reversing acute brain swelling, but its effectiveness in the ongoing management of severe head injury remains unclear. There is evidence that, in prolonged dosage, mannitol may pass from the blood into the brain, where it might cause increased intracranial pressure.

C. Objectives:

To assess the effects of different mannitol therapy regimens of mannitol compared to other intracranial pressure (ICP) lowering agents, and to quantify the effectiveness of mannitol administration given at other stages following acute traumatic brain injury.

D. Authors' Conclusions:

- Implications for practice:

  There is insufficient reliable evidence to make recommendations on the use of mannitol in the management of patients with traumatic brain injury.

- Implications for research:

  There are many unanswered questions regarding the optimal use of mannitol following acute traumatic head injury. The widespread current use of mannitol, and lack of clarity regarding optimal administration, presents an ideal opportunity for the conduct of randomized controlled trials.

3. MULTIPLE TRAUMA, CRITICAL CARE PATIENTS

A. Authors:

Jeffery C Metzger , Alexander L Eastman and Paul E Pepe Year in review 2008: Critical Care – trauma, 21 October 2009, 2009 BioMed Central Ltd.

B. Author's Conclusions:

- Traumatic brain injury:

  One of the challenges in treating patients with (TBI) is the identification of increased ICP. Increased ICP is known to affect outcomes, but often requires invasive catheters to determine its presence and monitor its course. One non-invasive modality that has been examined is the use of ultrasound to detect changes in the optic nerve sheath diameter (ONSD). Patients with (TBI) and an elevated ICP that is refractory to all other medical and surgical treatment modalities may very well benefit from high-dose barbiturate therapy, although this practice remains controversial in some centers given its associated high morbidity.

  Current practice management guidelines dictate the need for airway protection in patients with (TBI) and a Glasgow coma scale (GCS) score of 8 or less for respiratory protection and prevention of aspiration into the lungs. It has been suggested, however, that prolonged endotracheal intubation leads to increased risk of pneumonia, as well as, longer intensive care unit (ICU) and overall hospital stays. Research shows that none of the patients in the early extubation arm developed nosocomial pneumonia compared with 37% in the delayed extubation arm. Prevention of secondary injury is perhaps the most important goal in the critical care of those with (TBI).

  The recent critical care literature has demonstrated significant debate concerning the optimal range and method of glycemic control in many critical illnesses. The lower glucose range was associated with a trend toward increased mortality during the first two weeks.

- Burns:

  Two papers last year in Critical Care focused on burn care. Acute renal failure is a relatively uncommon occurrence in burn patients, but it carries a high mortality. As in the case of renal failure, inhalational injury is a harbinger of increased mortality in burn patients.

- Diagnostic issues:

  A basic tenet of current trauma care practice is that the cervical spine remains vulnerable to further injury in patients who have received significant blunt force trauma to neck and who may have underlying unstable cervical spine injuries. This rate of cervical spine injury may be as high as 34% in the subpopulation of patients with severe blunt trauma who require (ICU) admission. Although there are good data and resulting established criteria for ruling out a significant cervical spine injury in patients with normal mentation, there is still some debate with regard to the removal of cervical spine immobilization in those who remain comatose. Many sources recommend a CT scan of the cervical spine, but others have recommended that, in patients with a CT that indicates no cervical injury, an (MRI) may be necessary to detect injuries that may be missed by standard CT.

  Early post-traumatic mortality is determined by the initial traumatic impact and early resuscitation, whereas late mortality is usually associated with the development of septic inflammatory response syndrome and progresses to multiple organ dysfunction syndrome and death.

This pathophysiological pathway is thought to be responsible for up to 80% of trauma deaths in the ICU. Cardiac function has been described as having particular relevance to multiple organ failure, but the traditional methods of assessment, such as pulmonary artery catheterization or echocardiography, may be too invasive or may not be readily available in an ICU.

- Post-traumatic immunocompromise:

  Hemorrhagic shock states are known to produce a variety of immunosuppressive effects that can lead to increased susceptibility to infections and post-traumatic complications like multiple organ dysfunction syndrome, multiple organ failure, or adult respiratory distress syndrome. While the exact mechanisms of post-traumatic immunocompromise continue to be misunderstood, several studies have suggested that functional and immunological alterations in the spleen may play a major role.

- Conclusions:

  The 2008 volume of critical care contained several articles related to the care of injured patients. Proper care of patients with (TBI) is necessary to prevent secondary brain injury. New therapeutic interventions may be on the horizon. Consistent approaches to the identification of renal injury and inhalational trauma in patients with thermal injury may improve future research and care. Reports from that volume also suggest that the (CT) scan is an adequate method of identifying unstable cervical spine injuries in comatose or obtunded trauma patients and that prolonged immobilization or (MRI) studies may actually cause adverse outcomes. It was also shown that (NT)-proBNP may be helpful in identifying post-traumatic cardiac impairment and that the response of the splenic and lymphocytic systems to massive hemorrhage may provide clues to the pathophysiology (and future interventions) in post-traumatic immunocompromise.

4. BRONCHIAL ASTHMA

4.1. Intravenous Beta$_2$-Agonists for Acute Asthma in The Emergency Department

A. Authors:

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B. Authors' Conclusions:

- Implications for practice:
  - Intravenous beta$_2$-agonist used either as an adjunct to, or replacement of, inhaled bronchodilator therapy appears to offer no clinical benefit in acute asthma.
  - The benefit of IV therapy in ventilated patients has not been examined.
  - Efficacy in the pediatric population remains unclear since too few pediatric clinical trials were identified.

- The only recommendations for IV $beta_2$-agonists use should be for those patients in who inhaled therapy cannot be used; however there have been no tests of its efficacy in such situations.

- Implications for research:
  - The effectiveness of IV $beta_2$-agonists in pediatric patients with severe acute asthma exacerbation's that present to the ED remains to be determined.
  - Future methodologically sound clinical trials could be used to clarify whether IV $beta_2$-agonists improve the initial bronchodilator response when given in addition to nebulized bronchodilator ($beta_2$-agonists and anticholinergics) and corticosteroid therapy (intravenous, oral, or inhaled).
  - The evidence for subcutaneous routes of $beta_2$-agonists (both selective and non-selective) must be formally evaluated via a systematic review.
  - Most of the studies in this review did not provide data beyond the first hour of follow up and there were only small differences demonstrated at 12 and 24 hours. It is possible that aminophylline provides some additional late bronchodilator effect or benefits aside from airway relaxation. However, the magnitude of this effect would be clinically irrelevant in the emergency department and such small potential benefit in bronchodilation would not justify a new emergency department study.
  - Due to the small samples in certain subgroups, the conclusions from these analyses require further evaluation. For example, the most severe subgroup data may be the only area in which aminophylline treatment would justify additional trials.
  - However, it is also possible that aminophylline provides some benefit through other mechanisms. This possibility can only be addressed by choosing other outcomes which could identify these benefits.
  - Future research on acute asthma must concentrate on well defined outcomes which may lead to more informative overviews in the future. More specifically the following areas must be refined:
    - Statistical planning and sample size calculations must be more carefully considered. Trials should be large enough to protect against type II error, and when multiple statistical tests are performed the increased risk of type I error should be addressed.
    - Complete reporting of pulmonary function test (PFT) data in a systematic and standardized fashion would assist in further work (i.e. reporting of % predicted peak expiratory rate PEFR and changes in % PEFR).
    - The inherent variability of these PFTs, particularly in acute asthma, emphasizes the need for further research into alternative measures, particularly assessment of factors that are important to the patient.
    - Standardization and complete reporting of symptom data and universal descriptions of what defines a "clinical success".
    - Standardization and complete reporting of adverse reactions and side effects.
    - Implications for practice
    - There is insufficient evidence to support the routine use of aminophylline in the management of acute asthma when adequate beta-agonist treatment is provided.
    - The development of side effects is significantly higher with aminophylline treatment than $beta_2$-agonist therapy alone.

- Treatments of proven benefit should be encouraged before consideration is given to intravenous aminophylline therapy.

4.2. Inhaled Magnesium Sulfate In The Treatment of Acute Asthma

A. Authors:

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B. Authors' Conclusions:

- Implications for practice:
  - Nebulized ($MgSO_4$) appears to be effective and safe to administer to patients experiencing asthma exacerbations.
  - Treatment with nebulized ($MgSO_4$) should be considered in addition to inhaled β_2-agonists in asthma exacerbations, particularly in those patients with more severe exacerbations.
- Implications for research:
  - The role of nebulized ($MgSO_4$) in asthma exacerbations has not been conclusively resolved by this review, particularly with respect to ($MgSO_4$) alone versus ($MgSO_4$) with β_2-agonists. Further research should be encouraged.
  - In addition, studies of acute asthma should stratify patients by presenting severity of the exacerbation and specify outcomes which are clinically valid, such as relapse or hospital admission. Research should focus on short term outcomes such as change in pulmonary function.
  - There is a strong argument for asthma researchers to develop a consensus regarding the reporting of pulmonary function results.

4.3. Magnesium Sulfate for Treating Exacerbations of Acute Asthma in the Emergency Department

A. Authors:

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B. Authors' Conclusions:

- Implications for practice:
  - In this review, parenteral magnesium sulfate was provided as 2 gm IV over 20 minutes to adults and 25-100 mg/kg IV to children.
  - Many patients who present to the emergency department for asthma exacerbation assessment and treatment may not benefit from early treatment with magnesium sulfate.
  - Patients with severe acute asthma appear to benefit by using Magnesium sulfate, in terms of pulmonary function improvements and reduced admissions. In this context, severe asthma is defined as peak expiratory flow rates of less than 25-30% predicted after initial $beta_2$-agonist therapy in adults and/or non-response to treatment (adults and children), or peak expiratory flow rates of less 60% predicted (children).
  - A clinical approach may be to identify candidates for magnesium sulfate therapy among patients who do not respond to initial $beta_2$-agonists treatment.
  - Two studies examined the use of magnesium in children. Given the similarity of the findings in children, Magnesium Sulfate is used in children in the same manner that it is used in the adult population. Only one study examined its use in children aged less than six, but the numbers were small.
  - In addition to any magnesium intervention, standard acute asthma therapy must be administered to these patients early in emergency department treatment.
- Implications for research:
  Many questions regarding the treatment of acute asthma with magnesium remain unanswered.
  - Most importantly, additional research is required to determine the optimal dose and duration of therapy.
  - Additional studies are needed to confirm the sub-group findings from this review suggesting a beneficial effect of magnesium sulfate only in severe acute asthma. In future studies, severity must be clearly defined and based on presenting pulmonary function results AND response to initial beta-agonist therapy whenever possible.
  - Studies involving very young children need to be performed to determine the effect of magnesium sulfate in this age group.
  - Further studies are required to examine the effect of magnesium sulfate based on the prior inhaled steroid use in patients presenting to the emergency department with an asthma exacerbation. The effect of treatment may differ based on inhaled steroid use, and the answer to this question remains unclear. Inhaled steroids are increasingly employed and the development of high dose inhaled steroids with lower systemic activity suggests that this would be an important area for future research.
  - Future research on acute asthma must concentrate on well defined outcomes, which may lead to more informative reviews in the future. More specifically, criteria for discharge and reporting of lung function test data in a systematic fashion would assist in further work. Finally, better description of the methodology would also be beneficial.

4.4. Early Emergency Department Treatment of Acute Asthma with Systemic Corticosteroids

A. Authors:

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B. Authors' Conclusions:

- Implications for practice:
  - This overview reconfirms evidence from earlier systematic reviews (Rowe 1992; Engel 1991) and supports the use of steroids for treatment of emergency department patients assessed with acute asthma.
  - Therapy with high dose systemic steroids should be commenced within one hour of presentation to the emergency department.
  - Unless the patient fails to respond to early therapy, deteriorated, or presenting case delineate admission, the decision to admit may be delayed until 6 hours after treatment.
  - In children, oral therapy appears to be very effective, although there is no data to provide guidance as to the efficacy of oral therapy for adults in this setting.
- Implications for research:

  Further studies in this area will need to consider the results of the subgroup analyses. Studies which stratify patients into those recently receiving oral steroids vs. those receiving maintenance inhaled steroids would seem appropriate. Documentation of asthma severity at presentation needs to be standardized to permit generalization of trial results. Standardized assessment times would also be useful. A better description of admission criteria is required.

4.5. Early Use Of Inhaled Corticosteroids In The Emergency Department Treatment of Acute Asthma

A. Authors:

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B. Authors' Conclusions:

- Implications for practice:
  - Systemic corticosteroids should be given to all patients with acute asthma presenting to the emergency department.
  - Inhaled steroid therapy decreases admission rates in patients compared to treatment with placebo.

- The additive benefit of inhaled steroids when used with systemic corticosteroids remains uncertain, although the results of this systematic review suggest an additive effect.
- Inhaled steroids are well tolerated with few short term side-effects.
- There is insufficient evidence to determine whether the effect of (ICS) therapy is different in certain populations (e.g. children vs. adults, or mild vs. severe asthmatics).
- There is insufficient evidence that (ICS) therapy alone can be used to replace systemic (CS) therapy.

References