FEDERAL MINISTRY OF HEALTH, ETHIOPIA

NATIONAL INTEGRATED EMERGENCY MEDICINE TRAINING

PARTICIPANT’S MANUAL

October, 2015
The Federal Ministry of health of Ethiopia has been working towards standardization and institutionalization of in-service (IST) trainings at national level. As part of this initiative the ministry developed a national in-service training directive and implementation guide for the health sector. The directive requires all in-service training materials fulfill the standards set in the implementation Guide to ensure the quality of in-service training materials. Accordingly, the ministry reviews and approves existing training materials based on the IST standardization checklist annexed on the IST implementation guide.

As part of the national IST quality control process, this Integrated Emergency Medicine IST package has been reviewed based on the standardization checklist and approved by the ministry in October 2015.

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Human Resource Development & Administration

Directorate Director

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Foreword

The Ethiopia’s Federal Ministry of Health (FMOH) has been leading a sector wide reform effort aimed at significantly improving the quality and accessibility of health services at all levels of the country’s decentralized health system. As part of this reform, health facilities throughout the country have been streamlining their operational processes and building their capacities with a view to making their services more effective and efficient.

Recognizing the importance of strengthening Emergency Services at all level: pre facility and facility level is one of the areas priority given. Obtaining of huge number of ambulances and ongoing initiatives towards training of Emergency Medical Technicians (EMT) to promote pre facility health care and to improve accessibility to health facilities for mothers and acutely ill or injured patients are some of the activities on progress.

At health facility level reorganizing services into emergency and none emergency; staffing by case teams with a well-rounded skill mix, equipping emergency units in hospitals with triage and resuscitation equipment’s, supporting hospitals with on job emergency medicine trainings are areas getting focus on the improvement process of intra facility emergency services.

This National Integrated Emergency Medicine Training guideline contains; trainees reference, facilitator manual with its case scenario and pretest. It focuses on common emergency health problems of all age and sex, and aims on the primary emergency care approach, resuscitation and stabilization in emergency department or emergency rooms until patients will streamed to their respective departments, and both mid level and high-level health professionals working in health facilities can use this manual as a reference. While primarily intended as a reference for hospital personnel, it is hoped that health professionals across all level of national health facilities will also find this guideline useful.

At this venture, I would like to take this opportunity to express my profound appreciation to all partners that have participated in the development of this important reference and training document. Special thanks go to our colleagues at the Addis Ababa University Medical School Emergency Department staff for mobilizing appropriate resource personnel’s for the development of this reference guideline.
Dr. Abraham Endeshaw  
Director, Medical Services Directorate  
Federal Ministry of Health  

**Acknowledgement**

The Federal Democratic Republic of Ethiopia Ministry of Health would like to acknowledge the Emergency Medicine Department of Addis Ababa University School of medicine; for its immense technical and academic support. The resource the department has and the enthusiasm of the staff is an asset for establishing emergency medicine in Ethiopia. The FMOH would like also to acknowledge the following individuals for their participation on the development of this reference guideline.

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Acronyms

ABC = Airway Breathing Circulation
TTC = Tetracycline
RBS = Random blood sugar
IM = Intra muscular
IV = Intra venous
Po = per Os
JHR = Jarisch Herxheimer Reaction
GCS = Glasgow coma scale
Dx = Diagnosis
DDX = Differential diagnosis
BP = Blood pressure
PR = Pulse rate
RR = Respiratory rate
Hx = History
P/E = Physical examination
Bpm = Beats per minute
Mx = Management
Sxs = Symptom
Sns = Sign
Rx = Treatment
URT = Upper respiratory tract
ED = Emergency department
EMT = Emergency medical technician
EMS = Emergency medical service
ECG = Electrocardiogram
CXR = Chest x-ray
CPR = Cardio pulmonary resuscitation
PTE = Pulmonary thromboembolism
OFT = Organ function test
C/I = Contraindication
UFH= Unfractionated Heparin
SC=Subcutaneous
GFR=Glomerular filtration rate
ACEIs= Angiotensin converting enzyme inhibitors
LMWH = Low Molecular weight Heparin
UTI =Urinary tract infection
U/A=Urine analysis
CSF=Cerebrospinal fluid
LFTs=Liver function tests
AFB= Acid fast bacilli
WBCs=White blood cell
Hct= Hematocrit
Hgb= Hemoglobin
MAP=Mean arterial pressure
SIRS = Systemic Inflammatory Response syndrome
JVP= Jugular vein pressure
BUN= Blood urea nitrogen
PT  = Prothrombin time
INR= International normalized ratio
MDI- metered dose inhaler
SABA= short acting beta agonist
PEF= peak expiratory flow
ICP= intra cranial pressure
ETT= endotracheal tube
NSAIDs = non steroidal anti inflammatory drugs
DKA= diabetic Ketoacidosis,
ACLS =Advanced cardiac Life support.
BLS= basic life support,
MI = Myocardial infarction,
CVA= cerebrovascular accident,
HHS =Hyperglycemic hyperosmolar state
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Introduction

Emergency Patient care is a comprehensive and continuous care provided for those who are sick, injured or presented with obstetric emergencies, involving trained personnel in order to decrease morbidity and mortality using available and accessible technology and manpower.

According to 2005 (EFY) data, trauma was the 8th cause of morbidity (1,072,568 cases, 3.8 %), 4th cause of admission (31,952 cases, 4.2 %), 5th cause of mortality (706 cases, 3.7%) in Ethiopia. Particularly acute respiratory tract infection, acute febrile illness and diarrheal diseases are the leading causes of morbidity. The incidence of fatal acute cardio respiratory disease, obstetric emergencies are also among emergencies that kill the productive population.

The problem becomes challenging when the resources are limited and when there is no available organized emergency medical service system that can handle these mostly preventable but life threatening diseases or accidents. The human resource capacity and equipments and supplies are among the challenges faced while thinking of establishing such a system.

To solve such problem Federal Ministry of Health (FMOH) has been working on equipping health facility with necessary medical equipments, expanding specialty care like ICU and Trauma care, distribute ambulances to each woredas and develop guidelines and protocols for emergency medical service system to run efficiently and provide quality of care. Despite that the shortage of trained man power makes the system not to function fully. Therefore expanding the human resource is now the priority area by implementing short courses for emergency health professionals so that this training manual and Facilitator guide was developed to be used by participant for reference and Facilitators for guidance.

Once the health professionals had short course training on emergency medicine they will be able to work in Emergency room and departments as well as on pre hospital care.
Course Syllabus

Course description

- This Five-days National Integrated Emergency Medicine Training/course is designed to provide all levels of health care workers the basic emergency medicine knowledge and skills they need to use to save lives and practices in both pre hospital and hospital settings with available resources.

Course Goal

- To improve knowledge and skill of health professionals on emergency patients handling and resuscitation of critically sick/injured patients.

Course objectives

At the end of this course participants will be able to:

- Describe the organization of emergency units and their function
- Detect and manage the ABC of life
- Describe Medical surgical, gynecological, obstetric and pediatric emergencies
- Manage Medical surgical, gynecological, obstetric and pediatric emergencies

Course evaluation

Each day’s courses will be evaluated based on the developed format addressing the provided documents, Training contents, instructors, facilities, Time and Interactions. Feedbacks will be given based on the given comments immediately. End of course evaluation format will be used to assess the overall effectiveness of the course as perceived by the trainees at the end of the course.

Course duration

5 days with a total of 30 hrs courses with

- Introduction to Emergency Medical Service System - 1hrs
- Airway and Breathing assessment and Management - 2 hrs
- Basic Life Support - 3 hrs
- Approach to the Management of Common Medical Emergencies - 6 hrs
- Assessment and management of trauma - 6 hrs
- Obstetric Emergencies - 6 hrs
• Pediatrics Emergency Medicine (PEM)-6 hrs

Training/Learning Methods

➢ Interactive Lectures
➢ Demonstration
➢ Brain storming
➢ Small group discussions
➢ Individual and group exercise
➢ Role-plays and simulations
➢ Videotapes and discussions
➢ Site observations or facility visits

Instructional materials, supplies and equipment needed in the training

• Ethiopian Hospital Reform Implementation Guideline
• National integrated emergency medicine training Participant manual
• National integrated emergency medicine training Facilitator Guide
• Case study booklet
• PowerPoint slide
• Training videotapes
• Class rooms should include a space for the lecture presentations and a room for skill stations
• Mannequins, ABC and other trauma care equipment’s
• Pediatrics, neonatal and obstetric mannequins and supplies
• Laptop and LCD projectors for lecture presentations

Target audience

➢ Health professional (physicians, nurses, Heath officers) working in the emergency care

Facilitator/Trainer selection criteria

Instructors will be selected from National Integrated Emergency Medicine training Manual/NIEM/ developer or have Training of Trainers/TOT/. The NIEM trainer must have experience using the master learning approach to provide the training, which is conducted
according to adult learning principles—learning is participatory, relevant, and practical—and uses behavior modeling, is competency-based, and incorporates humanistic training techniques. NIEM trainers for this course must be aware of basic principles of transfer of learning to help the participants, transfer the new knowledge and skills in emergency management to their workplaces, and improve job performance.

**Facilitator’s responsibilities**

- Assign Course Director
- Schedule a daily meeting of all facilitators and the Course Director at the close of each day to review progress, solve problems, and to plan for the following day.
- Conduct pre and post test
- Develop norms on how to behave during the training period
- Encourage active participation of trainee
- At the end of the training course conduct training evaluation using pre prepared evaluation questioner

**Trainees’ assessment, qualification and criteria for certification**

- All the trainees will be assessed at the start and end of the course by pre test and post test respectively and will be certified with >70% of post test result and 100% of attendance.

**Core competencies**

Participants will be able to do tasks like emergency management and life saving procedures while working in Emergency room and departments as well as on pre hospital care. The followings are tasks that can be performed after this Training:

- Emergency unit organization
- Triage
- Assessment of ABC of life
- Basic Life Support for Adults and pediatrics including CPR, oxygen therapy
- Resuscitation and stabilization of medical and surgical emergencies
- Application of air way equipments
- Basic Neonatal and obstetric care
- Advanced Life Support; Advanced Trauma Life Support/ATLS/ and Advanced Cardiac Life Support/ACLS/
# Schedule of National Integrated Emergency Medicine Training

## Day 1

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:30-8:35</td>
<td>Registration</td>
<td>5 minutes</td>
</tr>
<tr>
<td>8:35-8:50</td>
<td>Welcome and introduction</td>
<td>15 minutes</td>
</tr>
<tr>
<td>9:00-9:10</td>
<td>Participants introduce each other and expectations</td>
<td>10 minutes</td>
</tr>
<tr>
<td>8:50-9:20</td>
<td>Pre Test</td>
<td>30 minutes</td>
</tr>
<tr>
<td>9:20-10:20</td>
<td>Introduction to Emergency medical service system/EMSS/</td>
<td>1 hour</td>
</tr>
<tr>
<td>10:20-10:30</td>
<td>Tea Break</td>
<td></td>
</tr>
<tr>
<td>10:30-11:30</td>
<td>Airway assessment and Management Practice on the above topics</td>
<td>1 hour</td>
</tr>
<tr>
<td>11:30-12:30</td>
<td>Breathing assessment and Management Practice on the above topics</td>
<td>1 hour</td>
</tr>
<tr>
<td>12:30-1:30</td>
<td>Lunch</td>
<td></td>
</tr>
<tr>
<td>1:30-3:00</td>
<td>Basic Life Support and Normal EKG and arrest rhythms</td>
<td>1 hour, 30 minutes</td>
</tr>
<tr>
<td>3:00-3:30</td>
<td>Tea Break</td>
<td></td>
</tr>
<tr>
<td>3:30-4:30</td>
<td>Practice on *CPR (AB, compression)*post resuscitation care</td>
<td>1 hour</td>
</tr>
<tr>
<td>4:30-5:00</td>
<td>General discussion and rap up</td>
<td>30 minutes</td>
</tr>
<tr>
<td>5:00-5:10</td>
<td>Daily Evaluation</td>
<td></td>
</tr>
</tbody>
</table>

## Day 2

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:30-9:00</td>
<td>Recap</td>
<td>30 minutes</td>
</tr>
<tr>
<td>9:00-10:30</td>
<td>Approach to a patient with cardio-respiratory distress</td>
<td>1 hour, 30 minutes</td>
</tr>
<tr>
<td>10:30-11:00</td>
<td>Tea Break</td>
<td></td>
</tr>
<tr>
<td>11:00 -12:30</td>
<td>Approach to a patient with shock</td>
<td>1 hour, 30 minutes</td>
</tr>
<tr>
<td>12:30-1:30</td>
<td>Lunch</td>
<td></td>
</tr>
<tr>
<td>1:30-3:00</td>
<td>Approach to a patient with Altered sensorium/coma</td>
<td>1 hour, 30 minutes</td>
</tr>
<tr>
<td>3:00-3:15</td>
<td>Tea Break</td>
<td></td>
</tr>
<tr>
<td>3:15-4:45</td>
<td>Approach to other medical emergency problem</td>
<td>1 hour, 30 minutes</td>
</tr>
<tr>
<td>4:45- 5:00</td>
<td>Daily Evaluation</td>
<td>30 minutes</td>
</tr>
</tbody>
</table>
### Day 3

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:30-9:00</td>
<td>Recap</td>
<td>30 minutes</td>
</tr>
<tr>
<td>9:00-10:00</td>
<td>Primary and secondary assessment Airway, chest and abdomen, pelvis injuries</td>
<td>1 hour</td>
</tr>
<tr>
<td>10:00-10:40</td>
<td>Trauma life support for head &amp; spinal injury: neuro-protection, prevention of 2ry insult, C-spine immobilization</td>
<td>40 minutes</td>
</tr>
<tr>
<td>10:40-10:50</td>
<td>Tea Break</td>
<td>Facilitators</td>
</tr>
<tr>
<td>10:50-11:50</td>
<td>Practice in small group -- - primary and secondary assessment -C-spine immobilization, patient lifting, extrication - Head injury – prevention of 2ry insult</td>
<td>1 hour</td>
</tr>
<tr>
<td>11:50-12:30</td>
<td>Approach to patients with fracture</td>
<td>50 minutes</td>
</tr>
<tr>
<td>12:30-1:30</td>
<td>Lunch</td>
<td></td>
</tr>
<tr>
<td>1:30-2:20</td>
<td>Wound care</td>
<td>50 minutes</td>
</tr>
<tr>
<td>2:20-3:00</td>
<td>Practice on fracture management, and wound care</td>
<td>40 minutes</td>
</tr>
<tr>
<td>3:00-3:30</td>
<td>Tea Break</td>
<td>Facilitators</td>
</tr>
<tr>
<td>3:30-4:30</td>
<td>Triage and ER functional organization with discussion</td>
<td>1 hour</td>
</tr>
<tr>
<td>04:30-5:00</td>
<td>Daily evaluation</td>
<td></td>
</tr>
</tbody>
</table>

### Day 4

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<tr>
<th>Time</th>
<th>Activity</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:30-9:00</td>
<td>Recap</td>
<td>30 minutes</td>
</tr>
<tr>
<td>9:00-9:45</td>
<td>Triage in pediatrics</td>
<td>45 minutes</td>
</tr>
<tr>
<td>9:45-10:45</td>
<td>Pediatric airway management</td>
<td>1 hour</td>
</tr>
<tr>
<td>10:45-11:00</td>
<td>Tea break</td>
<td></td>
</tr>
<tr>
<td>11:00-12:30</td>
<td>Pediatric respiratory emergency management</td>
<td>1 hour</td>
</tr>
<tr>
<td>12:30-1:30</td>
<td>Lunch</td>
<td></td>
</tr>
<tr>
<td>1:30-2:15</td>
<td>Pediatric circulatory problems and management</td>
<td>45 minutes</td>
</tr>
<tr>
<td>2:15-3:15</td>
<td>Pediatric BLS</td>
<td>1 hour</td>
</tr>
<tr>
<td>3:15-3:30</td>
<td>Tea Break</td>
<td></td>
</tr>
<tr>
<td>3:30-4:30</td>
<td>Neonatal resuscitation</td>
<td>1 hour</td>
</tr>
<tr>
<td>4:30-5:00</td>
<td>Management of burn</td>
<td>30 minutes</td>
</tr>
<tr>
<td>5:00-5:15</td>
<td>Daily Evaluation</td>
<td>30 minutes</td>
</tr>
</tbody>
</table>

### Day 5

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:30-9:00</td>
<td>Recap</td>
<td>30 minutes</td>
</tr>
<tr>
<td>Morning 9:00-10:00</td>
<td>Management of normal labor with skill session</td>
<td>1 hour</td>
</tr>
<tr>
<td>Time</td>
<td>Session</td>
<td>Duration</td>
</tr>
<tr>
<td>--------------</td>
<td>--------------------------------------------------------------------------</td>
<td>--------------</td>
</tr>
<tr>
<td>10:00-10:10</td>
<td>Tea Break</td>
<td></td>
</tr>
<tr>
<td>10:10-11:00</td>
<td>Vaginal bleeding during pregnancy with case discussion</td>
<td>50 minutes</td>
</tr>
<tr>
<td>11:00-12:00</td>
<td>Shoulder dystocia and breech deliveries with skill session</td>
<td>1 hour</td>
</tr>
<tr>
<td>12:00-12:30</td>
<td>Hypertensive disorders during pregnancy</td>
<td>30 minutes</td>
</tr>
<tr>
<td>12:30-1:30</td>
<td>Lunch</td>
<td></td>
</tr>
<tr>
<td>1:30-2:15</td>
<td>Obstructed labor and ruptured uterus</td>
<td>45 minutes</td>
</tr>
<tr>
<td>2:15-3:15</td>
<td>PPH assessment and mg</td>
<td>1 hour</td>
</tr>
<tr>
<td>Afternoon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3:15-3:30</td>
<td>Tea Break</td>
<td></td>
</tr>
<tr>
<td>3:30-3:45</td>
<td>Trauma during pregnancy</td>
<td>15 minutes</td>
</tr>
<tr>
<td>3:45-4:00</td>
<td>CPR during pregnancy</td>
<td>15 minutes</td>
</tr>
<tr>
<td>4:00-5:30</td>
<td>post-training assessment, final training evaluation and closing</td>
<td>1 hour and 30 minutes</td>
</tr>
</tbody>
</table>
Chapter I: Emergency Medical Service System

Duration - 1hr

Objectives

At the end of this session, participants will be able to

Describe different level, structure, and organization of EMSS

Describe the organization of emergency units and their function

Describe organization of pre facility emergency service organization

Emergency Medical Services System (EMSS): A network of services and resources coordinated to provide aid and medical assistance from primary response to definitive care, involving personnel trained in the rescue, stabilization, transportation, and advanced treatment of traumatic, obstetric and medical emergencies.

Major activities of EMSS: Prevention of injury and acute illness (public education and public health activities), Recognition of the event by bystanders, Activation of the EMS system, Bystander care (ideally with telephone instructions from the EMS dispatcher), Arrival of First Responders, who might be Fire/rescue personnel (paid or volunteer), Law enforcement personnel, Industrial response teams, Arrival of additional EMS resources, which may include EMT-Basics, Intermediates, or Paramedics, according to the level of services designed by the service provider, Emergency care at the scene, Transport to the receiving facility (hospital) and In-hospital care.

Components of EMSS include: Regulation and policy, Resource management, Human resources and training, Transportation, Facilities (different level of health facilities), Communications, Public information and education, Medical oversight (physician involvement), Trauma systems (organized network of resources and procedures for providing care to critically injured patients), Evaluation (quality assurance/quality improvement processes).

Pre-hospital EMS:
The type of medical care provided at the scene of a medical emergency and includes better communication and coordination with the ultimate goal of emergency medical services is to
transport the victim with appropriate care and support to the health facilities. It includes the following six steps:

1. **Detection** – The first rescuers on the scene, usually untrained civilians or those involved in the incident, observe the scene, understand the problem, identify the dangers to themselves and the others, and take appropriate measures to ensure their safety on the scene (environmental, electricity, chemicals, radiations, etc.).

2. **Reporting** – The call for professional help is made and dispatch is connected with the victims, providing emergency medical dispatch.

3. **Response** – The first rescuers provide First AID immediate care to the extent of their capabilities.

4. **On scene care** – The EMS personnel arrive and provide immediate care to the extent of their capabilities on-scene.

5. **Care in Transit** – The EMS personnel proceed to transfer the patient to a hospital via an ambulance for specialized care. They provide medical care during the transportation.

Ambulance: is used to transport and to render care for sick or injured people appropriate to the medical care needs. A pre hospital emergency medical services provider tends to the sick or injured occupant during transportation.

Dispatch center and Ambulance stations: The public must be able to notify the EMS system in a timely manner in order for the EMS system to be of value. The most efficient way to do so is through 3-digit system in which trained dispatchers collect information from the caller and activate the appropriate level of response. In a system, there should be one call or dispatch center, but many ambulance stations in order the ambulance will reach to the caller or scene in short period of time acceptable to save life.

1. **Pre-hospital EMS Providers**: There should be nationally recognized Levels of Training. First Responders are trained at the most basic level to provide initial emergency care at the scene until more highly trained personnel arrive. Ideally, in a system, there will be a high number of First Responders located throughout the community in order to arrive at the scene of the emergency quickly.

2. **EMT-Basics** are the first level of EMS personnel to provide patient care both at the scene and during transportation. The skills performed by EMT-Bs include such things as the use of specialized airway devices, spinal immobilization, and the use of certain medications.
3. EMT-Intermediates are EMT-Basics who have acquired additional training to provide more complicated care to patients. These higher-level skills are referred to as advanced life support (ALS) and include starting IVs and giving some medications.

4. EMT-Paramedics are the most highly trained pre-hospital care providers in the EMS system. Paramedics are EMT-Bs with extensive additional training and education that have a wider scope of knowledge of disease processes and provide advanced life support for patients with a variety of problems. One of the responsibilities of an EMT-Paramedic is providing patient education and community injury and illness prevention activities.

**Hospital EMSS:**

A coordinated Emergency medical support provided at health facility level with standard triage system and definitive care.

The emergency department (ED) or the emergency room (ER) is a hospital or primary care department that provides initial treatment to patients with a broad spectrum of illnesses and injuries which could be life-threatening and requiring immediate attention.

A typical emergency department has several different areas; each specialized for patients with particular severities or types of illness.

1. **The triage area**, patients are seen by a triage nurse who completes a preliminary evaluation, and treatment as necessary, before transferring care to another area of the ED or a different department in the hospital. Patients with life or limb-threatening conditions may bypass triage and to be seen directly by a physician. The triage nurse has to go to resuscitation area and complete the triage form, and patients relatively stable can sent to waiting area while re triaging and reassurance is maintained.

2. **The resuscitation area** is a key area of an emergency department. It usually contains several individual resuscitation inlets, usually with one specially equipped for pediatric resuscitation. Each bay is equipped with a defibrillator, monitors, airway equipment, oxygen, intravenous seats and fluids, and emergency drugs. Resuscitation areas also have ECG machines, and portable X-ray facilities to perform chest and pelvis films. Other equipment may include non-invasive ventilation (NIV) and portable ultrasound devices.

3. **The observation and treatment area** is an area for stable patients who still need to be confined to bed or an area to keep patients for 24hrs until transfer to respective wards or transferred/referred to other health institutions.

4. **Procedure room**: where different interventional activities undertaken
5. Other area such as stores, dispensary for emergency drugs, isolation rooms have to be considered.

**ED/ER Work Flow**

Patients arrive at emergency departments in two main ways: by ambulance or independently. The ambulance crew notifies the hospital beforehand of the patient's condition and begins Basic Life Support measures as needed. Depending on the patient's condition, the emergency department physician may direct the ambulance crew to begin specific interventions while still en route. These patients are taken to the emergency department's resuscitation area, where a team with the expertise to deal with the patients’ conditions meets them. For example, a trauma team consisting of emergency physicians and nurses and other relevant workers sees patients with major trauma.

Patients arriving independently or by ambulance are typically triaged by a nurse with training in emergency medicine. Patients are seen in order of medical urgency, not in order of arrival. Patients are triaged to the resuscitation area, observation and treatment area, or minor’s area. When patient arrives, the ED porters will take to the triage officers and the process will be triggered. The triage officer will be trained BSC nurses, health officers or other relevant workers in the hospital setup but any nurse can led this process in the health centers.

**Emergency Department Human resource**

The structural units available in the department are triage room including porters/runners, registration and recording room, procedures and resuscitation room, observation and treatment rooms, diagnostic, stores and ambulance units.

**Physicians:** different categories of physicians depending on the hospital level. It includes emergency physician, general practitioner or residents in teaching institutes. In rural and regional hospitals, nurses and health officers would take the responsibility.

**ED/ER Nurses**

- Nurse initiates care according the urgency in ED, consult doctors in difficulty and so they are integral part of the management Process.

- So the need to be a mechanism of training in place to improve nursing capability in triage and acute care modalities.
Runners/Porters

They transport only stable patients from ambulance to ED, move Patients from place to place for diagnostic and treatment procedures and to other hospitals in referrals. It is suggested that this workers be primary emergency Health care workers or emergency medical technicians or individuals who has BLS training so as to handle patients professionally and even assist nurses in delivering care.

Environment keeping/Cleaners

New categories of patients constantly visit emergency rooms and environment should be kept clean regularly. Therefore, cleaners should work in team with health care workers and to this goal, training and frequent sensitization is needed.

Guards

Crowding and security issues are threats to emergency care in number of ways and as a result cooperative team of security workers are needed in the ER.

Registration Rooms and officers

Registration and recording room must be adjacent and easily accessible .The system must be designed in modern way so that information can easily be retrieved and analyzed. Any patient should get privilege to be registered and evaluated in emergency situation, regardless of payment.

ED Service

- In higher level where space is not problem Majority of service should be available in the same place
- Basic laboratory, portable X ray and other necessities should be available in the emergency department if possible.
- Emergency drugs and supplies should always be available and accessible, emergency drug box always filled with essential drugs.
- Checklist of such items must be available with periodic revision and refilling.
- There must be standard of ED equipment’s and drugs to each levels and specialties, this must also be worked out and annexed.
Communication in ED

**ED of hospitals** needs to communicate with Dispatch center, pre-hospital care, and other health facility, RHB

- Vertically-with dispatch center and ambulances if Needed and also inpatient structures such as OR, ICU and wards
- Horizontal communications should be in place with House staff to facilitate patient care

Efficient emergency care plays a critical role in reducing mortality and morbidity/disability resulting from obstetric and medical or surgical emergencies or injuries sustained during an automobile accident, fire or any natural or manmade disasters. The survival of emergency patients depends on the quick and efficient emergency pre hospital care delivered at the scene and during transportation to the both public and private health facilities (health centers and hospitals).

The Federal Ministry of Health has a plan to overcome problem faced in delivering the right service to the community in all case including EMS by establishing emergency care in all health institutions and strengthen pre hospital care, which are on progress.

**Activities**

- Discussion on questions raised by participants
- Discussion on topic experiences
Chapter II: Airway assessment and Management

- Duration - 1 hr

Learning Objectives

At the end of this session, participants will be able to:
- Perform proper airway assessment and identify potential airway problems
- Perform manual airway assessment and management maneuvers
- Use adjuvant equipment properly

Introduction

The airway conducts gases between the atmosphere and the alveoli. Therefore, competence in airway management is a critical skill for safe emergency airway problem management and to play a key role in cardio pulmonary resuscitation. Maintaining the airway patent is a fundamental medical skill that no healthcare worker should be without. Airway management is a process of ensuring:

1. There is an open pathway between a patient’s lungs and the outside world.
2. The lungs are safe from aspiration

Airway Emergencies

Causes of upper airway obstruction include:

a) The patient's tongue, in patients with impaired consciousness
b) Secretions in patients who are unconscious with suppressed airway reflexes
c) Foreign body
d) Swelling (Anaphylaxis)
e) Infection (e.g. Epiglottitis)
Causes of potential upper airway obstruction include:

a) Trauma to the face / neck - due to progressive edema of the soft tissue  
b) Airway burns / Neck burns -  
c) Swelling (Anaphylaxis)  
d) Infection (e.g. Epiglottitis)

**Airway assessment in conscious patients**

- The patient’s airway history should be evaluated to determine whether there are any medical, surgical, factors that have implications for airway management.  
- If the patient is sitting up and talking normally, he/she have an adequate airway AT THAT TIME, Reassess regularly.  
- Look for **dyspnea**;  
- Hoarseness or weakness of the patient’s voice,  
- **Stridor**: an abnormal, high pitched sound produced by turbulent airflow through a partially obstructed upper air way during inspiratory phase  
- Previous History of trauma to the airway

**Airway assessment in unconscious patient**

**General approach**

- If Cervical Spine Injury is suspected (major trauma, unconscious patient, head injury), either, apply rigid cervical collar and head blocks or sandbags; or Maintain in-line stabilization manually, while attempting airway maneuvers.  
- Position of the patient is important. e.g.  
  - a. Left lateral position in unconscious patient with adequate spontaneous breathing (unless suspected cervical spine injury)  
  - b. Left lateral position (or wedge) in 3rd trimester of pregnancy  
  - c. Self-positioning in facial trauma or severe dyspnea  
- Remember that patients with a GCS ≤ 8 are unable to protect their airway, due to the absence of coughs, swallowing and gag reflexes.

**Steps in Airway Management**

**Basic Airway Skills**

- Open the airway using head tilt chin lift maneuver in non trauma patient and jaw thrust for trauma patients, **and**
• **See for the following findings and treat as you find**

  1. Presence of any foreign body or secretions – suction or remove manually if the foreign body is reachable
  2. See whether the tongue is falling back to obstruct the airway - apply head tilt chin lift of jaw thrust maneuvers and if patient is not maintaining patent airway insert oropharyngeal or nasopharyngeal airway
  3. For any facial bone deformity, progressive soft tissue swelling and with signs of airway obstruction - consider definitive airway management (intubations, cricothyrotomy) consult colleagues with such skill

**Manual methods**

**Head tilt/Chin lift**

- Used for lifting the tongue from the back of the throat.
- Contraindicated in pts with suspicion of cervical spine injury
- While tilting the head see for chest movement and air is coming in and out
- If no chest movement lift the chin see for any foreign body or secretions and manage accordingly
- If patient has adequate breathing with this maneuver, position patient in left lateral position and administer oxygen
- If there is no effort of breath deliver TWO RESCUE BREATH

![Image of Head Tilt/Chin Lift](image)

**Jaw thrust**

- The jaw thrust is a technique used on patients with a suspected cervical spinal injury and is used on a supine patient.
- The practitioner uses their thumbs to physically push the posterior (back) aspects of the mandible upwards –
- When the mandible is displaced forward, it pulls the tongue forward and prevents it from occluding (blocking) the entrance to the trachea, helping to ensure a patent (open) airway.

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- While maintaining this maneuver see for chest movement or breathing effort
- If no see for foreign body or secretions and manage
- If no effort give TWO FESCUE BREATH

Recovery position

Unconscious patients who have adequate breathing effort should turn into the recovery position, (left lateral position) as this allows prevent tongue from falling back and occluding the airway, and the drainage of fluids, secretions out of the mouth instead of down to the trachea. Therefore all unconscious patients with breathing effort has to be in left lateral position if no contraindications (C-spine injury)
Airway adjuncts

**Oropharyngeal Airway (OPA)**

- A curved piece of plastic inserted over the tongue that creates an air passageway between the mouth and the posterior pharyngeal wall.
- Useful in unconscious patients with GCS of less than 8, to decrease gag reflex.
- Technique of insertion: insert the oral airway upside down until the soft palate is reached. Rotate the device 180 degrees and slip it over the tongue.
- Be sure not to use the airway to push the tongue backward and block, rather than clear, the airway.
- Make sure proper size to the patient is used (measure from the angle of the mouth to the angle of mandible).
- Device greater than this measurement can obstruct or less size will not help the patient.
Nasopharyngeal airway

- Is inserted through one nostril to create an air passage between the nose and the nasopharynx.
- The NPA is preferred to the OPA in semi conscious patients because it is more tolerated and less likely to induce a gag reflex.
- The length of the nasal airway can be estimated as the distance from the nostrils to the meatus of the ears and is usually 2-4 cm longer than the oral airway.
- Any tube inserted through the nose should be well lubricated and advanced at an angle perpendicular to the face.
- NPA are contraindicated in patients who are on anti-coagulant, patients with basilar skull fractures, and with nasal infections and deformities

Laryngeal Mask Airway (LMA)

- It is particularly useful in maintaining an airway in unconscious patients with no breathing or difficulty of breathing
- In which mask ventilation is not possible or intubation fails.
- LMA is a wide bore tube, with a connector at its proximal end (that can be connected to a breathing circuit) and with an elliptical cuff at its distal end. When inflated, the elliptical cuff forms a low-pressure seal around the entrance into the larynx.
- The LMA comes in a variety of pediatric and adult sizes and successful insertion requires appropriate size selection.

Steps for laryngeal mask airway insertion:
• Attach an empty syringe to the valve. Fill the laryngeal mask airway with air, ensuring that it inflates. Ensure there are no leaks or bulges in the cuff.
• Remove all the air from the cuff, making it flat. Place a water-soluble lubricant to the posterior portion of the cuff.
• Pre-oxygenate the patient and induce general anesthesia or make sure patient is deeply unconscious. Once the patient is induced, open the patient’s mouth, and hold the laryngeal mask airway like a pen. Press the tip of the cuff against the hard palate, inserting the laryngeal mask airway into the hypo pharynx until it meets resistance.
• Inflate the laryngeal mask airway until there is an adequate seal. Do not put more than the maximum recommended amount of air into the cuff. Connect the laryngeal mask airway to the anesthesia circuit. Auscultate lung sounds, ensuring that they are equal and bilateral. If there is any difficulty in ventilation, deflate the cuff and reposition.
• A bite block is placed to prevent the patient from biting down on the laryngeal mask airway. Rolling up 4X4’s and placing it between the teeth usually create a bite block. An oral airway will not work.
• Ensure that the patient is breathing adequately before removing the laryngeal mask airway.
• This is a temporary airway device until patient starts to breath or definitive airway device is inserted
• Be aware, that LMA doesn’t prevent aspiration

**Cricothyroidotomy**

The cricothyroid membrane joins the thyroid with the adjacent cricoid cartilage. It is close to the skin, relatively avascular, and the widest gap between the cartilage of the larynx and trachea, so it provides the best access for per-cutaneous (cricothyrotomy) airway rescue techniques. *This technique is used in emergency condition when intubation and ventilation are impossible with the usual methods.*

![Cricothyroidotomy Diagram](image)

**Indications**

• Emergency airway not able to be secured by other means
• cannot intubate / cannot ventilate = Failed intubation, oxygenation unable to be maintained by bag-mask ventilation

**Contraindications**
Children < 8 years old - Needle cricothyroidotomy with jet insufflations is preferred.

**Complications**

**Immediate:** Haemorrhage, Creation of a false passage into the tissues, Hematoma formation, Laceration of the oesophagus, Laceration of the trachea,

**Longer term:** Sub glottic stenosis, Laryngeal stenosis, Vocal cord paralysis hoarseness

**Technique - Identify the anatomy**

1. Surgically prepare the neck, using antiseptic swabs.
2. Palpate the cricothyroid membrane, anteriorly, between the thyroid cartilage and cricoid cartilage. Stabilise the trachea with the thumb and forefinger of one hand to prevent lateral movement of the trachea during the procedure.
3. Local anaesthetic down to cricothyroid membrane if patient is conscious.
4. Puncture the skin in the midline with a 14 - 16 gauge cannula attached to a syringe, directly over the cricothyroid membrane (i.e., midsagittal).
5. Direct the needle at a 45 degree angle caudally, while applying negative pressure to the syringe.
6. Carefully insert the needle through the lower half of the cricothyroid membrane, aspirating as the needle is advanced.
7. Aspiration of air signifies entry into the tracheal lumen.
8. Remove the syringe, and needle then widen the puncture size with scalpel and insert a tube to facilitate breathing or use for jet insufflations

Summary

- The commonest cause of pharyngeal/airway obstruction in unconscious patients is tongue falling back and secretions
- Recovery position (left lateral) is used in unconscious patients who have breathing effort. This technique prevents aspiration and airway obstruction by the falling back tongue
- Avoid head tilt chin lift in trauma patients with suspicion of C-spine injury
- The jaw-thrust maneuver moves the mandible and attached relaxed soft tissue structures anteriorly which helps to make airway patent mainly used in trauma patients to minimize C-spine manipulations
- Oro-pharyngeal airway is used in patients with depressed (absent) gag, swallowing reflexes or GCS of less than 8.
- Patients with GCS less than 8 are considered as depressed gag, swallowing and cough reflexes therefore during insertion of oropharyngeal airway they tolerate without complications or vomiting will not induced
- Nasopharyngeal airway is used for all types of patients who are prone for airway obstruction and facilitates suctioning of the airway
- Laryngeal Mask Airway is a rescue device where mask ventilation is difficult or attempts to intubate is failed The cricothyroid membrane is close to the skin, relatively avascular, and the widest gap between the cartilage of the larynx and trachea, so it provides the best access for percutaneous (cricothyrotomy) airway rescue techniques. This technique is used in emergency condition when intubation and ventilation are impossible with the usual methods.

Self-assessment questions
1. Describe The commonest cause of airway obstruction in unconscious patients
2. Describe the different techniques of manual maneuvers of airway management
3. Describe the difference and function of oro-pharyngeal and naso-pharyngeal airways
4. Describe the anatomy, indications and technique of cricothyrotomy

Activities
- Discussion on case scenario #1
- Simulation&practice in small group
- General discussion on questions raised by participants
Chapter III: Assessment of Breathing and management

- Duration -1 hr

Learning Objectives:

At the end of this session, participants will be able to:
- Detect signs and symptoms of emergency breathing problems
- Manage breathing problems
- Use adjuvant equipment to rescue breath and oxygen administration

Assessment of Breathing

1. Work of Breathing
   - Respiratory Rate
     Increase due to lower respiratory or airway problem, pyrexia and metabolic acidosis
     Decrease due to fatigue: over dose of sedation drugs or opioids, exhaustion, poisoning
   - Use of accessory muscles
     Sternocleidomastoid, Intercostal, subcostal, sternal recession
   - Sounds of breathing
     Stridor: upper airway obstruction
     Wheeze: lower airway obstruction
     Grunting: sign of severe respiratory distress, characteristically in infants

2. Effectiveness of Breathing
   - Degree of chest expansion
   - Breath sounds on auscultation - beware the silent chest
   - Heart rate
   - Colour
   - Mental state
   - Signs of hypercarbia - warm peripheries, sweating, and decreased level of consciousness.
   - Pulseoximetry
Signs and symptoms of respiratory problems
- **Look for chest movement**: unilateral, bilateral,
- Look for **dyspnea**: a history of shortness of breath,
- Hoarseness or weakness of the patient’s voice,
- **Stridor**: an abnormal, high pitched sound produced by turbulent airflow through a partially obstructed upper air way during inspiratory phase
- Does the patient exhibit shallow respirations, increased rate of respiration, retractions with inspiration, or symptoms of being short of breath

Management of breathing difficulty
- The primary problem in airway management is an inability to oxygenate, ventilate, prevent aspiration, or a combination of these factors.
- Effective ventilation requires both a face-tight mask fit and a patent airway. In unconscious patients following opening of the airway using hand maneuvers and airway adjuvants if the breathing is inadequate or no breathing start assist/rescue breath mouth to mouth or with bag valve mask.

The bag-valve masks (BVM) ventilation:
- BVM device is used to manually deliver positive pressure through an applied facemask, extraglottic/LMA device or endotracheal tube.
- The former would be an initial step in an apneic or hypo ventilating patient, and is almost always indicated prior to, or during intubation of an ill patient.
- The clinician should be intimately familiar with the workings of the BVM device, as it has a number of valves, and needs proper assembly to work. Also
- These devices incorporate a self-inflating bag, a one-way bag inlet valve, and a no rebreathing patient valve.

**Indications:**
- For patients with inadequate ventilation BVM is meant to simply provide positive pressure ventilation.

**Technique:**
1. Select appropriate mask size that fits comfortably over the mouth and nose.
2. Place the mask strap beneath the occiput. Use the C and E method (see the picture below)
3. Apply the mask’s nasal groove to the low point of the nasal bridge to avoid pressure on the eyes.
4. Grip the left mandible with the third and fourth fingers of the left hand
5. Lower the mask so that its inferior rim contacts the face between the lower lip and the mental prominence.
6. If there is a leak between the mask and the cheeks, consolidate the seal by dragging mobile tissue of the left cheek toward and under the mask cushion, stabilizing the tissue with the ulnar margin of the left hand.

7. Bracing the mentum against the mask, pull the mandible up and forward with the third through fifth fingers, while the thumb and index finger grip the mask above and below the connector. C&E method

8. Maintaining the left-sided seal, tilt the mask toward the right cheek, consolidating the seal by dragging the mobile tissue forward to the cushion and by keeping it there with one limb of the mask strap.

Predictors of difficult mask ventilation

1. Age >55yr

2. Body mass index > 26kg/m2

3. History of snoring

4. Beards

5. Absence of teeth (the presence of two of the above factors has >70% sensitivity and specificity)

6. Facial abnormalities
7. Receding jaw
8. Obstructive sleep apnea

Oxygen therapy

- Oxygen is a life saving treatment. It should be treated like any other drug; it should be prescribed in writing, with the required flow rate and the method of delivery clearly specified.
- Failure to correct hypoxemia (PaO₂>60mmHg or s0₂>90%) for fear of causing hypoventilation and carbon dioxide retention is unacceptable clinical practice.
- Careful monitoring of treatment is essential and will detect those patients at risk.
- Intermittent oxygen therapy is particularly dangerous since the increased alveolar CO2 concentration which may then occur, results in an even lower O₂ concentration when the patient breaths air (o₂ supply is discontinued).
- The amount can be adjusted and regulated according to the results of pulseoximetry and arterial blood gas analysis if available

Causes of tissue hypoxia

Arterial hypoxemia

- Low inspired oxygen partial pressure (high altitude)
- Alveolar hypoventilation respiratory rate decreased (sleep apnea, opiate overdose)
- Ventilation-perfusion mismatches (acute asthma, atelectatic lung zones, ARDS)

Failure of oxygen-hemoglobin transport system

- Inadequate tissue perfusion,(shock), Low hemoglobin concentration(anemia), Abnormal oxygen dissociation curve (hemoglobinopathies, high carboxyhemoglobin), Histotoxic poisoning of intracellular enzymes (cyanide poisoning, carbonmonoxide poisoning septicemia,

Recommendations for instituting oxygen therapy

- Cardiac and respiratory arrest
- Hypoxia with pulseoxymeter measurement (saturation of oxygen <93%)
- Hypotension (systolic blood pressure <100 mm Hg)
- Low cardiac output and metabolic acidosis (bicarbonate<18 mmol/l)
- Respiratory distress (respiratory rate >24/min)in adult
Technique of Oxygen administration:

**Nasal prongs or catheter**-

- Suitable and better tolerated.
- Oxygen administration via nasal prongs range from 1-litter -5litter/minute.
- Rises inspired oxygen concentration to 30-40 percent.
- A humidifier should be used.
- If a flow rate greater than this amount is used it results on irritation to the nose and it doesn’t increase the oxygen delivery to the patient rather it is wastage.
- If the patient has fast breathing and the oxygen saturation is not improving change to face mask.

**Face mask**-

- There are different types of facemasks.
- They could be with reservoir or without.
- A flow rate of 6-10 liters/minute provides a rise in inspired oxygen concentration to 60-70 percent.
- The maximum concentration can be increased with masks, which has a reservoir bag.
- The flow rate shouldn’t be less than 6 litters/minute to avoid rebreathing
- If you decide patient is hypoxic start from the higher flow suitable to the device and titrate down ward according the patients response.
- If patients oxygenation and general conditions is not improving and signs of hypoxia or hypercarbia are persisting conceder the next technique of oxygen administration, non invasive respiratory support (CPAP) or invasive (intubation and ventilation with mechanical ventilator) respiratory support according the patient’s condition

**Adverse effects of high oxygen concentrations** (100% oxygen for >24hrs), which is achieved only with mechanical ventilator, may cause Retrolental fibroplasias, in premature babies. In adults Oxygen toxicity manifest as convulsions, damage to pulmonary epithelium and worsening of the respiratory failure

**Activities**

- Discussion on case scenario #2
- Simulation&practice in small group
- General discussion on questions raised by participants
Chapter IV: Basic Life Support

Duration: 3 hrs

Learning Objectives
At the end of this session, participants will be able to
- Describe the aims of BLS
- Describe the chain of survival
- Demonstrate the steps of BLS
- Perform effective CPR

DEFINITION: BLS is an initial ABC assessment and management of sick or injured patients. It can be provided by trained non-medical or medical workers, until definitive medical treatment can be accessed.

Aims: The 4 main aims of primary assessment “4 Ps” are:

- **Preserve life**– prompt ABCD assessment and management
- **Prevent further injury**– C- spine protection in trauma patients
- **Promote recovery**- administer ox, prevent aspiration, pain control
- **Protect yourself**- outside health institution- scene safety (electricity, blast, chemicals), and self-protective materials (apron, glove, mask…)
- The 5th P we have to consider is preparation / readiness with necessary lifesaving equipment and supplies

Key Initial assessment Skills

A- Airway: unconscious patients may Easley die due to airway obstruction. Just by opining the airways using manual maneuvers and proper positioning you can save life.
B- Breathing: Following an opening of the airway see, weather patient is breathing adequately or not. Look and listen for chest movement
C- Circulation: check for central pulse on the carotid, femoral and if central pulse is available see for peripheral on the arms
D- for
  - Disability -a rapid assessment of neurological function in trauma patients,
  - Deadly bleeding in trauma pts - stop bleeding
  - Defibrillation in patients with cardiac arrest
E- Exposure/Environment

  - Exposure- undress the patient fully during evaluation to minimize unseen injuries
• Environment: protect from hypothermia - keep patients warm

Preserving Life: In order to preserve life look for:

Airway - Maintain clear passage of air without obstruction.

• Determine unconsciousness, if unconscious
• Assess the airway for presence of any foreign body, secretion, the tongue is obstructing the airway

Action

• Patient unconscious - open the mouth using head tilt chin lift or jaw thrust maneuvers
• Look and remove/suction any foreign body or secretions and see for B - breathing

B- Breathing: Can patient talk and breathe freely? YES - this means patient has open airway, sufficient breathing and oxygenation, therefore go to C

C- Circulation: check for regularity, volume and rate of the pulse, measure blood pressure and saturation of oxygen using pulseoxymetry and act accordingly:

✓ Has peripheral pulse continue oxygen delivery
✓ Blood pressure low insert large bore canola start resuscitation to rich the lower optimal level of blood pressure that maintains the perfusion. Do not over load patients especially if you are not clear about the cause of low blood pressure.
✓ In trauma patients with low blood pressure stop bleeding, insert two large bore cannula resuscitate to maintain the optimum allowable blood pressure. Do not aim to raise the blood pressure to normal value because it will induce more bleeding.

• If the patient is unconscious but has adequate breathing effort place in recovery position, with the patient leant over his/her side (see the picture below).
• NB. Consider risk of trauma to cervical vertebrae before changing position of the patient in all unconscious trauma patients.
• If patient is unconscious and seems no breathing or agonal breathing-, confirm breathing by: looking for chest movement, feeling airflow around the nose and mouse, listening for breathing. When you confirm breathing is not there:

✓ Give TWO rescue breaths each for one second and start chest compressions 30 compressions (30:2 ratio)
If patient is a trauma case and has breathing difficulties see for tension pneumothorax/haemothrax, then decompress the lung with large bore needle or insert chest tube, or if it is open chest injury close the open injury, administer oxygen. For detail see trauma chapter.

3. **C- Circulation**, if there is no breathing or the patient is not breathing normally, such as agonal breathing, start CPR,

- activate emergency team if available or shout for help
- position patient properly on hard back board or to the floor
- start CPR with 2 rescue breath and 30 chest compressions (2:30) (ABC) or start immediately cardiac compressions (CAB) if you witnessed the collapse or arrest of the patient (for detailed description see the CPR chapter)

4. **D- Disability, Defibrillation**

- Assess patient for neurological deficiency using GCS, or AVPU
- Defibrillate patients with cardiac arrest as early as possible.

5. **Exposure**

- Undress patient and look for injury, If patient is suspected to having a neck or spinal injury do in-line immobilization
- Protect patients from hypothermia

**Prevent further injury** – while doing the primary survey or assessment in unconscious patients always protect the cervical spine until the diagnosis of cervical spine injury is ruled out.

**Promote recovery**-

- Oxygen administration,
- strict follow up for vital signs,
- aspiration protection,
- on time initiation of definitive management,
- Pain control…

**Protect yourself**-

- Outside hospital, consider the safety of the scene.
- Apply protective devices to protect yourself from contaminations,
- consider any possible legal issue
Prepare your working area, resuscitation equipment and emergency drugs during every shift change, refill regularly missing items.

Cardio Pulmonary Resuscitation (CPR)

Definition: is a skill, which includes artificial respiration to provide oxygen to the lungs and artificial circulation to maintain blood flow through the body enough to give a person a chance for survival.

1. Components:
   - **BLS** - can be done any were, any time, by anyone who is trained to do so, and most of time it doesn’t need special equipment’s.
   - **ALS** - needs trained medical workers, special equipment’s,

   The goal of resuscitation interventions for a patient in respiratory or cardiac arrest is to:
   - ✓ Restore effective oxygenation & ventilation
   - ✓ Restore circulation
   - ✓ Return of intact neurological functions

2. Chain of survival

   For effective result of resuscitation there should be:
   1. Early access to the patient or victim
   2. Early CPR initiation
   3. Early defibrillation
   4. Early & effective post resuscitation care

3. Causes of cardiac arrest

   - Cardiac origin: ventricular fibrillation, Pulses tachyarrhythmia’s due to ischemic heart disease, shock
   - Respiratory origin: airway obstruction, drowning, stroke, smoke inhalation, drug overdose, electrocution, physical trauma.
   - Metabolic disturbance: electrolyte imbalance, acid base imbalance

Cardio pulmonary arrest (CPA) out – of hospital

   - The primary rhythm for cardiac arrest is usually ventricular fibrillation, Pulseless tachyarrhythmia’s due to ischemic heart disease

CPA in Hospital –

   - Often have multi system abnormalities that are non-cardiac in origin,
   - Commonly suffering a gradual deterioration in physiological state, with hypotension, hypoxemia or both prior CPA.
   - The terminal event in these cases is usually pulseless electrical activity or asystole.
4. **Consequences of cardiac arrest**

- Rapid depletion of oxygen in vital organs.
- After 4-6 min of cardiac arrest brain damage can occur.
- Early CPR within 4 min and rapid ALS with defibrillation within 8 min are essential in improving survival and neurological recovery.

5. **Adult Basic Life Support (BLS)**

When patient is found unconscious or suspected to have cardiac arrest:

- **Determine un- responsiveness by shaking and shouting**
- **Call for help**
- **Contact EMSS if available**
- **Position the victim and start CPR.**

**Adult basic life support sequence**

```
Unresponsive
Shout for help
Open airway
Not breathing normally
Provider 2 rescue breaths
30 chest compressions
2 rescue breaths
30 chest compressions/5cycle and reassess
```
A-air way

- **Stabilize the neck**
  - To open the air way push backward on the forehead and lift the chin if the possibility of cervical spine injury is less
  - or use only jaw thrust in patients with suspicion of cervical spine injury
  - Keep the head tilted
  - Place your ear just above the casualty's nose and mouth
  - Look for chest movement
  - Listen for sounds of breathing
  - Fell for breath on your cheek

B-breathing

- Assume cardiac arrest if patient is unresponsive and apneic or has an abnormal breathing pattern.
- Give 30 chest compressions immediately before any rescue breaths are attempted.
- Breath in to the causality twice, each breath should take about 1 sec, and use enough air to make the chest rise.
- Avoid interruptions during CPR

If the chest does not rise when you blow air

a. Reopen the airway by tilting the head and lifting the jaw, see and remove any foreign bodies or secretions
b. Pinch the nose again
c. Make a better seal around the mouth
d. Try blowing again

C-circulation

- Carotid artery palpation has been found inaccurate in both untrained and health care workers and has been omitted. An absence of spontaneous breathing including agonal breaths is now taken as a sign of cardiac arrest.
- Start chest compression:
  - Depress and release the chest rhythmically,
  - Press the heels of the hands straight dawn on the center of the chest.
  - The pressure and release phases should take the same time,
  - Give compressions at a rate of 100 per min.,
  - Count compressions out loud,
  - Give 30 compressions to 2 breaths weather with one rescuer or **TWO**
  - Depress the chest 3.8-5cm depth
  - After every 5 cycle/2min. check for spontaneous breathing and circulation for 5 sec
Defibrillation

- The most frequent cause of cardiac arrest is said to be cardiac fibrillation.
- Start defibrillation as soon as possible.
- Treat VF/ or pulseless VT with a single shock followed by resumption of CPR.
- Reassess rhythm after 2 minute (5 cycles of 30:2 CPR) and give another shock if indicated.
- For out of Hospital settings, AEDs (automated electrical defibrillators) can be used. They are user friendly and analyze the patient’s rhythm and recommend shock delivery or not.
- **Remember after every defibrillation immediately continues cardiac compression.**
- The initial shock for all biphasic defibrillators is 150J with subsequent shocks at 200J. The initial and subsequent shocks for monophasic defibrillators are 360J.
- If there is difficulty differentiating between a rhythm of fine VF and asystole, the treatment should be as for asystole and no shock given. Defibrillation in these cases causes myocardial injury and chest compression is the preferred

Complications of CPR

- Gastric distention and regurgitation- insert NGT and deflate the stomach
- Rib and sternal fracture – see for pneumothorax

Activities

- Discussion on case scenario # 3
- Simulation&practice in small group
- General discussion on questions raised by participants
Chapter V: Advanced resuscitation of cardiac arrest

Learning Objectives
By the end of this session, participants will be able to
- Describe the assessment of a cardiac arrest
- List the management principles of cardiac arrest
- Identify and manage common ECG abnormalities
- List causes of bradycardia, tachycardia and their management

Definition of terms
- **SCD (sudden cardiac death)** is death due to cardiac causes heralded by abrupt loss of consciousness in an individual with/without known preexisting heart disease.
- **Cardiovascular collapse** is acute loss of effective blood flow to distal organs.

**Cardiac arrest**: refers to abrupt cessation of cardiac pump function which may be reversed by prompt intervention. Causes include:
1) Cardiac origin: VF, pulseless tachyarrhythmia due to ischemic heart disease, shock, stroke…
2) Respiratory origin: upper airway obstruction, drowning, smoke inhalation, drug overdose, trauma…
3) Metabolic disturbances: electrolyte imbalance, acid base imbalance

Always think of reversible causes for cardiac arrest: the 5 H’s (hypovolemia, hypoxia, hydrogen ion (acidosis), hypo/hyperkalemia, hypothermia) and 5T’s (tension pneumothorax, tamponade-cardiac, toxins, thrombosis-pulmonary/coronary
Consequences of cardiac arrest are rapid depletion of oxygen in vital organs; after 4-6 min of cardiac arrest brain damage can occur. Early CPR within 4 min and rapid ACLS with defibrillation within 8 min are essential in improving survival and neurological recovery.

ACLS is simply CPR plus better setup and expertise and should be considered as a continuation of BLS; otherwise the activity and goal is the same – facilitating recovery of cardiac arrest victim.

**Goals of ACLS:**
- Achieve adequate ventilation
- Control arrhythmias – revert to one that is hemodynamically effective
- Stabilize BP and cardiac output
- Restore organ perfusion and maintain it

Strategies of ACLS include:
- high quality CPR
- Defibrillation
- IV medications
- Advanced air way

So, survival from cardiac arrest is highly dependent on high quality CPR, ACLS & Post cardiac arrest care

**For high quality CPR,** effective chest compression skills are very essential.
- Push hard & fast (100/min) using heel of both hands (5 cm depth)
- Allow complete chest recoil after each compression. Do not apply pressure over the ribs
- Compression to ventilation ratio is 30:2 (for both one or two rescuer).
  - Avoid rapid & forceful breathes (to prevent hyperventilation).
  - If advanced air way inserted, 100 per minute without interruptions for ventilation.
- Minimize interruptions in compression (No pause in compression for delivery of ventilation). Any interruption should be less than 10 seconds

- Current ACLS algorithm follows a new circular format (see algorithm) to emphasize the importance of high quality CPR. The ACLS interventions (drug therapy, defibrillation) should be reorganized around 2 minute periods of uninterrupted CPR
  - CPR & defibrillation are of primary importance; drug therapy is of secondary importance.

* Note: CCR (Cardio cerebral resuscitation) is conducted for witnessed collapse. It begins from chest compressions to facilitate the circulation of the available oxygen in the lungs to the body
**Defibrillation**: is the definitive therapy for most cardiac arrests. It is passage of an electrical current of sufficient magnitude to depolarize a critical mass of myocardium and restoration of coordinated electrical activity for VF and Pulseless ventricular tachycardia. It is non synchronized delivery of energy, i.e., the shock is delivered randomly during the cardiac cycle while cardioversion refers to delivery of energy that is synchronized to the QRS complex

- Synchronized= i.e. timed with the QRS complex; eliminates the risk of VF
  - Indication: Unstable SVT/AF or monomorphic VT

- In witnessed arrest, defib should be attempted as early as possible & repeated as per the algorithm.
- In unwitnessed arrest, 5 cycles of CPR with simultaneous drug Rx may precede the first defibrillation.
- Initial doses of electric energy for defibrillation and cardioversion depend on the type of the defibrillator (Monophasic Vs Biphasic) and the particular rhythm disorder.
- Proper use of the defibrillator requires special attention to the following: Selection of proper energy, proper mode (asynchronous vs Synchronous), proper position of the paddles or electrode pads, adequate contact between paddles and skin, no contact with anyone other than the victim, rhythm assessment.
  (Preparation→select energy→charge→discharge)
- In AED (automated external defibrillators): turn on→apply the pad→wait while the machine analyzes the rhythm→then the machine advises shock if it detects a shockable rhythm.

*Note: Precordial thump, one or two blows to the junction of the middle & lower thirds of sternum, may be considered for termination of witnessed unstable ventricular tachyarrhythmias when defibrillator is not immediately available. (Anecdotal reports of successful "thump-version" of asystole, VF, and VT)

**Advanced air way**: endotracheal tube, supraglottic airway

Benefits:
- Eliminates need for interruption of chest compressions for ventilation
- Improves ventilation and oxygenation
- Reduces risk of aspiration
- Drug delivery when other routes fail

Disadvantage:
- Interruption of chest compression, misplacement of tube
- Apply continuous chest compressions at 100/min once advanced airway is in place
- Rescue breaths at 8-10 bpm / one breath every 6-8 seconds
- Confirm tube placement with clinical examination or if available with end tidal CO2
Drugs used in ACLS & management of different arrhythmias:

- Route of administration: All peripherally administered drugs should be given bolus followed by 20 ml bolus IV fluid, elevating the limb to improve central delivery. Central line, Intraosseous(IO) lines are better options. When IV/IO route is unsuccessful, one can use endotracheal tube(ETT)- for epinephrine, vasopressin or lidocaine (Dose should be 2- 2 ½ times the IV dose).

1. Vasopressors:
   - **Epinephrine**: indication: VF, Pulseless VT, Asystole, PEA, Symptomatic bradycardia
     Dose : 1mg IV/IO Q 3-5 minutes during CPR, followed with 20 ml flush
     (If IV/IO access not possible – 2-2.5mg via ETT diluted in 10 ml N/S)
   - **Vaspressin**: 40 U IV/IO is alternative

2. Anti arrhythmics:
   - **Amiodarone** = indication: VF/ Pulseless VT unresponsive to CPR, Vasopressor or Shock; Polymorphic VT, adjunct to cardioversion of SVT/PSVT. Dose : Cardiac Arrest: 300 mg IV/IO with 20-30 ml D5W push. If no response,150 mg IV push; repeat in 3-5 minutes.(for stable wide complex tachy-150mg iv )
   - **Lidocaine**: alternative to amiodarone in cardiac arrest from VF/VT, stable monomorphic VT with preserved LV function. Dose : 1-1.5mg/kg IV; if VF/VT persists 0.5-0.75mg/kg IV at 5-10 min.(max 3mg/kg)
   - **Magnesium sulphate**: for VF/Pulseless VT associated with torsades de pointes(Irregular/Polymorphic VT associated with long QT interval) ;hypomagnesemia or digitalis toxicity
     Dose : IV/IO bolus of 1-2 gram diluted in 10 ml D5W over 5-20 minutes
   - **Adenosine**=first drug for most forms of narrow tachyarrhythmias. Dose:6 mg rapid IV push, follow with 20 ml N/s; repeat dose of 12 mg in 1- 2 minutes, up to 3rd dose of 12 mg. S/E : hypotension, chest discomfort . C/I : Asthma, 2nd or 3rd degree AV block
   - **Diltiazem / Verapamil** = indication: control ventricular rate in Afib and A flutter. Dose:Diltiazem-15-20 mg IV over 2mins or Verapamil- 5mg may repeat in 15 mins at 20-25 mg.

- **Atropine**= indication: Symptomatic sinus bradycardia;2nd °(I) AV Block; 2nd line for asystole/PEA
  
  *Note: Atropine for PEA / Asystole is no longer recommended.

- **Digoxin**= for A fib/ flutter rate control. Dose: loading 0.25mg Q 2hr until 1mg total; then 0.125- 0.25mg/d
Monitoring of success
- Mechanical parameters: Rate and depth of compression, rate of ventilation
- Physiologic parameters:
  o check expected ECG changes
  o **Pulse** is not an ideal marker. Palpation of a pulse in femoral triangle during chest compression may give false impression of palpable pulse (Due to retrograde flow of blood to femoral vein)
    ▪ Attempt to check may lead to unnecessary interruptions in CPR
    ▪ Check in <10 seconds, and if not palpable resume CPR
  o **Pulsoximeter** doesn’t provide a reliable sign as pulsatile blood flow is inadequate to peripheral vessels.
    ▪ It may be used to monitor oxygenation after ROSC (Return of Spontaneous Circulation - i.e. return of pulse & BP
  o Arterial blood gas measurements are not reliable indicators
  o Others not available in our set up: End tidal CO2, central venous oxygen saturation
Adult Cardiac Arrest

Shout for Help/Activate Emergency Response

1. Start CPR
   - Give oxygen
   - Attach monitor/defibrillator

   Yes
2. VF/VT
   - Shock
3. Rhythm shockable?
   - CPR 2 min
   - IV/IO access
   - Epinephrine every 3-5 min
   - Consider advanced airway, capnography
   - CPR 2 min
   - Amiodarone
   - Treat reversible causes

4. CPR 2 min
   - IV/IO access
   - Epinephrine every 3-5 min
   - Consider advanced airway, capnography

5. Rhythm shockable?
   - Yes
   - Shock
   - CPR 2 min
   - IV/IO access
   - Epinephrine every 3-5 min
   - Consider advanced airway, capnography

6. CPR 2 min
   - Epinephrine every 3-5 min
   - Consider advanced airway, capnography

7. Rhythm shockable?
   - Yes
   - Shock
   - CPR 2 min
   - IV/IO access
   - Epinephrine every 3-5 min
   - Consider advanced airway, capnography

8. CPR 2 min
   - Amiodarone
   - Treat reversible causes

9. Asystole/PEA
10. CPR 2 min
    - IV/IO access
    - Epinephrine every 3-5 min
    - Consider advanced airway, capnography

11. CPR 2 min
    - Treatment of reversible causes

12. Rhythm shockable?
    - No
    - CPR 2 min
    - IV/IO access
    - Epinephrine every 3-5 min
    - Consider advanced airway, capnography

13. Rhythm shockable?
    - Yes
    - CPR 2 min
    - IV/IO access
    - Epinephrine every 3-5 min
    - Consider advanced airway, capnography

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in PrCO2 (typically >40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy
- Biphasic: Manufacturer recommendation (eg, initial dose of 120-200 J). If unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- Monophasic: 360 J

Drug Therapy
- Epinephrine IV/IO Dose: 1 mg every 3-5 minutes
- Vasopressin IV/IO Dose: 40 units can replace first or second dose of epinephrine
- Amiodarone IV/IO Dose: First dose: 300 mg bolus. Second dose: 150 mg.

Advanced Airway
- Supraglottic advanced airway or endotracheal intubation
- Waveform capnography to confirm and monitor ET tube placement
- 8-10 breaths per minute with continuous chest compressions

Reversible Causes
- Hypovolemia
- Hypoxia
- Hypercapnia
- Acidosis
- Hypo/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

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**Strategies in post cardiac resuscitation care**

- **Therapeutic hypothermia (32-34°C)**
- **Hemodynamic optimization (hypotension Mx)**
- **Ventilation optimization (Sao₂ ≥ 94%)**
- **Immediate coronary reperfusion with PCI (percutaneous coronary intervention) for MI**
- **Glycemic control: maintain glucose levels between 140-180 mg/dl to avoid hypoglycemia**
Management of Symptomatic brady- and tachyarrhythmias:

- Goal is to indentify & treat unstable, symptomatic patients. Decision on Mx should always be based on whole clinical assessment rather than rhythm records from the monitor
- **Unstable**: vital organ function is acutely impaired i.e acute altered mental status, ischemic chest pain, acute heart failure, hypotension, other signs of shock or cardiac arrest is imminent & calls for immediate intervention
- **Symptomatic**: Causing symptoms like palpitations, light headedness, or dyspnea, but the patient is stable and not in imminent danger \( \rightarrow \) Gives time to decide on most appropriate measure
- Decide if the unstable features are caused by the rhythm disorder or not. Eg. In septic shock, a rate of 140 is a physiologic response and electrical cardioversion doesn’t change the rhythm

**Description of normalelectrocardiogram (ECG )**

- ECG records electrical activity of the heart. It is important to evaluate cardiac & non cardiac problems like renal, pulmonary and electrolyte abnormalities.
- Ordinary ECG has 12 leads (recorded through 5 electrodes-one attached to each limb & one to the chest) -6 limb leads ("standard leads"): 3 bipolar: I, II, III and 3 unipolar: aVR, aVL and aVF
  -6 precordial (chest) leads: V1-V6

The contraction and relaxation of cardiac muscle result from the depolarisation and repolarisation of myocardial cells. These electrical changes are recorded via electrodes placed on the limbs and chest wall and are transcribed on to graph paper to produce an ECG

The ECG is recorded on to standard paper travelling at a rate of 25 mm/s. The paper is divided into large squares, each measuring 5 mm wide and equivalent to 0.2 s. Each large square is five small squares in width, and each small square is 1 mm wide and equivalent to 0.04 s.
The ECG grid

The Normal ECG:
**P wave:** represents atrial depolarization. Normal duration is <0.12 sec or < 3 small squares. Amplitude is <0.25mv(<2.5mm)

**PR interval:** represents conduction delay in the AV node. Duration is 0.12-0.2 sec

**QRS complex:** represents ventricular depolarization. Duration is <0.12 sec.

**ST segment:** begins with J point. Usually isoelectric and has upward concavity. ST elevation with upward convexity occurs in acute MI and suggests transmural injury or infarction. (See…ACS)

**T wave:** represents ventricular repolarization. Polarity is similar to preceding QRS.

- T-wave inversion is the most frequent finding in pulmonary thromboembolism (PTE). T-wave inversion is normal in leads with dominantly negative QRS complexes (aVR, V1, and sometimes lead III). Pathological T-wave inversion occurs as a non-specific response to various stimuli (e.g. viral infection, hypothermia). More important causes of T-wave inversion are ventricular hypertrophy, MI. Exaggerated peaking of the T wave is the earliest ECG change in ST elevation myocardial infarction. It also occurs in hyperkalaemia.

- **QT interval:** represents total ventricular activity. Corrected QT interval is calculated as \( QT_c = \frac{QT}{\sqrt{RR}} \). Normally it is < 0.44 sec.

  ![Image of QT interval](image)

  - Abnormal prolongation of the QT interval predisposes to ventricular arrhythmia eg. Torsades de pointes. It can be caused by some of the cardiac drugs, hypocalcemia, hypokalemia, MI or myocarditis. Shortening is caused by hyperkalemia, hypercalcemia and digitalis therapy.
Below is presented normal ECG.

Anatomical relations of leads in a standard 12 lead ECG

- II, III, and aVF: inferior surface of the heart
- V1 to V4: anterior surface
- I, aVL, V5, and V6: lateral surface
- V1 and aVR: right atrium and cavity of left ventricle

ECG analysis: for better understanding of an ECG, it requires systematic/stepwise interpretation of the following parameters.

1. Heart rate
2. Rhythm
3. Axis
4. P-wave morphology
5. PR interval
6. QRS wave duration, voltage, morphology
7. ST segment morphology
8. T wave morphology
9. U wave
10. QT interval
**Heart rate**: normal sinus rhythm in resting rate is 60-100bpm. <60 is bradycardia &> 100 is tachycardia. When the rhythm is regular and the paper speed is running at the standard rate of 25 mm/s, the heart rate can be calculated by counting the number of large squares between two consecutive R waves, and dividing this number into 300. Alternatively, the number of small squares between two consecutive R waves may be divided into 1500.

When an irregular rhythm is present, the heart rate may be calculated from the rhythm strip (lead II).

The heart rate per minute can be calculated by counting the number of intervals between QRS complexes in 10 seconds (namely, 25 cm of recording paper) and multiplying by six. See the following example:

A standard rhythm strip is 25 cm long (that is, 10 seconds). The rate in this strip (showing an irregular rhythm with 21 intervals) is therefore 126 beats/min.

**Rhythm analysis**:

**Step 1** = locate p wave. Is p wave visible? What is the rate of p waves? What is the morphology and axis of p wave? Eg. absence of P waves may occur secondary to atrial fibrillation (see below)

**Step 2** = relationship between p & QRS

- Are P & QRS related in 1 : 1 fashion? Do all p waves precede QRS?

E.g. If there are more P waves than QRS complexes, then some form of AV block is present

**Step 3** = analyze QRS- morphology and duration; QRS regularity. Eg If the QRS complexes are of normal duration (<0.12 sec) and morphology, then the rhythm is supraventricular and if wide QRS complex, it is infra ventricular rhythm.
Brady arrhythmias:

- **Definition:** HR <60bpm, symptomatic usually when below 50bpm.

- **Causes**
  - Hypoxemia is commonest cause
    - Look for signs of respiratory distress, pulse oxymetry reading
  - Drugs eg. B- blockers
  - Electrolyte disturbances
  - Myocardial infarction
  - Conduction abnormalities …etc

- Immediate treatment is needed if bradycardia is the cause of unstable symptoms and signs

- **Classification of bradyarrhythmia**
  - Sinus bradycardia
  - AV block: 1\textsuperscript{st}, 2\textsuperscript{nd} and 3\textsuperscript{rd} degree

**Mx:** Atropine; Pacing (Transcutaneous or transvenous pacing)

- Alternative drugs (Dopamine(2-10mcg/kg/min and titrate to response); Epinephrine(2-10 mcg/min)

![Severe sinus bradycardia](image-url)
Adult Bradycardia (With Pulse)

1. Assess appropriateness for clinical condition. Heart rate typically <50/min if bradyarrhythmia.

2. Identify and treat underlying cause
   - Maintain patent airway; assist breathing as necessary
   - Oxygen (if hypoxemic)
   - Cardiac monitor to identify rhythm; monitor blood pressure and oximetry
   - IV access
   - 12-Lead ECG if available; don’t delay therapy

3. Persistent bradyarrhythmia causing:
   - Hypotension?
   - Acutely altered mental status?
   - Signs of shock?
   - Ischemic chest discomfort?
   - Acute heart failure?

4. Monitor and observe
   - No

5. Yes
   - Atropine
     If atropine ineffective:
     - Transcutaneous pacing OR
     - Dopamine infusion OR
     - Epinephrine infusion

6. Consider:
   - Expert consultation
   - Transvenous pacing

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Tachycardia

- Definition: HR > 100 bpm, more likely to be due to arrhythmia if ≥ 150 bpm
  - Rates < 150 are usually secondary to the underlying condition rather than the cause of the instability.
- Hypoxemia is a common cause
- Provide oxygen, fluid, and assess for signs of instability. If unstable, determine if that is primarily due to the rhythm disorder.

Tachy arrhythmias:

- Classification
  - Based on appearance of QRS, rate and regularity
    - Narrow QRS complex (<0.12 second) (supraventricular tachyarrhythmias)
      - Regular: Sinus tachycardia (upper limit calculation: 220-age), Supraventricular tachycardias
      - Irregular: Atrial fibrillation, Atrial Flutter, Multifocal atrial tachycardia
    - Wide QRS complex (≥0.12 sec)
      - Ventricular tachycardia, ventricular fibrillation, others

E.g. **Supraventricular tachycardia**

Irregular narrow complex tachycardia

- **Atrial Fibrillation** – note the irregular R-R intervals, and no distinct P wave

- **Atrial Flutter** – note the saw toothed appearance & regular rate
- **Wide QRS tachycardias**
  
  o **Monomorphic ventricular tachycardia**

  ![Monomorphic ventricular tachycardia](image)

  o **Ventricular Fibrillation**

  ![Ventricular Fibrillation](image)

**Mx of tachyarrhythmias:**

- **Regular narrow complex**
  - Sinus tachycardia: No specific treatment. Identify and treat underlying cause
  - Supraventricular tachycardia (Reentry SVT)
    - Vagal Maneuvers: Valsalva maneuver, carotid massage
    - Adenosine
    - Ca – channel blockers – Verapamil, diltiazem
    - Beta blockers

- **Irregular narrow complex**
  - Atrial fibrillation and flutter
    - Rate control: IV Beta blockers; non dihydropyridine Ca-channel blockers
    - For concomitant heart failure: digoxin, amiodarone
    - Rhythm control – electrical/pharmacologic
    - AF >48hr has a risk of cardioembolism. Therefore; electrical/pharmacologic cardioversion is reserved only for unstable patients

- **Wide complex**
  - Unstable: immediate defibrillation, precordial thump if defibrillator not ready
  - Stable: IV anti arrhythmics
Adult Tachycardia
(With Pulse)

1. Assess appropriateness for clinical condition. Heart rate typically ≥150/min if tachyarrhythmia.

2. Identify and treat underlying cause
   - Maintain patent airway; assist breathing as necessary
   - Oxygen (if hypoxic)
   - Cardiac monitor to identify rhythm; monitor blood pressure and oximetry

3. Persistent tachyarrhythmia causing:
   - Hypotension?
   - Acutely altered mental status?
   - Signs of shock?
   - Ischemic chest discomfort?
   - Acute heart failure?

4. Synchronized cardioversion
   - Consider sedation
   - If regular narrow complex, consider adenosine

5. Wide QRS? ≥0.12 second
   - Yes
     - IV access and 12-lead ECG if available
     - Consider adenosine only if regular and monomorphic
     - Consider antiarrhythmic infusion
     - Consider expert consultation
   - No

6. Consideration for synchronized cardioversion

Doses/Details

Synchronized Cardioversion
Initial recommended doses:
- Narrow regular: 50-100 J
- Narrow irregular: 120-200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (NOT synchronized)

Adenosine IV Dose:
First dose: 6 mg rapid IV push; follow with NS flush.
Second dose: 12 mg if required.

Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia

Procainamide IV Dose:
20-50 mg/min until arrhythmia suppressed, hypotension ensues, QRS duration increases >50%, or maximum dose 17 mg/kg given.
Maintenance infusion: 1-4 mg/min. Avoid if prolonged QT or CHF.

Amiodarone IV Dose:
First dose: 150 mg over 10 minutes. Repeat as needed if VT recurs.
Follow by maintenance infusion of 1 mg/min for first 6 hours.

Sotalol IV Dose:
100 mg (1.5 mg/kg) over 5 minutes. Avoid if prolonged QT.

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Chapter VI: Triage

Objectives
At the end of this session, a trainee will be capable
- Describe triage during different situations
- Develop knowledge on how to categorize and dispose emergency
- Patients during different situations
- Understand and practice on triage documentation

Triage, from the French word “trier”, literally means “to sort”. Is a method of ranking sick or injured people according to the severity of their sickness or injury in order to ensure that medical and nursing staff facilities are used most efficiently, and Patients with the greatest need are helped first.

Types

➢ Patient to triage: when a patient appears relatively stable and is able to mobilize him/herself to the designated triage area. This will be the type of triage used in most of the cases.

➢ Triage to patient: here the patient is usually unstable. The patient is unable to mobilize him/herself to the designated triage area and should be referred directly to the resuscitation room. Triage should be performed at the bedside and documented in retrospect. This type of triage is used in critical patients.

Benefits of triage- The aim of an efficient triage system is

1. To expedite the delivery of time-critical treatment for patients with life-threatening conditions,

2. To ensure that all people requiring emergency care are appropriately categorized according to their clinical condition,

3. To improve patient flow

4. To improve patient satisfaction

5. To decrease the patient’s overall length of stay

6. To facilitate streaming of less urgent patients

7. To be user-friendly for all levels of health care professionals.
Triage area

- Triage area is the first point of clinical contact for all people presenting to the Emergency Department and the point at which care/treatment begins. **Triage is a brief ABCD assessment that determines the clinical urgency of the patient’s presenting problem and concludes with the allocation of an emergency category, which determines the time and sequence in which they receive emergency care.**

Minimum standards for the triage practice and the triage environment are:

**Standard 1: Clinical practice**

The Role of the Triage Nurse is to:

1. Undertake patient assessment and allocate the Triage Score category based on;
   - a. Findings of the primary survey and
   - b. Risk assessment
2. Initiate appropriate nursing interventions
   - ✓ First aid and emergency interventions to improve patient outcomes
   - ✓ Secure the safety of patients and staff of the department;
   - ✓ Maintain patient privacy
3. Ensure continuous reassessment and management of patients who remain in the waiting room
4. Provide patient and public education where appropriate to facilitate
   - A. Health promotion and education
   - B. Injury prevention
   - C. Community information resource during multiple casualties
   - D. Act as the liaison for members of the public and other health care professionals

**Standard 2: Equipment and Environment**

- ✓ The triage environment must provide safety for the public, the triage nurse, staff and patients of the Emergency Department and the hospital.
a. Must be immediately accessible and well sign posted

b. Must have access to an area for patient examination and primary treatment

c. Must be designed to maximize the safety of the triage nurse, staff and Patients

(e.g. threat alarms, access to security personnel)

- Must be equipped with emergency and protective equipment
- Air way equipment,
- Thermometer, Electronic/manual blood pressure & pulse analyze
- Pulseoximetry, ECG, Dry dressings/ bandages,
- Finger prick gluoses test& finger prick hemoglobin, Urine dipsticks & urine pregnancy tests
- Gloves, face masks & other barrier protective devices,
- Emergency drugs such as: Diazepam, Adrenalin, Atropine, 40%Glucose

d. Must enable to provide care for infection control and prevention

e. Should enable and facilitate patient privacy

Triage Scale (TS) or Emergent Severity Index (ESI)

ESI 1- (RED) - Immediately life threatening ----> disposition -->Resuscitation
E.g. ABCD unstable patients, Respiratory, facial, neck, chest injuries, severe hemorrhage, neck injuries, shock, coma with signs of airway obstruction, severe respiratory distress, convulsions, chest pain with unstable Vital Signs,

ESI 2 - (orange) - potentially life threatening --> to resuscitation WITHIN 10-15MIN
If care is not given within 10minutes(pending respiratory failure, altered consciousness without airway obstruction, moderate trauma with stable vital signs, such patients require frequent re-triage until they are seen by the physician and they are the second priority following the red and if any deterioration appears they may be re-categorized accordingly

ESI 3- (Yellow)- less urgent potentially serious, could be delayed up to 60minutes --> waiting area

E.g. Injuries to the Lower genitourinary tract, splinted fractures, soft tissue lesions, they also require re- triaging until they are assessed by the physician and if any deterioration appears they might be re-categorized accordingly

ESI 4- (Green)- non urgent, can be delayed up to 240minutes and can be sent to nearby health institution, regular OPDs, after counseling or 1st aid
Organization and Sequence of patient evaluation (triage)

1. The triage nurse should be available at the triage area at all times.

2. The triage nurse should organize her/his working area with necessary supplies before the start of her/his shift (self-protecting devices, airway equipment’s, bag valve mask, oxygen with accessories, materials to control bleeding, Diazepam, 40% glucose, ..), this is important especially when the resuscitation room is busy with other patients.

3. The triage nurse needs to be attentive to pick up the critical patient on arrival.

4. All unconscious patients should be evaluated for ABCD before any history and vital signs taken.

5. Critical patients can be transferred immediately to the resuscitation area while the triage nurses can do their triage documentation at bed side.

6. Whenever >5 patients are coming to the ER the coordinator should assign more than one triage personnel.

7. Whenever critical patient or injured patient is evaluated complete undressing is important to minimize pitfall.

8. During assessment/ triage of critical patients conduct primary assessment which includes: ABCD, vital signs, short history about the course of illness or mechanism of injury (see primary assessment protocol).

9. After the evaluation score the patient’s condition using the Triage Early warning Score (TEWS), Table 1.

10. Then add up your findings and categorize the patient according to the Emergent Severity Index (ESI Table 2).

11. According the color code distribute patients to the respective treatment/assessment area.
Table 1 TEWS: Triage Early Warning Score.

<table>
<thead>
<tr>
<th>ADULT TRIAGE SCORE</th>
<th>3</th>
<th>2</th>
<th>1</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mobility</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RR</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AVPU</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trauma</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
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<td></td>
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<td>0</td>
<td></td>
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<tr>
<td>1</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Mobility

RR

HR

SBP

Temp

AVPU

Trauma

over 12 years / taller than 150cm
### Severity Index (ESI Table 2)

<table>
<thead>
<tr>
<th>Colour</th>
<th>RED</th>
<th>ORANGE</th>
<th>YELLOW</th>
<th>GREEN</th>
<th>BLUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>TEWS</td>
<td>7 or more</td>
<td>less than 10 mins</td>
<td>less than 60 mins</td>
<td>less than 240 mins</td>
<td>DEAD</td>
</tr>
<tr>
<td>Target time to treat</td>
<td>Immediate</td>
<td>High energy transfer</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mechanism of injury</td>
<td>Shortness of breath - acute</td>
<td>Coughing blood</td>
<td>Chest pain</td>
<td>Haemorrhage - uncontrolled</td>
<td>Haemorrhage - controlled</td>
</tr>
<tr>
<td>Seizure - current</td>
<td>Seizure - post ictal</td>
<td>Focal neurology - acute</td>
<td>Level of consciousness reduced</td>
<td>Psychosis / Aggression</td>
<td>ALL OTHER PATIENTS</td>
</tr>
<tr>
<td>Presentation</td>
<td>Threatened limb</td>
<td>Dislocation - other joint</td>
<td>Fracture - compound</td>
<td>Fracture - closed</td>
<td>DEAD</td>
</tr>
<tr>
<td>Burn - face / inhalation</td>
<td>Burn over 20%</td>
<td>Burn - electrical</td>
<td>Burn - circumferential</td>
<td>Burns - other</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Burn - chemical</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypoglycaemia - glucose less than 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>Severe</td>
<td>Moderate</td>
<td>Mild</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Example:**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Temperature</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>With help — scores 1</td>
<td>36.5</td>
<td>Orange</td>
</tr>
<tr>
<td>Respiratory rate = 18</td>
<td>Patient Alert = scores 0</td>
<td></td>
</tr>
<tr>
<td>Heart rate = 118</td>
<td>No Trauma = Scores 0</td>
<td></td>
</tr>
<tr>
<td>BP = 208/112</td>
<td>scores 2</td>
<td></td>
</tr>
</tbody>
</table>

**Activities**

- Discussion on case scenario # 4
- Simulation & practice in small group
- General discussion on questions raised by participants
Chapter VII: Approach to the Management of Common Medical Emergencies

- Duration - 6 hrs

Respiratory emergencies

Learning Objectives

After reading this chapter, participants should be able to

- List life threatening causes of respiratory distress
- Describe initial approach to respiratory distress
- Describe initial management plan for a patient in respiratory distress
- Describe classification and initial approach to asthma
- Describe approach and treatment of pneumonia

Approach to a patient in respiratory distress

Introduction

Patients in respiratory distress require rapid but careful assessment. Initiation of life saving treatment precedes definitive diagnosis. The aim is to stabilize the patient and prevent further deterioration.

Primary/ Initial assessment

- Observe the general appearance of the patient- is patient alert, responsive, anxious, drowsy, check if patient is diaphoretic, pale, grey, signs of dehydration

- Airway:
  - look for foreign bodies obstructing the airway, listen for stridor
  - management
    - Oral/nasopharyngeal airway, head tilt/jaw thrust,
    - Foreign body sweep if visualized
    - Prepare for advanced airway management if necessary

- Breathing:
  - Check respiratory pattern and rate, accessory muscle use and supra clavicular retractions, cyanosis, ability to speak without difficulty (talking in sentences/words)
  - Management-
    - Administer oxygen
- Put patient on pulsoximeter – monitor oxygen saturation, target saturation is 92% and above.
- Use Bag valve mask- for apnea, low respiratory rate, extreme tachypnea, poor saturation
- Prepare for intubation if necessary

- **Circulation** –
  - Check pulse rate and quality, measure blood pressure
  - Auscultate – heart sounds, murmurs
  - **Management**
    - Put patient on cardiac and BP monitor
    - Establish IV access

**Secondary assessment**

Following primary assessment and correction of any immediate life threats, focus should be directed toward identifying underlying causes

- History and physical exam
- Do ECG, get urgent chest x-ray

**Differential diagnosis**

- Asthma / Chronic Obstructive Pulmonary Diseases/COPD/exacerbation
- Acute coronary syndrome
- Pulmonary edema
- Pneumonia
- Pulmonary embolism
- Tension pneumothorax
- Pericardial tamponade
- Anaphylaxis
- Upper airway obstruction

**Asthma**

**Introduction**

Asthma is a chronic inflammatory condition of the airways resulting in hyper responsiveness of the airways to various stimuli. This leads to excessive narrowing of the airways with reduced airflow and symptoms of dyspnea and wheezing.
Triggering factors

Several stimuli trigger airway narrowing, wheezing, and dyspnea in asthmatic patients

- Allergens, upper respiratory tract infections, exercise and hyperventilation, chest infections (viral or bacterial), cold air, irritant gases, sulfer dioxide, drugs ( B blockers, aspirin ), Stress, irritants - household sprays, paint fumes
- No clear precipitating factor is identified in over 30% of patients

Risk factors for severe Asthma

Factors increasing the risk of severe life-threatening asthma include

- previous ventilation
- hospital admission for asthma in the last year
- heavy rescue medication use
- >3 classes of asthma medication
- repeated attendances at emergency room for asthma care

Presentation

- Characteristic symptoms are dyspnea, cough productive of whitish sputum, chest tightness and wheezing
- Acute attacks may build up over minutes, hours, or days and the patients may deteriorate very rapidly and present as respiratory or cardio-respiratory arrest

Initial assessment

✔ Patients presenting with an asthma attack may be at imminent risk of death
✔ Rapid and accurate assessment is vital

1. Assess for signs of imminent respiratory arrest. If present start treatment immediately
   ❖ Characteristics defining a patient in imminent arrest
     i. Unable to walk
     ii. Drowsy or confused
     iii. Has paradoxical chest movements
     iv. No wheezing
     v. Bradycardia
     vi. (Unable to perform PEF measurement)

2. If no signs of imminent arrest, assess for signs of clinical distress
3. If the patient is not in imminent arrest, proceed with assessment and treatment

**Classification of severity of an asthma attack**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Imminent respiratory arrest</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathless</td>
<td>Walking</td>
<td>Talking</td>
<td>At rest</td>
<td>Hunched forward</td>
</tr>
<tr>
<td></td>
<td>Can lie down</td>
<td>Prefers to sit up</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Talks in</td>
<td>Sentences</td>
<td>Phrases</td>
<td>Words</td>
<td>Unable to speak</td>
</tr>
<tr>
<td>Alertness</td>
<td>May be agitated</td>
<td>Usually agitated</td>
<td>Always agitated</td>
<td>Drowsy or confused</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>increased</td>
<td>increased</td>
<td>Often &gt; 30/min</td>
<td></td>
</tr>
<tr>
<td>Accessory muscles and</td>
<td>Usually not</td>
<td>usually</td>
<td>usually</td>
<td>Paradoxical thoracic and</td>
</tr>
<tr>
<td>suprasternal retractions</td>
<td></td>
<td></td>
<td></td>
<td>abdominal movements</td>
</tr>
<tr>
<td>Wheeze</td>
<td>Moderate,</td>
<td>Loud</td>
<td>Usually loud</td>
<td>Absence of wheeze</td>
</tr>
<tr>
<td></td>
<td>often only end-expiratory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulse rate</td>
<td>100</td>
<td>100-120</td>
<td>&gt;120</td>
<td>Bradycardia</td>
</tr>
<tr>
<td>PEF after inhalation of</td>
<td>≥80%</td>
<td>60-79%</td>
<td>&lt;60% or (&lt;100/l/min in adults)</td>
<td>Impossible to measure</td>
</tr>
<tr>
<td>salbutamol</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Where signs from several grades of severity are present, the highest grade of severity is used to classify the attack, even if not all the signs for that grade are present

**Acute severe asthma: Immediate therapy**

Priorities of treatment

- Treat hypoxia
- Treat bronchospasm and inflammation
- Assess need for intensive care
- Treat any underlying cause if present
Severe or life threatening attack

Initial treatment

- Oxygen – the highest percentage available
  - Maintain O2 saturation > 92%
- Bronchodilators
  - SABA- Short acting beta agonist
    - Salbutamol/Albuterol
      - Puff: 4-8 puffs Q 20 min for up to 4 hrs, then Q 1-4 hrs as needed
    - Technique of salbutamol puff
      - Test the inhaler: shake well and release one puff into the air
      - Breathe out gently & place the mouth piece in the mouth and close lips around it
      - Tilt head slightly backwards, breathe in slowly and press down the canister to release one dose
      - Remove the inhaler and hold breath for 10 seconds and breathe out slowly
    - Nebulization: 2.5-5mg every 20 mins for 3 doses then 2.5-10 mg Q 1-4 hrs as needed
    - MDI - 4 puffs every 10 mins, 8 puffs every 20 mins
      - Add Ipratropium bromide 0.5 mg 4-6 hourly if initial response to B agonist is poor
        - 500 mcg via nebulizer every 20 minutes for three doses then as needed
- Obtain IV access
- Start steroids
  - Hydrocortisone 200 mg IV, continue with either hydrocortisone 100 mg QID IV or prednisolone 30-50mg Po daily. (IV steroid treatment is not more effective than oral treatment)
- If no improvement
  - Add magnesium sulfate 2 gm administered over 20 minutes or
  - Aminophilline
    - Loading dose- 5mg/kg or 250 mg over 20 minutes (dilute with IV fluid to a concentration of 1mg/ml) followed by continuous infusion
    - Do not give loading dose in patients taking oral theophylline
- Maintenance dose- 0.5mg/kg/hr Q 12 hr (increase dose in smokers & decrease in elderly, corpulmonale, CHF& liver failure)
  - Adrenaline -0.3-0.5 mg (1:1000 solution) Q 20 min for 3 doses subcutaneously
- Antibiotics – only given if there is evidence of chest infection (fever, purulent sputum, abnormal CXR, raised WBC count )
- Adequate hydration –essential and may help prevent mucus plugging.

**Management of moderate attack**

- Salbutamol 4-8 puffs every 20 minutes for the first hour
- Oral prednisolone =40-60mg Po per day for 5-10 days (no tapering)
- The patient is then reassessed
  - Complete response –disappearance of clinical signs (and PEF> 80%)
    - The patient is kept for one more hour. If stable, the patient can be discharged to continue treatment at home.
  - No response/incomplete response – no or incomplete disappearance of clinical signs (or PEF<80%)
    - After the first or second hour, the patient should be treated as for a severe attack and be kept in the emergency room for at least 6 hours to continue treatment.

**Management of mild attack**

- Salbutamol 2-4 puffs every 20 minutes for the first hour
- The patient is then reassessed
  - Complete response – disappearance of clinical signs (and PEF≥ 80%)
    - Patient is kept for one more hour. If stable, the patient is discharged to continue treatment at home.
  - No response/incomplete response – no or incomplete disappearance of clinical signs (or PEF<80%)
    - After the first or second hour, the patient should be treated as for moderate attack.

**Assessment of response**

**Clinical improvement**

- Patient is less distressed
- Decreased respiratory rate and heart rate
- Able to talk in sentences
- Louder breath sounds on auscultation (may be more wheeze)

Pulse oximeter- aim O2 saturation of 94-98%
Monitor heart rate and Oxygen saturation continuously and measure BP frequently
**Pneumonia**

**Definition:** Pneumonia is an acute infection of the pulmonary parenchyma

**Diagnosis**

- **Symptoms**
  - Cough, either nonproductive or productive of mucoid, purulent, or blood-tinged sputum.
  - Pleuritic chest pain.
  - Dyspnea - Depending on severity, the patient may be able to speak in full sentences or may be very short of breath
  - Fever, chills and/or sweats.
  - Fatigue, headache, myalgia, and arthralgia.
    - Atypical presentation in elderly and immunocompromised
- **Signs**
  - Tachypnea, tachycardia, use of accessory muscles of respiration, cyanosis
  - Focal signs – dullness, crackles, bronchial breathing, pleural rub

Community acquired pneumonia/CAP/ can vary from indolent to fulminant in presentation and from mild to fatal in severity.

**Likely underlying cause**

<table>
<thead>
<tr>
<th>Type</th>
<th>Organism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical</td>
<td>S. Pneumonia</td>
</tr>
<tr>
<td></td>
<td>H. Influenza</td>
</tr>
<tr>
<td></td>
<td>Influenza virus</td>
</tr>
<tr>
<td></td>
<td>S. Aureus</td>
</tr>
<tr>
<td></td>
<td>Gram negatives ex Klebsiella</td>
</tr>
<tr>
<td>Atypical</td>
<td>M Pneumonae</td>
</tr>
<tr>
<td></td>
<td>C. Psittica</td>
</tr>
<tr>
<td></td>
<td>L. Pneumophilia</td>
</tr>
</tbody>
</table>

**Assessment of severity**

**CURB -65**

- C-New onset confusion
- U-Urea >7mmol/L
- R-Respiratory rate >30/min
- B-SBP <90mmHg and/or DBP<60mmHg.
- Age > 65 years
  - Patients may be treated in an outpatient setting or may require hospitalization according to their CURB 65 score-score of 0-1, outpatient treatment, score of 2-admit to medical ward, score of 3 or greater-admit to ICU
Additional features

- Hypoxia (Saturation <92%)
- Co morbidity
- WBC <4,000 or >20,000 or bilateral or multi-lobar involvement on CXR

Management of pneumonia:

General resuscitation and investigations

- Check ABC (airway, breathing, and circulation).
- If patient is in respiratory distress, give oxygen
- Secure venous access: if there are signs of dehydration, start Iv crystalloids; examine regularly for signs of fluid overload.
- Send blood: CBC, OFTs
- Monitor O2 saturation.
- Arrange for urgent CXR.
- Culture blood and sputum: Urgent sputum microscopy with Gram-stain is occasionally useful but should not be relied upon routinely.
- Pain relief: Paracetamol or a NSAID usually suffice.

Initial antibiotic Management

Initial therapy is usually empirical and is designed to cover the most likely pathogens. In all cases, antibiotic treatment should be initiated as expeditiously as possible

- Modify therapy in the light of subsequent investigations or positive cultures.
- Start on iv therapy for at least 48 hours; adjust according to clinical condition and response

Choice of antibiotics:

1. Outpatients

- **Previously healthy and no antibiotics in the past three months**
  - Clarithromycin (500 mg PO bid) or Azithromycin (500 mg PO once, then 250 mg qd)

- **Co morbidities or antibiotics in the last three months**
  - high-dose amoxicillin (1 g tid) or amoxicillin/clavulanate (2 g bid);
  - alternatives: ceftriaxone (1–2 g IV qd), cefuroxime (500 mg PO bid)] plus a macrolide
2. Inpatient – non ICU
   - Ceftriaxone (1–2 g IV qd), Cefotaxime (1–2 g IV q8h), Ampicillin (1–2 g IV q4–6h), plus a macrolide(oral clarithromycin or azithromycin)

3. Inpatients, ICU
   - Cefotaxime (1–2 g IV q8h), Ceftriaxone (2 g IV qd) plus Azithromycin
     - Monitor response to therapy with
       o Clinical parameters
       o CBC
       o Pulse oximetry or blood gases
       o CXR: if deteriorating sooner at day 3 & 5.
   - Total duration of therapy usually is 7-10 days

Activities
   - Discussion on case scenario # 5
   - General discussion on questions raised by participants
Common abnormalities of circulation

Learning Objectives
After the end of this session, you will be able to:

- List components of assessment of the circulatory system
- List common circulatory abnormalities
- Describe the approach to a patient with heart failure
- List underlying causes, and precipitating factors for heart failure
- List the management principles of heart failure
- Outline the management principles of pulmonary edema
- Define hypertensive emergency, urgency and understand their differences
- Outline the management approach differences between hypertensive urgency and emergency
- Distinguish ischemic chest pain from non ischemic causes
- Diagnose acute coronary syndrome promptly
- Manage ACS timely

Introduction
The circulatory system is an essential component of our body’s homeostasis. The assessment of the circulatory system includes taking appropriate history, doing physical examination, ordering laboratory tests to reach at a diagnosis, and doing the appropriate interventions to manage the identified problem. The order of doing these critical steps depends on the emergency situation. At times, one could be forced to do quick physical examination and decision on management while taking a short history.

Common circulatory system disorders are heart failure and pulmonary edema, cardiac arrest, hypertensive crises, acute coronary syndrome, and shock. These topics will be described in detail later in this chapter.
Evaluation of the patient:

1. The primary survey
   a. General appearance

   The following questions should be answered.
   - Is the patient acutely sick looking?
     o Features suggestive include patient in pain, signs of cardiorespiratory distress, patient with change in mentation
   - Is the patient in cardiorespiratory distress?
     o Signs of cardiorespiratory distress include: cyanosis (central & peripheral), flaring of ala nasi, intercostal retraction, neck vein distension tachypnea, tachycardia, unable to take recumbent position.
   - Patients in distress should be initiated on immediate measures aimed at stabilizing the patient.

b. Vital signs
   1. **Blood pressure**:
      - Techniques
        o Use appropriate cuff size (length 80% & width 40% of arm circumference)
          ▪ Larger cuff lowers and smaller cuff exaggerates BP
        o Preferably in seated/ supine position with arms supported at the heart level
        o Best after 5-10 minutes of rest
        o Both arms should be measured and compared ( difference >10mmHg is abnormal)
      - Evaluate for orthostatic hypotension
        o Defined as a fall in blood pressure of ≥ 20/10 mmHg within 3 minutes of standing from an initial supine position.)
        o It indicates the presence of fluid deficit or can be seen in some diseases like diabetes.
      - Normal blood pressure: systolic 90 – 140 mm Hg and diastolic 60-90 mm Hg.
        o Hypotension : BP <90/60 mm Hg,
        o Hypertension : BP ≥140/90mmHg on two different occasions
      - Correlate blood pressure findings with pulse rate.

   2. **Pulse rate**
      - Normal range: 60 – 90 beats per minute.
      - It is preferably recorded at the radial site. There are situations where one is unable to feel the radial pulse and can measure at brachial, carotid or from the precordium.
- Assess for pulse volume and rhythm.
  - Common volume abnormalities: feeble pulse (barely palpable pulse eg. During shock, heart failure), bounding pulse (a very strong pulse eg. In anemia, thyrotoxicosis, and pregnancy).
  - The rhythm pattern should be assessed if it is regular or irregular (regularly irregular, or irregularly irregular).
    - The commonest arrhythmia in our setup is atrial fibrillation and it presents as irregularly irregular rhythm.

3. **The other vital signs** – Respiratory rate and temperature, oxygen saturation are also vital in the assessment of the circulatory system. (see general assessment of the emergency patient.)

Check for capillary refill; attach pulsoximetry and cardiac monitor if available.
Timely intervention based on the identified problems in a prioritized fashion has a significant impact in reducing morbidity and mortality.

2. **The Secondary Survey**
   - This follows stabilization of the patient and includes taking detailed history and physical examination to reach at a diagnosis

**Heart Failure and Pulmonary Edema**

**Heart Failure**
Acute decompensated Heart failure is one of the common emergency department presentations of patients with circulatory system affection. It is part of the spectrum of the progressive, complex clinical syndrome of heart failure resulting from structural and/or functional cardiac disorders that impair systolic and/or diastolic function. The body’s neurohumoral system tries to compensate for this state through various compensatory mechanisms initially beneficial but later on ending up in deleterious effects. It leads to fluid buildup in the lungs, liver, gastrointestinal tract, and the limbs and weight gain.

Heart failure can be broadly categorized as:

- **systolic heart failure** (occurring when the heart is unable to pump blood)
  - Causes: valvular heart disease, dilated cardiomyopathy, Ischemic heart disease, hypertensive heart disease.

- **Diastolic heart failure** (when the heart muscles are very much stiff and prevent filling of the heart, but the contractility remains to be normal).
  - Causes: Hypertrophic cardiomyopathy, Restrictive cardiomyopathy, chronic hypertension, Ischemic heart disease, diabetes.
Another schema for classifying heart failure patients is as right or left sided heart failure.

- **Right sided heart failure** presents with bilateral leg swelling, ascites, and hepatomegaly
- **Left side heart failure** presents with pulmonary edema. In general they occur together though they could as well present separately.

**Etiologies of heart failure**

- **Heart failure with depressed Ejection Fraction**
  - Coronary heart diseases
  - Chronic pressure overload
  - Hypertension
  - Valvular obstruction
  - Chronic volume overload
  - Valve Regurgitation
  - Dilated cardiomyopathy
  - Toxic/drug induced
  - Viral

- **Heart failure with preserved Ejection Fraction**
  - Hypertrophic cardiomyopathy
  - Hypertension
  - Restrictive cardiomyopathy

- **Pulmonary heart diseases**
  - Cor pulmonale

- **High output states**
  - Chronic anemia
  - Thyrotoxicosis

• Common presentation of patients with heart failure includes:
  - Progressive dyspnea, orthopnea (shortness of breath in lying position),
  - Palpitation, Paroxysmal nocturnal dyspnea (waking up from sleep due to severe shortness of breath)
  - Swelling of the body starting from the feet progressing upwards, swollen abdomen,
  - Cough productive of pink frothy sputum,
  - Fatigue, weakness, angina, syncope.

• Other diseases could present in a similar ways and should be considered in the differential.
• Look for evidences of other comorbidities
  o E.g. Diabetes, hypertension, dyslipidemia, chronic kidney disease

• Look for evidence and severity of complications
  - Pulmonary edema, cardiogenic shock, acute renal failure

The severity of the dyspnea and other symptoms should be graded by the **New York Heart Association** (NYHA) classification.
- Class I: symptoms* elicited only at levels of exertion that would limit normal individuals
- Class II: symptoms elicited at ordinary exertion
- Class III: symptoms elicited on less than ordinary exertion
- Class IV: symptoms elicited at rest

  *symptoms: dyspnea/ Fatigue / palpitation / angina pain

**Physical examination**

- Evaluate for any evidence of cardiorespiratory distress as part of the primary survey
- Look for evidences of reduced cardiac output
  o Diaphoresis, Resting tachycardia, narrow pulse pressure (<25mmHg), cool, pale and cyanotic limbs, delayed capillary refill (>2 sec)
- Vital signs
- Neck for any neck vein distention,
- chest - lower lung field crepitation, evidence of pleural effusion
- CVS – S3 gallop, murmurs
- Abdomen – Right upper quadrant tenderness and hepatomegaly, any sign of fluid collection (shifting dullness, fluid thrill)
- Musculoskeletal – pitting edema in limbs, sacral area for bed ridden patient

Through a quick history and physical examination, we should look for evidences for possible underlying cause and precipitating factor for the heart failure.

- **The underlying cause:** Imagine the anatomy of the heart and which structures could be affected to lead to heart failure. Common underlying causes of heart failure include:
  o **Valvular heart disease** – commonest cause of heart disease in our set up, with the affection of the different valves of the heart, either alone or in combination
  o **The cardiomyopathies** – hypertrophic, dilated, restrictive cardiomyopathies
Ischemic heart disease – due to narrowing of small blood vessels of the heart (coronary vessels) as a result of atherosclerosis. This results in compromised blood and oxygen supply to the heart.

Congenital heart disease
- Examples: Atrial septal defect, Ventricular septal defect etc.

Pericardial diseases – Effusive pericarditis, constrictive pericarditis
- Viral pericarditis, TB pericarditis

Precipitating Factors: Common precipitating factors include anemia, thyrotoxicosis, arrhythmia, drug discontinuation, salt intake, infection, Spontaneous bacterial endocarditis, uncontrolled hypertension, acute myocardial infarction, and other drugs.

Patients with a specific underlying cause of heart failure may remain asymptomatic for a long period of time until the natural course of the disease or a precipitating cause unmasks it to become decompensated. Identifying what precipitated the heart failure is an essential step later guiding the management of the patient.

Patients with acute heart failure can be classified into four based on status of perfusion and congestion.

Work up
The following investigations can be ordered to reach a diagnosis. Work up should be individualized.
- CBC, Urinalysis, Renal function test
- CXR, ECG, Echocardiography
- Serum electrolytes (potassium, sodium)
- Other tests depending on the patient’s presentation

A quick emergency ultrasound evaluation could help to evaluate left ventricular contractility, Inferior venacaval distension and change of size with respiratory
movement, and pericardial effusion. Detail Echocardiography can be sent after patient stabilizing.

Due to issues of availability of detailed echocardiographic evaluation, the emergency practitioner needs to develop his/her skills of physical examination and performing emergency ultrasound.

Management
Goals of management:

- Establish Diagnosis, etiology and precipitating factors
- Treat life threatening abnormalities eg. Oxygenation, hemodynamic stability
- Initiate therapy to rapidly provide symptom relief – revert them back to profile A

In the acute setting, management of systolic and diastolic heart failure share similar properties except for minor differences like requirement of inotropic drug in systolic failure as opposed to diastolic failure.

The general principles of management of heart failure are mentioned here below:

1. Decision on Hospital admission
   Decision on admission should be made on individual basis considering different factors. A general guide is presented here but the emergency practitioners’ clinical judgment comes first.
   - Priority of admission should be given to the following sub groups
     - Evidence of severely decompensated heart failure
       - Hypotension, worsening renal function, Altered mentation
     - Dyspnea at rest
     - Hemodynamically significant arrhythmia eg. Atrial fibrillation with rapid ventricular response
     - Acute coronary syndromes
   - A second subgroup for whom admission is recommended include
     - Worsened congestion despite oral treatment even if without dyspnea
     - Electrolyte disturbance
     - Concomitant comorbidities eg. Pneumonia, DKA, stroke etc.
     - Newly diagnosed patient with congestion

2. Initial stabilization
   - Assessment of the airway, oxygenation and prescription of oxygen
     - Oxygen support
- A decision should be made based on clinical clues of hypoxia (respiratory distress, cyanosis) and oxygen saturation (<90%). Routine supplemental oxygen in those with normal oxygen saturation is not recommended.

- Put the patient on Continuous cardiac monitoring
- Elevate the head of the bed to improve comfort
- Salt restriction <2g sodium / day
  - This can generally be achieved by avoiding the addition of table salt.
- Fluid restriction (<2L/d) though not routinely employed for milder forms of heart failure remains an essential component of the management of patients with concomitant heart failure and hyponatremia and in pulmonary edema.
  - IV fluids in general should be withheld from such patients

3. **Addressing symptoms and signs related to congestion or low perfusion**
   a) **Diuretics**
      - A Potent diuretic, preferably parenteral loop diuretic should be initiated early to manage the fluid overload state
      - Why IV route preferred over oral route?
        - PO drugs have reduced absorption due to edema of gastrointestinal tract.
      - Dosing: The dosage of the loop diuretics eg. Furosemide is decided based on
        - the patients’ urine output response and
        - The urgency of alleviating the congestion.
          - Unlike Peripheral congestion (ascites, limb swelling) where slow diuresis is recommended, early aggressive diuresis in patients with pulmonary edema saves lives.
          - Usual starting dose for Furosemide is 40mg IV and should be escalated (doubled) every hour till one finds the dose that produces adequate urine (>0.5ml/kg/hr). That dose can be given on a twice or three times a day doses.
      - If no adequate response to the loop diuretic alone
        - Continuous IV infusion of the loop diuretic.
        - Add a thiazide diuretic prior to the loop diuretic dose to further potentiate the action of the loop diuretic
          - Monitor for hypotension, worsening renal function, electrolyte abnormalities
      - Add potassium PO or a potassium sparing diuretic or spironolactone to prevent hypokalemia.
b) Vasodilators
   - Nitrates
     - Improve pulmonary congestion, ↑ coronary blood flow, ↓ afterload
     - Nitroglycerin – 20mg/min with 20mg/min increment every 5-15 minutes
     - Target Mean arterial pressure reduction of 10mm Hg, with systolic blood pressure remaining above 100mm Hg
     - Unable to use after 24 hrs due to tolerance
     - S/E : Headache, hypotension
   - Other vasodilators : Isosorbide dinitrate, Nitroprusside

   - Some medications to treat chronic heart failure like Beta Blockers, and ACEI’s have deleterious effect in the acute setting and should be discontinued or used very cautiously.
   - NSAIDS should be avoided - they reduce efficacy of diuretics
   - Approach to management of cardiogenic shock is discussed under the topic shock.
     - Inotropic agents like Dobutamine, Dopamine are used.
   - The approach of management of pulmonary edema is discussed separately later in this chapter.

4. Identification and management of precipitating factors
   - Eg. In atrial fibrillation with fast ventricular response, Digoxin or low dose Beta blockers could be used for rate control. Cardioversion can be attempted for those with new onset atrial fibrillation with hemodynamic instability.
   - Antibiotics for infection, etc.

5. Monitoring
   - Close patient monitoring is very important for optimal outcome. Important parameters to follow are :
     - Vital signs – frequently depending on the severity of the heart failure. Eg. A patient with cardiogenic pulmonary edema might need frequent V/S monitoring (every 30 minutes) to adjust the diuretic dose
     - Monitoring for optimal diuresis
       - Assessment of urine volume
       - Daily weight measurement – better done with the same weight scale, at a fixed time during the day to make comparisons
       - Assessing for resolution of edema – marking the upper border of the crepitation in the lung helps to assess the response to diuretics.
       - Monitoring BUN, Cr, vital signs
o Renal function test, serum electrolytes (especially potassium if high
dose diuretics are being used) – this can be done daily or even more
frequently on individualized basis.
o Others depending on the specific underlying cause of heart failure
o Monitoring for drug side effects
  • Loop diuretics, Thiazide diuretics
  • Worsening renal function, hypokalemia, hypotension, hyponatremia, hypomagnesemia

- A flow sheet should be prepared for use including the most important
parameters to be followed. A sample flow sheet is presented as follows:

<table>
<thead>
<tr>
<th>Date</th>
<th>Vital signs</th>
<th>Mental status</th>
<th>Input</th>
<th>Output</th>
<th>Insensible loss</th>
<th>Diuretic dose</th>
<th>BUN Cr</th>
<th>Daily Weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>PR</td>
<td>BP</td>
<td>T°C</td>
<td>RR</td>
<td>Sao2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

6. Management of the underlying cause

7. Discharge and Enroll to chronic care for Mx following principles of chronic heart failure Mx

Pulmonary edema
Pulmonary edema occurs due to fluid leak into the interstitium of the lungs and
alveoli during severe heart failure, most frequently in left sided heart failure. Patients
have profound dyspnea and orthopnea, hemoptysis. It is a life threatening situation
and timely intervention has a great impact on the outcome.

Auscultatory findings are fine crepitations more prominent in the lower part of the
chest. The level of the upper border of the crepitation should be marked to assess for
the patients response to our interventions.

At times non-cardiac sources of pulmonary edema might mimic cardiogenic
pulmonary edema. They occur in situations like severe pneumonia and do not respond
to the measures done for heart failure. The role of history and physical examination
cannot be over emphasized to differentiate them.

Chest X ray feature : bilateral, perihilar, bat wing (butterfly) shaped interstitial
infiltrates more prominent in lower lung fields.

Management
- General Measures
  - Admit to the emergency room
  - Immediately administer oxygen via appropriate route
  - Keep the patient in a propped up position with the legs dangling
  - Morphine 2-4mg IV boluses for reduction of preload and anxiety

- Urgent diuresis
  - Parenteral loop diuretics: Furosemide are an ideal choice (venodilator reducing preload in addition to its diuretic effect)
  - Use frequent dosing provided that blood pressure, renal function permit
  - There is no standing dose of furosemide that is universal for every patient and decision on frequency and dosage must be individualized.
  - Furosemide can be started at <0.5mg/kg or 40 mg IV, with 40 mg increments every hour. The dose that gives urine output ≥0.5ml/kg/hr should be used frequently (every hour) with adequate monitoring. The maximum recommended dose of Furosemide as a single IV bolus is 160-200 mg.
  - In very severe pulmonary edema not responding to the above measures, the following measures can be taken
    - Addition of a thiazide diuretic eg. Hydrochlorothiazide
    - Use a perfuser to administer continuous Lasix infusion at 10-40mg/hr. If the urine output remains below 1ml/kg/hr, the infusion rate can be increased each hour as necessary till a maximum dose of 80-160 mg/hr.

- Vasodilators
  - Nitrates
    - Sublingual Nitroglycerin (0.4 mg x 3 every 5 min) – first line for cardiogenic pulmonary edema
    - If pulmonary edema persists and no evidence of shock
      - IV Nitroglycerin 5-10 microgrm/min infusion

- Monitoring
  - Resolution of symptoms (dyspnea, orthopnea)
  - Vital signs, saturation
  - Mark the level of upper border of crepitation to see for resolution of lung congestion
  - BUN, Cr
  - Serum potassium – hypokalemia is an important adverse effect of Furosemide.
    - Start oral Potassium chloride, or spironolactone to prevent hypokalemia

- Management of underlying cause and precipitating factor for the heart failure
Hypertensive Crises

Hypertension is among the leading causes of cardiovascular morbidity and mortality. It is often associated with other cardiovascular risk factors. In a majority of cases it is asymptomatic and remains undiagnosed presenting with one or more of the chronic or acute complications.

**Definition:** The level of blood pressure at which the institution of therapy reduces blood pressure related morbidity and mortality. It has been defined as a blood pressure $\geq 140/90$ mm Hg on two different visits.

Hypertensive emergency and urgency are common presentations of hypertension in our emergency departments due to the lack of awareness of the general population and poor drug compliance. It is important to differentiate among these two conditions. The degree of end organ damage determines the rapidity of lowering the blood pressure because precipitous lowering of blood pressure has deleterious effects. The brain has an auto regulatory mechanism which allows it to adapt to a wide range of blood pressures. This auto regulatory set point is shifted to a higher level in patients having hypertension in attempt to maintain normal cerebral blood flow.

For practical purposes the following definitions are used for the hypertensive crises

- **Hypertensive urgency** (severe asymptomatic hypertension) is defined as Blood pressure measurement $\geq 180/120$ mmHg without any evidence of end organ damage.
- **Hypertensive emergency:** blood pressure $\geq 180/120$ mm Hg with evidence of end organ damage. It must be noted that in some patients with newly diagnosed hypertension, end organ damage can even be present at diastolic pressure as low as 100 mm Hg
  - End organ damages in hypertensive emergency is described in the following table:

<table>
<thead>
<tr>
<th>Miscellaneous</th>
<th>Malignant Hypertension*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebrovascular</td>
<td>Hypertensive encephalopathy, Intracerebral hemorrhage, Ischemic stroke, subarachnoid hemorrhage</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Acute left ventricular failure, Myocardial infarction, acute aortic dissection</td>
</tr>
<tr>
<td>Renal</td>
<td>Acute renal failure</td>
</tr>
<tr>
<td>obstetric</td>
<td>Preeclampsia / Eclampsia</td>
</tr>
<tr>
<td>Surgical</td>
<td>Severe hypertension prior to emergency surgery, post-operative hypertension</td>
</tr>
<tr>
<td>miscellaneous</td>
<td>Severe epistaxis</td>
</tr>
</tbody>
</table>

*Malignant hypertension* refers to sudden increase in blood pressure with associated hemorrhages, exudates, papilledema and fibrinoid necrosis in small arteries of the
kidney, brain, and retina. It presents clinically as impaired renal function, retinopathy, and encephalopathy

Evaluation
- Evaluate all systems for any evidence of end organ damage
  - Eyes – Fundoscopy to evaluate the retina
  - Chest – any evidence of pulmonary edema
  - CVS, abdomen – Evidence for heart failure,
  - CNS – GCS, any facial deviation, weakness of the limbs

Work up
Objective: to look for any evidence of end organ damage.
- Urinalysis, BUN and Cr, Peripheral blood morphology
- Other Investigations on individualized basis – eg. CT of the brain for those with weakness

Management
Because of the auto regulatory readjustment of the cerebral, renal and coronary blood flow in hypertensive patients, any attempt to decrease blood pressure to a level below the lower limit of this new set point leads to reduction of blood flow with deleterious effects on different organs. Therefore the management of such patients should balance between the benefits and risks of rapid blood pressure lowering.

Hypertensive emergency
- Initial goal is to reduce the mean arterial pressure by not more than 25% in the first 2 hours or to a level around 160/100 mmHg. This gives time for healing of necrotizing vascular lesions in the end organs without increasing the risk of ischemic events.
- Choice of drug: An ideal drug to achieve this should be parenteral, rapid onset, and short acting to help adjust the dose based on response.
  - Common drugs used for hypertensive emergencies include Labetalol, Nitroprusside, Nitroglycerin, and Hydralazine. Specific recommendations exist for the choice of antihypertensive for each of the hypertensive emergencies.
  - In our setup, Hydralazine is more readily available. Usual dosage range is 10 – 50 mg at 30 minute intervals. Oral antihypertensives are not preferred due to their slower onset of action and difficulty to monitor response minute to minute.
- Once the blood pressure is controlled, oral therapy can be initiated with a goal to gradually reduce the diastolic pressure to 85 – 90 mmHg over weeks to months.
- Continued monitoring should be made with emphasis on the initial target organ affected. Some patients may continue to have a progressive end organ disease, which should be addressed in the chronic care of the patient.
Provide Patient education

Hypertensive urgency (severe asymptomatic hypertension)

- Goal is to reduce the blood pressure to <160/100 mmHg over hours to days. There is no benefit of rapidly reducing the blood pressure and it rather has deleterious effects.
  - Rest
  - Already on Antihypertensives
    - Counsel on adherence and restart initial antihypertensive if noncompliant
    - add another drug for difficult to control with one drug
    - Enroll to chronic care
  - Newly diagnosed
    - Initiate oral antihypertensive
    - Enroll to chronic care

Any of the long acting antihypertensive can be used for the management of these patients. The patient should be observed for a few hours to ascertain that the blood pressure is improving. They can then be enrolled into chronic care.
MX of suspected Acute Coronary Syndrome (ACS) in the ED

Introduction:

- Coronary artery disease (CAD) is commonly defined as a >50% luminal stenosis of a coronary artery by atheromatous plaque. This produces a mismatch between myocardial oxygen supply and demand. CAD can present as stable angina, ACS, CHF, sudden cardiac death or silent ischemia. ACS represents a continuum of myocardial ischemia (UA) or infarction (NSTEMI/ STEMI).

Definition:

*Stable angina pectoris* = chest discomfort <10 min associated with exertion/stress, relieved by rest or sublingual nitroglycerin.

*Unstable Angina* = angina pain with at least 1 of 3 features:

- occurs at rest/with minimal exertion, lasting>10 min; severe & of new onset; crescendo pattern (i.e. more severe, prolonged, or frequent than previously).

*NSTEMI (Non ST segment elevation MI) = UA with evidence of myocardial necrosis (elevated cardiac biomarkers)

*STEMI (ST segment elevation MI) = chest pain >20 to 30 min occurring at rest (not relieved by nitroglycerin), serologic evidence of myonecrosis, and persistent ST segment elevation.

Initial Assessment of ACS:

Patient evaluation (Hx,P/E & diagnostic tests) & interventions to stabilize the hemodynamic status should occur simultaneously!!

First **Distinguish** between ischemic Vs non-ischemic causes of chest pain: i.e. ACS Vs others (pericarditis, aortic dissection, PTE, pneumonia, pneumothorax…etc)

Assess **Clinical stability**, before trying to arrive at a definite dx…secure the ABCs

Take brief Hx & do P/E PLUS quick ECG interpretation& cardiac enzymes

Is **ACS is likely** …?-Hx: Chest or left arm pain as the main Sx; Known CAD

P/E: transient MR murmur, low BP, diaphoresis, CHF or pulmonary edema

New ST deviation (≥1 mm) or T- inversion and/or elevated troponin or CK-MB
If ACS is likely → **Prompt initial Rx** with aspirin plus clopidogrel; nitroglycerine; morphine/pethidine; O2, B-blocker is essential.

Note: If the patient presents early within few hours and can afford or the setup allows, early referral of high risk patients for fibrinolysis, PCI/CABG is recommended.

Monitor & immediately treat arrhythmias & other complications (e.g. pulmonary edema, cardiogenic shock…see Mx of CHF)

Pay attention to exacerbating factors, such as disturbances in electrolytes (K+, Mg++), hypoxemia, drugs, or acidosis & manage accordingly.

Stratify risk: if high risk & time allows → consult for more invasive Mx (If affordable)

**Killip class for STEMI:**
- Class I – no HF
- Class II - mild to moderate HF (S3, basal rales, raised JVP)
- Class III - overt pulmonary edema
- Class IV - cardiogenic shock

Always **monitor patients condition & assess Rx response** (pain, ECG, cardiac enzymes)

Following clinical stabilization, proceed with thorough clinical evaluation:

**History** → Characterize the chest pain … retrosternal; heaviness, squeezing, pressure, tightness; radiation (left shoulder, jaw, arm, abdomen); duration; onset & aggravating/relieving factors

→ Associated Sxs (and at times the only manifestations of ACS without chest pain called “angina equivalents”): heartburn, nausea/vomiting OR dyspnea, weakness, dizziness, syncope (Beware of "Atypical" presentations in the elderly, diabetic&women)

PLUS look for additional historic features which increase likelihood of ACS:

- Prior history of CHD or other vascular disease; risk factors for CHD (old age, male sex, DM, HTN, CKD, dyslipidemia, obesity, smoking, ? chat chewing); recent cocaine use

**P/E:** cardiopulmonary distress, diaphoretic, anxious, restless

   Low BP, ↑ RR, irregular pulse; crepitations in lung fields; raised JVP, gallop, MR murmur, quite precordium; and check distal pulses & bruits
Diagnostic tests:

1. **ECG:** done at presentation; repeat at 6–12 h& with any change in Sx

   - Ischemia → T wave inversion
   - Injury → S-T segment elevation
   - Infarction → Pathologic Q wave

   *UA/NSTEMI= ST depression/transient elevation or deep T inversion (>=0.3mV)*

   * STEMI=New ST elevation in 2 contiguous leads >= 0.2 mV in men or 0.15 mV in women in leads V2-3 and/or 0.1mV in other leads OR new LBBB

   N.B the ECG must also be analyzed for rate, rhythm etc (look for arrhythmias)

2. **Cardiac biomarkers:** serial testing at presentation & 6–12 h after sx onset

   *Cardiac troponins ( T&I) -rise 20 -50 Xs Upper normal limit/UNL/ in acute MI; rise 4-8 hr after injury; may remain elevated for 7-10 days; more Specific & Sensitive than CK-MB

   *Creatine kinase (CK) -rises in 4–8 hr; normalize by 48–72 h; lacks specificity

3. Echocardiography: may show new wall motion abnormality

4. RBS, electrolytes, OFTs, lipid profiles

5. CXR: to look for pulmonary edema; R/o other DDx (PTE, pneumonia, Pneumothorax…)

6. Coronary angiography if indicated

**Management of ACS:** should focus on stabilizing the patient's condition, relieving ischemic pain, and providing antithrombotic therapy to reduce myocardial damage and prevent further ischemia. The goal is early revascularization.

1) **General measures:** Continuous ECG monitoring for arrhythmia & ST changes

   - V/S: Q 2 hr until stable, then Q 4hr & as needed
   - O₂ (2-4 lit/min) if SaO₂<90% - Bed rest, Sedation, VTE (venous thromboembolism) prophylaxis
   - NPO except for sips of water until stable; IV fluid – eg. for inferior MI
   - Glycemic control-goal is RBS of 140-180mg/dl (if > 180mg/dl, give regular insulin-1-2IU for each 50mg/dl increase above 180mg/dl, by measuring RBS Q 6hrs)
• Treat comorbidities like DM (for both types-standing doses of lente/NPH insulin at A.M & P.M with correctional doses of Regular Insulin in between 6 hrly); and HTN (see Mx).etc

2) Medications:

- Nitroglycerin (NTG): sublingual 0.4 mg Q 5 min as needed or chest pain (can also use buccal spray) *IV NTG for persistent ischemia. C/I= low BP, sildenafil use
- Morphine sulphate: 2–5 mg IV , may be repeated Q 5–30 min as needed to relieve Sxs (can also use morphine syrup, pethidine or tramadol)
- Aspirin(ASA): loading:162–325 mg chewed, then 75–162 mg/d plus Clopidogrel-loading: 300mg Po, then 75 mg/d for at least 1 yr (but ASA lifelong)
- Metoprolol :25–50 mg PO q 6 h (If HTN, ongoing pain,tachycardia: give IV over 1–2 min by 5mg increments)
  If not available- use atenolol, propranolol/carvedilol. C/I- CHF,bradycardia
- UFH: Bolus 60–70 U/kg (max. 5000 U) IV then infusion of 12–15 U/kg/ h (initial maximum 1000 U/h) titrated to aPTT 50–70 s * If no perfuser,12,500U SC BID is possible; OR LMWH (Enoxaparin):1 mg/kg SC Q 12 h(if GFR< 30,1mg/kg once daily)
- Warfarin: initially 2.5mg titrated to INR goal of 2-3(eg. for extensive anterior STEMI with severe left ventricular dysfunction, CHF, atrial fibrillation or LV thrombus)
- Statins: atorvastatin 80mg po/d is preferred. Others options are pravastatin/simvastatin/lovastatin 40mg Po/day
- ACEIs: start low dose eg. Enalapril / lisinopril 2.5 to 5mg po/d OR captopril 6.25-12.5mg TID; then escalate gradually to clinically effective dose.

Invasive therapy in ACS: for high risk patients who present early, referral to a better set up is recommended (If the patient can afford)

- STEMI : fibrinolysis Vs PCI/CABG
- Unstable angina/NSTEMI: PCI/ CABG; but fibrinolysis is not indicated.

Activities
- Discussion on case scenario # 6-10
- General discussion on questions raised by participants
Emergency approach to a Bleeding patient and Shock

Learning objectives:

By the end of this chapter, participants will be able to

- Identify causes of upper GI bleeding
- List the management principles of upper GI bleeding
- Identify causes of Epistaxis and list management principles
- Identify the different types of shock
- List management principles of shock

Upper GI(UGI) BLEEDING

Upper GI(UGI) bleeding is defined as bleeding originating proximal to the ligament of Treitz.

As with all true emergencies, the traditional triad of medical history, physical examination, and diagnosis must be accomplished simultaneously with resuscitation and stabilization.

Factors associated with an increased morbidity and mortality are increasing age, coexistent organ system disease, and recurrent hemorrhage.

Etiology of significant GI bleeds in Adults (in decreasing frequency)

- Upper:- peptic ulcer disease, gastric erosions, varices, Mallory-Weiss tear, esophagitis, duodinitis
- Lower:- upper GI bleeding, diverticulosis, angiodysplasia, cancer/polyps, rectal disease, inflammatory bowel disease

DIAGNOSIS

History

Hematemesis (vomiting blood) - approximately 50% of patients with UGIB (bleeding from esophagus, stomach, or proximal small bowel).

Melena (black tarry stool):- occurs from 150 – 200 ml of blood in the GI tract. It is present in ~70% of patients with UGIB and a third of patients with LGIB.
Hematochezia (bloody stool- bright red or maroon colored):- most often signifies LGIB, but may be due to brisk UGIB with rapid transit time through the bowel. Because UGIB is much more common than LGIB, a more proximal source of bleeding must be excluded before assuming the bleeding is from the GI tract.

Specific questions include the duration and quantity of bleeding, associated symptoms, previous history of bleeding, current medications, alcohol, NSAIDs, long-term aspirin ingestion, allergies, associated medical illness, previous surgery, and the response to any previous treatment. Symptoms of hypovolemia, such as dizziness, weakness, or loss of consciousness, most often after standing up. Other non-specific complaints include dyspnea, confusion, and abdominal pain. An elderly patient may present with ischemic chest pain from significant anemia.

**Physical examination**

Vital signs: - hypotensive, tachycardic, or sustained postural changes of >20 beats/minute in PR should be assumed to have significant hemorrhage.

General examination: important to make a diagnosis and assessing the severity of blood loss and patient’s response to that loss. Give attention to general appearance, vital signs, mental status (including restlessness), skin signs (e.g., color, warmth, and moisture for shock, bruises, petechiae to assess vascular lesions, or hypercoagulable states). Pulmonary and cardiac findings, abdominal, and rectal examination is important. Frequent reassessment is very important.

- Rectal examination, looking for: Hemorrhoids, Rectal mass, Rectal tenderness,
- Stool examination for evidence of frank blood, stool color, melena. Guiac test for occult blood.
- Nasogastric lavage, looking for: Bright red blood or coffee grounds indicate UGI bleed, Clear bilious drainage, indicating acute UGI bleed as an unlikely source
- Additional laboratory tests:- HCT/HGB( changes may lag behind the actual blood loss), coagulation studies (PT, platelet count), type and cross-match, CBC, Endoscopy, Bleeding scan, Arteriography

**Management:-**

Depends on etiology and severity, but quick identification, aggressive resuscitation, and prompt consultation are the keys to appropriate emergency management.

1. **Primary treatment**

**Patients who are hemodynamically unstable**

- Undress quickly, placed on cardiac and oxygen saturation monitors
- Establish IV access with two large-bore IV catheters (18 gauge or higher).
- Evaluate patient for hemodynamic instability: Hypotension, Tachycardia, Orthostatic hypotension
- Definitive airway control for patients with massive UGIB, to prevent aspiration of blood/gastric content
- Draw Blood for Hct, Type and cross-mach, CBC, coagulation studies
- IV fluid resuscitation( 2 L bolus in adults) until the patients vital sign has stabilized, or patient has received 40ml/kg,
- Administer blood based on the clinical finding of volume depletion or continued bleeding, not based on initial Hct
  - GENERAL GUIDELINES - CONTINUED ACTIVE BLEEDING AND FAILURE TO IMPROVE PERFUSION AND VITAL SIGNS AFTER THE INFUSION OF 2 L OF CRYSTALLOID
  - THE THRESHOLD FOR BLOOD TRANFUSION SHOULD BE LOWER IN THE ELDERLY
    - Replace coagulation factors as needed
- Vasopressors if BP does not respond to aggressive fluid resuscitation
- Immediate consultation for persistently unstable patients.

2. Secondary treatment

- Early UGI Endoscopy - is the treatment of choice for significant UGIB (bleeding ulcers and varices).
  - Injection with ethanol, epinephrine, thrombine, and others
  - Coaptive therapies; thermocoagulation, multipolar electrocoagulation, and laser electrocoagulation
  - Endoscopic band ligation or injection therapy for varices
- IV proton pump inhibitors - reduce rebleeding and the need for surgery. Are best used as adjunct to endoscopic therapy
  - Esomeprazole/pantoprazole 80 mg bolus, then 8mg/h
- Most Mallory–Weiss tears resolve spontaneously.
- Somatostatin, or octreotide infusion (inhibits gastric, intestinal, and biliary motility, de-crues visceral blood flow):- inferior to endoscopic technique, but considered when endoscopy is unsuccessful, contraindicated, or unavailable.
  - Dose:- 25-50micg iv bolus, followed by IV infusion of 25-5-micgm/h
- Vasopressin- mostly used for variceal bleeds, but adverse reactions are more common, and replaced by other drugs
- Histamine 2 antagonists- are not beneficial in acute UGI hemorrhage
- Other drugs which are important, but not utilized in acute ED management, including
  - Beta- blocker therapy-beneficial in variceal bleeds which prevent the initial bleed, and risk of rebleeding.
- Treatment of H. pylori infection reduces the recurrence of PUD, and re-bleeding.
- IV erythromycin (200mg IV) to prepare for endoscopy as it accelerates gastric emptying and reduce duration of endoscopy
  - Discuss with the endoscopist before administration
- Consider balloon tamponade (rarely used at present) for massive, acute hemorrhage.
- Early surgical consultation for lower GI bleeds

**Epistaxis**

**Causes:**
- **Local Causes:** Trauma: Nose picking of a dried mucosa is most common. Barometric pressure changes can also traumatize mucosa, Septal perforations as with cocaine use, Polyps and tumors, Infections: Rhinitis, vestibulitis, and sinusitis, Angiofibroma of the nasopharynx.
- **Systemic Causes:** Inflammation and infectious diseases such as scarlet fever, malaria, and typhoid fever, Vascular lesions such as arteriosclerosis, hereditary hemorrhagic telangiectasia (Osler–Weber–Rendu disease—autosomal dominant), and coarctation of aorta, Bleeding disorders such as hemophilia and platelet disorders, Overdose with anticoagulants or antiplatelet agents

**Management**
- Patient will usually present with an anxious and bloody appearance, making diagnosis of underlying cause impossible until bleeding is controlled.
- Secure bilateral, wide bore IV and run crystalloids
- Have patient pinch nostrils against nasal septum with head flexed anteriorly for at least 5 minutes. This will stop bleeding originating from Kiesselbach’s plexus or any origin anterior to the pinch. If bleeding is behind the pinch, blood will collect in the nasal cavity and start spilling over into the nasopharynx. This blood will be coughed up.
- Next, try to make a geographic diagnosis of the bleed. If bleeding origin is identified, it is usually in Kiesselbach’s plexus and will stop with the 5-minute pinch, cauterization, or an anterior nasal pack. If the origin is not identified, must treat as a diffuse bleed by packing.
- Bleeding posterior to Kiesselbach’s plexus will need a speculum and good light to locate, and possibly an endoscope.
- Bleeding can be controlled temporarily by applying pressure with a cot-ten pledget impregnated with a vasoconstrictor, such as phenylephrin/epinephrine, and a topical anesthetic, such as lidocaine, until the site is anesthetized.
• Cautery of the hemorrhagic point: When visible, the bleeding site maybe cauterized with silver nitrate or electrocautery. Do not use cautery for bleeding due to a bleeding disorder.
• For epistaxis due to a bleeding disorder, use petrolatum gauze to apply pressure asatraumatically as possible.
• If bleeding is in the anterior nasal cavity, an anterior nasal pack shouldbe used.
• If bleeding is in the posterior nasal cavity, an anterior–posterior pack should be used.
• Ligation of arteries such as the sphenopalatine should be reserved for the most severe cases in which packing does not stop the bleeding.
• Arterial embolization can be considered instead of ligation.

Shock

Definition: - physiologic state characterized by a significant reduction in systemic tissue perfusion, resulting in decreased oxygen delivery to the tissues, which creates an imbalance between oxygen delivery and oxygen consumption. In Adults

- Systolic BP < 90 mmHg, or
- MAP <= 60 mmHg
- Reduction of systolic BP > 30 mm Hg from the patient’s baseline

Physiologic determinants

- Systemic vascular resistance (SVR): Vessel length, Blood viscosity, Vessel diameter
- Cardiac output (CO): Heart rate (HR), stroke volume (preload, myocardial contractility, after load)

Stages of shock

- Pre-shock (warm/compensated shock): regulatory mechanisms compensate for diminished perfusion
  □ Tachycardia, warm extremity, low/normal BP
- Shock: overwhelmed compensatory mechanisms. Eg. with loss of 20 – 25% of effective blood volume
- End organ damage: irreversible state due to untreated shock
Types of Shock

- **Hypovolemic**: hemorrhage, vomiting, diarrhea, third-space loss, burns...
  - Causes: hemorrhage, vomiting, diarrhea, third-space loss, burns...

- **Cardiogenic**: due to abnormality in cardiac function, resulting in pump failure and decreased cardiac output
  - Causes: cardiomyopathies, arrhythmias, mechanical causes, extracardiac/obstructive

- **Distributive**: sepsis, anaphylactic, acute adrenal insufficiency, neurogenic, resulting mainly in decreased systemic vascular resistance

- **Obstructive**: cardiac tamponade, tension pneumothorax, massive pulmonary embolus, etc… resulting in decreased CO and increased SVR

**Definition and criteria for septic, hemorrhagic and cardiogenic shock**

1. **Septic shock**
   - a. SIRS: two or more of the following:
     i. Temperature >38°C or <36
     ii. HR >90 bpm
     iii. RR>20 bpm or PaCO₂<32 mmHg
     iv. WBC>12,000/mm³, <4000/mm³, or >10% band neutrophilia
   - b. Sepsis syndrome: SIRS associated with organ dysfunction or hypotension; lactic acidosis, oliguria, or altered mental status (AMS)
   - c. Septic shock: SIRS with hypotension despite adequate fluid resuscitation; septic shock still be diagnosed if vasopressor therapy has normalized BP

2. **Hemorrhagic shock**
   - a. Simple hemorrhage: suspect bleeding with pulse <100 bpm, normal RR, normal BP, and normal base-deficit
   - b. Hemorrhage with hypotension: suspected bleeding with persistent pulse >100 bpm
   - c. Hemorrhagic shock: suspected bleeding with at least 4 criteria listed below

3. **Cardiogenic shock**
   - a. Cardiac failure: clinical evidence of impaired forward flow of the heart, including dyspnea, tachycardia, pulmonary rales, peripheral edema, or cyanosis
   - b. Cardiogenic shock: cardiac failure and four criteria listed below

Clinical presentation: Varies according to the type of shock, its cause, and its stage of presentation. Several features are common among all types of shock (cardinal features), while others may suggest a particular type of shock
1. Cardinal findings:
   a. hypotension (absolute/relative),
   b. oliguria,
   c. abnormal mental status (the continuum of agitation confusion, delirium, obtundation or coma),
   d. metabolic acidosis (decreased clearance of lactate by the liver, kidneys, and skeletal muscle), and
   e. cool and clammy skin
2. Suggestive findings:
   - **Hypovolemic shock:**
     o History:- hematemesis, hematochesia, vomiting, diarrhea, or abdominal pain, blunt/penetrating trauma
     o P/E:- decreased skin turger, dry skin, dry axillae, tongue/ buccal mucosa, postural hypotension, decreased JVP
   - **Cardiogenic shock:**
     o History:- Dyspnea, chest pain, or palpitation
     o P/E:- diffuse crackles on lung examination, new murmur, elevated jugular and central venous pressures
     o Investigations:- evidence of pulmonary congestion/edema on CXR, evidence of old/recent ischemia on ECG, elevated cardiac enzymes
   - **Distributive shock:**
     o History:- dyspnea, productive cough, dysuria, hematuria, chills, myalgias, rashes, fatigue, etc
     o P/E:- fever, tachypnea, tachycardia, leukocytosis, abnormal mental status, flushing

**Diagnostic approach**

Diagnostic evaluation should occur at the same time as resuscitation. Resuscitative efforts should not be delayed for Hx, P/E, and lab.

- **History:-** baseline medical status, recent complaints and recent activities, food and medical allergies, etc
- **Physical exam:-** neither sensitive nor specific, should be directed to uncover the type, severity, and cause of the shock
- **Laboratory:-**
  o To identify the cause and early organ failure.
  o Especially performed early in undifferentiated shock.
  o CBC, Basic chemistry- Na, K, Cl, serum bicarbonate, BUN, creatinine, LFTs, amylase, lipase, PT, INR, PTT, fibrinogen, FSP, ABG, Type and cross-match, CXR, abdominal x-ray, CT/MRI,
Empiric criteria for diagnosis of circulatory shock:
- ill appearance or altered mental status (AMS)
- HR > 100 beats/minute
- RR >22bpm, or PaCO2 < 32 mmHg
- Arterial base deficit <-5 mEq/L or lactate >4 mM/L
- Urine output <0.5 mL/kg/hr
- Hypotension> 20minutes duration
  - Regardless of the cause, 4 criteria should be met

MANAGEMENT

1. Hemorrhagic shock
   a. Ensure adequate ventilation/oxygenation
   b. Provide immediate control of hemorrhage, when possible (e.g., traction for long bone fracture, direct pressure)
   c. Initiate judicious infusion of Normal Saline or lactated ringer’s solution (10-20 ml/kg)
   d. With evidence of poor organ perfusion and 30-minutes anticipated delay to hemorrhage control, begin PRBC infusion (5-10 ml/kg)

2. Cardiogenic shock
   a. Put the patient on cardiac and pulse oxymetry monitoring
   b. Ameliorate increased work of breathing: provide oxygen; pain control, eg. Morphine for acute MI; PEEP for pulmonary edema
   c. Preload augmentation : give fluid : 250ml of NS
   d. Begin inotropic support: dobutamine (5micro gm/kg/min) is common empiric agent for a border line BP (SBP between 90 – 100 mmHg), dopamine/norepinephrine are choices for significantly reduced BP (SBP < 80 mmHg). If no option, epinephrine can be considered.
      i. The most important part is titration of the dose of inotropes and vasopressors based on patients response.
   e. Diuress after inotropic support if there are signs and symptoms of pulmonary edema
   f. Reverse the underlying pathology (e.g. treatment of arrhythmias, MI etc….see MX of ACS)

3. Septic shock
   a. Remove work of breathing - Ensure adequate oxygenation by giving oxygen via face mask, if possible or through nasal catheter or nasal prongs.
   b. Administer 20 ml/kg of crystalloid as bolus, and titrate infusion to adequate urine output, upto 5 -6 Lts, until you see evidence of lung congestion.
c. Initiate broad spectrum antibiotic therapy early, usually based on the possible focus of infection/septic focus.
d. Surgical drainage or debridement of an abscess or dead and necrotized tissue.
e. If volume restoration fails to improve organ perfusion, begin vasopressor support:
   - initial choice includes dopamine, infused at 5-15 mcg/kg/min and titrated every 10 – 15 min until response is achieved, or norepinephrine, infused at 0.5-1 mcg/min, and titrated based on response.
f. Corticosteroid administration - for refractory vasopressor-dependent shock.
g. Blood transfusion if HCT is < 30% to keep adequate O2 saturation

4. Anaphylactic shock
   a. Control airway and ventilation
      i. Put the patient on monitor and give oxygen via high pressured face mask
      ii. Consider Early definitive airway control for those evidence of significant airway edema or if you fail to correct oxygenation with face mask.
   b. Secure bilateral wide bore needle IV lines and administer 20ml/kg of crystalloids as fast as possible.
   c. Administer Epinephrine for control of acute symptoms.
      i. 1 mg of epinephrine IM on anterior or lateral thigh as quick as possible
      ii. then mix 5 mg of epinephrine in 500 ml of normal saline. Infuse at 10 cc/hr, and titrate to arterial BP response.
   d. Administer 5-10 mg/kg of hydrocortisone or 1-2 mg/kg of methylprednisolone for late control of symptoms
      i. Don’t have any role in the control of acute symptoms.
   e. H2 receptor blockers – do not have a role in ED management of patients with anaphylactic shock.

Activities
   - General discussion on questions raised by participants
Common Neurologic Emergencies

Learning Objectives

At the end of this chapter, participants will be able to:

- Identify common emergency presentations of neurologic system affection
- List causes of coma and its management principles
- List causes of seizure and its management
- Explain management principles of status epilepticus

Coma:

Coma is a state of reduced alertness and responsiveness from which the patient cannot be aroused. The Glasgow coma scale is widely used and also the FOUR (Full Outline of Unresponsiveness) score is used widely in ICU (see annex).

Clinical feature

History

✓ Exploit all available historical sources (EMS personnel, caregivers, family, witnesses, medical records, etc)
✓ Onset of symptoms
✓ Any history of fever, medication, seizure

Physical examination

✓ Vital signs including RBS
✓ Look for signs of trauma
✓ Neurologic examination (cranial nerves, motor examination, posturing or meningeal signs)

The clinical features of coma vary with the depth of coma and the cause.

Based on the clinical findings the cause of coma can be categorized in to two

✓ Toxic-Metabolic coma –
  ✓ characterized by diffuse CNS dysfunction and no focal neurologic findings
Structural Coma- characterized by focal CNS dysfunction further classified to
- Hemispheric(supra tentorial)
  - Progressive hemiparesis or asymmetric muscle tone and reflex
  - On the contrary coma without lateralizing sign may result from increased ICP
- Posterior fossa (infratentorial)
  - An expanding lesion, such as cerebellar hemorrhage or infarction, may cause abrupt coma, abnormal extensor posturing, loss of papillary reflexes, and loss of extraocular movements.

Investigation

- CBC, RBS, serum electrolytes, renal and liver function tests, blood film etc.
- Brain CT, CXR, ECG, Brain MRI
- Lumbar puncture
  - Contraindications- although no absolute C/I, caution should be taken in patients with
    - Focal neurologic deficits
    - Possible raised intracranial pressure
    - Thrombocytopenia
    - Suspected spinal epidural abscess

Differential Diagnosis

1. Coma from causes affecting the brain diffusely
   - Encephalopathies
     - Hypoxic encephalopathy
     - Metabolic encephalopathy (hypoglycemia, hyperosmolar state, electrolyte abnormalities e.g. hyper/hyponatremia)
     - Hypertensive encephalophathy
     - Organ system failure (hepatic encephalopathy, uremia/renal failure, endocrine (addison disease, hypothyroidism), hypoxia, carbondioxide narcosis)
   - Toxins
   - Drug reactions (e.g. neuroleptic malignant syndrome)
   - Environmental causes (e.g. hypothermia / hyperthermia)
   - Sepsis

2. Coma from primary CNS disease or trauma
   - Direct CNS Trauma
     - Diffuse axonal injury
     - Subdural hematoma
Epidural hematoma
✓ Vascular disease
✓ CNS infections
✓ Neoplasms
✓ Seizures
  i. non convulsive status epilepticus
  ii. postictal state

**Approach to the patient**

The goal of the physician is to rapidly determine if the CNS dysfunction is from diffuse impairment of the brain or if signs point to a focal (and perhaps surgically treatable) region of CNS dysfunction.

Treatment of coma involves identification of the cause and initiation of specific therapy directed at the underlying cause. Evaluation for readily reversible causes of coma, such as hypoglycemia and opioid toxicity, demands priority.

1. Secure airway, breathing and circulation, take vital signs including RBS, secure IV line
   ✓ Indication for intubation
     i. Deep coma - GCS<9
     ii. Status epilepticus
     iii. Anticipated clinical worsening or rapidly deteriorating GCS despite initial management
     iv. If suspected poisoning and gastric lavage is planned.
2. Immobilization of C-spine if there is any concern of trauma
3. Coma cocktail
   ✓ Thiamine 100mg IV prior to dextrose in alcoholic patients
   ✓ Dextrose 50gm IV push
   ✓ Naloxone 0.01mg/kg if opiates suspected
   ✓ Flumazenil 0.2mg IV if benzodiazepine suspected (routine use in coma of unknown etiology is not recommended)
4. If concern for increased ICP and herniation
   ✓ Elevate head 30degrees
   ✓ Consider mannitol 0.5-1gm/kg bolus
   ✓ If mass lesion- consider dexamethasone
5. Send lab examinations and do LP if no contraindication. If patient is febrile give empiric antibiotic ceftriaxone 2gm IV stat with dexamethasone without waiting for result.
6. Send the patient for investigation after stabilization
### Glasgow coma scale

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<td>Spontaneous</td>
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<tr>
<td>Response to verbal command</td>
<td>3</td>
</tr>
<tr>
<td>Response to pain</td>
<td>2</td>
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<tr>
<td>No eye opening</td>
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<th>Best verbal response</th>
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<td>Oriented</td>
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<tr>
<td>Confused</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
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<td>Incomprehensible sounds</td>
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<td>No verbal response</td>
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<td>Obeys commands</td>
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<td>Localizing response to pain</td>
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<td>Withdrawal response to pain</td>
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<tr>
<td>Flexion to pain</td>
<td>3</td>
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<td>Extension to pain</td>
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Seizure

Definition:

Seizure is an episode of abnormal, paroxysmal, excessive discharge of central nervous system/CNS/ neurons.

Epilepsy is recurrent seizure due to an underlying cause

Classification of seizures

1. Generalized seizures: involves brain diffusely
   a. Tonic-clonic (grand mal)
   b. Absence seizures (petit mal)
   c. Others (myoclonic, tonic, clonic, or atonic seizures)
2. Partial or focal seizures: electrical discharges begin in a localized region of the cortex
   a. Simple partial: without impairment of consciousness
   b. Complex partial: with impairment of consciousness
   c. Partial with secondary generalization

Clinical features

History

- When a patient presents after the event, the first step is to determine whether the attack was truly seizure.
- Suggestive histories include presence of preceding aura, abrupt or gradual onset, progression of motor activity, loss of bladder or bowel control, whether the activity is local or generalized and symmetric or not, duration of the attack

Clinical features that help to distinguish seizure from other kinds of mimicking attacks

1. Abrupt onset and termination. (most lasting 1 to 2 minutes)
2. Lack of recall. Except simple partial seizure
3. Purposeless movements or behavior during the attack
4. Post-ictal confusion and lethargy

Physical Examination

- Check vital signs including RBS
• Check for injuries (head or spine, look for posterior dislocation of the shoulder, laceration of tongue and mouth, dental fracture and pulmonary aspiration)
• Perform detailed neurologic examination
• Todd’s paralysis is a transient focal neurologic deficit following a simple or complex seizure which should resolve within 48 hours.

Differential diagnosis

1. Syncope
2. Pseudoseizures
3. Hyperventilation syndrome
4. Migraine headache
5. Movement disorders
6. Narcolepsy/cataplexy

Laboratory examination

In a patient with a well documented seizure disorder who had a single unprovoked seizure, the only tests that may be needed are a glucose level and an anticonvulsant level.

In case of an adult with a first seizure or when the history is unclear more extensive studies are needed e.g. (RBS, electrolytes, BUN, Cr, calcium, magnesium, a pregnancy test and toxicology studies)

Lumbar puncture - in a febrile or immunocompromized if no contraindication.

Lab investigations helpful in distinguishing seizure from pseudo seizure

1. Wide gap metabolic (lactic acidosis) - majority will clear within 30min
2. Serum prolactin level - may be elevated briefly 15 to 60 minutes

TREATMENT OF UNCOMPPLICATED SEIZURES

1. Patients with active seizure
   ✓ Protect the patient from injury; do not try to restrain
   ✓ If possible turn the patient to the side
   ✓ Do not try to insert bite block or to ventilate during seizure attack
   ✓ Once attack subsides, ensure a clear airway
   ✓ There is no indication for IV anticonvulsant medication during the course of an uncomplicated seizure
2. Patients with a history of seizure
   ✓ Identify and treat seizure precipitants
   ✓ In the known epileptic patient non-compliance is the main cause of acute onset of seizure so if possible send for serum drug level.
   ✓ If serum levels are very low, supplemental doses may be appropriate, and the regular doses may be adjusted or restarted. E.g. Phenytoin 18mg/kg Po as a single dose or divide in to three doses given every three hours will achieve therapeutic serum level within 2 to 24 hrs.
   ✓ If serum level is normal and patient has single attack additional treatment is not needed because even patients with well controlled seizure might have breakthrough attacks.
   ✓ If seizures are too frequent dose adjustment, adding another antiepileptic drug or even changing of medication should be considered but should be done in consultation with a neurologist or primary care physician.

3. Patient with a first seizure
   ✓ In general patients with a first seizure who have a normal neurologic examination, no acute or chronic medical comorbidities, normal diagnostic testing including normal imaging and who have normal mental status can be discharged from the ED without initiation of antiepileptic medication.
   ✓ Patients with secondary seizure due to an identifiable neurologic condition should generally be treated as the risk of seizure recurrence is high.
   ✓ The ideal initial antiepileptic regimen is a single-drug therapy that controls seizure with minimum toxicity.
   ✓ Selection of antiepileptic drugs
     i. Generalized tonic clonic seizure
        1. First line- Valproic acid, lamotrigine, topiramate
        2. Alternatives- Phenytoin, carbamazepine, Phenobarbital
     ii. Focal seizure
        1. First line- carbamazepine, phenytoin, lamotrigine
        2. Alternative- valproic acid, Phenobarbital
     iii. Absence, myoclonic, tonic, clonic
        1. First line- valproic acid
4. **Seizures in the HIV positive patient**

- Mass lesions, HIV encephalopathy and meningitis are seen more frequently.
- If there is no evidence of increased intra cranial pressure, and focal neurologic deficit Lumbar puncture should be done.
- If CT is available and cost is not an issue non-contrast head CT scan can be used initially.
- If no explanation for seizure, a contrast-enhanced head CT or MRI should be obtained.

**Status Epilepticus**

Status epilepticus is continuous or intermittent seizures for more than 5 minutes without recovery of consciousness. The most common causes of status epilepticus include subtherapeutic antiepileptic levels; preexisting neurologic conditions such as prior CNS infection, trauma, hemorrhage, or stroke; acute stroke; hypoxia; metabolic abnormalities; and alcohol or drug withdrawal.

**Types of Seizure Activity**

Seizure activity may be generalized tonic clonic type which is associated with higher mortality and complications. In non-convulsive status epilepticus the patient is comatose or has fluctuating abnormal mental status or confusion, but no overt seizure activity or only subtle activity and the diagnosis is made by EEG.

Epilepsia partialis continua is a focal tonic-clonic seizure activity with normal alertness and responsiveness.

**Treatment**

The goal of treatment is seizure control as soon as possible and within 30 minutes of presentation. Examination, identification of precipitating cause, application of the ABCs and treatment begin simultaneously.

**Approach to the patient**

- ABC of life
- Place the patient in semi prone or lateral position to decrease risk of aspiration.
- Large bore IV line should be established and RBS should be determined
- Thiamine 100mg IV prior to dextrose infusion
- Dextrose 50g IV push.
- Administration of anticonvulsant (see below)
If IV line is difficult to establish give diazepam 5 to 10 mg (0.15 mg/kg) diluted in 10 ml NS per rectum.

- Phenytin should not be mixed with any glucose containing IV fluid
- Phenytin should be infused no faster than 25 mg/min
- An oral loading dose of phenytin will achieve therapeutic serum concentration in 2 to 24 hours, where as IV phenytin achieves anticonvulsant level in 1 to 2 hours.

Remember; if status epilepticus is diagnosed, the patient should be started with both benzodiazepine and loading of phenytin simultaneously and ideally intubation should be considered.

**Refractory Status Epilepticus**

It is defined as persistent seizure activity despite the IV administration of adequate amounts of two antiepileptic agents.

**Drug therapy of status epilepticus**

1. Diazepam 5-10mg Iv stat/ lorazepam 0.1- 0.15mg over 1-2min and repeat dose if no response after 5 min
2. Phenytoin 20mg/kg IV at 50mg/min or fosphenytoin 20mg/kg at100-150 mg/min
3. If seizure continues repeat phenytin 7-10mg/kg at 50mg/kg or fosphenytoin 7-10mg/kg at150mg/min
4. If seizure continues and no ICU, Phenobarbital 20mg/kg IV at 60mg/min.
5. If no response with the first dose repeat Phenobarbital 10mg/kg IV at 60mg/kg
6. If no response general anesthesia with propofol, midazolam or pentobarbital. If the above medications are not available ketamine 1.5mg/kg bolus then 0.01-0.05mg/kg/hr can be infused.
   - Ketamine is considered as an agent of last resort as it has neuroprotective properties. Its major contraindication is the presence of intacranial mass lesion.

Valproic acid 20-40mg/kg iv at 5mg/kg/min is considered as a second line instead of phenytin if the patient was already on valproic acid or if the patient cannot respond to phenytin instead of Phenobarbital.

Note : The above medications are usually not available in the Iv form in most centers; so the respective Po preparations with the same dose can be used.

**Activities**
- General discussion on questions raised by participants
Approach to an acute febrile illness (AFI) in the emergency department

Objectives: By the end of this session, you will be able to:

- Describe initial assessment of a suspected AFI
- Explain clinical spectrum & differentials of an AFI
- List diagnostic work up of a febrile illness
- Mention management approach of common AFIs

INTRODUCTION:
Fever is an elevation in core body temperature above the daily range for an individual, due to altered hypothalamic set point. Mean oral temperature is 36.8° ± 0.4°C (98.2° ± 0.7°F) with low levels at 6 A.M. & higher levels at 4–6 P.M. A temperature of >37.2°C (>98.9°F) at a.m. or >37.7°C (>99.9°F) at p.m. defines fever. If > 41.5°C, it is called hyperpyrexia. Axillary temperature is < oral by 0.5°C; and oral temperature is < rectal temperature by 0.4-0.5°C.

The normal daily temperature variation is typically 0.5°C (0.9°F). During a febrile illness, the diurnal variation usually is maintained, but at higher, febrile levels. An AFI has varied presentations with long DDX but majority are self-limited infections, commonly of viral origin. Others may be life threatening febrile illnesses like cerebral malaria, relapsing fever, pyogenic meningitis etc.

Assessment of a patient with an acute febrile illness in the ED:

- Assess severity of the illness including the ABCs & manage accordingly.

If toxic or severity Sxs/Sxs present :( i.e. hypotension, change in mentation, inability to eat/to take drugs, organ dysfunction, Or hyperparasitemia as in P.falciparum/p.f/ >2% for non endemic areas or >5% for endemic areas) → prioritize initial stabilization of the clinical condition.
Look for localizing Sxs/ Sns…if suspected focus of infection is present → septic work up ( see septic shock) → take culture & start empiric antibiotic Rx without delay.

N.B In critically ill patients, if DX is uncertain or while waiting for lab results, consider empiric drug Rx based on a brief Hx,P/E or epidemiologic data.

*Pay attention to cover all possible etiologies, so as not to miss lethal but treatable febrile illnesses (severe malaria, meningitis, relapsing fever...etc) early drainage & surgical consultations should be made for suspected abscess or intra abdominal fluid collections on top of drug Rx.
Search & manage complications: seizure, hypoglycemia, sepsis, anemia, raised ICP… etc

Assess Rx response: resolution of fever & other parameters (BP, GCS, urine output, feeding ability…)

- Stable patients with mild cases can be treated as an outpatient. Some AFIs can be managed only with supportive cares, as in common URT infections, so that avoiding unnecessary antibiotic use.

**History:** An AFI can have varied presentations & may present as non-specific symptoms like headache, joint pain, chills, rigor, sweating, anorexia. Flu like Sxs are common in URT infections

A patient may have change in mentation or coma as in malaria, meningitis, relapsing fever, sepsis, encephalopathy….etc. Meningeal Sxs and abnormal body movement are also possible presentations

Some patients might present with localizing symptoms like

- Chest Sxs. (cough, pleuritic chest pain)...pneumonia or TB

- Urinary Sxs. (urinary frequency,urgency,hesitancy, dysuria or hematuria)...UTI/pyelonephritis

- GI Sxs....diarrhea, nausea, vomiting, abdominal pain, anorexia...gastroenteritis, intestinal parasites, food poisoning; or Yellowish eye ,RUQ pain, aversion to food...acute viral hepatitis, cholangitis or liver involvement in other infectious diseases

- Musculoskeletal Sxs: septic arthritis, osteomyelitis, myositis/myonecrosis

Also ask dental sxs/procedures, earpain/discharge, perianal complaints & genitourinary manipulations

Attention must be paid to:

- Geographic area, travel hx(for malaria, leishmaniasis);living condition (crowd, prison..) for relapsing fever, typhus; exposure to ill patient or animal/animal products, personal hygiene

- Recent dietary intake or drug use including cancer therapy, sexual history & recent surgery

- Underlying illness e.g. cardiac patient…for infective endocarditis; neutropenia..for neutropenic fever & level of immunosuppression (HIV, malignancy, DM etc)
**Physical exam:** On general appearance, the patient may appear acutely sick looking (in distress, sweating, shivering, convulsing, restless…)

- Vital sign: fever is always present but elderly & compromised patients (e.g. uremic or cirrhotic & those taking glucocorticoids or antipyretics) may be afebrile despite serious underlying infection; and hypothermia is possible as in late stage of septic shock. BP, PR & RR help determine degree of hemodynamic & metabolic compromise.

- Look for icteric sclera, pale conjunctiva; oral lesions including periodontal areas & inflamed tonsils

- Enlarged lymph nodes, nasal bleeding…also to rule out acute leukemia as a cause of fever.

- Cardiac murmurs, gallop…R/o infective endocarditis

- Chest exam for evidences of pneumonia, pleural effusion/empyema or TB

- Abdominal tenderness …for peritonitis, intraabdominal collections or hepatitis(RUQ location)

- Costovertebral tenderness as in pyelonephritis

- Joint swelling & other evidences of inflammation (in the musculoskeletal system)

Pay attention to look for: Surgical or other wound sites/decubitus ulcer, skin lacerations or burn;

- Local areas of erythema, edema or tenderness….cellulitis, necrotizing fasciitis, myositis, or myonecrosis.

- Perianal areas, Sinus tenderness, periodontal swelling, mastoid tenderness

- Skin lesions in viral illnesses or petechial rash in meningococcemia

*Neurologic exam including GCS & evidences for raised ICP; if meningeal Sns +ve… do LP, if no C/I

**Diagnostic Workup:**

- CBC with ESR including differential: for leukocytosis, neutropenia

- Culture & sensitivity: blood, urine, CSF, discharge-Sputum Gram stain, AFB

- Blood chemistries

- Blood film: for hemoparasites (malaria, borrelia, leishmania) - Stool examination: for ova or parasites

- Urinalysis: for WBCs, bacteria, leukocyte esterase , nitrite, casts, hematuria: for UTI/pyelonephritis
-Hepatitis serology (if LFTs abnormal) -CSF analysis for cell count, organisms (Gram stain, AFB, Indian ink), VDRL

-HIV test

-Serology studies with titer: widal, weifelix (both have limited clinical utility)

-CXR, abdominal ultrasound: if indicated

*Diagnostic Procedures: Lumbar puncture: for suspected meningitis

-Aspiration & drainage of infected collections / abscesses

Treatment of an AFI: supportive cares, specific Rx & Mx of complications.

1) General measures:

Control of fever- with sponging, fanning, cooling blankets

-antipyretics (if $T^0>38.5^0C$) - paracetamol Q 4hrs

Oxygen supplementation: if SaO2< 90%

Fluid Mx: Encourage Po fluid intake if possible; IV fluids: as maintenance fluid 2-3 bags over 24 hrs with replacement of ongoing loses. If in shock: 2-3lt NS over 1-2 hrs then assess V/S & proceed accordingly (see shock Mx) - Monitor fluid balance

Coma care (maintaining the airway, positioning, NG tube feeding, catheterization....)

2) Specific therapy: given if a certain Dx is confirmed or is strongly suspected

➢ Drug Rx of selected common AFI in the ED is presented here:

Uncomplicated P.falciparum/p.f/ malaria or spp. not identified:

A. P.f chloroquine resistant/unknown resistance: Coartem = Dose: 25-<35 kg=3 tabs/dose ; > 35 kg = 4 tabs/dose(bid for 3 days) or

-Quinine sulfate = 1tab (600mg) po TID plus one of the following: Doxycycline 100mg po bid or TTC 250mg po qid or Clindamycin 150-450 mg/dose Q 6-8 hrs(total of 7 days)

B. P.f chloroquine sensitive: chloroquine 600mg base po then 300mg base po at 6, 24 & 48 hrs or (4,4,2 regimen of 150mg base)

*Rx of p.vivax/p.v/ malaria-Chloroquine plus Presumptive antirelapse Rx with Premaquine

-coartem can also be used for p.v or mixed p.f/p.v
**Rx of Severe P.falciparum malaria** (refer case definition if needed)

Pre-referral Rx options: IM quinine/artemether or IM or rectal artesunate

*Artesunate=2.4 mg/kg IV at 0, 12 and 24 h, then daily. It is Rx of choice.

*If unavailable:* Artemether = 3.2 mg/kg stat IM then 1.6 mg/kg Q day OR

*QUININE: Loading - 20 mg/kg in 5% dextrose over 4 hrs, followed by 10 mg/kg in 5% dextrose over 2-4 hrs Q 8 hr. No loading dose, if patient took quinine within the previous 12 hrs (to avoid cardiotoxicity)-Decrease dose by 1/3 to1/2 after 48 hrs.

N.B. Iv Rx with quinine is given for minimum of 24 hrs once started (irrespective of patient’s ability to tolerate oral medications earlier) and complete Rx by giving: complete course of - Quinine + doxycycline/clindamycin OR coartem alone.

S/E of quinine: hypoglycemia (monitor RBS Q 6 hrs); hypotension (with rapid infusion- never give IV bolus); cinchonism & cardiac toxicity

**Relapsing fever**: louse born in our set up

- Doxycycline 100 mg or TTC/Erythromycin/ Chloramphenicol 500 mg ( po/iv) or Procaine penicillin 600,000 IU IM (all single dose)

* Follow patient’s condition & V/S in the first few hours of therapy and watch for worsening of Sxs due to a reaction called JHR (supportive care including adequate volume support is recommended)

**Pyogenic meningitis**: Ceftriaxone 2 gm iv BID (10-14 days) but the choice of antibiotics depends on the specific etiology and may be modified after CSF results. Dexamethasone 10 mg IV QID for 4 days is given 20 min prior to or with the antibiotic.

**Typhoid fever**: Ciprofloxacin 500 mg po BID x7-10 days (first line) or Amoxicillin, Cotrimoxazole OR IV Ceftriaxone = 1-2 gm /day for 7-14 days

**Epidemic typhus**- louse-borne Typhus: Doxycycline 200mg po/d X 2-3 days OR Chloramphenicol 500mg orally or IV QID x 5 days.

Note: in any case a patient with an AFI might present with coma and/ or other organ dysfunction; so supportive cares should be applied accordingly.

**Activities**

- General discussion on questions raised by participants
Emergency management of Common Endocrine Emergencies

Learning objectives
By the end of this session, participants will be able to:

- Define diabetic ketoacidosis/DKA/, HHS, Hypoglycemia
- Outline the clinical features of DKA, HHS, Hypoglycemia
- Able to diagnose DKA HHS, Hypoglycemia
- Manage DKA HHS, Hypoglycemia according to the standard protocol

Diabetic Ketoacidosis

Definition
DKA is a metabolic disorder characterized by the triad of hyperglycemia, anion gap metabolic acidosis (increased anion gap), and ketonemia.

Precipitating factors
The most common precipitating factors are infection and discontinuation of insulin treatment. Other less common factors include:

- Acute major illnesses such as MI, CVA, or pancreatitis.
- New onset type 1 diabetes
- Drugs (glucocorticoids, higher dose thiazide diuretics, sympathomimetic agents (e.g., dobutamine and terbutaline).
- Cocaine use
- Factors that may lead to insulin omission in younger patients include fear of weight gain, fear of hypoglycemia, rebellion from authority, and the stress of chronic disease.
- Poor compliance with the insulin regimen.

Clinical presentation
The clinical manifestations of DKA are directly related to the three primary metabolic derangements- hyperglycemia, volume depletion and acidosis. DKA usually evolves rapidly, over a 24-hour period.
 Symptoms
Nausea/vomiting, Thirst/polyuria, Abdominal pain, Shortness of breath

 Precipitating events
Inadequate insulin administration, Infection (pneumonia/UTI/gastroenteritis/sepsis), Infarction (cerebral, coronary, mesenteric, peripheral), Drugs (cocaine, Pregnancy

 Physical Findings
Tachycardia, Dehydration/hypotension, Tachypnea / Kussmaul respirations/respiratory distress, Abdominal tenderness (may resemble acute pancreatitis or surgical abdomen), Lethargy/obtundation/cerebral edema/possibly coma

 Lab. Abnormalities
DKA is characterized by hyperglycemia, ketosis, and metabolic acidosis (increased anion gap) along with a number of secondary metabolic derangements. Occasionally, the serum glucose is only minimally elevated. Serum bicarbonate is frequently <10 mmol/L, and arterial pH ranges between 6.8 and 7.3, depending on the severity of the acidosis. Total-body stores of sodium, chloride, phosphorus, and magnesium are reduced in DKA but are not accurately reflected by their levels in the serum because of dehydration and hyperglycemia. Elevated blood urea nitrogen (BUN) and serum creatinine levels reflect intravascular volume depletion. Interference from acetoacetate may falsely elevate the serum creatinine measurement. Leukocytosis, hypertriglyceridemia, and hyperlipoproteinemia are commonly found as well. Hyperamylasemia may suggest a diagnosis of pancreatitis, especially when accompanied by abdominal pain. However, in DKA the amylase is usually of salivary origin and thus is not diagnostic of pancreatitis. Serum lipase should be obtained if pancreatitis is suspected. Ketonemia is a consistent finding in DKA and distinguishes it from simple hyperglycemia.

 Treatment
- Stabilize ABC of life
- Fluid management
- Insulin
  +
- K repletion
- Treatment of precipitating factors
- Monitoring
- Long term management
General measures

- Stabilize the ABC of life
- Obtain IV access
- Monitor RBS every hour, urine ketone every 2-4 hrs
- Identify and treat Precipitating cause of DKA

Repletion of fluid deficit

- Give as much NS rapidly for a patient in shock
- Change the fluid to DNS when blood sugar falls to below 200
- Replace ongoing fluid loss
- The usual fluid deficit is about 3-6 liters

Repletion of $K^+$ deficit

- If baseline $K^+$ is $<3.3$ mEq/L, avoid insulin and administer 20 to 30 mEq/hour $K^+$ IV until $[K^+]$ is above 3.3 mEq/L.
- If baseline $K^+$ is 3.3-5.3 mEq/L or is unknown, administer 40 mEq/L to run over 4-8 hrs after confirming adequate urine output ($\geq 50$ ml/hr)
- If baseline $k^+$ is above 5.3 mEq/L, don’t administer $k^+$
- The target is to keep it between 4-5 mEq/L

Give insulin

- If perfuser and trained staff for monitoring of the rate of infusion is available:
  - Administer short-acting insulin: IV (0.1 units/kg), then 0.1 units/kg per hour by continuous IV infusion
  - Increase two- to three fold if no response by 2-4 h.
  - If the initial serum potassium is $<3.3$ mmol/L (3.3 meq/L), do not administer insulin until the potassium is corrected.
- If not:
  - Give initial bolus of 10IU IV and 10 IU IM of regular insulin
  - Then give 5 IU IV every one hour until blood sugar falls below 200 and urine ketone is twice negative
If RBS doesn’t drop by at least 50mg/dl or is persistently above 350-400, double the dose of insulin i.e. give 10 IU IV

- Overlap the last dose of regular insulin with the standing dose of long acting insulin

- In 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, insulin regimen should be started at a dose of 0.5 to 0.8 U/kg per day

- Measure RBS every 4-6hrs and give correctional dose of regular insulin (1-2 IU for every 50mg/dl rise above 200mg/dl)

**DKA follow up sheet**

<table>
<thead>
<tr>
<th>Date/Time</th>
<th>Vital signs</th>
<th>Mental status</th>
<th>Investigations</th>
<th>Management</th>
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<td>RBS (mg/dl)</td>
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<td>PR RR BP T° Input Output WT GCS Urine ketone</td>
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**Total**  **Total**
Hyperglycemic hyperosmolar state/HHS/

DKA and HHS differ clinically according to the presence of ketoacidosis and the degree of hyperglycemia. In HHS, 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, and neurologic abnormalities are frequently present (including coma in 25 to 50 percent of cases). Most patients with HHS have an 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. The absence of ketosis in HHS is not understood. It is possible that the liver is less capable of ketone body synthesis or that the insulin/glucagon ratio doesn’t favor ketogenesis. The patient with HHS is usually older, more likely to have mental status changes, and more likely to have a life-threatening precipitating event with accompanying comorbidities.

Treatment

The treatment of DKA and HHS is similar, including the administration of insulin and correction of the fluid and electrolyte abnormalities that are typically present, including hyperglycemia and hyperosmolality, hypovolemia, metabolic acidosis (in DKA), and potassium depletion.

Emergency management of hypoglycemia

Hypoglycemia

Hypoglycemia is a clinical syndrome with diverse causes in which low serum (or plasma) glucose concentrations lead to symptoms and signs. In patients with diabetes, hypoglycemia symptoms and signs occur as a consequence of therapy.

Causes of Hypoglycemia

1. Drugs (Insulin or insulin secretagogue, Alcohol, quinine)
2. Critical illness (Hepatic, renal or cardiac failure, Sepsis, severe malaria, Inanition)
3. Hormone deficiency (Cortisol, Glucagon and epinephrine (in insulin-deficient diabetes))
4. Non–islet cell tumor
5. Endogenous hyperinsulinism (Insulinoma), Functional beta-cell disorders (nesidioblastosis), Non-insulinoma pancreatic hypoglycemia, Post–gastric bypass hypoglycemia, Insulin autoimmune hypoglycemia, Antibody to insulin, Antibody to insulin receptors
6. Accidental, surreptitious or malicious hypoglycemia

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Clinical manifestations

Symptoms: Hypoglycemia causes neurogenic (autonomic) and neuroglycopenic symptoms.

Neuroglycopenic symptoms are those caused by CNS glucose deprivation and include behavioral changes, confusion, fatigue, seizure, loss of consciousness, and, if hypoglycemia is severe and prolonged, death.

The neurogenic symptoms include tremor, palpitations, and anxiety/arousal (catecholamine-mediated, adrenergic) and sweating, hunger, and paresthesias (acetylcholine-mediated, cholinergic). They are the results of the perception of physiologic changes caused by the CNS-mediated sympathoadrenal discharge triggered by hypoglycemia.

Signs: diaphoresis and pallor are the commonest signs of hypoglycemia. Tachycardia and systolic blood pressure elevations also occur. Occasionally, transient focal neurologic deficits may be seen. Permanent neurologic deficit may occur in patients with diabetes mellitus or prolonged hypoglycemia.

Diagnosis

It should be considered in any patient with episodes of confusion, an altered level of consciousness, or a seizure.

Whipple’s triad

(1) symptoms consistent with hypoglycemia,

(2) a low plasma glucose concentration measured with a precise method (not a glucose monitor), and

(3) relief of those symptoms after the plasma glucose level is raised.

* The lower limit of the normal fasting plasma glucose value is typically 70 mg/dL (3.9 mmol/L).

Glucose levels <55 mg/dL (3.0 mmol/L) with symptoms that are relieved promptly after the glucose level is raised document hypoglycemia.
**Treatment**

Oral treatment with glucose tablets or glucose-containing fluids, candy, or food is appropriate if the patient is able and willing to take these. A reasonable initial dose is 20 g of glucose.

If the patient is unable or unwilling, because of neuroglycopenia, to take carbohydrates orally, Initial management is administration of 1g/kg dextrose as 50% dextrose in water followed by the infusion of 10% dextrose at a rate to maintain the serum glucose above 100mg/dl.

Repeat bedside glucose determination should be done Q 30 minutes for the first 2 hrs to detect rebound hypoglycemia.

If intravenous therapy is not practical and glucagon is available, subcutaneous or intramuscular glucagon (1.0 mg in adults) can be used, particularly in patients with Type 1 Diabetes. Because it acts by stimulating glycogenolysis, glucagon is ineffective in glycogen-depleted individuals (e.g., those with alcohol-induced hypoglycemia)

**Activities**
- Discussion on case scenario # 11
- General discussion on questions raised by participants
**Toxicology**

**Learning Objectives**

By the end of this session, participants will be able to:

- Identify common toxicologic emergencies in our setup
- Develop a systematic approach to evaluating poisoned patients.
- Quickly identify and manage life threatening abnormalities in poisoned patients
- Describe the manifestations (toxidromes) of common poisons in our setup.

**General Management of Poisoned Patients**

A poisoning occurs when exposure to a substance adversely affects the function of any system within an organism. The setting of exposure may be occupational, environmental, recreational, medicinal or suicidal.

Poisoning may result from varied portals of entry including, inhalation, ingestion, cutaneous Exposure and injection. But most poisonings occur when substances are tasted or swallowed. Accidental or intentional poisonings or drug overdoses constitute a significant source of morbidity and mortality, so we should have a high index of suspicion.

The most commonly implicated poisoning exposures were due to analgesics, cleaning substances, cosmetics, sedative – hypnotics and antipsychotics, cough & cold preparations. Fatalities most commonly result from carbon monoxide poisoning, ingestion of analgesics, sedative – hypnotics and organophosphate compounds.

We will see the general approach to poisoned patient subsequently and common poisonings in our setup.

**General approach**

- We should have a consistent and systematic approach to evaluation and management of poisoned patients. Diagnosis and resuscitation proceed simultaneously.
- The first priorities are always the ABCs (Airway, Breathing & Circulation)
- Once the airway and respiratory status are secured abnormalities of blood pressure, pulse, temperature, oxygen saturation and hypoglycemia must be corrected. Vital Signs, mental status and pupillary size should be briefly assessed.
- Four possible etiologies of altered mentation in such patients can be corrected easily, Hypoxia, Opioid intoxication, hypoglycemia, and Wernick’s...
encephalopathy. Supplemental oxygen, Naloxone (for symptoms of opioid toxicity), 50 ml of D50W and 100 mg of thiamine known as the ‘coma cocktail’ should be administered.

- Identify the substance ideally through obtaining the original toxic substance container; ask detailed history about the type of exposure and amount of substance and route of exposure. (getting accurate history may be difficult)
- Plasma concentration, when available are essential for paracetamol, salicylate, carboxyhemoglobin for carbon monoxide poisoning, lithium, Digoxin and the likes.
- Useful investigations in most poisonings include serum electrolyte, blood glucose, arterial blood gas, liver and kidney function tests, INRs, urinalysis and an ECG.

Decontamination
- Remove all contaminated cloths from the patient and dispose it.
- Wash skin and hair with soap and water while wearing gloves
- Eye exposure: irrigate with copious amounts of water or saline for 10-15 minutes.

Gastric lavage
Indicated for ingestion of large amounts of tablets and capsules with a high inherent toxicity within 2hrs

Method:
- Insert a large bore orogastric tube, 32-40 F in adults & 24-28F in children.
- Place patient in left lateral decubitus position
- Aspirate fluid from stomach prior to fluid lavage
- Install water or saline 200 – 300 ml in to stomach for adults, 10ml/kg in children.
- Aspirate fluid back, repeat lavage until aspirate clear of debris or pill fragments.

Contraindications: patients with decreased Level of Consciousness/LOC/, unprotected airway, ingestions of corrosive agents, hydrocarbons, and patients at risk of gastrointestinal hemorrhage.

Charcoal: minimizes systemic absorption from the Gastro Intestinal Track.

- Consider use if within 1hr of ingestion of the poisonous substance
- Given orally or via NG tube, 1-1.5g/kg as slurry in 400-800 ml of water.

Pediatric dose: Less than 6 years, 10g in 50-100 ml water, older children, 20-50g in 200-300ml water.

  o  shake vigorously to ensure adequate dispersion of charcoal
Has no value in strong acids, alkali, corrosives, heavy metals, lithium, organophosphate, paraffin, methanol and ethylene glycol ingestion.

- **Contraindication**: decreased LOC or unprotected airway.

- Multiple doses can enhance elimination of drugs already absorbed into the body by interrupting enterohepatic circulation of drugs excreted into the bile.

- After first dose of activated charcoal, follow up dose of 25g every 2 hours, or 50g every 4hrs until clinical condition improves.

- E.g. ingestion of life threatening amounts of carbamazepine, dapsone, quinine, phenobarbitone, Digoxin & sustained release formulations.

**Organophosphate & Carbamates poisoning**

- Organophosphates & carbamates are potent cholinesterase inhibitors capable of causing severe cholinergic toxicity following cutaneous exposure, inhalation or ingestion.

- These compounds are used as insecticides worldwide, common examples include Malathion & parathion.

- They inhibit the enzyme cholinesterase in the nervous system leading to accumulation of ach in the CNS, Autonomic nervous system & neuromuscular junction.

- Toxic dose: depends on substance

- Symptoms and signs may appear from within minutes up to 12 hrs (rarely longer) after exposure.

- Muscarinic effects: hyper secretion (increased sweating, salivation & bronchial secretion) constricted pupils, bradycardia, hypotension, bronchoconstriction, vomiting, diarrhea & urinary incontinence.

- Nicotinic effects: muscular weakness, fasciculation and weakness of respiratory muscles.

- CNS effects: restlessness, anxiety, headaches, convulsions, difficulty of breathing and coma

- Patients will also have characteristics garlic like odor that may assist in the diagnosis.

  - The clinical features can be remembered by the following pneumonics

  **SLUDGE**: Salivation, Lacrimation, Urination, Defecation, Gastric Emesis

  **DUMBELS**: Defecation, Urination, Miosis, Bronchorrhea, Emesis, Lacrimation, Salivation

  **Killer Bees**: Bradycardia, Bronchorrhea & bronchospasm.
Treatment
- Treatment consists of airway control, intensive respiratory support, general supportive measures, decontamination, prevention of absorption, and the administration of antidotes.
- Protective clothing must be worn to prevent secondary contamination of health care workers.
- Early post ingestion activated charcoal is indicated for patients presenting within one hour.

Antidotes
- **Atropine IV**: used for reversal of Muscarinic effects including pulmonary symptoms
- Initial IV test dose of 1mg provides a measure of severity, followed by 2-4 mg every 15 mins until full atropinisation is achieved. Maintenance therapy: continuous IV infusion of 0.05mg/ kg/ hr.
- The criterion of adequate therapy is control of excessive bronchial & oral secretions.
- As the patient improves, reduce the dose of atropine slowly, over 24 hours or longer.

Pralidoxime: a cholinesterase reactivating agent is used to treat both Muscarinic & nicotinic symptoms in severe poisoning.

Symptomatic and supportive
- Seizures: IV benzodiazepines
- Bronchospasm: inhaled ipratropium
- Hypotension: IV fluids, dopamine, noradrenalin

Monitoring:
- It is better to monitor such patients using a chart for all the parameters we should follow (you can find the attached follow up sheet in the annex part)
  - Muscarinic effects: hyper secretion, pupil size
  - Cardiovascular: ECG, blood pressure and heart rate
  - Pulmonary function: vital capacity, arterial blood gas if required.

2, 4 D poisoning:
- These compounds are herbicides used as weed killers on lawns and grain crops.
- Metabolic pathway or mechanism of toxicity is unknown,
- Toxicity results from dermal contact, inhalation or ingestion.
- Local exposure leads to eye and mucous membrane irritation, ingestion leads to nausea, vomiting and diarrhea.
- Hypotension, tachycardia, dysrithmias and also tachypnea form pulmonary edema are seen, muscle toxicity manifests as muscle tenderness, fasiculations, myotonia and rhabdomyolysis.
- Diagnosis is based on history of exposure
- Treatment is supportive, including decontamination, and respiratory support.

**Corrosives**
- Corrosives are chemicals primarily acids and alkali that cause tissue injury similar to a burn. Acids cause coagulation necrosis with eschar formation that limits penetration and depth of injury.
- Alkalis cause liquefaction necrosis and penetrate more deeply. Some corrosives can cause severe systemic toxicity and profound electrolyte disturbance.
- Common examples of alkali include bleach (Sodium Hypochlorite) & Ammonia found in disinfectants, heavy cleaners.
- Symptoms & signs usually appear within minutes and up to 12 hrs after exposure and are usually confined to the GIT.
- Gastrointestinal: chemical burns of oral cavity, esophagus or gastric mucosa with associated nausea and vomiting, epigastric pain, dysphagia or odynophagia.
- Significant injury: airway compromise or gastrointestinal perforation complicated by peritonitis, mediastinitis, infection, sepsis and shock.
- Investigations: guided by signs & symptoms
  - Endoscopy indicated within 24-48 hrs for severe or deliberate ingestions.
  - Gastric lavage & induction of vomiting are contraindicated.
  - Consider small amounts of milk or water orally to dilute corrosive.
  - Neutralization with PPI or H2 receptor antagonists not shown to reduce injury
  - Antacids are used for subsequent ulcer treatment;
  - Use of steroids & antibiotics has no benefit.

**Carbon monoxide poisoning**
- CO is an odorless, tasteless, colorless, non irritating gas formed by HC combustion. Atmospheric concentration is generally below 0.001%, but higher in urban areas & closed environments.
- CO poisoning is one of the leading causes of poisoning deaths. Smoke inhalation is responsible for most inadvertent cases of CO Poisonings; it demonstrates both seasonal & regional variation being most common drugs cold climates.
- CO binds to Hb with much higher affinity than oxygen (240×) forms carboxyhemoglobin and tissue oxygenation is impaired. Clinical findings are harshly variable and largely nonspecific, headache, malaise, nausea and dizziness commonly seen. We should also ask about loss of consciousness.
- Patients may manifest symptoms ranging from mild confusion to coma.
- Acute myocardial injury is common among CO poisoned patients & is associated with increased mortality.
**Diagnosis**: is based on a compatible Hx and physical exam in addition with an elevated carboxyhemoglobin level measured by cooximetry of a blood gas sample.

**Management**: The most important interventions in the management are removal from the source and administration of oxygen by face mask.
- Comatose patients should be intubated & mechanically ventilated using 100% oxygen.
- Hyperbaric oxygen: involves exposing patients to 100% oxygen under supr atmospheric conditions to decrease the half life of carboxyhemoglobin, used in potentially severe intoxication.

**Barbiturates**
- From the group of `sedative -hypnotics` that lower excitement and induce sleep.
- Most commonly used are amobarbital, butabarbital, pentobarbital & Phenobarbital which is long acting.
- Have a narrow therapeutic index.
- Clinical features f acute intoxication includes slurred speech, in coordination, unsteady gait and impaired attention or memory.
- Severe overdose leads to coma.
- Most common vital sign abnormalities are hypothermia, respiratory depression and hypotension.

**Treatment**
- Airway stabilization, intubation in severe overdose before GI decontamination.
- Volume expansion by rapid infusion of 1 – 2 L of isotonic fluid.
- Gastric lavage if within 1hr of ingestion.
- activated charcoal in multiple doses to reduce serum concentration.
- Forced diuresis with fluid loading and diuretic therapy is most effective for Phenobarbital.
- Hemodialysis and hemoperfusion are used to maximize barbiturate elimination reserved for patients who are deteriorating despite institution of aggressive supportive care.

**Snake bites**

The clinical manifestations of patients bitten by snakes are very different with different species of snakes.

When patients are bitten by snakes, it may just cause wound (dry bite), may produce bacteria & spores, tetanus & secondary infections or may inject venoms with systemic effects.
There are three main groups of envenomations:

- Cytotoxic: causes tissue loss & swelling, may lead to compartment syndrome.
- Neurotoxic: causes descending type of paralysis, patient may die of respiratory depression.
- Hematotoxic: Causes DIC, uncontrolled bleeding and organ dysfunction.
- Or a combination of the above groups.
- Knowledge of toxicity profiles of local snake species is vital.

**Diagnosis:**

- Diagnosis of snake bite is based on the presence of fang marks and a history consistent with exposure
- To a snake.
- Snake envenomations involve the presence of snakebites plus evidence of tissue injury.

**Management**

- Primarily assess the ABCs and then a complete head to toe exam:
- Airway: may be unable to clear secretions and exhibit hyper salivation
- Breathing: may be compromised by respiratory muscle weakness
- Assess for shock and signs of unusual bleeding (hematuria/ oozing from the wound)
- Neurological examination and cranial nerves
- Neurovascular status of limb, swelling and dermal necrosis
- In our country, the Pasteur institute is working on two antivenom for the species prevalent in
- Selected areas of the country, the details of their use and related topics will be discussed by the institute.

**Activities**

- Discussion on case scenario # 12-13
- General discussion on questions raised by participants
Chapter VIII : Assessment and management of trauma

- Duration -6 hrs

Learning objectives
By the end of this session, participants will be able to:

- List the initial assessment of trauma
- Describe Management principles of trauma patient

Introduction
Trauma or injury is defined as damage to the body caused by an exchange with the environmental energy that is beyond the body’s resilience.

- Polytrauma refers to injury to several physical regions or organ systems, where at least one injury or a combination of several injuries is life threatening.

- Trauma is considered the leading cause of morbidity and mortality globally for individuals between the ages of 1 and 44 yrs old.

- And is the third overall cause of death among all age groups.
  
  – Common causes were RTA, violent trauma, suicides and falls.

TRIMODAL DEATH DISTRIBUTION PATTERN
Deaths following injury occur in 3 distinct patterns:

✔ 1st peak: immediate deaths: 50%
  
  – Occur at the scene of the accident
  – Due to unsalvageable lethal injuries, which kill in seconds to min.
  – Can only be reduced by preventive measures

✔ 2nd peak: early deaths: 30%
  
  – Occur in the first few hours
  – The so called “golden hour” where by an emergency primary trauma care can salvage the patient
  – The deaths occur as a function of time

✔ 3rd peak: late deaths: 20%
  
  – Generally due to sepsis or multi-organ failure

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— Usually arise as a continuum of inadequate early care

The need for a systematic approach to trauma patients

- Injury kills in a certain reproducible time frame,

- The basic concepts of systematic approach to a trauma patient:

  1. To identify and treat the greatest threat to life first

  2. Lack of a definite diagnosis should never impede instituting life saving measures

❖ These principles translated to the *initial assessment and management of the trauma patient*
Mechanical Trauma Classifications:

Blunt traumas
- High energy transfer
  - High risk for multiple injuries
- Low energy transfer
  - Localized injuries with substantially lethal net energy transfer

Motor vehicle collisions
- Head on, rear-end, lateral….blunt

Penetrating traumas
- GSW’S
  - High velocity
  - Low velocity
- Stab wounds
- Impalements

Gunshot wounds – result in passage of a missile thru the body dissipating high amounts of kinetic energy to the surrounding tissues resulting in cavitations:
  - Permanent cavity formed by the missile tract
  - Temporary cavity formed by pressure wave of the passing missile – results in a wide area of damage beyond the missile tract.

Stab wounds - cause damage only to tissues it comes in contact with

Impalements – involve passage of sharp stakes through the body of the victim

☐ When dealing with penetrating injuries keep in mind 3 factors:
  1. Body part involved
  2. Character of the penetrating object
3. Amount of energy transferred

Preventive measures in trauma

**Primary prevention:** - By creating awareness and education of the public

**Secondary prevention:** - By various engineering methods like air bag, seat belt, properly built roads and bridge and building.

**Tertiary prevention:** - By provision of effective medical care at the scene, during transport and in hospital as well.

“Initial assessment and management” of a trauma patient

In severe trauma, resuscitation and assessment should be performed simultaneously. The initial evaluation and management diagnose and address life-threatening problems which can cause death or serious morbidity if not treated early. This is called primary survey.

- Refers to the systematic and practical approach in the management of a seriously injured patient that allows a rapid assessment and institution of life preserving therapy

- This process includes:

  I. **Preparation**

  II. **Triage**

  III. Primary survey and resuscitation

  IV. Adjuncts to primary survey

  V. Secondary survey

  VI. Adjuncts to the secondary survey

  VII. Continued post resuscitative monitoring and reevaluation

  VIII. Definitive care
I. Preparation

➢ Has two phases:

a. Pre-hospital phase:-

✓ Aim: rapid and smooth transfer from the scene of the accident to a hospital equipped and staffed to handle them

✓ We can either use a “load and go” principle or a “stay and play”

b. In-hospital phase:-

✓ planning and preparation

✓ Readily available equipment

✓ Good communication means with an identified team leader

✓ Standard precautions should be in place

II. Triage

• Refers to the process of prioritizing/ sorting trauma victims based on the severity of their injuries, likelihood of survival and available resources to provide treatment.

• Triage can be performed at various levels:

  – Pre-hospital triage : field triage

    • Aim:- identifying the most severely injured patients

    - establishing the most appropriate order for evacuating to hospitals

  – In hospital triage:

    • Aim: prioritizing access to resuscitation rooms and operating rooms

• Over-triaging vs. under-triaging

• Two types of triage situations occur in trauma:

  • Multiple casualty scenario – number and severity of the injuries proportional to the resources and ability at hand to render care

  • Priority is given to life threatening injuries followed by those with poly-traumas
• **Mass casualty scenario** – number and severity of injuries does exceed the capacity of the facilities and staff

• **In this case, those with the greatest chance of survival with the least expenditure of time, equipments and supplies are prioritized**

• **Common in natural disasters**

❖ Principles of triage:

- degree of life threat posed by the injury
  - *As per the principles of the primary survey (ABCDE’s of care....)*
- Injury severity
- Salvageability
  *Consider likelihood of survival*
- Resources available
- Time, distance and environment

**PRIMARY SURVEY**

The primary survey includes 5 components which should always be followed in strict order.

A. Airway Maintenance with Cervical Spine Protection
B. Breathing and Ventilation
C. Circulation and Hemorrhage Control
D. Disability /Neurological Status
E. Exposure/Environmental Control

After the 5 main component of the Primary Survey, continue with F.F.H.:

F. Foley Catheter
G. Gastric Tube
H. Hertz – Trauma Ultrasound (E-FAST)

**A. AIRWAY/CERVICAL SPINE PROTECTION**

1. • Clear the oropharynx of blood, mucus and foreign bodies.
   - Lift the angle of the jaw or the chin to prevent the tongue from falling back and obstructing the airway chin lift and jaw thrust maneuvers. (Don’t overextend the neck; the patient might have a spinal injury!).
Use of oropharyngeal tubes in patients with gag reflexes may induce vomiting and aspiration. Remember that oropharyngeal tubes have limited use! Perhaps their only use is in patients with orotracheal tubes, to prevent the patient from biting the endotracheal tube. Choose the correct length oropharyngeal tube. The distance between the angle of the mouth and the earlobe is an easy way to choose the right size tube.

2. If the above measures are not sufficient or if the patient is unconscious, endotracheal intubation is the next step. (Size 8 for adult males, size 7 for females, or the size of the patient’s small finger irrespective of age).
   - Apply cricoids pressure during intubation to prevent aspiration. Keep applying the pressure until the cuff of the tube has been inflated. Make sure that the tube is in the correct place by checking for CO₂ return, listening for bilateral breath sounds and obtaining a chest x-ray.

3. If endotracheal intubation is impossible (e.g. in severe facial trauma), the next step is a cricothyroididotomy. In emergencies there is no place for tracheotomy. In patients with short, fact necks, the procedure can be difficult. If the anatomy is difficult a vertical incision may be more appropriate.

4. In desperate cases, two or more large-bore needles in the cricothyroid space may be lifesaving.

5. Monitor oxygen saturation (pulseoximetry)

Cervical spine protection
- High index of suspicion depending on the history of the accident: (traffic accidents, falls, certain sports)
- Avoid rough manipulation of the head and neck. Use sand bags or hard collars to immobilize the neck. Immobilize the whole body on a long spinal board.
- Obtain adequate x-rays, which should an always included T₁. If the patients is unconscious, maintain the neck collar even if the x-rays are normal. Radiological evaluation should be done only after the patient has been stabilized, if necessary after an emergency operation.
- Nexus criteria

B. BREATHING AND VENTILATION
- Put the patient on oxygen
- Inspect for symmetrical chest movements. Auscultate for breath sounds bilaterally. Palpate the trachea for deviation and the chest wall for fractures or emphysema.
- Life-threatening problems to be identified during primary survey:
- Life -threatening problems to be identified during primary survey:
1. Flail chest: monitor pulse oximetry and blood gases, intubate and ventilate if there is hypoxia or respiratory distress. Consider early intubation in elderly patients.

2. Open, sucking/blowing wound in the chest wall: Do not suture or pack before thoracostomy tube insertion. Danger of tension pneumothorax! A square gauze taped on only 3 sides can be applied while preparing for chest tube insertion.

3. Massive hemothorax

4. Tension pneumothorax: the diagnosis should be clinical patient should not be sent to x-ray department before decompression initial decompression with needle insertion thought the 2nd or 3rd intercostals space anteriorly, mid-clavicular line. Thoracostomy tube.

C. CIRCULATION AND HEMORRHAGE CONTROL

1. Assess level of consciousness, skin color, pulse, BP, capillary refill
2. Control any external bleeding by direct pressure.
3. In penetrating injuries of the neck, where venous injuries are suspected, put the patient in the Trendelenbrug position, (head down) to prevent air embolism.
4. If there is shock, insert two large intravenous lines and start fluid resuscitation.

Following trauma there are 3 groups of conditions which can cause shock: Hypovolemic Shock, Cardiogenic Shock, Obstructive shock, Neurogenic shock

1. **Hypovolemic Shock**

   - In by far the commonest
   - This could be due to external or internal blood loss.
Vascular access and volume replacement are of critical importance. Two or more large-bore intravenous lines are established. Assess to central veins can be achieved by means of subclavian or jugular or femoral vein catheterization. In patients with neck or arm injuries, the intravenous line should be inserted on the opposite side to avoid extravasation of the infused fluid from a proximal venous injury.

In children younger than 6 years consider intra-osseous infusion, if a peripheral vein is not available.

The infusion rate depends on the length and diameter of the catheter and NOT on the size of the vein.

Give a fluid challenge of 2-3 liters of Ringer’s Lactate/NS (or 20 ml/kg for children). If more fluids are needed, consider blood transfusion and perhaps operation. However, if the patient has a clear indication of surgery no time should be wasted for fluid resuscitation! There is evidence that in penetrating trauma with active bleeding some degree of mid hypotension until the bleeding is surgically controlled may be beneficial!

<table>
<thead>
<tr>
<th>Blood Loss</th>
<th>Heart rate</th>
<th>Blood Pressure</th>
<th>Capill Return</th>
<th>Resp Rate</th>
<th>Mental State</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;750</td>
<td>&lt;100</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>750-1500</td>
<td>&gt;100</td>
<td>Systolic Normal</td>
<td>Prolonged</td>
<td>20-30</td>
<td>Mildly Anxious</td>
</tr>
<tr>
<td>&gt;1500-2000</td>
<td>&gt;120</td>
<td>Decreased</td>
<td>Prolonged</td>
<td>30-40</td>
<td>Anxious Confused</td>
</tr>
</tbody>
</table>

**Blood**

- O Rh negative: No need for typing or cross-matching for life-threatening blood loss only. Available in refrigerator in the Emergency Room and Operating Rooms.
- Typing but no cross-matching. (“Type specific blood”) ready in about 10 minutes.
- Fully typed and cross matched. Ready in about 30 minutes.
- Always use micro-filter to prevent micro-embolization to the lungs. Use blood warmers. Hypothermia may aggravate acidosis, induce arrhythmias, shift the oxyhemoglobin dissociation curve to the left, and impair platelet function.
2. **Cardogenic Shock, obstructive Shock**

This should be suspected in trauma patients with shock in the absence of blood loss. The blood pressure is low and the neck and peripheral veins are distended.

- The following condition may be associated with cardiogenic shock: cardiac tamponade, myocardial contusion, tension pneumothorax, air embolism, and myocardial infarction.
- Air embolism may follow injuries to major veins, lungs, or the low-pressure cardiac chambers. Occasionally it may be iatrogenic, during insertion of a central venous line. Sudden deterioration of a patient in the presence of one of the above injuries should alert the doctor to the possibility of air embolism. Something “sloshing” sounds may be heard over the heart. The treatment consists of positioning the patient in the Trendelenburg position, Thoracotomy and direct aspiration of the air from the heart. In lung injuries, cross-clap the hilum to control the source of air embolism.
- In cardiac tamponade-pericardiosynthesis
- Myocardial infarction should be suspected in elderly patients presenting in cardiogenic shock. ECG and Troponins should be performed routinely.

3. **Neurogenic shock**

This is the result of loss of vascular tone following cervical cord or upper thoracic spinal cord injury. Patients will have low pulse rate and warm skin temperature in contra disticion to hypovolumic shock.

D. **DISABILITY (NEUROLOGICAL EVALUATION AND MANAGEMENT)**

1. Assess level of consciousness (Glasgow coma Scale/AVPU)
2. Assess pupils (size, reactivity).
3. Lateralizing signs

E. **EXPOSURE/ENVIRONMENT CONTROL**

1. Undress the patient completely for thorough examination.
2. Keep the patient warm with blankets and warm IV fluids. Trauma patients become hypothermic very quickly. Severe blood loss, elderly patients and pediatric trauma patients are at high risk for hypothermia.
3. Log roll the patient for complete examination of the back and the spine

Re-evaluate for response to resuscitative measures before going to the secondary survey

SECONDARY SURGERY

- The secondary server is done only after the primary survey (ABC’s) is completed and resuscitation is initiated. Sometimes the secondary survey is performed after operation for life-threatening injuries.
The AMPLE history is important (A-Allergy, M-Medication, P-Past illness, L-Last meal, E-Event)

Complete examination from head to toe (head and neck, chest, abdomen, back rectal and vaginal examinations, and musculoskeletal).

COMMENTS

1. Examination of the trauma patient:
   - Often this is very difficult because of intoxication, shock or head injury.
   - Undress the patient completely and always examined the back. Serious injuries may otherwise be missed. Cover the patient with warm blankets to prevent hypothermia.
   - The presence of an obvious wound should not distract from another less obvious but perhaps more dangerous injury elsewhere.

2. Head injuries:
   - Correct any condition which aggravates an existing brain injury (e.g. shock or hypoxial).
   - Cervical spine injury is a commonly associated problem. Apply a semirigid collar. Keep the head and neck in a neutral position, and apply precautions during transportation, until and cervical injury has been excluded. The cervical spine clearance is not an emergency as long as protection is maintained.
   - Closed head injuries alone rarely produce hypotension, except in the terminal stages or in neonates. If the patient is in shock, look for a source of bleeding or cardiogenic shock or associated cervical spine injury. Scalp lacerations can bleed profusely and may cause hypotension.

3. Fractures:
   - Immobilize all severe fractures at an early stage, before moving the patient to CT scan or other investigations. This will reduce pain, decrease bleeding, reduce fat embolism, and minimize neurovascular damage.
   - Fracture of the pelvis or the femur may be associated with significant blood loss.
   - Early operative fixation of major fracture decreases morbidity, mortality, and hospitalization. However, in the presence of sever associated head or chest trauma prior stabilization of the patient is advisable.
   - Pelvic binding or wrapping helps in reducing bleeding and pain from pelvic...
Common mistakes

- Insertion of an oropharyngeal tube in the presence of brisk gag reflexes.
- Tracheostomy in emergency situations. Problem: It takes a few minutes even in the hands of experienced surgeon! Procedure of choice: cricothyroidotomy.
- Cervical spine protection: soft collars offer no protection. Hard collars offer some protection. Always apply total body immobilization with spinal board during transportation. C-spine clearance is not an emergency as long as spinal precautions are maintained.
- External cardiac massage in traumatic cardiac arrest due to blood loss or cardiac tapenade. Procedure of choice: Thoracotomy and internal cardiac massage.
- Pack or suture open sucking/blowing wounds before Thoracotomy tube insertion. Problem: Tension pneumothorax! If a dressing is needed, use square gauze and tape it on to skin in only 3 sides!
- Examine a severely injured patient without removing his clothes. Problem: Serious injuries may be missed!
- Omit rectal or vaginal examinations, especially in pelvic fractures. (Do not perform routine vaginal exam in children) Problem: Serious injuries may be missed!
- The 3 most commonly missed injuries: a) Spinal injury; b) Spinal injury; c) Spinal injury.

Never admit directly a patient with suspicious mechanism of injury (traffic injuries, falls from significant height) to an orthopedic or neurosurgical unit. It is a disaster waiting to happen! Serious injuries may be missed. The trauma surgeon should be in charge from at least first 24 hours.

BASIC PRINCIPLES OF EMERGENCY WOUND EVALUATION

Introduction

A wound is a disruption of the normal structure and function of the skin and skin architecture. An acute wound has normal wound physiology and healing is anticipated to progress through the normal stages of wound healing, whereas a chronic wound is defined as one that is physiologically impaired.

To ensure proper healing, the wound bed needs to be well vascularized, free of devitalized tissue, clear of infection and moist. Wound dressings should eliminate dead space, control exudate, prevent bacterial overgrowth, ensure proper fluid balance, be cost-efficient, and be manageable for the patient and/or nursing staff. Wounds that demonstrate progressive healing as evidenced by granulation tissue and epithelialization can undergo closure or coverage.
**Principles of Initial Evaluation**

Evaluation of the patient with a traumatic wound begins with overall patient assessment—Airway, Breathing and circulation. Less obvious but more serious life-threatening injuries need care before directing attention to wound management.

- Determine the patient's past medical history and circumstances surrounding the injury.
- Remove rings or other jewelry that encircle the injured body part as soon as possible so they do not act as constricting bands when swelling progresses. Remove clothing over the injured area to reduce the potential for contamination.
- External bleeding can usually be controlled by direct pressure over the bleeding site. When possible, replace skin flaps to their original position before applying pressure in order to avoid exacerbating vascular compromise.
  - Tourniquet application is rarely needed. Two exceptions are when an arterial tourniquet is necessary to stop life-threatening exsanguination or when a tourniquet is needed for a short period to create a "bloodless" field for wound inspection.
- Amputated fingers or extremities should be covered with a moist, sterile, protective dressing, placed in a water proof bag, and then placed in a container of ice water for preservation and consideration for future reattachment.
- Before wound exploration, cleansing, and repair, most patients will need some form of anesthesia.

**History and Comorbidities**

Proper wound management begins with a pertinent patient history.

A variety of patient factors have adverse effects on wound healing and increase the rate of wound infection—extremes of age, diabetes mellitus, chronic renal failure, obesity, malnutrition, the use of immunosuppressive medications, the presence of connective tissue disorders such as Marfan syndrome, osteogenesis imperfecta, and protein and vitamin C deficiencies.

The most predictive factors for infection are the wound characteristics of location, age, depth, configuration, and contamination.

- Obtain a detailed history of allergies or prior adverse reactions to anesthetic agents or antibiotics. -Determine the status of prior tetanus immunization and the need for further tetanus vaccination
- Review the mechanism of injury to identify the presence of potential wound contaminants and foreign bodies.

- Bite wounds are at high risk for infection and generally managed differently than other lacerations.

- Foreign bodies are common in puncture wounds, wounds associated with broken glass, and motor vehicle collisions. Ask about the presence of a foreign body sensation. **In adults, those reporting a foreign body sensation are more likely to have a retained foreign body than those who do not.** This question has little utility in children. Both foreign body retention and visible contamination increase the risk of infection. Organic and inorganic components of soil can cause infection even from very small doses of bacterial inoculum. **Clay** is the major inorganic soil component responsible for infection. Conversely, sand grains and black dirt from roadways are relatively inert.

- The likelihood of wound infection varies according to the forces applied as the time of injury. The most common mechanism for traumatic wounds is blunt force. The skin is crushed against underlying bone and tears or splits from the subsequent tension.

- Sharp objects produce shear forces that cut skin cleanly.

- Crush injuries produce more tissue devitalization and are more susceptible to infection than wounds from shear forces.

- Low-energy impact injuries may not result in lacerations, but instead may disrupt vessels, leading to ecchymosis or hematoma formation. Some hematomas spontaneously resorb. Those that become encapsulated may eventually require aspiration or incision and drainage.

- Determine the time that the injury occurred:

  - The growth of the bacterial inoculum is directly related to the time interval from injury to laceration repair. But, time from injury until presentation is only one element to be considered, in addition to the **wound etiology, location, degree of contamination, host risk factors, and the importance of cosmetic appearance**, before determining whether or not to perform primary wound closure.

  - **Wounds that are not closed primarily because of a high risk of infection should be considered for delayed primary closure after 4 days.** After 4 days of open wound management, the risk of infection after closure substantially decreases.
- The anatomic location of the injury helps predict the clinical outcome, both in terms of infection risk and cosmetic result. The risk of infection is determined largely by the interplay between baseline bacterial colonization and vascular blood supply.

- The density of the bacterial population is low on the upper arms, legs, and torso. Conversely, moist areas of the body, such as the axilla, perineum, toe webs, and intertriginous areas, harbor millions of bacteria per square centimeter, including anaerobes. Obviously, any wounds with human or animal fecal contaminants run a high risk of infection, even with therapeutic intervention.

- Wounds located on highly vascular areas, such as the face or scalp, are less likely to be infected than wounds located in less vascular areas. The increased vascularity of the area more than offsets the high bacterial inoculum found in the scalp. Lacerations of the scalp and face have a very low infection rate regardless of the intensity of cleansing.

**Wound examination**

Thorough wound examination should be conducted when the patient is calm and cooperative and positioned appropriately, with optimal lighting conditions, and with little or no residual bleeding. Cursory examination under poor lighting or when the depths of the wound are obscured by blood will occasionally result in poor detection of foreign bodies, tendon, nerve, and vascular injuries. If bleeding is a problem, epinephrine-containing anesthetic solutions may be helpful, when not contraindicated. *Finger tourniquets may be used to obtain a bloodless field, but they should not be used for more than 30 minutes.*

Lacerations over joints may have penetrated the joint capsule, and sometimes it is necessary to inject the joint to ensure that there is no communication between the joint space and the laceration. Evaluate the wound in neutral position and also in the position present during injury. Repositioning the joint or extremity in the position assumed during injury can better reconstruct the mechanism of injury and identify injured structures. Lacerations over the metacarpophalangeal joints are suspicious for having occurred during a fight (clenched fist injury) and should be treated as though they are human bites.

**Adjunctive Testing**

Although most lacerations will not require any diagnostic testing, on occasion, wound imaging for detection of foreign bodies may be necessary. Most foreign bodies commonly found in wounds are much denser than the surrounding tissue and are readily apparent on plain radiographs. Metal, bone, teeth, pencil graphite, certain plastics, glass, gravel, sand, some fish bones, some painted wood, and most aluminum are visible on plain radiographs.
Almost all (>95%) glass fragments are visible on radiographs if they are 2 mm or larger in size. If the wound was caused by metal or glass and no foreign body is found on wound exploration or on plain films, it is unlikely that a foreign body is present. Radiopaque skin markers, such as paper clips, can be placed around the wound entrance so the position of the object can be determined relative to these markers.

Some objects will not be identifiable with plain radiography. CT and MRI are useful for identifying and locating objects that have densities similar to soft tissue. Sonography may also be useful, particularly for wooden foreign bodies, although the sensitivity of sonography is inadequate to reliably exclude small (<2.5 mm) wood fragments.

**Wound preparation**

Wound preparation is the single most important step in treating a traumatic wound. Proper ED wound management can help restore integrity and function of injured tissue, minimize the risk of infection, and assure the best possible cosmetic result. The majority (80% to 90%) of wounds treated in EDs heal with a good outcome. However, careful preparation is particularly important when underlying medical conditions (DM, malignancy, etc.) affecting wound healing is present.

**Sterile Technique**

Full sterile technique, with the physician wearing hair cap and face mask in addition to sterile gloves, does reduce the incidence of postrepair infections.

**Anesthesia**

In general, pain control should be provided before extensive wound preparation. Not only is this more humane, the administration of anesthesia and analgesia will enable better preparation and treatment if patients are relaxed and able to cooperate without undue anxiety and pain. Prior to the administration of local or regional anesthetic, the sensory, motor, and vascular examination should be performed at, and distal to, the wound site.

Sensory examination should include evaluation of pain, temperature, touch, pressure, and/or position. Motor examination should assess movement and strength of tendons and muscles around the wound site as well as muscles that are innervated by nerves traversing the site. Vascular examination should assess distal perfusion by noting skin color, temperature, capillary refill time, and quality of pulses.

Two additional assessments may be required before local or regional anesthesia:

- testing of two-point discrimination on the volar pads of the thumb and fingers and
- Comparison of the systolic blood pressure in the injured extremity with the non-injured one.
Two-point discrimination (<6 mm) checks for possible injury to the digital nerve. Systolic blood pressure comparison (using a Doppler stethoscope and pneumatic cuff) assesses for hemodynamically significant arterial obstruction.

Hemostasis

Control of bleeding is necessary for proper evaluation of a wound. Diffuse bleeding most often occurs from the subdermal plexus and superficial veins.

Direct pressure with saline-soaked sponges or gauze is usually effective in stopping this type of bleeding.

Once bleeding from a minor extremity vessel is halted, more permanent control can be achieved by clamping the involved vessel, isolating a short length, and ligating it with absorbable synthetic suture (typically 5-0).

Major arteries of an extremity should not be ligated, and surgical consultation is needed for further hemorrhage control if this type of bleeding is present. Exercise caution clamping vessels in facial wounds to avoid damaging facial nerves. Scalp lacerations can bleed extensively from the wound edges due to the highly vascular subcutaneous layer.

For bleeding wounds where the involved vessel is not visible, a figure-of-eight or horizontal mattress suture applied adjacent to the wound edge near the site of bleeding will sometimes achieve control. However, this technique may impair blood flow and leave nonviable tissue in the wound.

Chemical means of hemostasis is typically done using epinephrine mixed with local anesthetics in concentrations of 1:100,000 or 1:200,000 and injected into the wound area. This will induce local vasoconstriction that will allow a longer duration of anesthesia and a larger total local anesthetic dose due to the depot effect of the vasoconstriction. The use of epinephrine mixed with local anesthetics is safe for digital nerve blocks and in procedures on the nose and ears in patients without small vessel disease.

Extremity wounds that are refractory to direct pressure, ligation, or cautery may require an arterial tourniquet. Tourniquets may compress and damage underlying blood vessels and nerves, reducing tissue viability. The simplest tourniquet to use in an ED is a blood pressure cuff placed proximal to the wound and inflated above the patient's systolic pressure. Elevating the extremity to reduce venous blood volume prior to cuff inflation is useful. If an extremity tourniquet is needed to control bleeding, the best course of action is exploration and repair in the operating room.
Foreignbody removal

Obvious foreign debris should be carefully removed from the wound, using forceps. Probing wounds with a gloved fingertip to detect foreign bodies by palpation is discouraged. Clinical clues to the presence of a foreign body include foreign body sensation, point tenderness, or increased pain on range of motion. Visual wound inspection, down to the full depth and along the full course of the wound, is the most important method for detecting foreign bodies.

Skin disinfection

A common practice is to disinfect intact skin around the wound with either a povidone-iodine–based or chlorhexidine-containing agent. Although these agents suppress bacterial growth on intact skin, they impair host defenses and promote bacterial growth in the wound itself. Skin disinfectants should be applied from the wound edges outward and care taken to avoid spillage into the wound.

HairRemoval

Though wounds in well-perfused locations (i.e., scalp and face) may be closed without prior hair removal and with no apparent increase in infection, in general, hair can interfere with wound closure, becoming entangled in sutures or staples, and/or act as a foreign body, potentially increasing the risk of wound infection.

Shaving the area with a razor damages the hair follicle, allowing bacterial invasion, and is associated with an increase in infection rates when compared with clipping or a depilatory cream. Therefore, hair is best removed by clipping it 1 to 2 mm above the skin with scissors.

An alternative method to clipping is to use ointment or saline to allow hair to be parted away from wound edges. Hair should never be removed from the eyebrows or at the hairline because of the potential for impaired or abnormal regrowth.

Simple scalp wounds, without contamination or active bleeding, may be closed via the hair-apposition technique. Surrounding hair (>3 cm long) on either side of the laceration is brought together, twisted, and secured with tissue adhesive, thereby closing the wound. This technique is an alternative to more traditional methods of closure, with potentially fewer complications, less pain to the patient, and less overall cost.

Irrigation

Effective irrigation decreases bacterial count and helps to remove debris and foreign bodies, thereby reducing the risk of wound infection
- Low pressure irrigation is sufficient for uncontaminated wounds and for loose tissues around the scrotum or eyelids. It is achieved with a slow, gentle, wash with saline or water.
- High pressure irrigation should be used for wounds with high levels of contamination, especially in areas of the body that are at higher risk of infection such as the extremities. It can be easily obtained by any combination of syringes and 18 gauge intravenous catheters.

Although the exact volume of irrigant required is not known, a common recommendation is to use 60 mL per cm of wound length. Another recommendation is to use at least 200 mL for wound irrigation.

Sterile normal saline, the most commonly used irrigant, also has the lowest toxicity. However, tap water irrigation of acute and chronic wounds is as safe and efficacious as sterile normal saline. In addition, tap water is easily obtained in large quantities at almost no cost. Regardless of the irrigant, there is no added benefit to the addition of an antiseptic such as povidone-iodine or hydrogen peroxide.

**Debridement**

Devitalized tissue may increase the risk of infection and delay healing by acting as a culture medium and inhibiting leukocyte phagocytosis. Debridement not only removes foreign matter, bacteria, and devitalized tissue, but it also creates a clean wound edge that is easier to repair. After debridement is completed, wounds should be re-irrigated.

There are three methods of debridement

- **Autolytic debridement**: refers to the body's natural methods of wound healing through phagocytosis and lysis, a process that can be promoted by a moist wound environment. In the ED, it may be encouraged by the placement of occlusive dressings, which accelerate wound healing, are less likely to get infected, are less painful, and result in better cosmesis. Of note, this method of wound management is not recommended for infected wounds.

- **Mechanical debridement**: when applied to the ED refers mainly to irrigation (discussed above in Irrigation) and wet-to-dry dressings, which are useful mainly in the setting of necrotic, exudative wounds. Wet gauze is allowed to dry within the wound bed and then removed, thus taking viable and nonviable tissue nonselectively from the wound. This method may require the use of analgesia.

- **Excision**: the most effective type of debridement, because it converts a contaminated wound into a clean surgical wound.
Prophylactic antibiotics

There is no clear evidence that antibiotic prophylaxis prevents wound infections in most patients whose wounds are closed in the ED.

For wounds contaminated by debris or feces or caused by punctures or bites, wounds with tissue destruction or in avascular areas, and neglected wounds, sufficient bacteria may be present to cause infection, and prophylactic antibiotics are often administered.

- Prophylactic antibiotics are recommended for all human bites to the hands and feet as well as to those overlying joints or cartilage.
- For human bites in all locations and for mammalian bites on the hands, amoxicillin-clavulanate should be used to cover both Pasteurella and Eikenella.
- Prophylactic antibiotics do not reduce the incidence of wound infection after dog or cat bites on areas other than the hands. Antibiotics have an inconclusive role in intra-oral traumatic wounds.
- Wounds contaminated by fresh water and plantar puncture wounds through athletic shoes should include Pseudomonas coverage.
- The duration for antibiotic prophylaxis is unknown; most physicians use 3 to 5 days for nonbite wounds and 5 to 7 days for bite wounds. Patients with established wound infections usually require longer treatment

Management of Burns

Introduction

The commonest types of burns in our community are:

- Flame burns with associated burns caused by melted synthetics
- Liquids (i.e. scalds)

Although copious amounts of fluids and electrolytes can be lost, the commonest cause of death in the first hour is smoke inhalation. Hence early attention to Airway, Breathing then Circulation is vital.

Pathophysiology

Severity of Burns

- Dependent on temperature & duration of contact
- Scalds rarely cause more than partial thickness burns because the temperature of water is usually below boiling point and contact is brief.
Exceptions include

- Very hot liquids (e.g. fat)
- Prolonged contact seen in young infants unable to move away.

Flame burns involve high temperatures and often prolonged contact, which lead to severe burns.

**Classification of Depth**

1. **Superficial burns:**

   **Superficial epidermal burns**
   - To epidermis only
   - Skin pink or red, painful, no blisters
   - Not included in evaluation of burn area

   **Superficial dermal burns or Partial Thickness**
   - Injury to epidermis and dermis
   - Painful, blisters, oedema, hairs intact
   - Non-blanching indicates a more severe burn
   - Healing usually occurs without scarring

2. **Deep burns:**

   **Deep dermal burn**
   - May have some blistering
   - Base of the blister demonstrates an appearance of a blotchy red colouration = the loss of the capillary blush phenomenon. This demonstrates that the burn has destroyed the dermal vascular plexus.
   - The dermal nerve endings are also situated at this level and so in these burns sensation to pinprick will be lost.

   **Full Thickness**
   - Injury to epidermis, dermis and extending into subcutaneous tissue
   - Painless, white or charred, no hairs
   - Leathery to touch
   - Healing only occurs by epithelial migration or contracture
Primary Survey and Resuscitation

Airway and Cervical Spine

Airway compromise is due to:

- Inhalation
- Severe burns to face

Signs of inhalation injury include:

- Carbonaceous sputum/ Sputum containing Soot
  - Singed nostril hairs, oral erythema, blistering
  - Upper airway oedema
- Mechanism of Injury e.g. Burn / Explosion in confined space
- Burns above inter nipple line
- Singed nasal hairs
- Burnt red oral mucosa, Burns to Mouth, Nose and Pharynx
- Dysphagia
- Change in voice / dysphonia/hoarse voice
- Stridor
- Brassy cough
- Bronchospasm
- Respiratory Difficulty
  - Tracheal Tug
  - Rib Retraction
  - Indrawing of Supraclavicular Fossae
  - Nasal flaring

An unconscious patient has carbon monoxide intoxication until proven otherwise

Management:

Airway

Assess patency and support airway as needed
Oedema can rapidly develop therefore early intubation must be considered.
Rapid sequence using Suxemethonium can be performed if burns are less than 5 days old.
Consider performing intubation before signs of respiratory obstruction become evident particularly before transfer to upgraded medical care if inhalation injury suspected.

**Beware concurrent cervical spine injuries** – immobilise as necessary

**Breathing**

- Once airway is secured, breathing is assessed.
- All patients should initially receive high flow oxygen (humidified if possible and high concentration will wash out the excess carbon monoxide) via bag-valve-mask with reservoir or mask with reservoir
- If breathing inadequate, assist with bag-valve-mask ventilation with high flow oxygen.
  - Intermittent positive pressure ventilation with bag-valve-mask or with ventilator if saturation not adequate
- Attend to serious (life-threatening) respiratory conditions if present.

**Circulation**

- Insert I/V line x 2 into non-burnt area if possible
- Consider intraosseous route if above not possible
- Take blood concurrently for FBE, Coags, biochemistry, Group and Match, glucose.
- Treat shock if present (see below).

**Secondary Survey and Assessment of Burns**

**Surface Area**

**Rule of 9’s**

Applicable >14 years old.
- 9% each upper limb
- 9% head
- 18% each lower limb
- 18% back and front of torso
- 1% perineum
Infants and children
Lund-Browder Chart
• below are for 1 year olds.
  19% head
  12.5% each lower limb
  Other percentages as above
• For each additional year subtract 1% from the head and add to lower extremities.

Alternative method
The palm and adducted fingers of a patient constitute 1% of the body surface.

Depth
(1) Superficial
  • Epidermal burns
  • Superficial dermal/partial thickness burns
(2) Deep
  • Deep dermal burns
  • Full thickness

Special Areas
  • Face and mouth   airway compromise
  • Hands and feet functional loss if scarring
  • Perineum  prone to infection

Investigations
Determined by severity and extent of burns.
Consider :
  • CXR, ABG
  • FBE, U&E, CO level if available
  • Cross match

  • Urinalysis; haemoglobinuria/myoglobinuria

Secondary Survey and Management of Burns
Analgesia
• Use I/V opioids: e.g. Morphine titrated to effect

Running tap water or a Cool Saline pack and apply on the area
• Good symptomatic relief
• At least 20 minutes
• Beware of hypothermia
Fluid therapy
- Treat shock with fluid boluses (in paediatrics = 20ml/kg)
- I/V required if > 10% burns in children

>10% - 20% burns in adults
Fluid volume required:
(1) Maintenance fluid requirements, plus
(2) % burn X weight (kg) X 4 (in ml)
- 50% given in first 8 hours following time of burn, remainder in next 16 hours.
(Estimate time since burn and not since arrival of patient)
- This is a guide only and should be monitored.

- Aim for a urine output of:
  a) Adult > 50ml/hour
  b) Paediatrics 1-2ml/kg/hour
- Treat as per trauma patient with monitoring of vital signs rather than with blind formulae but is a good starting point

Wound Care
- First aid:
  – Running tap water for at least 20 min to cool wound.
  – Running tap water for at least 45 min to clear chemicals in chemical burns
  – Visible chemicals to wipe off with dry cloth before wetting.
  – Prevent hypothermia
- For transport and after first aid all burns should be dressed with Burnshield, gauze, sheets or non-stick dressings and crepe bandages.
- Wet dressings predispose to hypothermia.
- Glad wrap can be used but often slides off predisposing to infection. (Beware of circumferential glad wrap – rather in longitudinal strips and wrap then with a crepe bandage
- Facial burns should be dressed with Vaseline or sterile emollient like Flamazine.
- Chloromycetin ointment should be applied to eyes and eyelids.
Management of orthopaedic emergencies

General principles

- Orthopaedic emergencies on their own are rarely life threatening, but associated injuries can be. They should be dealt with only after attention to assessment of Airway, Breathing, Circulation, Disability and Exposure with basic and advanced life support measures instituted as necessary. Patients with orthopaedic emergencies should be assessed like any other multi trauma patient.

Life-threatening orthopaedic emergencies can occur in the following situations:

• Where there is major uncontrolled haemorrhage
  – Pelvic fractures
  – Bilateral fractured femurs
  – Multiple fractures where the cumulative blood loss is enough to cause circulatory collapse

• Crush injuries
  – The breakdown of large amounts of necrotic tissue can lead to renal failure.

• Open fractures
  – Can be potentially fatal due to overwhelming sepsis.

Limb threatening injuries can occur in the following situations:

• Vascular compromise
  – Distal ischaemia
  – Compartment syndromes
- Localised necrosis and ischaemia
- Open fractures with associated vascular injury
- Dislocations of major joints

**Principles of Management of Fractures and Dislocations**

The management of life-threatening orthopaedic emergencies due to bleeding usually takes place as part of the primary survey. Management usually entails the stop of the bleeding by immobilization, traction, tourniquet, direct pressure, etc.

The management of non-life-threatening orthopaedic emergencies takes place as part of, and following, the secondary survey and includes the following:

- **Assessment of perfusion**
  - Colour
  - Capillary refill
  - Distal pulses
- **Identification of open fractures**
- **Identification of closed fractures**
  - Swelling
  - Deformity
  - Loss of function
- **Assessment of neurological function**
  - Sensation
  - Movement
- **Identification of abnormal joint mobility**

- Initial management involves analgesia with reduction of gross deformities and where appropriate the application of traction and immobilisation, prior to definitive treatment.

- Initial management of compound fractures in addition to the above involves application of sterile water or Saline dressings after irrigation with copious water to the wound or bone ends, antibiotics (Kefsol 2g IVI stat and 1g 6 hourly in adults) and tetanus prophylaxis and then referral for definitive treatment and debridement in theatre.
Fractured Clavicle

This is usually caused by a fall onto the outstretched hand. The lateral fragment is distracted downwards by the weight of the arm.

Clinical features

- There is usually tenderness at the junction of the lateral third and medial two-thirds of the clavicle. There is often an obvious tender lump present at this site.

Treatment

- Support the weight of the arm in a broad arm sling.
- Analgesia
- Early mobilisation

Complications

- Rare apart from persistent lump in the region of the fracture
**Fractured Neck of Humerus**

The fracture of the neck of humerus is commonly caused by a fall onto the outstretched hand. The shaft of the humerus impacts into the humeral head.

**Clinical features**

- Swelling and tenderness of the shoulder
- Attempted movement is painful; less so with impaction

**Treatment**

- Traction and immobilisation in a collar and cuff sling. Initially or for transport, the arm may feel more comfortable if it is bound to the chest with a crepe bandage.
- Analgesia
- Patients are generally most comfortable sitting upright when transported
- Definitive treatment may involve internal fixation

**Complications**

- Easily missed (especially when there is impaction)
- Nerve injury - radial or circumflex humeral

**Fractured Shaft of Humerus**

These injuries usually occur as a result of a fall onto an outstretched hand or from a direct blow

**Clinical features**

- Often gross swelling and deformity
- The arm may be unstable and crepitus may be elicited
- The patient is usually unwilling to move the arm and often supports it with the opposite arm
- Radial nerve damage may lead to impaired finger and wrist extension as well as decreased sensation over the dorsum of the hand (it must be tested and documented)
Treatment

• Analgesia

• Traction and immobilisation using a collar and cuff sling, with stability provided by bandaging the arm to the chest wall with a crepe bandage.

• Definitive treatment may involve internal fixation

Complications

• Radial nerve palsy

Supracondylar fracture (kids)

These injuries usually occur in kids as a result of a fall onto an outstretched hand/arm (can also occur in adults)

Clinical features

• Gross swelling and deformity of distal humerus

• Tenderness distal humerus

• Medial nerve damage may lead to weakness of the abductor pollicis as well as decreased sensation over the radial three and a half fingers (must be tested and documented)

• Posterior displacement of the distal fragment can cause damage to the brachial artery or produce pressure due to swelling. This can lead to limb loss!

Treatment

• Analgesia

• Arterial damage/risk indicated by pain, pallor, paralysis, paraesthesiae, pulselessness and cold needs immediate reduction (in theatre)!

• Immobilisation in a plaster back slab and broad arm sling

• Definitive treatment involves reduction and closed/internal fixation

Complications

• Medial nerve palsy

• Brachial artery damage or occlusion
**Fractured Forearm**

Forearm fractures may be due to a direct blow or more commonly a fall onto the outstretched hand

**Clinical features**

- Deformity of the forearm
- Soft tissue swelling
- Movement of the elbow may be very painful
- Pulses may be compromised if deformity is marked

**Treatment**

- Analgesia
- Immobilisation with a plaster back slab initially
- Broad arm sling
- Definitive treatment involves open reduction and internal fixation

**Complications**

- Nerve and vascular injury

**Fractured Wrist**

This injury is one of the commonest of fractures. The Colles fracture is one of the commonest wrist fractures. Wrist fractures occur most commonly after a fall into the outstretched hand in geriatric patients

**Clinical features**

- Tenderness over the distal radius or ulna
- Deformity—the classic Colles fracture deformity being “dinner fork“ in nature—representing dorsal displacement, dorsal angulation, radial displacement and radial angulation.
- Limited wrist movement
**Treatment**

- Analgesia
- Immobilisation in a plaster back slab
- Broad arm sling
- Reduction of displaced fractures - if appropriate to the operator’s skill level. This may be carried out under regional anaesthesia; either Bier’s block or haematoma block if done under sterile conditions
- Immobilisation in a below elbow cast (once adequately reduced). Backslab initially for one week due to the swelling
- Open reduction and internal fixation are sometimes required

**Complications**

- Nerve and vascular injury are uncommon
- Complications may be secondary to immobilisation in plaster - Wrist stiffness from prolonged immobilisation - especially in the elderly.
- Wrist stiffness may occur with a fracture of the joint
- Reflex sympathetic dystrophy

**Management of Fractures - Lower Limb**
**Pelvic Fractures**

Pelvic fractures may result from a direct blow or crushing injury or from violence transmitted along the femoral shaft such as motor vehicle accidents or falls from a height.

**Clinical features**

- Depend on the type of fracture
- Bruising may be seen early especially around the perineum
- Severe pain may be present and may not be well localised
- Blood loss may be significant and result in hypovolaemic shock
- There may be pain on leg movement depending on the site of injury
- Tenderness may be elicited when the iliac crests are sprung or when pressure is applied to the pubic symphysis
- Crepitus may be felt

**Treatment**

- Shock should be anticipated, with large bore I/V access X 2 obtained
- Treat hypovolaemia comprehensively
- Analgesia
- Immobilisation - one of a few situations where a MAST suit may be useful or a sheet around the pelvis and tied tight (wet the knot to keep it tight)
- Urinary catheterisation should be considered, though care should be taken to assess for urethral rupture
  - Binding of “open book “ fracture with sheet or equivalent
  - Hypotensive resuscitation (need to discuss)

**Hypotensive resuscitation:**

“The goal of resuscitation is to restore organ perfusion. This is accomplished by the use of resuscitation fluids to replace lost intravascular volume, and has been guided by the goal of restoring normal blood pressure. It has been emphasized that if blood pressure is raised rapidly before the haemorrhage has been definitely controlled,
increased bleeding may occur. This may be seen in the small subset of patients in the transient or non responder categories. Persistent infusion of large volumes of fluid in an attempt to achieve a normal blood pressure is not a substitute for definitive control of bleeding. Fluid resuscitation and avoidance of hypotension are important principles in the initial management of blunt trauma patient. Although complications associated with resuscitation on injury are undesirable, the alternative of exsanguinations is even less so. A careful balanced approach with frequent re-evaluation is required. Balancing the goal of organ perfusion with the risk of rebreeding by accepting a lower than normal blood pressure has been called “controlled resuscitation” or “permissive hypotensive resuscitation”. The goal is the balance and not the hypotension.”

Complications

- Hypovolaemic shock
- Ruptured bladder
- Ruptured urethra

**Fractured Hip - Fractured Shaft of Femur**

The femur is the largest bone in the body and therefore requires significant force to break it. Fractures of the femur typically occur in high energy impacts such as motor vehicle accidents. There is usually significant associated blood loss

Clinical features

- Shortening and lateral rotation of the leg
- Deformity of the thigh
- Swelling of the thigh due to haemorrhage
- Loss of function - the patient is usually unable to move the leg

Treatment

- Shock should be anticipated, with large bore IV access X 2 obtained
- Treat hypovolaemia
- Analgesia
- Femoral nerve block provides excellent analgesia for this injury
- Traction and immobilisation by application of a traction splint e.g. Thomas splint, Trac-3 or skin traction (3 kg) for transport to definitive care
Definitive treatment involves internal fixation

Complications

- Hypovolaemic shock
- Vascular damage

Fat embolism if not immobilised

**Fractured Tibia and Fibula**

- Lower leg fractures commonly occur as a result of motor vehicle and sporting accidents.
- They are varied in their nature.
- Patterns of injury include spiral fracture of the tibial shaft and fibula, resulting from a twisting injury, or a transverse fracture that usually results from a direct blow.

**Clinical features**

- Marked tenderness and swelling of the lower leg
- Crepitus and instability
- Compound fractures are common at this site

**Treatment**

- Analgesia
- Immobilisation in a plaster back slab - initially or for transport
- The application of gentle traction during splinting will often significantly reduce pain
• Undisplaced fractures may be treated with a long leg cast, but open reduction and internal fixation is becoming an increasingly common form of management for this injury

Complications

• Vascular injury with resultant circulatory compromise to the foot
• Skin compromise if severely displaced fracture

Fractured Ankle

These fractures are common sporting injuries. They commonly occur as a result of a twisting, inversion or eversion injury

Clinical features

• Often marked swelling
• Tenderness maximal over the fracture site
• Loss of function - patient usually unable to weight bare

Treatment

• Initial management involves immobilisation with a plaster back slab
• Undisplaced stable fractures may be treated with the below knee cast.
• Unstable fractures require open reduction and internal fixation e.g. bi- and trimalleolar fracture, med malleolus fracture on it’s own etc

Complications

• Osteoarthritis of the ankle joint may be late complication following inadequate reduction of intra-articular fractures.

Management of Dislocations - Upper Limb

Dislocated Shoulder-Anterior

This is a common sporting injury and is due to a fall on the hand, arm or elbow where the head of the humerus is driven forward. The head of the humerus tears through the anterior joint capsule and ends up inferior to the clavicle - antero-inferior to its usual position.
Clinical features

• Pain may be variable but is usually moderately severe

• The patient is usually supporting the affected arm with the opposite arm

• There is a loss of the normal contour of the shoulder

• The acromion is more prominent and there is a depression below it where the humeral head once was

• The displaced humeral head may be palpable antero-inferiorly in the region below the clavicle

• There is often loss of sensation in the distribution of the axillary nerve over the lower deltoid (Test and document it)

Treatment

• Analgesia

• Reduction is best achieved as soon after the injury is possible. Delay often makes reduction more difficult due to muscle spasm.

• Where possible x-ray examination of the shoulder should take place prior to attempts at reduction. This may not be necessary if there is little doubt about the diagnosis and where there is likely to be an inordinate delay in obtaining an x-ray. Care must be taken in the middle aged / elderly patient where the possibility of fracture / dislocation is more likely.

• I/V analgesia and sedation may be required - Diazepam or Midazolam as sedation
and Morphine as analgesis (diluted and titrate to effect - remember to wait 15 min for morphine to take effect before sedation is given). Blood pressure should be monitored regularly.

• There are a variety of reduction techniques. The commonest is the Kocher’s method and involves:
  - Traction with arm flexed at elbow
  - External rotation
  - Adduction

• The shoulder usually reduces with a ‘clunk’

• Failure of reduction under sedation is an indication for general anaesthesia

• Once reduced the shoulder should be supported in a broad arm sling for three to four weeks with moderate mobilization during that time (if can by physiotherapist).

**Complications**

• Associated fracture of the greater tuberosity of the humerus - this is usually of little significance

• Nerve injury - commonly involving the axillary nerve but possibly the posterior cord of the brachial plexus

• Vascular injury - damage to the axillary artery

• May become recurrent.

**Dislocated Elbow**

This painful injury usually results from a fall onto the outstretched hand

**Clinical features**

• The patient is usually in moderate to severe pain

• The elbow is swollen and deformed and there may be a step palpable on the extensor aspect of the joint

• There is loss of function with no movement of the joint possible

• There may be associated median or ulnar nerve impairment

**Treatment**

• Analgesia

• I/V sedation may be required prior to reduction

- Morphine and diazepam or Midazolam – see above
• Reduction - Requires two operators - Traction is applied in the line of the forearm - Two handed counter traction is applied to the upper arm by the second operator while applying forward pressure on the olecranon process with both thumbs.

**Following reduction:**
• Support in a collar and cuff sling
• Plaster Backslab X 1 week and then mobilization

**Complications**
• Ulnar and median nerve injury. This should be assessed for both before and after injury as reduction may cause entrapment of nerves
• Elbow stiffness is a common sequel

**Management of Dislocations - Lower Limb**

**Dislocated Hip**

– This is most commonly a posterior dislocation and results from a high-energy impact where a force is applied to the femur when the hip is flexed.
– There is potential for damage to the sciatic nerve.
– Dislocation of the hip compromises the circulation to the femoral head.
– There is an increasing probability of avascular necrosis of the femoral head the longer the hip remains dislocated.
– The likelihood of this outcome is greatly increased with dislocations of greater than six hours duration.

**Clinical features**
• Patient presents in severe pain
• The hip is flexed, the thigh internally rotated and adducted
• There may be signs of sciatic nerve injury
• There may be signs of associated fractures to the pelvis

**Treatment**
• Analgesia
• Immobilise by supporting the leg on the affected side
• Reduction - requires general anaesthesia and should be carried out as soon as possible after the time of injury – may also do so under procedural sedation in the ED

**Complications**
• Sciatic nerve injury
• Avascular necrosis of the femoral head
• Osteoarthritis of the hip joint
**Dislocated Patella**

This is a common injury especially in young women. The patella dislocates laterally while tracking during extension of the knee.

**Clinical features**

- The patient is usually in moderately severe pain
- The knee cannot be moved
- The patella is palpable lateral to its normal position

**Treatment**

- Analgesia
- Reduction - Gentle medial pressure is applied to the patella as the knee is extended - The patella should “pop” back into place Post reduction the knee should be supported as per a soft tissue injury in a firm bandage like a Robert Jones bandage

**Complications**

- Osteochondral fractures of the under surface of the patella are uncommon but should be looked for on subsequent X-rays
- May become recurrent

**Activities**

- Discussion on case scenario # 16-20
- Simulation&practice in small group
- General discussion on questions raised by participants
Pain Management in the Emergency Department

Learning Objectives

- outline patho physiology of pain
- List principles of assessment of pain
- Outline the management principles of pain

Definition of pain-
- Acute Pain—an unpleasant experience with emotional and cognitive, as well as sensory, features that occur in response to tissue trauma.

I. Pathophysiology

- Classic descriptions of pain typically include four processes
- Transduction: the conversion of the energy from a noxious thermal, mechanical, or chemical stimulus into electrical energy (nerve impulses) by sensory receptors called Nociceptors.
- Transmission: the transmission of these neural signals from the site of transduction (Periphery) to the spinal cord and brain.
- Perception: the appreciation of signals arriving in higher structures as pain.
- Modulation: descending inhibitory and facilitory input from the brain that influences (Modulates) Nociceptive transmission at the level of the spinal cord.

II. Classification of pain on the basis of its presumed underlying path physiology

- Nociceptive pain—is caused by the ongoing activation of A-d and C-nociceptors in response to a noxious stimulus. The nervous system associated with nociceptive pain is functioning properly.
- Neuropathic Pain—caused by aberrant signal processing in the peripheral or central nervous system.

III. Assessment of pain

- It is an integral to effective pain management.
- Underassessment of pain is a major cause of inadequate pain management.
COMMON PAIN ASSESSMENT TOOLS

- Used to quantify pain or pain relief.
- The tool should be appropriate for the patient’s developmental, physical, emotional and cognitive status as well as reliable, valid, and easy to use.
- 1-uni-dimensional (rating scales) - usually assesses a single dimension of pain, patient self-report of pain intensity.
- Numerical Rating Scales-(NRS):
  - Patients rate their pain on a 0-to-10 scale or a 0-to-5 scale, with 0 representing “no pain at all” and 5 or 10 representing “the worst imaginable pain.
  - Pain intensity levels are measured at the initial encounter, following treatment, and periodically.
- Visual Analogue Scales:
  - Consists of a 10-cm line, one end is marked “no pain” and the other end is marked “the worst imaginable pain.
  - The patient marks the place on the line to indicate his or her pain intensity.
    The clinician then measures the line with a ruler and assigns a score.

Verbal Rating Scales
- No pain, mild pain, moderate pain, and severe pain to describe pain levels. A score from 0 to 3 is assigned to measure the pain level.

Wong-Baker FACES Pain Rating Scale- particularly useful for children who may not have verbal skills to express their pain level. Six faces are used that are numbered 0 to 5.

**Figure 4. Pain intensity scales**

![Pain Intensity Scales](image)
Assessment of Patients with Barriers to Communication

- Patient Populations:
  - Infants and children, Individuals of advanced age (e.g., older than 85 years),
  - Adults with emotional or cognitive disturbances,
  - Patients with cultural, educational, or language barriers to communication,
  - Intubated patients,
  - Patients who are seriously ill

General Approach

- Allow sufficient time for the assessment
- Give patient the opportunity to use a rating scale or other tool appropriate for that population.
- Use indicators of pain according to the following hierarchy of importance:
  - Patient self-report
  - Pathological conditions or procedures known to be painful
  - Pain-related behaviors (e.g., grimacing, restlessness, vocalization)
  - Reports of pain by family members or caretakers
  - Physiological measures (vital signs)
  - Rely on behavioral or objective indicators of pain (e.g., vital signs) only when no suitable alternative exists.

VI. Management of pain.

- Any type of pain should be managed without delay.

**Why do we manage pain?**

**Physiological Consequences of Unrelieved Pain**

- Endocrine/metabolic Stress Responses to Pain, Altered release of multiple hormones (e.g., ACTH, cortical, catecholamine increased and insulin release decreased) with Associated metabolic disturbances:

\text{Wong's Essentials of Pediatric Nursing, ed. 6, St. Louis, 2001, p. 1301. Copyrighted by}
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Hyperglycemia, sodium & water Retention, Protein catabolism Weight loss, fever.

- Respiratory response: Decreased air flow due to involuntary (reflex muscle spasm) and voluntary mechanisms that limit respiratory effort and coughing, that can complicated to accumulation of secretion with clinical manifestation of Atelectasis, Pneumonia, Arterial hypoxemia

- Cardiovascular response: Increased sympathetic outflow may lead to increased vascular resistance and cardiac workload, \(O_2\) demand and supply mismatch, HPN, Tachycardia, Myocardial Infarction, cardiac dysrhythmias

- Immune system response: Decreased reserve, increased catabolic state, Decreased immune function, Infection

- Coagulation system response: Increased platelet adhesiveness, hyper coagulation, Decreased fibrinolysis, immobility-DeepVein Thrombosis.

- Gastrointestinal system response: Decreased intestinal motility, Delayed gastric emptying: Ileus, constipation, Anorexia,

- Genitourinary system: Abnormal release of hormones that affect urine output fluid volume and Electrolyte balance Hypertension (fluid retention Electrolyte disturbances, Urine retention

Pain management approach

1. None pharmacological and/or
2. Pharmacological

1. Non pharmacological management

A. Psychological- cognitive behavioral therapy, biofeedback, relaxation, and psychotherapy.

B. Physical rehabilitative- reduces fear and anxiety, improve physical function, and alter physiological responses to pain
- Thermotherapy (application of heat)- cryo-therapy application and electro-analgesia (e.g. transcutaneous electrical stimulation)
- Stretching: Acupuncture & Acupressure (trigger point Rx)
- Relaxation Techniques: Biofeedback, Music, Hydro bath

C. Surgical treatment

2. Pharmacological management

Non-opioid analgesics---inhibit prostaglandin production responsible for pain, used for mild to moderate pain.

- E.g. Acetaminophen -analgesic antipyretic has no anti-inflammatory effect, elevate the pain threshold in the (brain) switching off the perception of pain.
- Non-steroidal anti-inflammatory drugs (NSAIDs) - anti-inflammatory, antipyretic, and analgesic effect.
- E.g. ibuprofen, diclofenac, aspirin, Ketorolac. Ketorolac- has the distinction of being the only non-narcotic analgesic available in a parenteral formulation that can be administered for the relief of acute pain.

**Opioid analgesics**
- Act directly on the opioide- receptors, which are found in the brain and spinal cord.
- E.g. morphine, codeine , meperidine (Demerol), tramadol, fentanyl, sufentanil, alfentanil.
- Have a higher analgesic potency and wider range of indications than any of the other currently available medications for pain control.
- Used to relieve severe pain. They inhibit pain-transmitting neurons and stimulate pain-inhibitory neurons thus changing the brain’s interpretation of pain.

**Adjuvants**-
- Commonly used adjuvant analgesics-tricyclic antidepressants- (e.g. Amitriptyline), antiepileptic (eg.carbamazepin (Tegretol),)
- Commonly used to treat neuropathic pain.
- Ketamine –is an anesthetic drug with analgesic effect used for minor operative procedures, wound dressing, and short diagnostic procedures like biopsy, analgesic and sedation in ICU etc.

**Combined analgesics**-
- Small amount of a mild opioid and a simple analgesic such as aspirin or paracetamol, all within one tablet or capsule are prescribed if the non-opioid painkillers are not enough.
- E.g. Aspirin with codeine, Dextropropoxyphene with paracetamol, dihydrocodeine with paracetamol, Paracetamol with codeine

**Pain management approach according to pain intensity level after assessment**
- Mild pain (score1-3)-reassures the patient and gives acetaminophens or NSAIDS if no response to the nonpharmacologic method
- Moderate pain (score4-6)-give NSAID or acetaminophen and add mild opioid if pain is not well controlled
- Severe pain (score 7-10) start immediately with potent analgesic opioids.
Analgesic delivery system

- Oral, IM, IV, infiltration of local anesthetics, central neuraxia (epidural, intratecal), peripheral nerve block
- Side effects of NSAIDs: stomach bleeding, stomach ulcers (with long-term use), and kidney or liver problems.
- Opioides and sedatives should be started in lower dose and be titrated to patient response and monitored for life threatening side effects such as respiratory depression.
- Communicate with the patient and encourage breathing
- Monitoring respiratory rate and if low <10bpm assist breathing and oxygenation
- Use Pulseoximetry-if saturation is low <90% administer oxygen

Summary-

- Pain assessment is the cornerstone for pain management
- Pain intensity levels should be assessed at the initial encounter, following treatment, and periodically.
- The main objective of pain management is to prevent its adverse physiological effect
- Pain medications should not be prescribed on PRN basis.
- Patient should be monitored for drug side effect, clinically, monitors, and laboratory according to the clinical condition

Activities
- Discussion on case scenario # 14-15
- General discussion on questions raised by participants
Chapter x: Obstetric Emergencies

- Duration-6 hrs

Learning Objectives

By the end of this session, participants will be able to:

- Describe the management approach to obstetric emergencies
- List the classification and management principles of hypertensive disorders of pregnancy
- Outline management of normal labour, breech, shoulder dystocia
- Describe the assessment and management of obstructed labour and ruptured uterus
- Describe management of bleeding during pregnancy
- List the management approach to trauma during pregnancy
- List causes and management of PPH

1. Hypertensive Disorders During Pregnancy

(A) Definition

Hypertension (HTN): Systolic blood pressure (SBP) ≥140 mmHg or diastolic blood pressure (DBP) ≥ 90 mmHg on two occasions 6 hours to 7 days apart; Or one BP ≥ 160/110 mmHg.

Proteinuria: 24-hour urine specimen with 0.3 g protein or ≥1+ dipstick or ≥100 mg/dl two random urine sample, collected at least 4 hours apart.

(B) Classification and Clinical features:

1. Gestational HTN: HTN without proteinuria or other signs of preeclampsia
2. Pre-eclampsia: HTN + proteinuria after 20 weeks. Can be mild or severe. Severe if:
   - BP≥160/110mmHg or urine protein ≥ +3 or >5 gm/24 hr urine
   - severe symptoms: headache, visual changes, epigastric pain, severe edema, IUGR, abruptio placenta, oliguria, DIC (bleeding, petechia), HELLP syndrome (hemolysis, elevated liver enzymes and low platelets), severe nausea and vomiting
• low platelets < 100,000, altered liver and renal function tests.

3. **Eclampsia**: grand mal seizures or coma in a woman with preeclampsia **EMERGENCY**

4. **Chronic HTN**: HTN antedates pregnancy. Get superimposed preeclampsia/eclampsia if:
   - new proteinuria after 20 weeks GA; or sudden increase in preexisting proteinuria
   - An exacerbation of blood pressure to the severe range or severe symptoms.

(C) **Diagnostic tests and Procedures**

Helps in diagnosis and determine severity of the disease.

- Hematocrit, urine protein, serum creatinine, uric acid, ALAT, ASAT, bilirubin, platelet count, LDH, PT, aPTT, type and crossmatch
- Nonstress test and ultrasound.

(D) **Management of different stages of pregnancy induced hypertension/PIH/**

**Gestational Hypertension**
Check blood pressure and protein. Refer for outpatient management. If BP remains in mild range, proceed with normal labour and childbirth before post term.

**Eclampsia and Pre-eclampsia**

<table>
<thead>
<tr>
<th>When to deliver</th>
<th>Severe Pre-eclampsia</th>
<th>Eclampsia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>If mild, deliver at 37 weeks (refer for outpatient follow up)</td>
<td>Immediately (should be in &lt; 12 hours); deliver by C-section if vaginal delivery not possible in this time.</td>
</tr>
<tr>
<td></td>
<td>If severe, GA &gt; 34 weeks or end organ damage: deliver immediately. If &lt; 34 weeks: give steroids, observe as inpatient, deliver at 34 weeks</td>
<td>Transfer patient if needed</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Initial Measures</th>
<th>ABCs</th>
<th>ABCs; apply oxygen; give prophylactic IV antibiotics</th>
</tr>
</thead>
</table>

<p>| When to give seizure | During evaluation, labour and continued for 24 hrs after delivery | Immediately; continue until 24 hours after |</p>
<table>
<thead>
<tr>
<th>prophylaxis</th>
<th>delivery or last seizure</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Measures when admitted</td>
<td>Monitor urine output (goal &gt;30ml/hr), fluid balance; vital signs, FHB, reflexes and signs of pulmonary edema.</td>
</tr>
</tbody>
</table>
| Acute blood pressure control (goal DBP 90-100 mmHg and SBP 140-150 mmHg) | **1st line: Hydralazine:** 5-10 mg IV every 5 minutes until DBP <110mmhg. Repeat hourly as needed (or 12.5mg IM every 2hrs).  
**2nd line: Nifedipine:** 5-10 mg sublingually, then 5-10mg in 30 minutes if response inadequate. Then 10-20 mg PO every 6 hours (10-40mg PO bid maintenance).  
**2nd line: Labetolol:** 20 mg IV push over 2 minutes. Repeat as needed every 10 minutes, doubling the dose up to 80 mg for desired effect. Maximum total cumulative daily dose is 300mg IV. |
| Seizure Prophylaxis: **Note:** Magnesium (MgSO4) is critical to stopping and preventing convulsions. | Magnesium (MgSO4) Before administration: ensure RR ≥ 16/minute, DTRs present.  
Loading dose: MgSO4 (20% solution) 4 g IV over 5 minutes. Then 10 g 50% MgSO4 IM, 5 g in each buttock. If convulsions recur after 15 minutes, give 2 g MgSO4 (50% solution) IV over 5 minutes.  
Maintenance: 5 g MgSO4 (50% solution) + 1 mL lignocaine 2% IM every 4 hours into alternate buttocks.  
If respiratory arrest: assist ventilation, give calcium gluconate 1 g (10 mL of 10% solution) IV slowly until respiration begins.  
**Diazepam: Second line.** Risk fetal or maternal respiratory depression  
Loading dose: 10 mg IV over 2 minutes, if convulsions recur, repeat loading dose and 40 mg in 500 ml IV fluids; drops titrated to keep woman sedated but arousable.  
Can give rectally when IV access is difficult: Loading dose of 20 mg followed by maintenance dose of10mg/hr. |
2. Management of Labour and Delivery

(A) History and Physical Examination

History: Gravidity, parity, Gestational age/GA/ (in weeks), time contraction started, frequency; leakage of liquor, bleeding, past medical illnesses.

Physical: Leopold maneuver for GA and fetal presentation; sterile digital vaginal examination if no vaginal bleeding to assess the stage of labor.

First stage: until cervical dilation completes (10 cm), divided into:
- **Latent phase**: Until faster rate of cervical change, (usually when cervix is 3 cm)
- **Active phase**: from start of faster rate until complete dilatation.

Second stage: delivery of the baby

Third stage: delivery of the placenta

Fourth stage: one hour after placenta delivery.

(B) Investigation

If not already done, Hgb, Blood group and Rh, U/A and microscopy, RVI

(C) Management during 1st stage

Record all observations and findings on the partograph.

(1) Maternal wellbeing monitoring

If patient in 1st stage or has high risk pregnancy (requiring obstetrician), immediately transfer to OB delivery unit. If unable to transfer or imminent delivery, do the following:
- Vital signs: every half an hour.
- Pain management - continuous emotional support, use analgesia safe for mother and fetus without effects on progress of labour. IV, IM or SC opioids or epidural analgesia.

(2) Fetal Wellbeing monitoring

- Fetal heart rate/FHR/ : Pinnard stethoscope or continuous FHR monitoring. Do for 1 min after contraction; every 30 min if low risk pregnancy, 15 min if high risk.
- Monitor liquor for meconium: if moderate meconium likely fetal distress; if thick, definite fetal distress
(3) Monitor Progress of labour:

1. Uterine contractions – track frequency, duration and intensity (by palpation or toco-
dynamometer); monitor q1 hr for latent phase and q30 min for active phase.
2. Descent of fetal head: determine by abdominal palpation and vaginal exam q4
   hours.
3. Vaginal examination (every 4 hours). Assess: cervical dilation, (normally
   progresses ≥1 cm/hr), station, position, caput and molding.

(D) Management of 2nd stage

Normal duration of 2nd stage: Nullipara: < 2hrs without or 3hrs with epidural
anesthesia. Multipara: < 1hrs without or 2hrs with epidural anesthesia. If longer, is
prolonged 2nd stage.

(4) Maternal wellbeing monitoring.

Evaluate general condition, pain, hydration, vital signs every 30 minutes
Empty bladder, avoid early pushing, Left lateral position/ LLP/ until head is visible.

(5) Fetal Wellbeing Monitoring.

Every 15 min and 5 minutes for low and high risk fetuses respectively.

(6) Monitoring of Labour Progress.

Evaluate uterine contraction every 10 minutes and the descent every 1 hour.

(7) Assistance of spontaneous delivery.

- Do episiotomy only if perineal resistance or if expedited delivery indicated.
  - Timing: When head distends vulva 2-3cms. Types: median or mediolateral
    (mediolateral is preferable). Local anesthesia used.

- Assist delivery of head using modified Ritgen’s maneuver if extension does not
  occur easily i.e., hand protected with sterile towel placed on the perineum, fetal
  chin palpated and pressed upward gently effecting extension.

- Check for cord around neck: if present gently move over the head. If not
  reducible: deliver without reduction or clamp at two sites and cut in between.

- After delivery of head: wipe mouth and oro-pharynx (routine suctioning not
  recommended). Allow for restitution of head. Then, place a hand on each parietal
eminence. Apply gentle downward pressure of the head toward the maternal sacrum to deliver anterior shoulder. Then, deliver posterior shoulder by upward traction.

- Put fetus at level of introitus for 3 min. Immediately dry the body. Clamp cord 4-5 cm from fetal umbilicus. Delayed clamping (after a few minutes) associated with increased neonatal hematocrits. Take cord blood if indicated.

- If second stage prolonged, manage the causes:
  - Poor maternal pushing effort: consider vaccume or forceps delivery.
  - Poor uterine contraction: oxytocin.
  - Cephalo-pelvic disproportion/CPD/: cesarean delivery.

(E) Management of 3rd stage of labour.

Provide active management of third stage of labour (AMTSL) for all patients:

- Administer uterotonic agents immediately after fetal delivery (1st choice: oxytocin 10 IU IM; 2nd choice: Misoprostol 600 mcg orally; 3rd choice: ergometrine 0.2mg IM or syntometrine (1 ampoule) IM)
- Apply controlled cord traction
- Use uterine massage (immediately after the placental delivery).

3. Shoulder dystocia

(A) Definition

Shoulder dystocia is inability to deliver the shoulders after the fetal head has been delivered despite the performance of routine obstetric maneuvers. It is an acute obstetric emergency requiring prompt, skilful management to avoid significant fetal damage and death. Higher risk of occurrence during delivery of macrosomic (>4kg) babies, but Shoulder dystocia may not be predicted. Be prepared for shoulder dystocia at all deliveries, especially if a large baby is anticipated and in women with diabetes mellitus, previous history of macrosomic babies and obesity. Asphyxia, birth injuries, injury to the brachial plexus and maternal Post Partal hemorrhage/PPH /are some of the complications.

(B) Diagnosis

- The fetal head is delivered but remains tightly applied to the vulva,
- The chin retracts and depresses the perineum. Traction on the head fails to
deliver the shoulder, which is caught behind the symphysis pubis.

(C) Management

- In the lithotomy position, ask the woman to open and flex both thighs, bringing her knees as far up as possible towards her chest. Ask two assistants to push her flexed knees firmly up onto her chest (McRobert’s maneuver).

- Apply firm (not excessive), continuous traction downwards (towards the floor) on the fetal head while an assistant simultaneously applies suprapubic pressure downwards to assist delivery of the anterior shoulder. If not successful, continue with maneuvers below. Episiotomy should be performed at any point if needed to create adequate space for maneuvers.

- Insert a hand into the vagina and apply pressure on the back surface of the anterior shoulder in the direction of the baby’s sternum to rotate the shoulder and decrease the width of the shoulders; if needed, apply pressure to the back of the posterior shoulder in the direction of the sternum (Rubin’s maneuver).

- Alternatively, one can try applying pressure to the anterior surface of the posterior shoulder until the baby turns and the anterior shoulder emerges from underneath the pubic symphysis. (Woods screw maneuver)

- If the shoulder still is not delivered despite the above measures, insert a hand into the vagina; grasp the humerus of the posterior arm. Keeping the arm flexed at the elbow, sweep the arm across the chest. This will provide room for the shoulder that is anterior to move under the symphysis pubis.

- If the posterior arm cannot be reached, apply traction with a hook in the axilla to extract the shoulder that is posterior; this may bring the posterior arm down sufficiently to be grasped and delivered.

- In patients with only local or pudendal anesthesia, an effective initial maneuver may be to rotate the woman to a position of hands and knees and attempt delivery in this manner. (Gaskin all-fours maneuver)

- If all of the above measures fail, the last option is to fracture the clavicle (will decrease the width of the shoulders and free the anterior shoulder); apply traction with a hook in the axilla to extract the arm that is posterior. Replacing the head and doing C/S is rarely successful.

- After this is done, repeat all above maneuvers with the newly shortened biacromial diameter.
4. Breech Presentation and Delivery

(A) History and Physical Examination

- The history may reveal discomfort under the rib (due to the hard head)

- On abdominal palpation (Leopold's maneuvers), may feel a round, hard, smooth mass (head) occupying the fundus. A soft, broad, indefinite and non ballotable mass (the breech) occupying the lower pole of the uterus. FHB loudest just above the umbilicus.

- On vaginal examination: three types of breech presentation can be identified:
  1. Frank breech: buttocks in the pelvis, both legs extended.
  2. Complete breech: one or both feet are felt along side the buttock.
  3. Footling breech: one or both feet are inferior to the buttock.

- Ultrasound confirms the clinical diagnosis of breech and predisposing factors.

(B) Investigation

- Hgb and Blood group and Rh, Ultrasound.

(C) Treatment Plan

- If preterm and not in labour, refer to OBGY for expectant management; may turn cephalic.

- If GA ≥ 37 weeks of gestation and if there is no contraindications for external cephalic version (ECV), consider ECV. If the ECV fails, consider breech vaginal delivery.
  - ECV: baby manipulated through mother’s abdominal wall into cephalic presentation.

- If in advanced labour do breech vaginal delivery. Do cesarean delivery only for footling breech (increased risk cord prolapse + arrest head) or obstetric indications like APH, etc.

- Induction or augmentation of labour is contraindicated in breech presentation.

Types of vaginal breech delivery:

1. Spontaneous breech delivery: The baby is expelled entirely spontaneously.
2. Total breech extraction: Deliver by extracting the entire body of the fetus from the uterus; rarely indicated except to expedite the emergent delivery of a second twin.

3. Assisted vaginal breech delivery: Partial breech extraction if no cord prolapsed or entanglement

**Delivery of the breech, abdomen and shoulder:**

- Keep in lithotomy position and empty her bladder. Provide emotional support, pudendal block and local infiltration, do episiotomy when the fetal anus is visible, instruct the mother to bear with contraction and allow the buttocks delivered spontaneously and shoulder blades are seen (no other manipulation until delivered up to umbilicus)
  - If the legs are not delivered spontaneously, assist delivery of one leg at a time, by lateral rotation of thighs and flexion of knees using a finger.

- Hold the baby by the hips with towel (not by the flanks or abdomen as this may cause organ damage) with fingers over anterior superior iliac spine and the sacrum. Apply gentle, steady downward traction with good maternal pushing until the lower half of the scapula is delivered.

- Allow the arms to disengage spontaneously one by one; only assist if necessary. After spontaneous delivery of the first arm, lift the buttock towards the mother’s abdomen to enable the second arm to deliver spontaneously. If the arm is not spontaneously delivered, place one or two fingers in the elbow and bend the arm, bringing the hand over the baby’s face.

- If arms are stretched above the head or folded around the neck, use the Lovset’s maneuver.

**Delivery of the head:**

- **Mauriceau Smellie Veit Maneuver (MSV):** Lay the baby face down with the length of its body over your hand and arm, with the mid and forefingers of your hand on the baby’s cheekbones. Pull down to flex the head while the other hand grasps the baby’s shoulders. With two fingers and middle finger placed and pushing over the subocciput (hooking round the neck), gently flex the baby’s face towards the chest, while applying downward pressure on the shoulder. Pull gently downward until the hairline is visible while an assistant applies suprapubic pressure. Once the head gets into the pelvis, following the pelvic curve, raise the baby until the mouth and nose are delivered.

- Forceps delivery if MSV fails.
5. Obstructed Labour and Ruptured Uterus

(A) Definition

Obstructed labour (OL) is failure of descent of the fetus in the birth canal for mechanical reasons in spite of good uterine contractions. OL is a neglected labour and should not occur in a labour ward. OL is an emergency condition and requires a concerted team approach.

(B) Causes

- Cephalopelvic disproportion (CPD): small/abnormal pelvis or large fetus.
- Congenital fetal abnormalities, locked twins, shoulder dystocia, fetal malpresentations and positions, or abnormal reproductive tract.

(C) Diagnosis

- History of labour for days, maternal exhaustion, anxiety, confusion, or unconsciousness, low BP, rapid PR and RR.
- Abdominal examination:
  - Fetal head above the pelvic brim, abnormal or no fetal heart tone.
  - Distended and tender abdomen, Bandl’s ring.
  - The ‘three tumor abdomen” indicates an impending uterine rupture.
- In ruptured uterus (common in multiparous women), the findings include:
  - Shock, hemoperitoneum, tender abdomen, easily palpable fetal parts.
  - On vaginal examination: foul smelling discharge, significant caput and molding, edema of the vulva and the causes of OL.

(D) Complications:

- Maternal: PPH, sepsis, urinary and rectal fistula, nerve injuries, uterine rupture, etc.
- Fetal/neonatal: fetal death, asphexia, sepsis, etc.

(E) Treatment

- Simultaneously start resuscitation while identifying the cause and treating infection
- In case of ruptured uterus, laparotomy is indicated.
- Once OL is diagnosed, C/S is the rule. No place for instrumental vaginal delivery.
  - Destructive delivery (craniotomy) is an exception used only when the fetus is dead, there is no sign of uterine rupture or impending rupture,
OL is in 2\textsuperscript{nd} stage with engaged head, and a place with no operating room facility.

6. Vaginal Bleeding During Pregnancy

6.1 First trimester vaginal bleeding

Vaginal bleeding in early pregnancy is common, but may indicate life-threatening disease. Women of child-bearing age with abdominal pain or vaginal bleeding should be presumed pregnant.

Obtain vital signs. If haemodynamically unstable (HR>100, SBP<90), give 1-2 L IV Fluid/IVF/

[A] History and physical exam

- Last menstrual period /LMP/, current pregnancy, prior ultrasounds this pregnancy

- Degree and duration of bleeding, abdominal pain, cramps, fever, dizziness, syncope

- **Ectopic risk factors:** tubal surgery, PID, prior abortion, IUD

- Exam: abdominal tenderness; internal cervical os open or closed, clots or products of conception (POC), adnexal mass, adnexal or uterine tenderness, lacerations or masses

[B] Possible Causes and Differential diagnosis

- **Ectopic pregnancy:** Gestational sac outside of uterus. Peritonitis or abnormal vitals suggest rupture (life threatening). Rule out in all pregnant women with vaginal bleeding. Unlikely if Intra uterine pregnancy/IUP/ on ultrasound (US).

- Recent induced abortion or complications

- **Spontaneous abortion:** May be inevitable (bleeding with open internal os), Incomplete (open os, POC visualized), complete (closed os, fetus and placental materials fully expelled)

- **Septic abortion:** fever, abdominal pain; can complicate any abortion.

- **Threatened abortion:** bleeding + closed internal os + US with IUP. Risk of abortion 35-50%.

- **Molar pregnancy:** Abnormal trophoblastic tissue. Bleeding at 12-16 weeks, uterus larger than expected, passage of grape-like material, US with 'snowstorm' pattern.
• Non-pregnancy-related vaginal or cervical pathology: laceration, ulcer, cervical mass

[C] Investigations:

• Laboratory: urine hCG, Hb/hct, type and Rh screen, crossmatch if heavy bleeding/haemodynamic instability, quantitative serum hCG,
  ◦ Pelvic ultrasound: transvaginal or transabdominal.
• Culdocentesis: If US not available. Detects hemoperitoneum, but less sensitive and specific. If positive, treat as ruptured ectopic.
• Examine passed uterine contents: rinse and place in saline/tap water. Blood dissolves, chorionic villi will be fluffy and fingerlike. Presence of villi excludes ectopic.

[D] Management

[B] General

IVF/blood as needed for HD instability, anti-D immunoglobulin if Rh-negative (50ug in first trimester, 300ug after first trimester), analgesics

[B] Ectopic: consult obstetrics or transfer

• Laparotomy/ Laparoscopy: Unstable, peritoneal signs, evidence of rupture
• Medical management: if stable, unruptured.

[B] Spontaneous abortion: incomplete or inevitable:

• If heavy bleeding, haematocrit, transfuse as needed.
• Gently remove fetal tissue if visualized in cervical os
• If febrile, doxycycline 100mg, ceftriaxone 1 gm IV and Metronidazole 500 mg IV or ampicillin 2 gm IV, Gentamicin 2mg/kg IV and Metronidazole 500 mg IV
• If prolonged bleeding, HD unstable, or febrile, needs manual vacuum aspiration(MVA) or evacuation and curettage.

[B] Threatened abortion: bleeding with live IUP on ultrasound, closed os
• Expectant management; close follow up with obstetrician/OB/

[B] Molar pregnancy
• Consult OB if no marked bleeding, Suction curettage if bleeding, ensure OB follow up to trend hCG

[E] Disposition

• Admit for ruptured ectopic, haemodynamic instability, falling haematocrit, severe pain, fever

• Discharge if abortion with minimal blood loss and good OB follow up. Instructions to return for fever, worsening abdominal pain, or significant increase in bleeding.

6.2 Second Trimester Vaginal Bleeding (14-28 Weeks):

In general, bleeding prior to 28 weeks should be treated like 1st trimester bleeding. Bleeding after 28 weeks should be treated like 3rd trimester bleeding In Ethiopia< 28 weeks = abortion

6.3 Third Trimester Vaginal Bleeding, Ante partum Hemorrhage (APH)

(A) History and physical Examination

Estimate gestational age (APH Is vaginal bleeding from the 28th week of gestation until fetus is delivered.), assess amount of visible bleeding. No digital vaginal or speculum exam until placenta previa ruled out.

(B) Investigation

Hgb, Blood group and Rh, Ultrasound exam

(C) Causes

2. Uterine rupture
3. Local causes of the cervix, vagina and vulva
4. Preterm labour /bloody show
5. Indeterminate: no cause identified even after delivery and examining the placenta.

1. Abruptio Placentae

Abruptio placenta is a premature separation of the whole or part of a normally implanted placenta. Complications include hemorrhagic shock, DIC, and utero-placental insufficiency (UPI) that may lead to IUGR, fetal distress or IUFD.

Diagnosis (Table 1).
Treatment plan

Delivery is the definitive management. In milder abruption and remote from term expectant management (admission, steroid administration) to prevent prematurity. Moderate and severe abruption (irrespective of gestational age) and abruption at term (irrespective of degree) immediate delivery. Mode of delivery is vaginal. Cesarean section is indicated for severe bleeding endangering maternal life and fetal distress, when vaginal delivery seems unlikely within a reasonable time and for other obstetrics indication. Coagulation defect must be corrected early.

2. Placenta Previa

Placenta previa is the presence of placental tissue lying adjacent to or overlying the internal cervical. Placenta previa is classified based on nearness of the placental edge to internal-os of the cervix: Low lying placenta, Marginal placenta previa, Major placenta previa (may be partial or total).

Diagnosis: Clinical (See table 1) and ultrasound if available.

Treatment

Delivery is the definitive management of placenta previa. In cases of mild and non-recurrent bleeding, do conservative management to prevent prematurity.

Mode of delivery: Cesarean section. Vaginal delivery may be considered if low lying placenta.

3. Local Causes

All local causes of APH have minimal spotting or bleeding. An exception to such a presentation is the occasional profuse bleeding of ruptured vaginal varicose vein. Once placenta previa is excluded, digital and speculum examination may confirms the specific local cause.
Table 1 Clinical Findings in Placenta Previa and Abruptio Placenta

<table>
<thead>
<tr>
<th>Clinical Findings</th>
<th>Placenta Previa</th>
<th>Abruptio Placentae</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaginal bleeding</td>
<td>Painless Causeless Recurring(often) Bright red</td>
<td>Painful Presence hypertension, trauma, etc Non-recurring Menstrual like</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Proportion to vaginal blood loss</td>
<td>Degree of hypotension out of proportion to amount of vaginal bleeding</td>
</tr>
<tr>
<td>Uterus</td>
<td>Quite or relaxed between labour contractions</td>
<td>Irritable, not relaxing between labour contractions (tetanic contraction),</td>
</tr>
<tr>
<td>Fetal presentation</td>
<td>Mal-presentation (transverse, breech), unengaged head</td>
<td>Difficult to palpate fetus Engaged head</td>
</tr>
<tr>
<td>Fetal condition</td>
<td>Usually normal fetal Condition</td>
<td>Fetal distress, Fetal death, IUGR</td>
</tr>
</tbody>
</table>

7. Trauma during Pregnancy

1. Major

Management

- Lie patient flat in left lateral position or at least have right hip elevated if possible (can tilt the trauma board as a whole if on a trauma board and manipulate the uterus to the left side)
- Check airway and immobilize cervical spine if necessary
- High flow oxygen via mask with reservoir
- If breathing inadequate, assist with bag-valve-mask ventilation with high flow oxygen
- Assess circulation: pulse, BP, capillary refill.
- Pulse oximetry if available
- Insert I/V line x 2
• Take blood as indicated
• If shock present give fluid bolus.
• Test neurological disability

2. Minor

Up to 22 weeks - Routine trauma care with confirmation of FHTs
After 22 weeks (including falls, whether abdomen was hit or not)
- Routine trauma care. Continuous fetal monitoring/tocograph for 4 hours after trauma.
- Patient may be discharged after this time if above is reassuring.
- Patient should return if “tightening” or back pain.
- Patient should also return for repeat monitoring at 24 hours (bleeding caused by mild/small marginal separations that are the result of trauma can dissect into myometrium or under placenta and cause PTL and/or fetal distress after initial trauma, usually 24-48 hours after)

Considerations:
- Trauma to the Uterus
- Shocked patient with fetus-in-utero. - Intrauterine resuscitation: High flow Oxygen by mask with reservoir, I/V Ringers lactate bolus, maternal repositioning (left or right lateral) and consult early

8. Post Partum Haemorrhage (PPH).

Vaginal bleeding > 500ml after singleton vaginal delivery of >28 weeks. (if cesarean delivery or multiple vaginal birth, bleeding >1000ml)

(A) History and Physical Examination

Vital signs, estimate the blood loss, uterine size and extent of contraction, completeness of the placenta.

(B) Causes

(1) Atonic Uterus (Uterus not contracted)

• The most common cause of primary PPH.
• Hypotonic uterus leads retention of the placenta and excessive bleeding.
- Diagnose if: soft, not contracted uterus with fundus above the umbilicus.

(2) Retained placenta

- The common cause of placental retention is poor uterine contraction.
- In retention of the placenta without bleeding, pathological adherence (accreta, increta and percreta) should be considered.
- Manual removal of the placenta has to be done in the operating room with all the preparation for laparotomy and possible hysterectomy.

(3) Traumatic causes

- Risk factors for tears of the birth canal (including uterine rupture) and PPH: Fetopelvic disproportion (leading to obstructed labour), instrumental deliveries and scarred uterus.
- Diagnosis: bright red (arterial) bleeding with a contracted uterus.

(4) Coagulation defects

- Risk factors: abruption placenta, intrauterine fetal death, infection etc.
- Physical examination: gross haemostatic failure is revealed.
- Bed side clotting tests and deranged laboratory coagulation profiles support the diagnosis.

(5) Acute inversion of the uterus

- The uterus may rarely turn inside-out during delivery. Causes shock by bleeding or neurogenic shock due to increased vagal tone from stretching of the pelvic parasympathetic nerves.
- With the placenta detached, is described as cherry red mass.

(C) Management of PPH

Once PPH is diagnosed, shout for help and gather the team, immediately initiate resuscitation, and perform diagnostic and treatment activities promptly. ABCs first and while stabilizing initiate specific treatment. If the cause is not known, do the following:
1. Retained placenta: PPH with undelivered placenta:
   - Apply controlled cord traction (CCT)
   - If CCT fails, manual removal of the placenta in operating room
   - Consider laparotomy for possible pathological adherence if both fails.

2. Atonic uterus: if PPH with delivered placenta and atonic uterus:
   - Stimulate contraction by massaging the uterus.
   - Start oxytocine infusion(20IU/1000ml, 30drops/minute).
   - If there is no response, perform bimanual compression of the uterus; consider compression of the abdominal aorta.
   - Administer other uterotonics such as misoprostol (800mcg sublingual) or ergometrin 0.2mg IM. If persistent bleeding, consider uterine tamponade with intrauterine balloon or condom tamponade (condom tied to end of foley catheter, inserted into uterus, and filled with 350ccs NS).
   - If there is no response subsequent management involves laparotomy uterine or utero-ovarian artery ligation, or hysterectomy.

3. Genital trauma: if PPH with delivered placenta and well contracted uterus:
   - Explore the genital tract manually and using speculum and repair vaginal/cervical tear; if uterine rupture detected laparotomy is indicated.

4. Clotting abnormality: Correct with fresh frozen plasma or whole blood.

5. PPH after acute inversion of the uterus: under appropriate analgesia, apply:
   - Immediate gentle upward transvaginal pressure.
   - The Johnson technique calls for lifting the uterus and the cervix into the abdominal cavity with the fingers in the fornix and the inverted uterine fundus on the palm.
   - Gently push the fundus back through the cervix. The operator’s hand should be kept in the uterus until the fundus begins to climb up. If the placenta is still attached, it should not be removed until after the uterus is replaced through the cervix. Tocolysis may be used. Oxytocin only after successful replacement. If this fails unsuccessful (in delayed recognition) laparotomy for abdominal replacement is indicated.
Activities
- Discussion on case scenario # 21-25
- Simulation&practice in small group
- General discussion on questions raised by participants
Chapter XI: PEDIATRICS EMERGENCY MEDICINE (PEM)

- Duration-6 hrs

Learning objectives:

By the end of this session, participants will be able to:

- How to triage
- How to assess and manage Pediatrics Airway
- Management of Pediatrics respiratory Emergency
- Identifying Pediatrics circulatory problem
- Over view of basic life support in infant and children
- Proper way of Neonatal resuscitation

Triage

Objective

At the end of the attachment, the trainee will be able to:

- Outline the principle of triaging
- Identify the three steps of triaging

Definition

Triage is the process of rapidly examining all sick children when they first arrive in hospital in order to place them in one of the following categories:

- Those with EMERGENCY SIGNS who require immediate emergency treatment.
  
  If you find any emergency signs, do the following immediately:- Start to give appropriate emergency treatment
  - Call a senior health worker and other health workers to help.
  - Carry out emergency laboratory investigations.
➢ Those with PRIORITY SIGNS, indicating that they should be given priority in the queue, so that they can rapidly be assessed and treated without delay.
➢ Those who have no emergency or priority signs and therefore are NON-URGENT cases.

These children can wait their turn in the queue for assessment and treatment. The majority of sick children will be non-urgent and will not require emergency treatment. After these steps are completed, proceed with general assessment and further treatment according to the child’s priority.

The ABCD concept
Triage of patients involves looking for signs of serious illness or injury. These emergency signs relate to the Airway-Breathing-Circulation/Consciousness-Dehydration and are easily remembered as “ABCD”.
Each letter refers to an emergency sign that, when positive, should alert you to a patient who is seriously ill and needs immediate assessment.

Priority signs
Besides the group of emergency signs described above, there are priority signs, which should alert you to a child who needs prompt, but not emergency assessment. These signs can be remembered with the symbols

TPR - MOB:
- Tiny baby: any sick child aged under two months
- Temperature: child is very hot
- Trauma or other urgent surgical condition
- Pallor (severe)
- Poisoning
- Pain (severe)
- Respiratory distress
- Restless, continuously irritable, or lethargic
- Referral (urgent)
- Malnutrition: Visible severe wasting
- Oedema of both feet
- Burns

The frequency with which children showing some of these priority signs appear in the outpatient department depends on the local epidemiology. The signs might need to be adapted accordingly, for example by including signs for common severe conditions which cannot wait in your setting and treatment.

The triaging process

Triaging should not take much time. For a child who does not have emergency signs, it takes on average 20 seconds. The health worker should learn to assess several signs
at the same time. A child who is smiling or crying does not have severe respiratory distress, shock or coma. The health worker looks at the child, observes the chest for breathing and priority signs such as severe malnutrition and listens to abnormal sounds such as stridor or grunting.

**HOW TO TRIAGE?**

Keep in mind the ABCD steps: Airway, Breathing, Circulation, Coma, Convulsion, and Dehydration.

To **assess if the child has airway or breathing problems** you need to know:

- Is the child breathing?
- Is the airway obstructed?
- Is the child blue (centrally cyanosed)?

Look, listen and feel for air movement. Obstructed breathing can be due to blockage by the tongue, a foreign body, a swelling around the upper airway (retropharyngeal abscess) or severe croup which may present with abnormal sounds such as stridor.

- Does the child have severe respiratory distress?

Is the child having trouble getting breath so that it is difficult to talk, eat or breastfeed? Is he breathing very fast and getting tired, does he have severe chest indrawing or is he using auxiliary respiratory muscles?

A **Airway**

B **Breathing**

C **Circulation**

Cm **Coma**

Cn **Convulsion**

D **Dehydration (severe)**

E **Emergency**

P **Priority**

Q **Queue**

**Airway**

**Objective**

At the end of this chapter, the trainee should be able to:

- Identify the knowledge and skills on how to open the air way
- Describe the Unique feature of pediatrics airway
- Outline the Management of foreign body in the air way

**Introduction**
The letters A in “ABCD” represent “airway”.
- It is evident that an open (patent) airway is needed for breathing.
- An airway problem is life threatening and must receive your attention before you move on to other systems.

It is therefore convenient that the first letter of the alphabet represent the most important areas to look for emergency or priority signs

**Unique features of the pediatric airways:**

1. Tongue relatively large in proportion to oral cavity
2. Infants <2 months of age are obligate nose breathers
3. Trachea is smaller and shorter than that of adults
4. Smallest diameter of trachea is at the cricoid ring, below the cords
5. Chest wall of infants relatively weak and unstable
6. Larynx is relatively anterior and high: C2 in neonate, C3-4 in child, C5-6 in adult
7. Immunologic immaturity leads to increased susceptibility to respiratory infections
8. Developmental immaturity leads to increased susceptibility to foreign body aspiration.

**Air way emergency:**

1. The patients tongue in a patient who is unconscious
2. Foreign body obstruction

**Management**

- Open air way head tilt chin lift in a patient who has no trauma if there is trauma jaw thrust
- Suction the air way
- Insertion of oro-pharyngeal air in unconscious
- In conscious nasopharyngeal air

Remove the foreign body if suspected
Foreign-Body Airway Obstruction (Choking)

- Signs of Foreign-Body Airway Obstruction include a sudden onset of respiratory distress with coughing, gagging, stridor, wheezing
- If there is foreign body obstruction:
  - For an infant, deliver 5 back blows (slaps) followed by 5 chest thrusts repeatedly until the object is expelled or the victim becomes unresponsive
  - For a child, perform sub-diaphragmatic abdominal thrusts (Heimlich maneuver) until the object is expelled or the victim becomes unresponsive
- If the victim becomes unresponsive, lay should perform CPR but should look in to the mouth before giving breaths
Breathing

**Objective**

At the end of this chapter, the trainee should be able to:

- List steps in assessment of breathing?
- Manage upper air way, lower air way and lung parenchyma
- Demonstrate Ways of administering of oxygen

**Introduction**
If there is no problem with the airway, you should look for signs:

- To assess if the child has a breathing problem, you need to know:
  - Is the child breathing?
  - Is the airway obstructed?
  - Is the child blue (lips and fingers)?
  - Does the child have difficulty of breathing?

If the child is not breathing or if the airway appears obstructed, you must first open the airway.

### Pediatrics respiratory emergencies

- **Acute Sever Asthma**
- **Bronchiolitis**
- **Croup**
- **Epiglottis**

#### 1. Severe Asthma

Disease characterized by hyper-reactive small airways and reversible obstruction of those airways.

- Three components of obstruction: B
  - Bronchoconstriction,
  - Mucosal edema,
  - Increased secretions.

**Management:**

- Position as comfortable
- Check the air way
- Administer oxygen high flow O2 to maintain saturation >95%
- Hydration has to be maintained
- Check for possible complication
- Salbutamol puff 0.5-1.5 mg 3-4 puff has to be repeated every 20 minute tree times if no improvement
- Hydrocortisone 4-5 mg/per dose every 6 hour Iv or po
- Adrenaline 0.1ml/kg 1:10000 sc in sever case
2. **Bronchiolitis**

- A viral infection causing obstruction of lower airways and symptom complex similar to asthma.
- Most common in children <2 years old.
- Epidemics occur in winter months.
- Characterized by diffuse crackles, wheezing, and increased work of breathing

**Management Bronchiolitis**

- High-flow oxygen and expedite transport.
- Goal is to improve air exchange and maintain adequate oxygenation (>90%).

  **Inhalation Therapy:**
  - 1 ml of adrenalin in 5 ml of Normal saline via nebulizer.
  - If no improvement with patient in moderate to severe distress:
  - 1 ml of adrenalin in 5 ml of Normal saline via nebulizer May repeat x 1.
  - maintain hydration status

3) **Croup**

- Viral infection causing edema of vocal cords and adjacent trachea (upper airway obstruction)
- Accounts for approximately 90% of infectious upper airway problems in children.
- Occurs more commonly in cold season
- Children 6 months -3 years most commonly affected
- Clinical syndrome consists of cold symptoms and fever for several days, followed by respiratory distress, stridor, and barking cough.
- Symptoms often worse at night

**Croup Management:**

- Positioning
- High flow O2
- Nebulizer 1ml of adrenaline 3ml of N/S
- Dexamethsone 0.6 mg/kg iv
- If no improvement intubation has to be consider

4) Epiglottitis:
Life-threatening bacterial infection causing inflammation and edema of the epiglottis and/or adjacent structures above the larynx
- has associated with fever.
- Respiratory distress occurs
- Fever and drooling within 12 hours of appearance of fever
- Muffled voice or refusal to talk
- Difficulty swallowing suggests upper airway obstruction.
- High pitched noise heard on inspiration
- Children are usually older than 12 months.

Epiglottises Management:
- Minimize interventions if child is conscious and maintaining own airway.
- Do not try to visualize the oral cavity
- Administer 100% O2 only as tolerated.
- If no improvement consider needle cricothyrotomy

**Oxygen administration**

1. **Nasal prongs** Nasal prongs - are short tubes inserted into the nostrils. Place them just inside the nostrils and secure with a piece of tape on the cheeks near the nose.

Set a flow rate of 0.5-1 litres/min in infants and 1-2 litres/min if older in order to deliver 30-35% oxygen concentration in the inspired air.

2. **Nasal catheter** - is made from tubing of 6 or 8 FG size such as a nasogastric tube or suction catheter.

Set a flow rate of 0.5-1 litres for infants and 1-2 litres/min for older children, which delivers an oxygen concentration of 45-60% in the inspired air.

1. **Face Mask**- rate of 0.5-1 litres for infants and 1-2 litres/min for older children, which delivers an oxygen concentration of 45-60% in the inspired air.
Circulation

Objective
At the end of attachment, the trainee is expected to:

- Identify early sign of circulatory problem
- Manage shock in children

If A and B has no problem you can assess C

Assess the circulation
First in this section we will look at the assessment of circulation and signs of shock. To assess if a child has a circulation problem you need to know:

1. ARE THE CHILD’S HANDS WARM?
   - If the child’s hands are warm, there is no problem with the circulation and you can move to the next assessment.
   - If they are cold, you need to assess the circulation further.
   - If it feels warm, the child has no circulation problem and you do not need to assess capillary refill or pulse.
   - If the child’s hands feel cold, you need to assess the capillary refill.

2. IS THE CAPILLARY REFILL TIME LONGER THAN 3 SECONDS?
   - Capillary refill is a simple test that assesses how quickly blood returns to the skin after pressure is applied.
   - It should be less than 3 seconds. If it is more than 3 seconds the child is shocked.
   - Capillary refill is prolonged in shock because the body tries to maintain blood flow to vital organs and reduces the blood supply to less important parts of the body like the skin (peripheral vasoconstriction).
   - So you will need to check the pulse only if the room is cold.
   - The pressure is applied for 3 seconds and then released. Time the capillary refill from the moment of release until total return of the pink colour.
If the refill time is longer than 3 seconds, the child may have a circulation problem with shock.

To confirm, it is necessary to check the pulses.

3. IS THE PULSE WEAK AND FAST?

- The radial pulse should be felt. If this is strong and not obviously fast, the pulse is adequate; no further assessment is needed.
- If the radial pulse is difficult to find, you need to look for a more central pulse (a pulse nearer to the heart).
- In an infant (less than one year of age) the best place to look is at the middle of the upper arm, the brachial pulse.
- If the child is lying down you could look for the femoral pulse in the groin.
- In an older child you should feel for the carotid pulse in the neck.
- The pulse should be strong.
- If the more central pulse feels weak, decide if it also seems fast.
- If the central pulse is weak and fast, the child needs treatment for shock.

Note that we do not recommend blood pressure to assess for shock because of two reasons:

1) Low blood pressure is a late sign in children and may not help identify treatable cases
2) the BP cuff necessary in children of different age groups is mostly unavailable in many district hospitals.

Shock

- The most common cause of shock in children is due to loss of fluid from circulation, either through loss from the body as in severe diarrhoea or when the child is bleeding, or through capillary leak in a disease such as severe infection.
- In all cases except obstructive, it is important to replace this fluid quickly.
- An intravenous line must be inserted and fluids given rapidly in shocked children without severe malnutrition carcinogenic shock.
- The recommended volumes of fluids to treat shock depending on the age/weight of child 20ml/kg every 20 min three times
- If the child has severe malnutrition, you must use a different fluid and a different rate of administration and monitor the child very closely.
- Therefore, a different regime is used for these children.
Treatment of shock

- Treatment of shock requires teamwork.
- The following actions need to be started simultaneously:
  - If the child has any bleeding, apply pressure to stop the bleeding, give oxygen. Make sure the child is warm feeling the brachial pulse in an infant.
  - Weigh the child.
  - Insert an intravenous line (and draw blood for emergency laboratory investigations).
  - Fix the cannula and immobilize the extremity with a splint.

- 15 ml/kg give over 1 hour

- Stay with the child and check the pulse and breathing rate every 5-10 minutes.

- Discontinue the intravenous infusion if either of these increase (pulse by 15, respiratory rate by 5/min).

If there is improvement:

pulse and breathing rate fall.

Repeat 15ml/kg over 1 hour

If the child has NO severe malnutrition

- Insert an intravenous line (and draw blood for emergency laboratory investigations).
- Fix the annual and immobilize the extremity with a splint.
- Attach Ringer’s lactate or normal saline, make sure the infusion is running well.

Infuse 20 ml/kg as rapidly as possible. The circulation should be reassessed as described before

If there is NO improvement:

- Reassess the circulation again, and if there is still no improvement
- Give another 20 ml/kg of Ringer’s lactate or normal saline, as quickly as possible.
- The circulation should be assessed again.
If there is still NO improvement:

- Give 20 ml/kg of blood over 30 minutes unless there is profuse watery diarrhea.
- The circulation should be assessed again.

After three bolus of 20ml/kg of fluid give blood and refer

**Basic life support**

**Objectives**

At the end of this chapter the students expected to

- List steps in the assessment and opening airway
- Assess breathing (Identify respiratory emergency)
- Assess circulatory problem
- Identify When and how to initiate CPR
- Describe the difference in infant, child and CPR

**Basic life support sequence**

**Initiate CPR** — The actions that constitute cardiopulmonary resuscitation (CPR) are opening the airway providing ventilations and performing chest compression.

The sequence in which the actions of CPR for infants and children should be performed as follow:

- Open airway and check breathing
- If there is no breathing, give two rescue breaths
- If there is no response, check pulse
- If pulse 60 beats per minute (bpm), continue ventilation
- If there is no pulse or pulse is <60 bpm, begin ventilation and chest Compressions

**Ventilation** — Breathing support can be provided with mouth-via-devise to mouth, mouth-to-nose via devise, or with a bag and mask. Each rescue breath should be delivered over one second. The volume of each breath should be sufficient to see the chest wall rise.
A child with a pulse 60 bpm who is not breathing should receive one breath every 3 to 5 seconds (12 to 20 breaths per minute). Infants and children who require chest compressions should receive 2 breaths per 30 chest compressions for a lone rescuer 2 breaths per 15 chest compressions.
CHEST COMPRESSIONS —

Essential elements for effective chest compressions:

- Hard
- Fast chest compression
- With full chest recoil
- Minimal interruptions

Chest compressions should be performed over the lower half of the sternum. Compression below the sternum can cause trauma to the liver, spleen, or stomach, and must be avoided.

The effectiveness of compressions can be maximized by attention to the following:

- The chest should be depressed by one-third to one-half of its anterior posterior diameter with each compression
- The optimum rate of compressions is approximately 100 per minute
- Each compression and decompression phase should be of equal duration.
- The sternum should return briefly to its normal position at the end of each smooth compression-decompression rhythm with minimum interruption

Infants — Chest compressions for infants (under one year) may be performed with either two fingers or with the two thumb-encircling hands technique. Two Techniques is recommended when there is a single rescuer.

Figure 8 two fingers chest compression
Two thumb encircling hands — the two thumb-encircling hands technique provides optimum chest compressions when there are two rescuers.

Figure 9 Encircling chest compression

Children — for children (from one year until the start of puberty), compressions should be performed over the lower half of the sternum with either the heel of one hand or with two hands, as for adult victims.

COMPRESSION TO VENTILATION RATIO — Chest compressions in infants and children should always be accompanied by ventilation.

For one rescuer, two ventilations should be delivered during a short pause at the end of every 30th compression.

For two rescuers, two ventilations should be delivered at the end of every 15th compression.

Coordination of compression and ventilation may be facilitated by counting compressions aloud or using an audio-prompted rate guide.
Management of Burn

Objective
At the end of the attachment the trainee are expected to:

- Classify burn
- Describe Initial assessment of a major burn
- Describe burn fluid management

Burn
A major burn is defined as a burn covering 25% or more of total body surface area, but any injury over more than 10% should be treated similarly. Rapid assessment is vital. The general approach to a major burn can be extrapolated to managing any burn. The most important points are to take an accurate history and make a detailed examination of the patient and the burn, to ensure that key information is not missed.

Classification of burn depths
Burns are classified into two groups by the amount of skin loss. Partial thickness burns do not extend through all skin layers, whereas full thickness burns extend through all skin layers into the subcutaneous tissues. Partial thickness burns can be further divided into superficial, superficial dermal, and deep dermal:

Superficial—The burn affects the epidermis but not the dermis (such as sunburn). It is often called an epidermal burn

Superficial dermal—The burn extends through the epidermis into the upper layers of the dermis and is associated with blistering

Deep dermal—The burn extends through the epidermis into the deeper layers of the dermis but not through the entire dermis.

Initial assessment of a major burn
Perform an ABCDEF primary survey

A—Airway with cervical spine control, B—Breathing,
C—Circulation, D—Neurological disability, E—Exposure with environmental control, F—Fluid resuscitation
Assess burn size and depth (see later article for detail)
Establish good intravenous access and give fluids
Give analgesia
Catheterize patient or establish fluid balance monitoring
Take baseline blood samples for investigation
Dress wound
Perform secondary survey, reassess, and exclude or treat associated injuries
Arrange safe transfer to specialist burns facility

**Fluids**

Calculate resuscitation formula based on surface area and time since burn

**F—Fluid resuscitation**

1) *Total fluid requirement for first 24 hours*

\[4 \text{ ml} \times (\text{total burn surface area}) \times (\text{wt in kg})/24\text{hours}\]

2) *Half to be given in first 8 hours, half over the next 16 hours*

3) *Subtract any fluid already received from amount required for first 8 hours*

4) *Calculate hourly infusion rate for first 8 hours*

5) *Calculate hourly infusion rate for next 16 hours*

**Maintenance fluid required for a child**

A 24 kg child with a resuscitation burn will need the following maintenance fluid:

- 4 ml/kg/hour for first 10 kg of weight = 40 ml/hour *plus*
- 2 ml/kg/hour for next 10 kg of weight = 20 ml/hour *plus*
- 1 ml/kg/hour for next 4 kg of weight = 1×4 kg = 4 ml/hour

Total = 64 ml/hour

**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 easily
titrated against pain and respiratory depression. The need for further doses should be assessed within 30 minutes.

**Investigations**
The amount of investigations will vary with the type of burn Hematocrit /Hct/, Total Serum Protein/TSP/

**Secondary survey**
At the end of the primary survey and the start of emergency management, a secondary survey should be performed. This is a head to toe examination to look for any concomitant injuries.

**Dressing the wound**
Once the surface area and depth of a burn have been estimated, the burn wound should be washed and any loose skin removed. Blisters should be de-roofed for ease of dressing, except for palmar blisters (painful), unless these are large enough to restrict movement. The burn should then be dressed. For an acute burn which will be referred to a burn centre, cling film is an ideal dressing as it protects the wound, reduces heat and evaporative losses, and does not alter the wound appearance. This will permit accurate evaluation by the burn team later.

**Snake bite and poisoning**

**Snakes Bites:** Several different types of snakes must be differentiated due to the varying effects of their venoms.

Many snake bites are provoked and thus involve the upper extremities some the snakes venom is voluntarily injected by venom gland contraction.

Snakes type has a neurotoxin which may lead to paralysis and respiratory arrest. There may be varying hemotoxins which profoundly decrease platelet and clotting factors.

**Treatment**

First Aid Management

- Initiate BLS as necessary (ABCs) - Move the patient to a health care facility as rapidly as possible
- Minimize movement of an affected extremity and keep the extremity below the level of the heart
- Avoid ice, aspirin (coagulopathies possible), alcohol or sedatives
Tourniquets not universally recommended; although constriction band in experienced hands may be useful when incision and suction are indicated or a long transport anticipated. Incision and Suction these should be considered only if:

- Patient is more than one hour from a medical facility
- The only incisions to be made are extensions of not more than 1 cm long and 0.5 cm deep.

DO NOT MAKE AN X SHAPED CROSS INCISION, and always keep in mind underlying structures.

**Tourniquets**

A varying portion of venom may be absorbed via the lymphatic system. Given this, if a medical facility is not nearby wide constricting band may be placed around an extremity.

Use of a tourniquet is a controversial topic. A BP cuff at 15-20 mmHg is adequate. Otherwise the band should be wide and two fingers able to pass freely under it. The band should be tight enough to occlude lymphatic flow but loose enough to palpate pulses distal to the bite. If swelling occurs, place a second tourniquet above the first one before removal of the first band.

Before this is done it is recommended that 2 IV’s are in place, fluid resuscitation is underway, and the antitoxin is given.

**POISONING**

A poison is any substance that causes harm if it gets into the body. Harm can be mild (for example, headache or nausea) or severe (for example, fits or very high fever), and severely poisoned people may die. Almost any chemical can be a poison if there is enough in the body.

*Acute exposure* is a single contact that lasts for seconds, minutes or hours, or several exposures over about a day or less. *Chronic exposure* is contact that lasts for many days, months or years.

**Routes of Exposure**

*Through the mouth by swallowing (ingestion)*

*Through the lungs by breathing into the mouth or nose (inhalation)*

*Through the skin by contact with liquids, sprays or mists*
By injection through the skin

Epidemiology

Poisoning is divided into accidental poisoning and non accidental or self poisoning.

Most cases of accidental poisoning occur within the home. The poisoning agents are usually household agents, medicaments and plant material.

Non-Accidental Poisoning (Self-poisoning)

Self poisoning is usually seen in older children and adolescents suffering from depression, serious illness, or alcohol dependence in an attempt to commit suicide or to attract attention.

Consequences of Poisoning

The effects of poisoning maybe none, mild or severe depending on:
- The amount of poison ingested.
- The nature of the substance
- The age of the child.
- The nutritional status of the child.
- The state of the stomach—whether empty or full of food.

The effects of poison

The effects of poisons can be local or systemic. A local effect is limited to the part of the body in contact with the chemical A systemic effect is a more general effect that occurs when a poison is absorbed into the body.

Local effects

On the skin chemicals can cause itching, rash, pain, swelling, blisters or serious burns. Inside the air passages and lungs irritation from vapors and gases can cause coughing, choking and lung edema.

Systemic effects
There are many ways in which poisons can cause harm by damaging organs such as the brain, nerves, heart, liver, lungs, kidneys, or skin. Poisons can also lead to muscle paralysis.

**Common Substances Causing Poisoning in Children**

The commonest substances causing poisoning in East and Southern Africa are household chemicals followed by drugs.

**Management**

The management of the poisoned child is at two levels; at home where first aid is administered and in the hospital where specific treatment is given.

*First aid at home*

First aid treatment should be administered by the person who finds the child after the poisoning episode. Care should be taken so that the first aid treatment does not cause severe complications that may be worse than the original poisoning.

*Treatment in hospital*

The clinician should take a brief history, examine the child thoroughly and rapidly and then do the following:
1. Ensure a clear airway and support respiration.
2. Treat shock if present.
3. Remove poison from the body before it is absorbed, by inducing vomiting or doing a gastric lavage except when kerosene or a corrosive has been ingested.
4. Reduce absorption by administering activated charcoal which absorbs many toxins and prevents subsequent absorption.
6. Anti-dotes should be used but these are available for very poisons.
7. General supportive measures are important to ensure adequate hydration, temperature control, fluid and electrolyte balance, nutrition intake and control of convulsions

**Newborn Resuscitation**

**Objective**

At the end of this chapter the participants are expected to:

1. Handle new born immediately after delivery
2. Recognize air way obstruction and their management

3. Identify the peculiarity of new born resuscitation

Resuscitation efforts should focus on improving respiratory status and maintaining body temperature.

**A. Evaluation and Treatment Priorities**

- During delivery, suction mouth then nose before delivery of body.
- This is especially important if there is meconium in the amniotic fluid.
- Dry infant and maintain warm environment.
- Wrap the baby in a thermal blanket.
- Cover the infant’s head to preserve warmth.

**B. Open and position the airway in the “sniffing” position.**

- Suction airway again using bulb syringe, mouth first then nasopharynx.
- Avoid hyperextension of the neck.

**If thick meconium is present in apneic and/or hypotonic infant:**

- Initiate suctioning before the infant takes first breath.
- Suction the airway while withdrawing the suction tube.
- Repeat suction only if meconium is not cleared and infant remains apneic and/or hypotonic, then ventilate infant with BVM.
- If infant becomes bradycardic (<60), discontinue suctioning and provide ventilation immediately.
- Assess Breathing and adequacy of ventilation.
- Stimulate the infant by rubbing the back or flicking the soles of the feet.

**If evidence of central cyanosis:**

- Oxygen 100% via blow-by administration.

**If infant is apneic:**

- BVM at 40-60 breaths/minute with 100% oxygen.
- Assess Heart Rate – Auscultation or palpation of brachial artery or umbilical cord stump.
- If heart rate is <60 and signs of poor perfusion are persistent after 30 seconds of assisted ventilation with 100% oxygen initiate the following:

**Continue ventilation**

- Begin chest compressions and CPR: ratio of 1 to 3, rate of 100 compressions per minute (hard and fast)
- Stop CPR when heart rate >60 with signs of improved perfusion
- If heart rate is 60 – 100/minute

**Continue ventilation**

- Assess skin color – If cyanosis use blow-by oxygen
- If heart rate is >100/minute Continue assisted ventilation until patient is breathing adequately on own and is vigorous.

**Reassess the infant frequently**

- Pulse, respiratory rate, tone, color, and response.
- Contact direct medical control for additional instructions
- Continued care of mother.
- Place two clamps 6 and 8 inches from baby, cut umbilical cord between clamps.
- Transport delivered placenta to hospital with the baby

**Overall Activities**

- Discussion on case scenario #26-31
- Simulation&practice in small group
- General discussion on questions raised by participants
Annex

DRUGS COMMONLY USED IN THE ED

Adrenaline
Endogenous catecholamine with alpha and beta action:
1. Treatment of anaphylaxis
2. Bronchodilator
3. Positive inotrope
4. Given by nebuliser for croup
5. Prolongation of local anesthetic action
   - 1:1000 contains 1mg/ml; 1:10,000 contains 100ug/ml; 1:200 000 contains 5 ug/ml
   - Cautions and contraindications: Arrhythmias especially with halothane and in elderly.
   - Administer - Via central catheter whenever possible
   - Side-effects: Hypertension, tachycardia, anxiety, hyperglycemia, arrhythmias and reduces uterine blood flow
Dose
   - (Pediatric) 1-3 IV/IM/IO 0.1ml/kg of 1:10000(10µg/kg) endotracheal tube (ETT) 0.1ml/kg of 1:1000(100ug/kg)
   - Infusion 0.05-1 µg/kg/min, Nebulisation 0.5 ml/kg (up to 5ml) 1:1000.
     Maximum dose for infiltration 2 µg/kg
   - (Adult) 1-3 IV/IM/ET 1ml aliquots of 1:10 000 up to 5-10ml (0.5-1mg)
     Infusion 2-20µg/min (0.04-0.4ug/kg/min). Nebulisation 5 ml 1:1000.
     Maximum dose for infiltration 2 µg/kg

Aminophylline
   - Methylxanthine bronchodilator
   - Used in prevention and treatment of asthma.
   - Serum levels 10-20 mg/l (55-110 umol/l)
   - Cautions and contraindications: in patients already receiving oral or IV theophyllines. Where serum level known, aminophylline 0.6mg/kg should increase the level by 1 mg/l.
   - Side effects: Palpitations, tachycardia, tachypnoea, seizures, nausea, and arrhythmias
   - Dose: (pediatric): 5mg/kg over 30min, then 0.5-1mg/kg/hr
     (Adult): 5mg/kg over 30 min then 0.5mg/kg/hr infusion

Amiodarone
Mixed class 1C and III antiarrhythmic
Useful in treatment of supraventricular and ventricular arrhythmias
Cautions and contraindications: in patients with sinoatrial heart block, thyroid dysfunction, pregnancy, porphyria.
Administration: Dilute in dextrose 5%, not saline and give via central catheter
Side-effects Commonly causes thyroid dysfunction and reversible corneal deposits
Dose
(Pediatric) 25µg/kg/min for 4 hr, then 5-15 µg/kg/min
(Adult) 5 mg/kg over 1-2 hr. Maximum 1.2 g in 24 hr 300mg slow IV bolus for defibrillation resistant VF.

Atenolol
Cardio selective B-blocker Long acting
Cautions and contraindications: Asthma, heart failure, AV block, verapamil treatment
Side effects: Bradycardia, hypotension, and decreased contractility
Dose
(Pediatric) 0.05mg/kg every 5 min-max 4 doses
(Adult) 5-10mg over 10min. PO: 50mg/day

Atropine
Muscarinic acetylcholine antagonist. Vagal blockade at AV and sinus node increases heart rate. (Transient decrease at low doses due to weak agonist effect). Tertiary amine therefore crosses blood-brain barrier.
Side-effects: Decreases secretions, and lower esophageal sphincter tone relaxes bronchial smooth muscle, Confusion in elderly
Dose
(Pediatric) IV:10-20ug/kg. Control of muscarinic effects of neostigmine 10-20ug/kg, IM/SC :10-30 µg/kg PO: 40 µg/kg
(Agult) 300-600ug. Prevention of muscarinic effects of neostigmine: 600-1200µg. Cardiac arrest 3 mg

Bicarbonate (Sodium)
Alkaline salt used for correction of acidosis and to enhance onset of action of local anesthetics. 8.4% = (1000) mmol/l
Cautions and contraindications: Precipitation with calcium containing solutions, increased CO₂ production, and necrosis on extravasations.
Administer Via central catheter if possible
Side-effects Alkalosis, hypokalaemia hypernatraemia, hypocalcaemia
Dose: ( mmol) in acidosis: weight ( kg) x base deficit x 0.3
(Pediatric) 1ml/kg 8.4% solution (1mmol/kg)
(Agult)Dependent on degree of acidosis:
Resuscitation: 50 ml of 8.4% then recheck blood gases.
Bicarbonation of LA: 1ml 8.4% to 20ml Bupivacaine. 1ml 8.4% to 10ml lidocaine/prilocaine

**Calcium chloride**
Electrolyte replacement, positive inotrope, hyperkalaemia, hypomagnesaemia. Calcium chloride 10% contains Ca$^{2+}$ 680µmol/ml
Cautions and contraindications: Necrosis on extravasations, incompatible with bicarbonate
Side-effects: Arrhythmias, hypertension hypocalcaemia
Dose (pediatric) 0.1-0.2 ml/kg 10% solution
(Adult) 2-5ml 10% solution (10mg/kg, 0.07mmol/kg)

**Calcium gluconate**
Calcium gluconate 10% contains Ca$^{2+}$ 220 µmol/ml
Cautions and contraindications: Less phlebitis than calcium chloride
Side-effects: As calcium chloride
Dose
- (Pediatric) 0.3-0.5ml/kg 10% solution (max20ml)
- (Adult) 6-15ml of 10% solution (30mg/kg, 0.07mmol/kg)

**Chlorpromazine**
Antipsychotic. Mild alpha blocking action, potent antiemetic and used for chronic hiccups
Cautions and contraindications: Hypotension
Side-effects Extra pyramidal and ant cholinergic symptoms, sedation, hypotension
Dose
- (Pediatric) 0.1-1mg/kg over 20min
- (Adult) Up to 25 mg (at 1mg/min diluted in saline to 1mg/ml) Deep IM:25-50mg 6-8hourly

**Cimetidine**
Competitive H2 histamine receptor antagonist Reduction of gastric acid
Side-effects Hypotension and arrhythmias on rapid IV administration
Confusion in elderly
Dose
- (pediatric) IV/PO:10-15mg/kg bd
- (adult) 200mg over 2 min (diluted in saline) qds. PO: 400 mg bd

**Citrate (Sodium)**
Non-particulate antacid, oral premedication for Aspiration prophylaxis
Dose (adult) PO:30ml-0.3Msolution

**Clonidine**
Selective alpha2 agonist, Reduces requirement for opioids and volatile anesthetics, Enhances epidural analgesia
Cautions and contraindications: Rebound hypertension on acute withdrawal of chronic therapy
Side-effects Hypotension, sedation
Dose
  o (Pediatric)  3-5µg/kg slowly. PO premed: 4 µg/kg, Caudal: 1 µg/kg
  o (Adult)  150-300 µg over 5min. Epidural: 150 µg in 10ml saline

**Codeine phosphate**
Opioid used for mild to moderate pain
Side-effects Nausea, Vomiting, dysphoria, drowsiness, constipation
Dose
(Pediatric) PO/IM/PR: 1mg/kg 6-hourly (max 3 mg/kg/d)
(Adult) PO/IM 30-60mg 4-hourly (max 240mg/d)

**Dexamethasone**
Prednisolone derivative corticosteroid.
Less sodium retention than hydrocortisone.
Use: Cerebral edema, edema prevention anti-emetic
Cautions and contraindications: Interacts with anti cholinesterase agents to increase weakness in myasthenia gravis
Side-effects See prednisolone
Dose
  o (Pediatric) IV/IM/SC: 200-400 µg/kg bd  Cerebral edema 100 µg/kg qds.
  Croup: 250 µg/kg then 125/ µg/kg qds for 24hr. Anti-emetic 150µg/kg
  o (Adult)  IV/IM/SC4-8mg  Cerebral edema 4mg qds Antiemesis 8 mg

(Dexamethasone 0.75mg =prednisolone 5mg)

**Diazepam**
Long-acting benzodiazepine,
Use: Sedation or termination of status epilepticus, Alcohol withdrawal
Cautions and contraindications: Thrombophlebitis: emulsion, (Dizemuls) less irritant to veins
Side-effects Sedation, circulatory depression
Dose
  o (Pediatric)  0.2-0.3mg/kg. Rectal: 0. 5mg/kg as Stesolid or may use IV preparation
  o (Adult)  2-10mg, repeat if required

**Diclofenac sodium (Voltarol)**
Potent NSAID analgesic for mild to moderate pain
Cautions and contraindications: Hypersensitivity to aspirin, asthma, severe renal impairment, and peptic ulceration
Side-effects Gastrointestinal upset or bleeding, bronchospasm, tinnitus, fluid retention, platelet inhibition

Dose
- (Pediatric) PO/PR: 1mg/kg tds. Maximum 3 mg/kg/day (>1yr)
- (Adult) PO/PR: 25-50 mg tds (or 100mg 18-hourly) Maximum 150mg/d

Digoxin
Cardiac glycoside. Weak inotrope and control of ventricular response in supraventricular arrhythmia. Therapeutic levels 0.5-2µg/l
Cautions and contraindications: Reduce dose in elderly. Enhanced effect/toxicity in hypokalaemia. Avoid cardioversion in toxicity
Side-effects Anorexia, nausea, fatigue, arrhythmias

Dose
- (Pediatric) Rapid IV/PO loading: 15µg/kg stat, then 5 µg/kg qds, then 5 µg/kg bd PO: 4µg/kg bd
- (Adult) Rapid IV loading 250-500 µg over 30 min. Maximum 1mg/24 hr. PO loading 1-1.5mg in divided doses over 24hr. PO maintenance: 125-250 µg/d

Dobutamine
B1 adrenergic agonist, positive inotrope and chronotrope
Use: Cardiac failure
Cautions and contraindications: Arrhythmias and hypertension. Phlebitis, but can be administered peripherally
Side-effects: Tachycardia, Decreased peripheral and pulmonary vascular resistance

Dose (pediatric) Infusion: 2-20 µg/kg/min.
(adult) Infusion: 2.5-10 µg/kg/min

Dopamine
Naturally occurring catecholamine with a1,B1 and dopaminergic activity. Inotropic agent
Cautions and contraindications: administer Via central catheter, phaeochromocytoma (due to noradrenaline release)
Side-effects Tachycardia, dysrhythmias

Droperidol
Butyrophenone related to haloperidol. Neuroleptic anaesthesia and potent anti-emetic. Duration 4hr
Cautions and contraindications: Alpha adrenergic blocker, Parkinson`s disease
Side-effects: Vasodilatation, hypotension, dystonic reactions

Dose

(Pediatric) Anti-emetic: 25-75 µg/kg

(Adult) Anti-emetic: 0.5-2.5 mg. Neuroleptic anaesthesia: 0.2 mg/kg with fentanyl 4 µg/kg

10 mg Reversal: 0.5 mg/kg with anticholinergic

**Ephedrine**

Direct and indirect sympathomimetic (α and β adrenergic action).
Vasopressor, safe in pregnancy
Duration 10-60min
Cautions and contraindications: in elderly, hypertension and CVS disease.
Tachyphylaxis. Avoid with MAOI

Side-effects: Tachycardia, hypertension

Dose (adult) 3-6 mg repeated (dilute 30 mg in 10 ml saline, 1 ml increments)
IM: 30 mg

**Ergometrine**

Ergot alkaloid used to control uterine hypotony or bleeding

Cautions and contraindications: Severe cardiac disease or hypertension

Side-effects: Vasoconstriction, hypertension, vomiting

Dose IM: 1 ml. Not recommended IV

**Esmolol**

Short-acting cardio selective β-blocker, metabolized by red cell esterases.
Treatment of supraventricular tachycardia or intra-operative hypertension

Duration 10 min

Cautions and contraindications: Asthma, heart failure, AV block, verapamil treatment

Side-effects: Hypotension, bradycardia, May prolong action of suxamethonium

Dose

(Pediatric) SVT: 0.5 mg/kg over 1 min, then 50-200 µg/kg/min

(Adult) SVT: 0.5 mg/kg over 1 min, then 50-200 µg/kg/min.

Hypertension: 25-100 mg, then 50-300 µg/kg/min

**Etomidate**

IV induction agent. Cardio stable in therapeutic doses.

Cautions and contraindications: Pain on injection, adrenocortical suppression

Side-effects: Nausea and vomiting, myoclonic movements

Dose (pediatric) & (adult) 0.3 mg/kg

**Fentanyl**

Synthetic phenylpiperidine derivative opioid analgesic. High lipid solubility and cardio stability. Duration 30-60 min

Cautions and contraindications: Reduce dose in elderly, delayed respiratory depression and pruritus if epidural/spinal
Side-effects - Circulatory and ventilators depression. High doses may produce muscle rigidity.

Dose
(Pediatric) 1-5 µg/kg, up to 25 µg/kg if postop Ventilation.
Infusion: 2-4 µg/kg

(Agent) 1-5 µg/kg (up to 50µg/kg). Epidural: 50-100 µg (diluted in 10ml saline/local anesthetic). Spinal 5-20 µg

**Flumazenil**
Benzodiazepine receptor antagonist. Duration 45-90 min

Cautions and contraindications - Benzodiazepine dependence (acute withdrawal), Resedation if long-acting benzodiazepine is used.

Side-effects Arrhythmia, seizures

Dose
(Pediatric) 5 µg/kg, then repeat up to 40 µg/kg
Infusion: 2-10 µg/kg/hr

(Agent) 200 µg then 100 µg at 60 s intervals (up to maximum 1 mg).
Infusion: 100-400 µg/hr

**Furosemide (frusemide)**
Loop diuretic used in treatment of hypertension, congestive cardiac failure, renal failure, and fluid overload

Side-effects Hypotension, tinnitus, ototoxicity, hypokalaemia, and hyperglycemia

Dose (pediatric) 0.5-1.5mg/kg bd (adult) 10-40mg slowly

**Glucose**
Treatment of hypoglycemia in unconscious patient

Cautions and contraindications - 50% solution irritant therefore flush after administration

Dose
(Pediatric) 0.5ml/kg of 50% solution: use more dilute solutions

(Agent) 25-50g (50-100ml 50% solution). Can use more dilute solutions

**Heparin (unfractionated)**
Endogenous mucopolysaccharide used for anticoagulation.
Half-life 1-3 hr. 100U = 1mg

Cautions and contraindications - Monitor activated partial thromboplastin time (APTT). Reversed with protamine

Side-effects - Hemorrhage, thrombocytopenia, hyperkalaemia

Dose
(Pediatric) Low dose: 50-75U/kg IV then 10-15 U/kg/hr.
Full dose: 200U/kg IV then 15-30U/kg/hr

(Agent) Low dose SC: 5000U bd
Full dose IV: 5000U, then 24000-48000U per 24hr infusion

**Hydralazine**
Direct-acting arteriolar vasodilator used to control arterial pressure.
Duration 2-4 hr
Cautions and contraindications - Higher doses required in rapid acetylators.
Systemic Lupus Erythematosus
Side-effects - Increased heart rate, cardiac output, and stroke volume
Dose (pediatric) 0.1-0.5mg/kg
       (Adult) 5mg every 5min to a maximum of 20mg

**Hydrocortisone**
Endogenous steroid with anti-inflammatory and potent mineral corticoid action, Treatment of allergy. Hydrocortisone 20mg = prednisolone 5mg
Side-effects Hyperglycemia hypertension, psychic disturbance muscle weakness, fluid retention
Dose (pediatric) 4mg/kg then 2-4mg/kg qds
       (Adult) IV/IM: 50-200mg qds. Adrenal suppression and surgery: 25mg at induction then 25mg qds. PO:10-20mg/d

**Hydromorphone hydrochloride**
Opioid used for moderate to severe pain
Cautions and contraindications - As morphine
Side effects Nausea, vomiting, dysphoria, drowsiness
Dose (adult) PO:1.3-4mg 4-hourly increased as necessary PO slow release: 4mg bd

**Hyoscine (Hydrobromide Scopolamine)**
Antimuscarinic sedative anti-emetic agent used as premedication
Cautions and contraindications - See atropine.
Avoid in elderly –delirium
Side effects See atropine, Sedation
Dose (pediatric) IV/IM/SC: 10 µg/kg  (adult) IV/IM/SC: 200-600 µg PO: 300 µg qds

**Ibuprofen**
NSAID analgesic for mild to moderate pain
Cautions and contraindications - Hypersensitivity to aspirin, asthma, severe renal impairment, peptic ulceration
Side-effects- Gastrointestinal upset or bleeding, bronchospasm, tinnitus, fluid retention, platelet inhibition
Dose (pediatric) PO:10mg/kg tds or 5mg/kg qds(>7kg)
       (adult) PO:400mg qds

**Insulin (Actrapid)**
Human soluble pancreatic hormone facilitating intracellular transport of glucose and anabolism.
Use- Diabetes mellitus, Ketoacidosis and hyperkalaemia
Cautions and contraindications- Monitor blood glucose and serum potassium.
Store at 2-8°C
Side-effects- Hypoglycemia, hypokalaemia
Dose
(Pediatric) Ketoacidosis: 0.1-0.2U/kg then 0.1U/kg/hr
(Adult) Ketoacidosis: 10-20U then 5-10U/hr.

Ketamine
Phencyclidine derivative producing dissociative anesthesia.
Induction/maintenance of anesthesia in high-risk or hypovolemic patients
Cautions and contraindications-Emergence delirium reduced by benzodiazipines.
Caution in hypertensive patients.
Control excess salivation with an ant muscarinic agent

Side-effects- Bronchodilator. Increased ICP, blood pressure, uterine tone, salivation. Respiratory depression if given rapidly

Dose
(Pediatric) Induction: 1-2 mg/kg IV 5-10mg/kg IM    Infusion: 1-3mg/kg/hr Caudal: 0.5mg/kg
(Adult) Induction: 1-2mg/kgIV 5-10mg/kg IM Infusion:1-3mg/kg/hr (analgesia only 0.25mg/kg/hr)

Ketorolac (Tornado)
NSAID analgesic for mild to moderate pain.
Not licensed for perioperative use
Cautions and contraindications- Hypersensitivity to aspirin, asthma, severe renal impairment, peptic ulceration
Side-effects-Gastrointestinal upset or bleeding, bronchospasm, tinnitus, fluid retention, platelet inhibition

Dose       (pediatric)  Slow IV/IM: 0.5mg/kg up to 30 mg qds
            (Adult)    Slow IV/IM: 10 mg then 10-30 mg every 4-6 hr (maximum daily dose 90 mg. But 60mg in elderly)

Labetalol - Combined Alpha (mild) and Beta-adrenergic receptor antagonist. Blood pressure control without reflex tachycardia. Duration 2-4hr
Cautions and contraindications- Asthma, heart failure, AV block, verapamil treatment
Side-effects-Hypotension, bradycardia, bronchospasm liver damage

Dose
(Pediatric) 0.2mg/kg boluses up to 1mg/kg. Infusion:1-3mg/kg/hr
(adult) 5mg increments up to 100mg Infusion: 20-160 mg/hr (in dextrose)

Lidocaine
Amide type local anesthetic:
1. Treatment of ventricular arrhythmias
2. Reduction of presser response to intubations
3. Local anesthetic-rapid onset, duration 30-90 min (prolonged by adrenaline pKa 7.7)

Cautions and contraindications
Adrenaline-containing solutions contain preservative
Maximum dose dependent upon injection site 3mg/kg/4hr (6mg/kg with adrenaline)

Side-effects
Toxicity: tongue/circumpolar numbness, restlessness tinnitus, seizures, cardiac arrest prolongs action of neuromuscular blockers

Dose
(Pediatric) 1. Antiarrhythmic 1mg/kg then 10-50μg/kg/min
2. Attenuation of pressor response: 1.5mg/kg
3. Local anaesthesia: 0.5-2% solution

(Adult) 1. Antiarrhythmic: 1mg/kg then 1-4mg/min
2. Attenuation of pressor response: 1.5mg/kg
3. Local anesthesia: 0.5-2% solution

Lorazepam
Benzodiazepine: Sedation or premedication; Status epileptics.
Duration 6-10 hr.
Cautions and contraindications: Decreased requirement for anesthetic agents
Side-effects: Respiratory depression in combination with opioids. Amnesia

Dose:
(pediatric) Status epileptics. 0.1mg/kg; max 4mg
(Adult) PO: 2-4mg 1-2hr preop IV/IM: 1.5-2.5mg
Status epileptics: 4mg IV

Magnesium sulphate
Essential mineral used to treat: 1. Hypomagnesaemia 2. Arrhythmias
3. Eclamptic seizures 4. Severe asthma
Magnesium sulphate 50% = 500mg/ml = 2mmol Mg^{2+}/ml.
Normal plasma level Mg^{2+} 0.75-1.05 mmol/l Therapeutic level 2-4mmol/l
Cautions and contraindications: Potentiates muscle relaxants. Monitoring of serum level essential during treatment. Myasthenia and muscular dystrophy
Heart block. Magnesium sulphate 1g = mg 2+ 4mmol
Side-effects- CNS depression, hypotension, muscle weakness

Dose
(Pediatric)
1. Hypomagnesaemia: 0.2ml/kg 50% solution over 20 min
2. Arrhythmias: 0.1ml/kg 50% solution over 20min

(Adult)
1. Hypomagnesaemia 10-15mg/kg over 20min, then 1g/hr.
2. Arrhythmias/asthma: 2g (8mmol) over 10min
3. Eclampsia: 4g (16mmol) over 10min then 1g/hr for 24hr

Mannitol
Osmotic diuretic used for renal protection and reduction of intracranial pressure. 20% solution = 20g/100ml
Cautions and contraindications- Extra cellular volume expansion, especially in severe renal or cardiovascular disease
Side-effects Diuresis, ARF, hyper tonicity
Dose
(Pediatric) 0.25-0.5g/kg (adult)0.25-1g/kg (typically 0.5g/kg of 20% solution)

**Methoxamine (Vasoxine)**
Potent direct-acting alpha1-adrenergic sympathomimetic.
Treatment of hypotension. Duration 15-60min
Cautions and contraindications- Pregnancy. Caution in elderly and hypertensives
Extravasations can cause necrosis
Side-effects-Hypertension, reflex bradycardia, arrhythmias, decreased renal and placental perfusion
Dose (pediatric) 10 µg/kg increment
(Agent) 1-2mg. Dilute 20mg in 20ml saline and give 0.5-1ml increments
( increase dilution in elderly

**Metoclopramide**
Dopaminergic anti-emetic, which increases gastric emptying and lower esophageal sphincter tone
Cautions and contraindications- Hypertension in phaeochromocytoma.inhibits plasma cholinesterase increases IOP
Side-effects-Extrapyramidal/dystonic reactions (treat with benztropine or procyclidine)
Dose (pediatric) PO/IM/IV:0.15mg/kg up to 10mg tds
(adult) po/im/iv:10mg tds

**Metoprolol**
Cardio selective B-blocker
Cautions and contraindications- Asthma, heart failure, AV block, verapamil treatment
Side-effects- Causes bradycardia, hypotension, and decreased cardiac contractility
Dose (pediatric) 0.1mg/kg up to 5mg over 10min
(adult) 1-5mg over 10min

**Midazolam**
Short-acting benzodiazepine. Sedative, anxiolytic, amnesic, anticonvulsant.
Duration 20-60min. Oral administration of IV preparation effective though larger dose required
Cautions and contraindications- Reduce dose in elderly (very sensitive)
Side-effects Hypotension, respiratory depression, apnoea
Dose (Pediatric) 0.1-0.2mg/kg. PO:0.5mg/kg (use IV preparation in orange squash) Intranasal: 0.2-0.3mg/kg (use 5mg/ml IV preparation)
(Agent) Sedation:0.5-5mg.titrated to effect. PO:0.5mg/kg
(use IV preparation in orange squash). IM:2.5-10mg(0.1mg/kg)

**Morphine**
Opioid analgesic
Cautions and contraindications-

- Prolonged risk of respiratory depression
- pruritus, nausea when used via spinal/epidural

Side-effects- Histamine release. Hypotension, bronchospasm, nausea, vomiting, pruritus, dysphoria

Dose

(Pediatric)

PO: 0.3-0.5 mg/kg 4 hourly. IV boluses: 50-100 μg/kg For patient controlled analgesia (PCA) and, nurse controlled analgesia (NCA), Infusion 1 mg/kg in 50 ml saline i.e. 20 μg/kg/ml; Rate 1-2 ml/hr (20-40 μg/kg/hr)

(Adult)

IV: 2.5-10 mg IM/SC: 5-10 mg 4-hourly PO: 10-30 mg 4-hourly PCA: 1 mg 5 min lockout infusion 1-3.5 mg/hr. Epidural 2-5 mg preservative free. Spinal: 0.1-1 mg preservative free

Naloxone

Pure opioid antagonist, Can be used in low doses to reverse pruritus associated with epidural opiates and as depot IM injection in newborn of mothers given opioids

Cautions and contraindications-

- Beware renarcotisation if reversing long-acting opioid. Caution in opioid addicts—may precipitate acute withdrawal.

Duration of action 30 min

Dose

(Pediatric) 5-10 μg/kg Infusion 5-20 μg/kg/hr IM depot in newborn: 200 μg. Pruritus: 0.5 μg/kg

(Adult) 200-400 μg titrated to desired effect.

Treatment of opioid/epidural pruritus: 100 μg bolus plus 300 μg added to IV fluids

Nitroprusside (Sodium-SNP)

Nitric oxide generating potent peripheral vasodilator.

Use- for Controlled hypotension

Cautions and contraindications-

- Protect solution from Light. Metabolism yields cyanide, which is then converted to thiocyanate

Side-effects-

- Methaemoglobinemia, hypotension, tachycardia. Cyanide causes tachycardia, sweating, acidosis

Dose (pediatric) Infusion: 0.3-1.5 μg/kg/min

(adult) Infusion: 0.3-1.5 μg/kg/min(up to 6 μg/kg/min).

Maximum dose: 1.5 mg/kg (acutely)

Nor adrenaline

Potent catecholamine alpha adrenergic agonist Vasoconstriction

Cautions and contraindications

Via central catheter only. Potentiated by MAOI and tricyclic antidepressants
Side-effects  Reflex bradycardia arrhythmia. hypertension
Dose:  (pediatric)  Infusion 0.1-1 µg/kg/min
(adult) Infusion 2-20 µg/min (0.04-0.4 µg/kg/min)

Omeprazole
Proton pump inhibitor, for Reduction in gastric acid
Side effects- Headache, diarrhea
Dose  (pediatric)  PO:  0.7-1.4mg/kg up to 40mg/day
(adult)  PO/Slow IV20-40mg/day. Premedication PO:40 mg evening before
and morning of surgery

Oxycodone
Opioid used for moderate pain, often in palliative care IV preparation
available: dose 1-10mg 4-hourly
Side-effects  Nausea, vomiting, dysphoria, drowsiness
Dose  (adult)  PO: Oxynorm 5mg 4-6 hourly increased up to 400mg/d as
required

Oxytocin (Syntocinon)
Non-apeptide hormone, which stimulates uterine contraction.
  Induction of labour and prevention of postpartum hemorrhage
Side-effects  Vasodilatation, hypotension, flushing , and tachycardia
Dose  (adult)  Postpartum slow IV:5U, followed if required by infusion
( 30U in 500ml saline at 30-125ml/hr)

Paracetamol
Mild to moderate analgesic and antipyretic
Side-effects  Liver damage in overdose
Dose
Neonates:  10-15mg/kg 6-hourly (5mg/kg if jauniced) Max 60mg/kg/d
(Pediatric)  PO/PR:  20mg/kg 6-hourly Rectal loading dose 30-40mg/kg
(Adult)  PO:0. 5-1g qds slow IV:0. 5-1g qds

Pethidine
Synthetics opioid:  1. Analgesia (agent of choice in asthma).
  2. Postoperative shivering
Cautions and contraindications- Seizures possible in high dosage – maximum
daily dose 1g/d (20mg/kg/d). MAOI
Side-effects;  Respiratory depression, hypotension, dysphoria
Dose
(Pediatric)  IV/IM/SC:0. 5-1mg/kg. Infusion: 5mg/kg in 50ml 5% dextrose at
1-3ml/hr  (100-300μg/kg/hr
Phenytoin
Anticonvulsant and treatment of digoxin toxicity.
Serum levels 10–20 mg/l (40–80 µmol/l)
Cautions and contraindications - Avoid in AV heart block and pregnancy
Monitor ECG/BP on IV Administration. Porphyria
Side-effects Hypotension, AV conduction defects, ataxia. Enzyme induction
Dose (pediatric) Loading dose 15mg/kg over 1hr.
(Adult) 15mg/kg over 1hr (dilute to 10 mg/ml in saline), Then
100mg tds. Arrhythmia: 3.5-5mg/kg (rate <50mg/min)

Potassium chloride
Electrolyte replacement
Cautions and contraindications- Dilute solution before administration
Side-effects- Rapid infusion can cause cardiac arrest. High concentration
causes phlebitis
Dose (pediatric) 0.5mmol/kg over 1 hr. Maintenance: 2-4mmol/kg/d
(Adult) 10-20mmol/hr (max. concentration 40mmol/1
peripherally). With ECG monitoring: up to 20-
40mmol/hr via central line (max 200mmol/d)

Prednisolone
Orally active corticosteroid. Less mineral corticoid action than hydrocortisone
Cautions and contraindications - Adrenal suppression, severe systemic
infections
Side-effects- Dyspepsia and ulceration, osteoporosis, myopathy, psychosis,
impaired healing, diabetes mellitus
Dose (pediatric) PO: 1-2mg./kg /d. Croup: 4mg/kg then 1mg/kg tds
(Adult) PO:10-60mg /d, reduced to 2.5-15mg /d.

Promethazine
Phenothiazine, antihistamine, anticholinergic, antiemetic sedative. Pediatric
sedation
Side-effects Extrapyramidal reactions
Dose (pediatric) > 2yr. Sedation/premed PO:1-2mg/kg
(adult) PO/IM:25-50 mg

Propofol
Di-isopropylphenol IV induction agent. Rapid recovery and little nausea.
Agent of choice for day stay surgery, sedation or laryngeal mask insertion-can
be used for electro convulsive therapy (ECT)
Cautions and contraindications
Reduce dose in elderly or haemodynamically unstable. Not recommended for
Caesarean section. Allergy to eggs, peanuts, Soya and soybean oil. Caution
in epilepsy
Side-effects Apnoea. Hypotension pain on injection. Myoclonic spasms, rarely convulsions

Dose
(pediatric) Induction: 2-5mg/kg Infusion: 4-15mg/kg/hr
Not recommended (NR) induction <1 month. NR maintenance <3yr
(adult)
Induction: 2-3mg/kg. Infusion 6-10mg/kg/hr. Target controlled infusion(TCI): initially 4-8 µg/kg/ml then 3-6 µg/ml (reduce in elderly)

Propranolol
NO selective Beta-adrenergic antagonist. Controlled hypotension
Cautions and contraindications Asthma, heart failure, AV block, verapamil treatment
Side-effects Bradycardia, hypotension, AV block, and bronchospasm
Dose (pediatric) 0.1mg/kg over 5min. (adult) 1mg increments up to 5-10 mg

Protamine
Basic protein produced from salmon sperm. Heparin antagonist
Cautions and contraindications Weakly anticoagulant and marked histamine release. Risk of allergy
Side-effects Severe hypotension, pulmonary hypertension, bronchospasm, flushing
Dose: pediatric and adult Slow IV: 1mg per 1mg heparin (100U) to be reversed

Ranitidine
Histamine (H2) receptor antagonist. Reduction in gastric acid secretion
Cautions and contraindications Porphyria
Side-effects Tachycardia
Dose (pediatric IV: 1mg/kg slowly/ tid. PO: 2-4mg/kg/ bid
(adult) IV: 50mg (diluted in 20ml saline, given over 2min) qds.
IM: 50mg qds. PO: 150mg bd or 300mg/d

Salbutamol
Β2 receptor agonist treatment of bronchospasm
Larger doses now suggested in paeds: 15 µg/kg/min over 10min, then 1-5 µg/kg/min
Cautions and contraindications Hypokalaemia possible
Side-effects Tremor, vasodilatation, tachycardia
Dose (pediatric) 4 µg/kg slow IV then 0.1-1 µg/kg/min Nebuliser <5yr 2.5mg >5yr 2.5-5mg
(adult) 250 µg slow IV then 5 µg/min (up to 20 µg/min) Nebuliser: 2.5-5mg as required (prn)

**Suxamethonium**
Phase II block develops with repeated doses (>8mg/kg). Store at 2-8°C
Cautions and contraindications.
Prolonged block in plasma cholinesterase deficiency, hypokalaemia, hypocalcaemia. Malignant hyperthermia, myopathies increased serum K (normally 0.5 mmol/l greater in burns, trauma, and upper motor neuron
Side-effects Increased intraocular pressure. Bradycardia with second dose
Dose (pediatric) 1-2mg/kg (adult) 1-1.5mg/kg Infusion: 0.5-10mg/min

**Thiopental**
Short-acting thiobarbiturate, induction of anaesthesia, anticonvulsant, cerebral protection. Recovery due to redistribution
Cautions and contraindications
Accumulation with repeated doses. Caution in hypovolaemia and elderly.
Porphyria
Side-effects Hypotension Necrosis if intra-arterial
Dose
(pediatric) Induction: neonate 2-4mg/kg, child 5-6mg/kg
(adult) Induction/cerebral protection: 3-5mg/kg Anticonvulsant: 0.5-2mg/kg prn

**Tramadol**
Opioid analgesic thought to have less respiratory depression, constipation, euphoria, or abuse potential than other opioids. Has opioid and non-opioid mechanisms of action
Cautions and contraindications
Only 30% antagonized by naloxone. Caution in epilepsy. Previously not recommended for intra-operative use, MAOI
Side-effects
Nausea, dizziness, dry mouth. Increased side effects in conjunction with other opioids
Dose
(pediatric) 1-2mg/kg 6-hourly
(adult) PO: 50-100mg 4-hourly. Slow IV/IM: 50-100mg 4-hourly
(100mg initially then 50mg increments to maximum 250mg) Maximum 600mg/d

**Vasopressin (pitressin)**
Synthetic ADH used in treatment of diabetes insipidus, resistant vasodilatory shock
Cautions and contraindications  Extreme caution in coronary vascular disease
Side-effects  Pallor, coronary vasoconstriction, water intoxication
Dose  (pediatric) Diabetes insipidus SC/IM: 2-10U 4-hourly
(Adult)  Diabetes insipidus SC/IM: 5-20U 4-hourly. Sepsis 1-4 U/hr Shock infusion

**Warfarin**

Coumarin derivative oral anticoagulant.
DVT prophylaxis: INR 2.0-2.5
DVT, PE treatment, AF, mitral valve disease: INR 2.5-3.0.
Recurrent DVT/PE, prosthetic heart valve: INR 3.0-4.5
Cautions and contraindications
Pregnancy, peptic ulcer Hemorrhage disease. Reduce dose in elderly
Side-effects  Hemorrhage
Dose
(Pediatric)  PO: 0.2mg/kg up to 10mg/d for 2d, then 0.05-0.2mg/kg/d
(Adult)  PO: 10mg /d for 2d then 3-9 mg /d dependent on INR

Annex-Two : INFUSION REGIMES
<table>
<thead>
<tr>
<th>Drug</th>
<th>Indication</th>
<th>Diluent</th>
<th>Dose</th>
<th>Suggested regime (60kg adult)</th>
<th>Infusion range</th>
<th>Initial rate (adult)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>Treatment of hypotension</td>
<td>0.9% saline, 5%dextrose</td>
<td>2-20 µg/min (0.04-0.4 µg/kg/min)</td>
<td>5mg/50ml (10µg/ml)</td>
<td>1.2-12ml/hr</td>
<td>5ml/hr</td>
<td>Via central catheter Suggest 1mg/50 ml for initial intraoperative use (or 1mg/500ml if no central access)</td>
</tr>
<tr>
<td>Aminophylline</td>
<td>Bronchodilation</td>
<td>0.9% saline, 5%dextrose</td>
<td>0.5mg/kg/hr</td>
<td>250mg/50ml (5mg/ml)</td>
<td>1-6ml/hr</td>
<td>6ml/h</td>
<td>After 5mg/kg slow bolus</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Treatment of arrhythmias</td>
<td>5%dextrose only</td>
<td>Loading infusion 5mg/kg over 1-2hr, then 900mg over 24hr</td>
<td>300mg/50ml (6mg/ml)</td>
<td>25-50ml/hr</td>
<td>25ml/hr</td>
<td>Via central line (peripherally in extremis). Max 1.2g in 24 hr</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Rapid control of ventricular rate</td>
<td>0.9% saline, 5%dextrose</td>
<td>250-500 µg over 30-60min</td>
<td>250-500 µg/50ml</td>
<td>50-100ml/hr</td>
<td>50ml/hr</td>
<td>ECG monitoring suggested</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Cardiac failure/inotrope</td>
<td>0.9% saline, 5%dextrose</td>
<td>2.5 -10 µg/kg/min</td>
<td>250mg/50ml (5mg/ml)</td>
<td>2-7ml/hr</td>
<td>2ml/hr</td>
<td></td>
</tr>
<tr>
<td>Dopamine</td>
<td>Inotrope</td>
<td>0.9% saline, 5%dextrose</td>
<td>2-10 µg/kg/min</td>
<td>200mg/50ml (4mg/ml)</td>
<td>2-9ml/hr</td>
<td>2ml/hr</td>
<td>Via central line</td>
</tr>
<tr>
<td>Esmolol</td>
<td>B-blocker</td>
<td>0.9% saline, 5%dextrose</td>
<td>50-200 µg/kg/min</td>
<td>2.5g/50ml (50mg/ml)</td>
<td>3-15ml/hr</td>
<td>3ml/hr</td>
<td>ECG monitoring</td>
</tr>
<tr>
<td>Heparin</td>
<td>Anticoagulation</td>
<td>0.9% saline, 5%dextrose</td>
<td>24000-48000U per 24hr</td>
<td>50,000U/50ml (1000U/ml)</td>
<td>1-2ml/hr</td>
<td>2ml/hr</td>
<td>Check APTT after 12hr</td>
</tr>
<tr>
<td>Insulin(soluble)</td>
<td>Diabetes mellitus</td>
<td>Sliding scale</td>
<td>50U/50ml(1U/ml)</td>
<td>Sliding scale</td>
<td>Sliding scale</td>
<td>Sliding scale</td>
<td></td>
</tr>
<tr>
<td>Ketamine</td>
<td>Analgesia</td>
<td>0.9% saline, 5%dextrose</td>
<td>0.2mg/kg/hr</td>
<td>200mg/50ml (4mg/ml)</td>
<td>1-6ml/hr</td>
<td>3ml/hr</td>
<td>With midazolam 2-5mg/hr</td>
</tr>
<tr>
<td>Ketamine</td>
<td>Trauma mixture</td>
<td>0.9% saline</td>
<td>0.5ml/kg/hr</td>
<td>50ml mixture (4mg/ml ketamine)</td>
<td>15-45ml/hr</td>
<td>30ml/hr</td>
<td>200mg ketamine+ 10 mg midazolam+</td>
</tr>
<tr>
<td>Drug</td>
<td>Indication</td>
<td>Solution</td>
<td>Dosage</td>
<td>Rate</td>
<td>Interval</td>
<td>Notes</td>
<td></td>
</tr>
<tr>
<td>----------------------</td>
<td>---------------------------------</td>
<td>---------------------------</td>
<td>---------------------------------------------</td>
<td>---------------</td>
<td>----------------</td>
<td>-----------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Lidocaine (lignocaine)</td>
<td>Ventricular arrhythmias</td>
<td>0.9% saline</td>
<td>4mg/min for 30 min, 2 mg/min for 2hr, then 1mg/min for 24 hr</td>
<td>500mg/50ml (10mg/ml = 1%)</td>
<td>6-24ml/hr</td>
<td>24ml/hr After 50-100mg slow IV bolus. ECG monitoring</td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>Analgesia</td>
<td>0.9% saline</td>
<td>1-3.5mg/hr</td>
<td>50mg/50ml (1mg/ml)</td>
<td>1-3.5ml/hr</td>
<td>2ml/hr Monitor respiration and sedation hourly. Administer oxygen</td>
<td></td>
</tr>
<tr>
<td>Naloxone</td>
<td>Opioid antagonist</td>
<td>0.9% saline, 5%dextrose</td>
<td>&gt;1 µg/kg/hr</td>
<td>2mg/500ml (4µg/ml)</td>
<td>100ml/hr</td>
<td>Rate adjusted according to response</td>
<td></td>
</tr>
<tr>
<td>Nor adrenaline</td>
<td>Treatment of hypotension</td>
<td>5%dextrose</td>
<td>2-20 µg/min(0.04-0.4 µg/kg/min)</td>
<td>4mg/40ml(100 µg/ml)</td>
<td>1.2-12ml/hr</td>
<td>5ml/hr Via central line</td>
<td></td>
</tr>
<tr>
<td>Oxytocin (Syntocinon)</td>
<td>Prevention of uterine atony</td>
<td>0.9% saline, 5%dextrose</td>
<td>0.02-0.125U/min</td>
<td>30U in 500ml (0.06U/ml)</td>
<td>30-125ml/hr</td>
<td>125ml/hr Individual unit protocols vary</td>
<td></td>
</tr>
<tr>
<td>Phenytoin</td>
<td>Anticonvulsant</td>
<td>0.9% saline</td>
<td>15mg/kg</td>
<td>900mg/90ml (administer through 0.22-0.5 µm filter)</td>
<td>Up to 50mg/min</td>
<td>180ml/hr ECG and BP monitoring. Complete within 1 hr of preparation</td>
<td></td>
</tr>
<tr>
<td>Propofol</td>
<td>Sedation</td>
<td>Undiluted (10mg/ml)</td>
<td>10-20ml/hr</td>
<td>TCI: 1-2.5µg/ml</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Salbutamol</td>
<td>Bronchospasm</td>
<td>5%dextrose</td>
<td>5-20 µg/min</td>
<td>1mg/50ml (20 µg/ml)</td>
<td>15-60ml/hr</td>
<td>30ml/hr After 250 µg slow IV bolus</td>
<td></td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>Acidosis</td>
<td>[weight (kg)x base deficit x 0.3] mmol</td>
<td>Undiluted (8.4%solution)</td>
<td>8.4% = 1000mmol/I. Via central line if possible</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Annex III Airway equipment’s**

*Laryngeal mask airway size based on patient weight*
LMA size | Patient size and weight | Maximum air for cuff inflation
---|---|---
1 | Neonates and infants (up to 5 kg) | 4 ml
2 | Infants and children (10-20 kg) | 10 ml
3 | Children (30-50 kg) | 20 ml
4 | Small Adults (50-70 kg) | 30 ml
5 | Adults (70-100 kg) | 40 ml

**Appropriate laryngoscope size**

<table>
<thead>
<tr>
<th>Age</th>
<th>Straight Blade Size</th>
<th>Curved Blade Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Infant</td>
<td>0-1</td>
<td></td>
</tr>
<tr>
<td>Toddler 1</td>
<td>1-2</td>
<td></td>
</tr>
<tr>
<td>Child</td>
<td>2-3</td>
<td>2</td>
</tr>
<tr>
<td>Adult</td>
<td>2-3</td>
<td>3-4</td>
</tr>
</tbody>
</table>

Annex IV: Blood transfusion monitoring form

1. Name____________________ card no_________ age____ sex_____
2. ward ___________, bed number__________
3. Dx____________________
4. Indication for transfusion

5. Hct/Hb_______ Blood group_______ Rh_____

6. Condition of pt. Before transfusion:
   - blood pressure_______
   - pulse_________
   - RR_________
   - Tem_________
   - Level of consciousness_________

7. Amount of units of blood ordered for transfusion_______

8. Parameters cheked before transfusion (on the donor blood)

<table>
<thead>
<tr>
<th>Checked for</th>
<th>Unit 1</th>
<th>Unit 2</th>
<th>Unit 3</th>
<th>Unit 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full name/card number</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rh</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>X. match</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expire date</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consistency/color</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

9. Transfusion started/end time and date ______________

10. Complications/reaction observed: pain on the infusion site_______, skin rash______, fever/rigors ______, decreased blood pressure______, difficult of breathing______, chest and back pain______, others________________

11. Measures taken________________________________________________

12. Transfusion ordered by_____________________ signature____________

13. Transfusion started/followed by______________ signature_______

14. Transfusion completed by____________________, signature ________

Annex V: ER TRIAGE FORM /sample

Serial No_______

1. Name__________________________, age_______, sex_____

2. Time/date of arrival_______________

3. Duration of illness/injury_______________
4. Transportation: □ walking, □ taxi, □ carried, □ ambulance
5. Origen of referral:-----------------------------------------------
6. Time/ date of triage _____________________
7. Pre-hospital care given: □ yes □ no____
8. Main problem:
   □ medical □ surgical □ Gyn/obs
   □ Chest pain □ trauma □ non trauma □ V. bleeding
   □ Unconsciousness □ RTA □ A. abdomen □ convulsion
   □ Respiratory problem □ stab □ soft tissue infection □ L. abd. pain
   □ HPN □ fall accident □ spontaneous pneumotorax □ labor
   □ Hemoptisis □ suicide □
   □ GI □ gun shot
   □ DM □ burn
   □ Head ache □ animal bite
   □ other specify

   _______________________________

9. Triage assessment:
   Airway: □ patent, □ obstructed
   Breathing: □ adequate, □ gasping, □ apnea □ >35/min, □ <10/min
   Circulation: □ peripheral pulse present □ carotid pulse present, □ CPA
   D- □ conscious, □ semi-conscious, □ unconscious, □ GCS____
10. Vital sign: BP__________, P_______, RR_______, T____
11. Triage decision:
    □ red □ orange □ yellow □ green □ black
12. Desposition: to: ______________ transfer time____________
    □ Exam room, □ resuscitation room, □ procedure room, □ OPD,
    □ other health institution
13. Treatment/intervention
given:____________________________________________________
    ______________________________________________________
    ______________________________________________________

Responsible nurse:__________________________, sig____________

National Integrated Emergency Medicine Training
Case study booklet

Outline
- Airway, breathing, BLS and Triage management: Cases 1- 4
- Medical Emergencies
1. A 28 years old young man arrived to the ED after 1 hr of fall down incident. On arrival patient came on stretcher, non communicative, snoring, responding only to pain, RR is 30/min, and labored BP 100/60mmhg, P 120/min, oxygen saturation 80%.
   a. How do you approach to this patient
   b. List main clinical problems on this patient
   c. What maneuvers and equipment’s do you use to manage this patient and why?
   d. List possible causes of the tachycardia?
   e. After 1 hr stay in the ED patients BP, dropped and became an recordable, describe possible causes and your approach

2. A 60 years old female patient arrived to the ED, on arrival patient is conscious and talks with difficulty; and irritable, has fast and shallow breath with RR is 35/min, BP 160/94mmhg, P 130/min, no history of trauma she is febrile to 38oc, saturation you don’t have pulseoxymetre
   a. List main clinical findings
   b. List possible causes of these findings/abnormality
   c. Describe your steps of approach/management
   d. After 1 hr patient breathing became gasping type. Why this patient deteriorated? How are you going to proceed your management of this patient?

3. A 40 years old man arrived to the ED walking, while waiting to be seen by a physician collapsed and brought by the triage nurse and runner. There is no relative around him. When you assess him there is no breathing
   a. What is your next step? And why?
   b. What will be the possible reason for the sudden collapse?
   c. Describe the chain of survival and their importance
   d. What equipment’s and drugs has to be ready for resuscitation of such patients
   e. What is effective CPR
   f. After successful resuscitation patient started to breath and pulse became palpable, then after 30min you were called and you found the patient again on arrest. Explain why this patient went to arrest for the second time?
4. A 28 years old young man arrived to the ED after 1 hr of the incident. On arrival patient came on stretcher, non communicative, snoring, has edematous face, responding only to pain, RR is 20/min, BP 100/60 mmhkg. P120/min, oxygen saturation 80%.
   a. What is your triage score and code
   b. Where will be the patients disposition
   c. What are the next steps

**Respiratory Emergency-5**

5. A 35 years old known asthmatic patients presents to the emergency with severe shortness of breath at rest. She’s agitated and can only talk in words. Her PR is 130 and RR was 36 on presentation. On auscultation she has loud wheeze all over her chest.
   a. How would you classify the severity of the attack
   b. What is the approach to treatment

**Case scenario on CVS -6-10**

6. A 55 yrs old female, with no prior illness, comes to the ED with vague epigastric discomfort of 2 days duration. She had no such a complaint previously. She took maloxsyrup but no improvement. She has also whitish cough and dyspnea

P/E revealed posterior basal lung rales ; ECG is normal

A- What are the possible DDX?
B- What is the next step in evaluating this patient?
   a. Discharge with appointment after few days/weeks?
   b. Giving omeprazole, considering H pylori test & endoscopy?
   c. Sending for CXR & giving antibiotics?
   d. Repeat ECG after 4-6 hrs& sending cardiac enzymes?

7. A 60 yrs old male patient, known diabetic on daonil 10mg po/day, presented to the ED after he sustained syncopal attack of 02 hrs duration. He reported retrosternal heaviness for the past 4 days with worsening since 2 days.

P/E He was anxious & sweating. BP= 190/110 mmHg -PR= 150 bpm(irregular) - SaO2- 85%.Otherwise normal physical exam.

A- What are the likely causes of the pain &syncopal attack?
B- What are your initial management options?
C- Does he have risk factors for ACS?
D- Is ACS likely? If so, how do you confirm?
E- How do you investigate him?
F- How do you monitor Rx response?
G- How do you manage the co morbidities?
H- What complications do you anticipate?
8. A 24 years old female patient from Harar presented with progressive shortness of breath initially manifesting while walking long distance later worsened to manifest at rest. She has associated cough productive of whitish sputum sometimes blood tinged, and orthopnea of 3 pillows equivalent. She has noticed bilateral leg swelling and claims to have palpitations at rest. She was brought to you carried by her brother.
   a. What additional information would you like to have from history
      - Ans. Characterization of the dyspnea, past medical history (if she is a known cardiac patient)
   b. What should be your approach be in managing this patient?

Quick examination revealed she is in cardiorespiratory distress, BP= 100/60 mmHg, PR = 135 BPM irregularly irregular, RR = 35 BPM. Her neck veins are distended. She has bilateral lower 1/3rd lung field crepitations, and pedal and pretibial edema.
   c. What do you think this patient has? What could have precipitated the heart failure?
   d. List the management principles of this patient

9. A 45 years old known hypertensive patient for the last 6 years who has discontinued his drug (Hydrochlorothiazide 25mg/day) since 3 months back because he felt very well and his blood pressure measurements were normal presented to your emergency department with global headache of 1 day duration. He didn’t experience any weakness in his limbs, no loss of consciousness. On examination his BP = 230/150, PR=100, Quick emergency examination revealed no weakness, no neck stiffness.
   a. What are the possible causes for his presentation?
   b. What investigations would you like to order?
   c. How would you like to manage this patient?
   d. If this patient has hematuria with new elevation of Cr how will your management be?
   e. What education will you give this patient before discharge?

10. A 30 years old known cardiac patient on diagnosed to have Chronic rheumatic valvular disease on chronic follow up at your hospital taking digoxin 0.125mg/d, Furosemide 40mg PO/d, presented to your emergency department with progressive body swelling initially starting from the feet, later involving the abdomen. He also has pain in his right upper abdomen. He claims his body swelling worsened recently despite increasing his Furosemide dose to 80mg PO/d in his clinic visit a week back. He has occasional shortness of breath while doing strenuous activities but no orthopnea or PND. He has developed a low grade fever and night sweats over the past 2 weeks. On a quick examination he has distended neck veins,
Chest is clear and resonant, has right upper quadrant tenderness and bilateral leg swelling.

a. Will you admit this patient to the emergency unit? Why?
b. List important investigations you will do in the emergency setup?
c. How would you manage this patient? Can you manage him with oral Furosemide following admission? What will be your dose? How frequent

d. What precipitating causes could be responsible for his decompensation?

Endocrine emergencies – 11

11. An 18 years old female with a history of Type 1 DM is brought to the emergency room with nausea, vomiting, abdominal pain, high grade fever and cough. She is lethargic has dry mucus membranes and is confused. Her BP was 70/40 mmHg, PR-126, RR 36 bpm, T° 38.1° c, O₂ saturation -84% and has crackles on the right lower 1/3 lung field

a. How will you approach this patient
b. On investigation her RBS was 465mg/dl urine ketone was +3 and serum K⁺ was 3.0 meq/l, her WBC count was 15,600/mm³. How will you go about managing this patient?

After six hours stay in the hospital her RBS was recorded as 43mg/dl, what will you do next?

Toxicology – 12- 13

12. A 15 years old girl from Bale brought to ER by family on bus. She was found behind Barn 6 hours ago has decreased LOC, drooling, tears streaming, covered in vomit and urine, feces. Her Vital signs are as follows:
   Heart Rate, 101
   Respiratory Rate, 16
   Blood Pressure, 90/60 mmHg
   Temperature, 36.5 c

a. How should you proceed?
b. What management options are there for this patient?

13. A 28 yrs old woman is brought to the ED by her parents. She has been unresponsive for 8 hrs and has the following vital signs,
   Heart Rate, 118
   Respiratory Rate, 10
   Blood pressure 90/60 mmHg
   Temp, 34.1

a. What history would you like to know?
b. What universal antidote would you consider for this patient?
c. What Lab tests you want to do for this patient?
d. What decontamination strategy would you choose for this patient?
e. What are the management priorities after you identify the toxin she took?

**Pain management – 14, 15**

14. A 58 years old male patient brought to ER with history of right lower abdominal pain associated with vomiting of ingested matter of 6hrs duration has history of, mild HPN, V/S BP 140/90mmHg, PR 112 bpm RR 24bpm and shallow, T° 37ºC

- How do you assess his pain?
- Explain the cause and mechanism of this tachycardia, tachypnea,
- Describe your approach on the pain management on this patient?
- Explain why pain has to be treated?

15. A 35 yrs old male patient brought to ER by a police with poly-trauma to the chest, abdomen and right lower limb after road traffic accident of 12 hr duration. On P/E conscious but restless and shouting for pain. V/S BP 120/80 PR 120bpm, RR 22bpm T° 37.5ºC

a. Do you want to assess and score the pain?
b. How do you manage the pain?
c. What do you want to monitor while managing the pain?

**Case Scenarios on Trauma 16-20**

16. A victim of a motorcycle crash is comatose and has obvious head injuries. Vital signs are blood pressure 170/100, pulse 50 per minute; respirations, 24 per minute. What is the most likely explanation for these vital signs?

17. A 5-year-old girl is struck by a car. She is unconscious and has obvious head injuries. Her vital signs obtained in the ambulance are blood pressure 50/30, pulse, 156 per minute; and respirations 40 per minute. You estimate her weight at 15kg. Which of the following is the most appropriate initial fluid management?

18. A 45-year-old woman is found unconscious at the scene of a motor vehicle collision. Her vital signs are blood pressure, 80/40; pulse 130 per minute, and respiration, 30 per minute. Which of the following is the MOST likely cause for her vital signs?
19. During transport, a trauma patient develops severe difficulty breathing, distended neck veins, diminished breath sounds on the right, cyanosis, and deviation of the trachea to the left. Vital signs are blood pressure: 60/40, respirations, 36 per minute; and pulse, 130 per minute. Which of the following is the most appropriate next step?

20. A 56-year-old suffers a gunshot wound to the abdomen. Physical examination is unremarkable except for a non-bleeding bullet hole and diffuse abdominal tenderness. Vital signs obtained en route to the hospital are BP 70/40, pulse 136 per minute, respiration 30 per minute. What should be done next?

**Obstetric case scenario 21- 25**

21. A 36 years old PII, type II diabetic women presented to the ER at 9 months of pregnancy after 2 days of labor at home. She is in second stage of labor and estimated fetal weight is 5kg. Immediately after arrival in the ER, fetal head was delivered but shoulder was not coming out. What do you do?

22. A 20 years old primigravida at GA of 30 weeks is in the ER having tonic clonic convulsion. BP is 180/120mmHg, GCS of 8/15. Institute emergency care in the ER.

23. 25 years a rural laboring woman was brought to ER by a taxi. On arrival the fetal legs are delivered out of the vagina, while trunk and head was not delivered. Deliver the fetus in the ER.

24. A 40 years old P-V, women presented to the ER at 9 Months of pregnancy on arrival she is in active labor and complaining for abdominal pain, dizziness, bloody vaginal discharge. On P/E: BP is 80/50mmHg; pulse 110/min; uterus tense; patient looks pale.
   a. Discuss the possible causes of this symptom
   b. Describe your approach
   c. What is your differential DX
   d. Your hospital is rural hospital and if you are forced to refer this patient list your plan before referring of this patient

25. 35 years old multipara lady delivered a 4.5kg baby with assisted vaginal delivery. After 30min of the delivery of placenta progressively she started to feel weakness, sweating, and BP borderline. IV fluid started but no response rather progressively declining.
   a. Discuss the main causes
   b. Discuss the 4 Ts and their management
   c. Discus prevention of PPH
   d. Outline your approach during PPH
Pediatrics case scenario 26-31

26. How to triage

Case scenario I
A two years old male child who was brought to emergency unit with diarrhea of one day duration. On physical examination has sunken eye ball, skin pinch goes back slowly, the capillary refill less than 1.5 second. How do you triage this patient?

27. How to assess and manage Pediatrics Air way

Case scenario II
A mother left her three month old infant with her five years old sister accidentally the sister put something in her sister mouth suddenly he baby developed repetitive cough. When the mother entered to the room the baby is breath less. What is the immediate action?


Case scenario III
A 2 years old child presented with runny nose, fever and shortness of breath. On physical examination the child has RR 56/min, Blood pressure 80/50mmhg, pulse oxymeter 85% what will be your assessment and immediate management?

29. Identifying Pediatrics circulatory problem

Case scenario IV
A 12 years old boy has sustained car accident while crossing the road. He came with ambulance the child has difficulty of breathing on arrival, has bleeding from the lower leg, has loss of consciousness V/S RR 16/min, blood pressure 70/40mmhg. Capillary refill 4 sec. How you assess the child and what will be the management?

30. Over view of basic life support in infant and children

Case scenario V
A three years old child was having pneumonia the mother did not seek for medical advice, when the child developed gasping type of breath. This time the mother him. On examination RR 5/min absent pulse. What is the immediate action?

31. Neonatal resuscitation

You are called in labor ward the mother gave baby at gestational age of 36 weeks, membrane ruptured intera partum the labor was prolonged. The mode of delivery
SVD, the out come alive male neonate weighing 2000gm. Immediately after delivery the baby is breath less. What is your immediate management.