Structured Notes According to

EMERGENCY MEDICINE

Revision friendly **Fully Colored Book/Structured Notes**

For Best results, watch the video lectures along with reading notes

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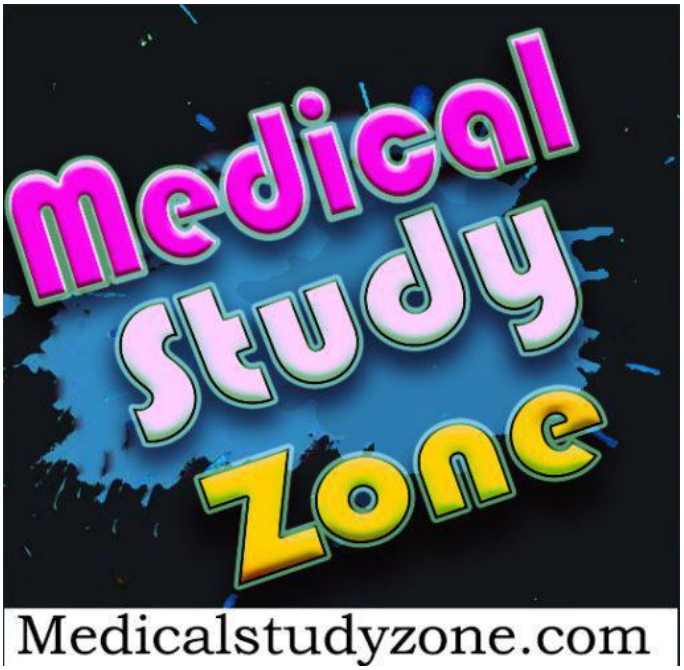


CONTENTS

EMERGENCY MEDICINE

1 BASIS OF EMERGENCY MEDICINE

APPROACH TO THE PATIENT

00:00:29

- Emergency differ from outpatient & impatient
- Emergency patients needs urgent medical attention for what is perceived to be either can emergent condition or a medical need that they cannot obtain help with elsewhere
- Emergency physicians function under the constraints of limited time, limited resources & seemingly endless line of patients waiting to be seen

Emergency patients

1. Triage, if time & patient condition allow
2. The patient's chief complaint, vital signs, past medical & surgical history, medication list, allergies & brief history of present illness are recorded
3. Decision of Triage nurse to move the patient where it is necessary

TRIAGE CODING

00:04:10

- MC: Emergency severity index
- Developed by Richard Uhery & David Eital in 1998
- Five Categories
  - Red (1) - Critical
  - Orange (2) - Emergent
  - Yellow (3) - Urgent
  - Green (4) - Minor
  - Blue (5) - Very minor

STEPS TO TREAT UNDIFFERENTIATED PERSON

00:06:30

Step 1: Initial actions & primary survey

- Airway
  - Is the patient able to speak?
  - Is their voice normal or high pitched?
  - Are they displaying signs of imminent airway compromise?
- Breathing
  - Is their breathing pattern rapid?
  - Are they grunting or audibly wheezing or do you hear stridor?
  - Are they displaying any other abnormal breathing pattern?
- Circulations
  - Feel for the patient pulse (PR, character of pulse)
  - Check blood pressure & observe skin color
  - Check for vascular access

Important Information

- For unresponsive adults in whom you suspect cardiovascular emergency, approach the patient using "CAB" (Circular, Airway, Breathing)
- ACLS guidelines

- A - Allergies
- M - Medications
- P - Past History
- L - Last Meal
- E - Events leading to the present condition

How to remember

- AMPLE

IMMEDIATE ACTIONS AFTER PRIMARY SURVEY

00:18:29

- Ventilation
- IV fluids for significant Hypotension
- Hemostasis for Hemorrhage
- Chest compressions if pulseless
- Glucose or Naltrexone for altered mental status
- Pain or fever control

Step 2 : Initial differential Diagnosis – Focus on the life presents

00:20:20

- Initial assessment of the patient
- List of possible diagnosis that are life or limb threatening
- This process initiated with the chief complaint & primary survey & refined after gathering more information

Important Information

- Depending on the primary survey, history, age, look of the patient, DDX will differ

Step 3 : Secondary Survey

- Ask the patient to give History, if possible
- Focused history "Cold Read"
  - **C** - Character
  - **O** - Onset
  - **L** - Location
  - **D** - Duration
  - **R** - Radiation
  - **E** - Exacerbating / Alleviating factors
  - **A** - Associated symptoms
  - **D**- Different today – what has changed in the patient's symptoms or presentation that prompted current visit?

How to remember

- COLD READ

- Entire past history, social & family history, medication & allergy history
- Focused History

Step 4 : Refine the differential diagnosis

- We can refine the DDX, by removing the diagnosis from the initial list

Step 5 : Diagnostic Evaluation 00:31:27

- To rule in or rule out the life threatening diseases, evaluate with investigations
- The investigations should help us to make the final diagnosis & help in the management & disposition of the patient

Step 6 : Patient Re-Evaluation 00:34:29

- Patient illness often manifesting, progressing & changing while in the emergency department
- Re-evaluate the differential diagnosis

Step 7 : Treatment & Disposition 00:36:42

- All patients in the emergency department have one of three dispositions:
  - Admit
  - Transfer
  - Discharge

Important Information

- It is important to know what are the policies & resources are available in hospital
- So that, we can transfer the patient to nearby hospital ahead of time in cases where particular resources or policy is not available in the hospital

2 BASIC LIFE SUPPORT

DEFINITION

00:00:17

- Out of hospital care to a person who has suddenly become unresponsive & pulseless.
- Any cardiac event occurring out of Hospital in western countries
  - Survival Rate ≡ 10%
- Any cardiac event occurring out of Hospital & Patient was provided By stander CPR
  - Survival Rate ≡ 20%

CARDIAC ARREST

00:03:32

- Unresponsive person (As there is no cerebral Blood flow)
- Pulseless (Carotid pulse is checked as it is closest to heart & last one to go in case of cardiac arrest)
- Apnea/ Gasping Respiration/ Agonal Rhythm

Pulseless conditions

- Ventricular Fibrillation
- Ventricular Tachycardia
- Asystole
- Pulseless Electrical Activity [PEA]

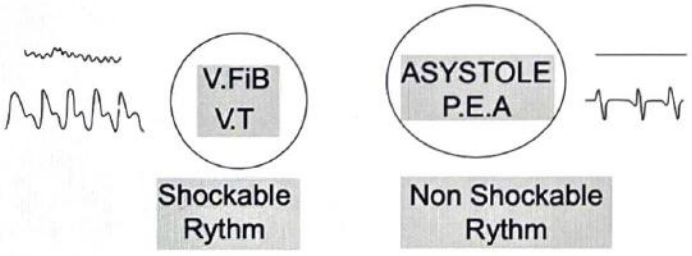
Shockable Rhythms

- Ventricular Fibrillation and Ventricular Tachycardia
- Treatment is Cardioversion
- Survival chances are higher

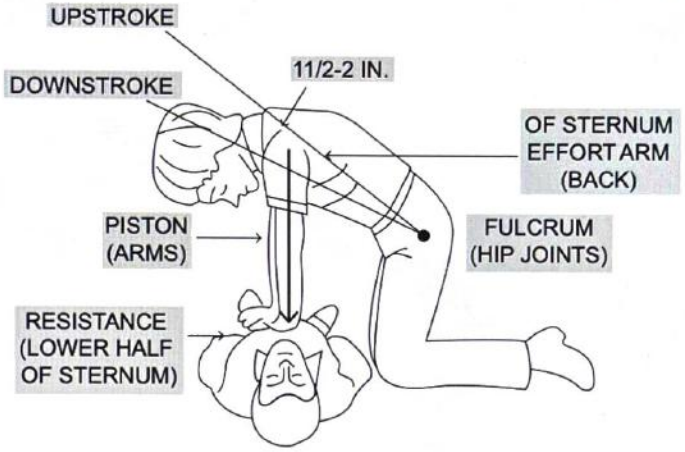
Non – shockable Rhythms

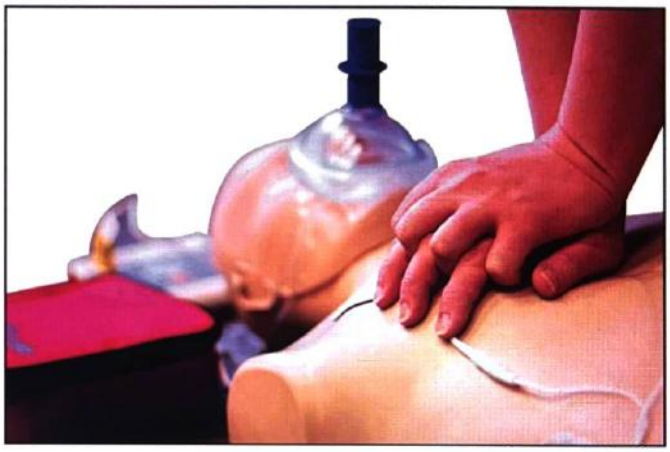
- Asystole and Pulseless Electrical Activity
- Survival chances are low

Both Shockable & non-shockable Rhythms are inter convertible. i.e. Ventricular Fibrillation may convert into Asystole & vice versa.



CARDIO PULMONARY RESUSCITATION (CPR)





- An elderly male collapsed suddenly in an airport at the boarding gate. What will be the correct management of the patient?

STEPS OF GIVING CARDIO PULMONARY RESUSCITATION (CPR)

00:06:48

- 1st step: Check scene safety (Take the patient to a flat surface)
- 2nd step: Check unresponsiveness of patient. (Tapping the shoulder)
- 3rd Step: Call for Help
- 4th step: Activate emergency response
- 5th step
  - Check pulse → carotid pulse
  - Check breathing effort → Look for chest rise and fall
  - Feel the breath sounds of the person

Case scenarios of a collapsed person

1. **Pulse**: Present
- Respiratory effort: Absent
  - Respiratory Arrest
- **Treatment**: Rescue breaths at the rate of 10-12/min (1 breath/second)

2. **Pulse**: Absent
- Respiratory effort: Absent
  - Cardio-Pulmonary Arrest
- **Treatment**: Cardiopulmonary Resuscitation at a rate of 30 : 2 (Chest compressions) ← 30 : 2 → (Rescue Breaths). One cycle of CPR is of 2 mins

3. **Pulse**: Present
- Respiratory effort: Present
- Treatment: Monitor the patient

Case 1: An elderly man, who is 60 years old collapsed suddenly at the airport boarding gate. How will you manage this case?

MANAGEMENT

00:19:02

Follow steps 1 to 5 of cardiopulmonary Resuscitation

↓

Give a CPR cycle for 2 mins

↓

Look for an Automated Electrical Defibrillator Device (AED)

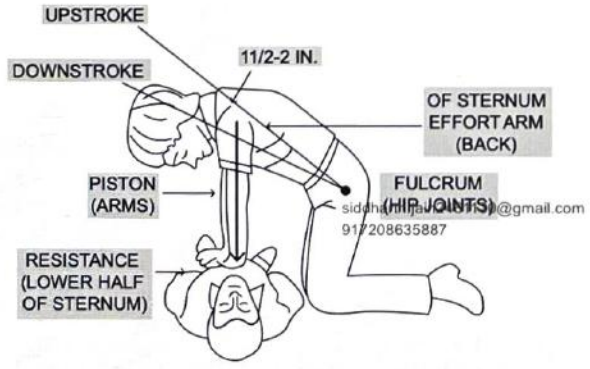
↓

If AED available

↓

Put chest leads & look for Rhythm

Shockable Rhythm	**Non-shockable**
↓	↓
Press button of AED	Continue CPR till Medical rhealth team arives
↓	
AED will give a DC shock of 200 J	
↓	
After shock is delivered, check for return of Pulse & Respiration	

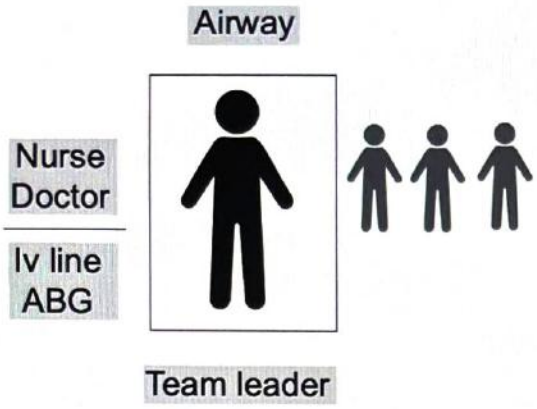


Ratio of CPR administration

• Adult	- 30 : 2	1 or 2 Rescuer
• Pediatrics (Child)	- 30 : 2	1 Rescuer
	- 15 : 2	2 Rescuer
• Neonate	- 3 : 1	2 Rescuer mandatory

Sternal Depression
- Adults → 5 cm
- Child → 2 cm
- Neonate → 1 cm
- Allow for proper recoil of chest so that
- Heart can get filled with blood
- Coronary blood flow can be ensured
- Chest compressions are given at a rate of 100 – 120 / min.
- Place hands on lower 1/3rd of sternum while giving chest compressions
- Very forceful & aggressive chest compressions can lead to pressure over Xiphisternum
- As Xiphisternum is pointed → It causes damage to Liver.
- Most common solid organ traumatized in CPR → Liver (Not lungs)
  - Lung is not a solid organ
- Hip joint acts as fulcrum while giving CPR
- Most common ribs damaged while giving CPR → 4th to 6th Ribs
  - Do not discontinue CPR even if the Ribs are fractured.

Airway management by Bag & Mask Ventilation (BMV)
- AMBU Bag
- Ensure
  - Head tilt
  - Chin lift
- Head tilt & chin lift ensure patency of airways so that air can enter r into lungs during BMV
- Position of patient → Sniffing position
- Correct application of mask: The mask should be held b/w thumb & an index finger of a left hand in a 'C' shaped manner so that air does not leak.
- Remaining 3 fingers are kept in an 'E' shaped manner, so as to stabilize the lower part of mandible
- With the right hand we press AMBU bag & give rescue breaths.
- The person who provides airway support stands at head end of patient
- Team leader stands at foot end of the patient
- On the right-hand side is a team of 2-3 doctors and paramedical staff to provide simultaneous chest compressions after every 2 minutes.
- On the left-hand side is the nurse/doctor to secure an I.V. line and collect blood samples for ABG
- Coordination of team members is an important component in CPR

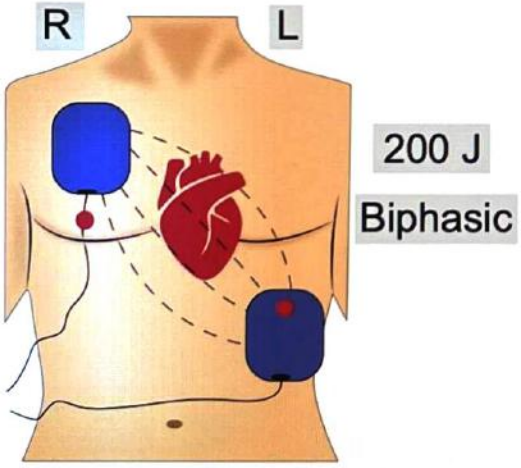


ADULT BLS ALGORITHM

00:22:29

Refer Image 2.1

- AED is used immediately upon arrival. We don't wait for CPR cycle to get completed. We attach AED electrode immediately.
- **AED:** Electrode placement
  - One electrode is placed below the right clavicle & superior to right nipple
  - The other electrode/paddle must be placed just covering the left nipple & going into the axilla.
  - AED gives a DC shock of 200J biphasic
  - Earlier times 360J of monophasic DC shock was used



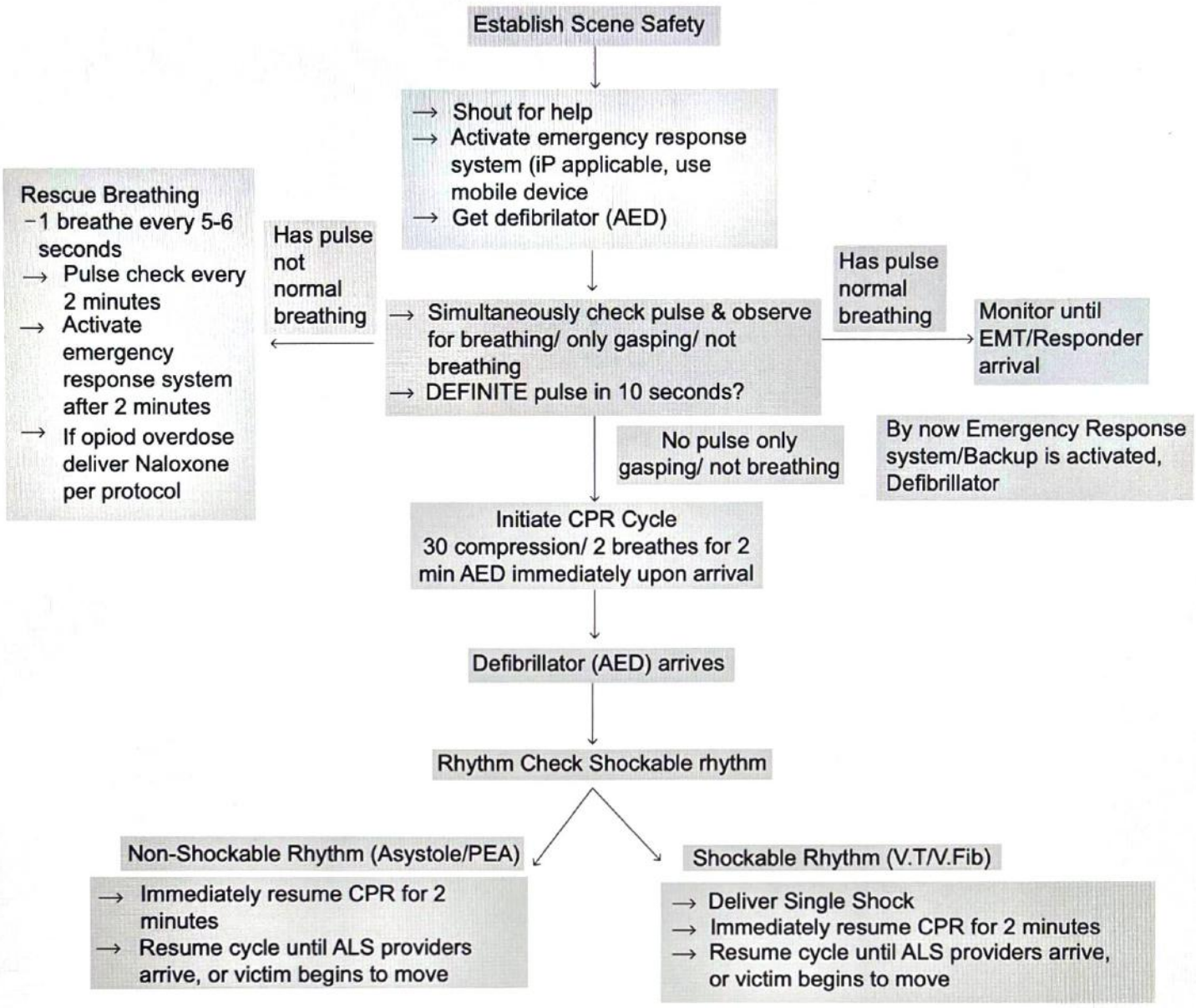
ETIOLOGY OF CARDIAC ARREST

- Coronary Artery Disease (CAD)→ Main/ Leading cause
- Causes
  - Cardiomyopathy e.g.
    - → Hypertrophic obstructive cardiomyopathy (HOCM)
    - → TakoTsubo cardiomyopathy (TTCM)
  - Valvular Lesions
    - → Aortic Stenosis, Mitral Stenosis, Mitral Regurgitation.
    - → Left Atrial dilation→ A. Fib→ Stroke
  - Bundle Branch Block
    - → BRUGADA Syndrome (Channelopathy)
- Na^+ channel defect
  - Predisposes to development of Torsades D pointes or Ventricular Tachycardia
- Gene involved
  - SCN5A
  - TDP
- Electrolyte Abnormalities
- Hyperkalemia
  - Toxins
  - TCA overdose
  - Organophosphate poisoning (OPC poisoning)
  - Cocaine/ Alcohol overdosage

Important Information

- Order of Resuscitation → CABD
  - Circulation > Airway > Breathing > Defibrillation (if needed)

Image 2.1


Establish Scene Safety
→ Shout for help
→ Activate emergency response system (iP applicable, use mobile device
→ Get defibrilator (AED)
→ Simultaneously check pulse & observe for breathing/ only gasping/ not breathing
→ DEFINITE pulse in 10 seconds?
Has pulse not normal breathing
Rescue Breathing
-1 breathe every 5-6 seconds
→ Pulse check every 2 minutes
→ Activate emergency response system after 2 minutes
→ If opiod overdose deliver Naloxone per protocol
Has pulse normal breathing
Monitor until EMT/Responder arrival
No pulse only gasping/ not breathing
By now Emergency Response system/Backup is activated, Defibrillator
Initiate CPR Cycle
30 compression/ 2 breathes for 2 min AED immediately upon arrival
Defibrillator (AED) arrives
Rhythm Check Shockable rhythm
Non-Shockable Rhythm (Asystole/PEA)
→ Immediately resume CPR for 2 minutes
→ Resume cycle until ALS providers arrive, or victim begins to move
Shockable Rhythm (V.T/V.Fib)
→ Deliver Single Shock
→ Immediately resume CPR for 2 minutes
→ Resume cycle until ALS providers arrive, or victim begins to move

3 ADVANCED CARDIAC LIFE SUPPORT

00:00:20

- How ACLS is different from BLS
  - In ACLS the differential diagnosis(d/d) of the causes of pulselessness are listed (we do Airway Breathing Circulation D/d i.e. ABCD) and manage the patient accordingly
  - BLS the main aim is to maintain brain perfusion by giving chest compressions

D/D of Pulselessness

- Ventricular Tachycardia
- Asystole
- Pulseless Electrical Activity
- Hypovolemia
- Tension Pneumothorax

- Defibrillator: Measures the rhythm, but the physician decides whether shock should be given
- AED (Automated): Decision is made by the machine
- Advanced Airway: 1 breath is given every 6 seconds (10 breaths /min)

defibrillation - V.FiB - pVT

PSVT
A.FiB
A.FLUTTER

DIFFERENCE B/W DEFIBRILLATION & CARDIOVERSION

00:04:02

Cardioversion

↓

Synchronized DC shock at the peak of R Wave i.e. Impulse is fired at the peak of R Wave

↓

As R wave is the time at which heart is contracting abnormally

↓

So, it makes the heart relax, so that, at the next beat SA node takes over

↓

Return of spontaneous circulation

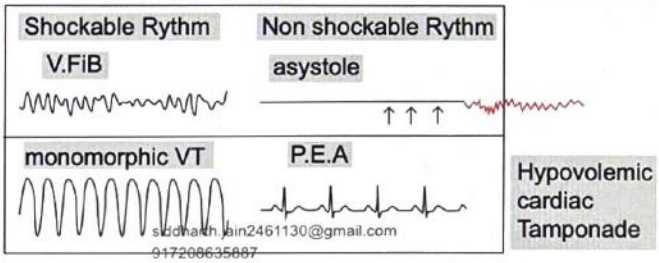
- Done for → PSVT, A. fibrillation, A. flutter

Defibrillation

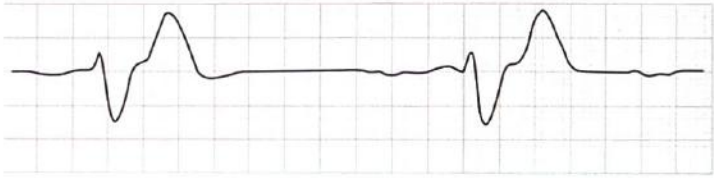
- Impulse will be fixed irrespective of peak of R wave
- Done for
  - V. Fibrillation
  - Pulseless Ventricular Tachycardia (PVT) Eg monomorphic V_T

RHYTHMS OF HEART

00:07:29



Agonal Rhythm



Important Information

Both Shockable & Non-shockable Rhythms are inter-convertible. i.e. Ventricular Fibrillation may be converted into Asystole & vice versa.

Agonal Rhythm

- Few last breaths of a person (efforts of brain stem)
- Irregular Broad QRS due to firing of bundle of HIS after this Asystole

TREATMENT OF REVERSIBLE CAUSES

00:12:36

- 5 H & 5 T

Refer Table 3.1

Refer Table 3.2

- Hypokalemia
  - Results in Torsades De Pointes
  - Diaphragmatic paralysis
- Hyperkalemia
  - Bradycardia Asystole
- Tamponade
  - Identified by electrical alternans of ECG
- Tension pneumothorax
  - Identified by absent breath sounds on ipsilateral side
- Coronary Artery Disease
  - ST elevation
- Massive pulmonary embolism
  - Have right ventricular failure [$S_1Q_3T_3$] & Kussmaul's sign

ALGORITHM OF ACLS

00:16:27

Mx of patient who had suddenly collapsed → CPR, O_2 and Defibrillator attached

- If PVT (premature ventricular tachycardia)/V.fib (ventricular fibrillation) ↓
  Non synchronized DC Shock
  ↓
  CPR x 2 min, I.V / I.O line
- Fails ↓ Repeat Non synchronized DC Shock
- CPR x 2 min, IV / IO epinephrine 1 mg in 1:10000 dilution
- Advanced Airway, Capnography
- Breathing: 1 breath / 6 Sec after
- Endotracheal Intubation
  - Fails ↓ 3rd Shock repeated
  - CPR x 2 min
  - IV amiodarone
- Treat reversible cause [hypoxia, toxins acidosis etc]

- If PEA (pulseless electrical activity) / Asystole ↓
  CPR x 2 min
  IV/IO [tibia /sternum]
  epinephrine
  Advanced Airway & Capnography
  ↓ fails
  CPR x 2 min
- Repeat epinephrine
- Treat reversible causes mainly Hypovolemia
- Epinephrine can be repeated every 3-5 minutes

Amiodarone 3 doses can be given

- 1st dose = 300 mg
- 2nd dose = 150 mg after 3-5 mins
- Amiodarone is useful for V as well as shock refractory V

ROSC (RETURN OF SPONTANEOUS CIRCULATION)

00:24:48

- TTM (Target Temperature Management) or Therapeutic Hypothermia (limit the progress but can't reverse damage)
- Cardioprotective
- Neuroprotective
- Prognosis of these patients isn't good

Table 3.1

Hypovolemia	Hypoxia	Hydrogen Ion (acidosis)	Hypo/Hyper-kalemia	Hypothermia
• Loss of fluid volume in the circulatory system. • Look for obvious blood loss. • Most important intervention Is to obtain IV access and administer IV fluids. • Use a fluid challenge to determine if the arrest is related to hypovolemia	• Deprivation of an adequate oxygen supply can be a significant contributing cause of cardiac arrest. • Ensure that the airway is open • Ensure adequate ventilation, and bilateral breath sounds. • Ensure oxygen supply is connected properly.	• Obtain an arterial blood gas to determine respiratory acidosis. • Provide adequate ventilations. • Use sodium bicarbonate to prevent metabolic acidosis if necessary.	• Both's thigh and low K^+can cause cardiac arrest. • Signs of high K^+ include taller, peaked T-waves, and widening of the QRS complex. • Signs of low K^+ include flattened T-waves, prominent U-waves and possibly widened QRS complex. • Never give undiluted intravenous potassium.	• If a patient has been exposed to the cold, warming measures should be taken. • Core temp. should be raised above 86 F and 30 C as soon as possible. • The patient may not respond to drug or electrical therapy while hypothermic,

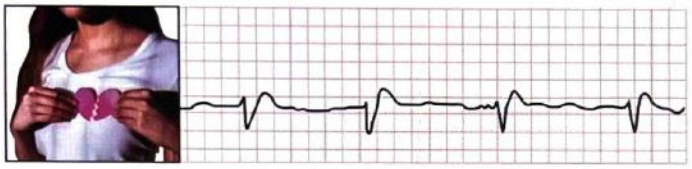
Table 3.2

Toxins	Tamponade	Tension Pneumothorax	Thrombosis (heart: acute, massive MI)	Thrombosis (lungs: massive PE)
•Accidental overdose: Some of the most common include: digoxln, betablockers, and calcium channel blockers). •Cocaine is the most common street drug that increases incidence of pulseless arrest. •Physical signs Include bradycardia, pupil symptoms, and other neurological changes. •Poison control can be utilized to obtain information about toxins and reversing agents.	•Fluid build-up in the pericardium results in Ineffective pumping of the blood which can lead to pulseless arrest. •ECG symptoms: Narrow ORS complex and rapid heart rate. •Physical signs: jugular vein distention (ND), no pulse or difficulty palpating a pulse, and muffled heart sounds. •Perform: pericardiocentesis to reverse.	•Tension pneumothorax shifts in the intrathoracic structure and can rapidly lead to cardiovascular collapse and death. •ECG signs: Narrow QRS complexes and slow heart rate. •Physical signs: JVD, tracheal deviation, unequal breath sounds, difficulty with ventilation, and no pulse felt with CPR. •Treatment: Needle decompression.	•Causes acute myocardial infarction. •ECG signs: 12 lead ECG with ST-segment changes, T-wave inversions, and/or Q waves. •Physical signs: elevated cardiac markers on lab tests, and chest pain/pressure. •Treatments: use of fibrinolytic therapy, PCI (percutaneous coronary intervention). •The most common PCI procedure Is coronary angioplasty with or without stent placement.	•Can rapidly lead to respiratory collapse and sudden death. •ECG signs of PE: Narrow ORS Complex and rapid heart rate. •Physical signs: No pulse felt with CPR. distended neck veins, positive d-dimer test, prior positive test for DVT or PE. •Treatment: surgical Intervention (pulmonary thrombectomy) and fibrinolytic therapy.

4 PULSELESS ELECTRICAL ACTIVITY (PEA)

00:00:18

- Electrical activity of heart i.e. SAN, AVN, Bundle of His are normal but pulse is absent



Code outcome by diagnosis (code blue)

Diagnosis	Survival	
Unstable V_T	65-70% (Pulseless)	shockable- rhythm
V-Fib	25-40%	
PEA	11%	non shockable rhythm
Asystole	0.2%	

Rx: Cause specific Rx.

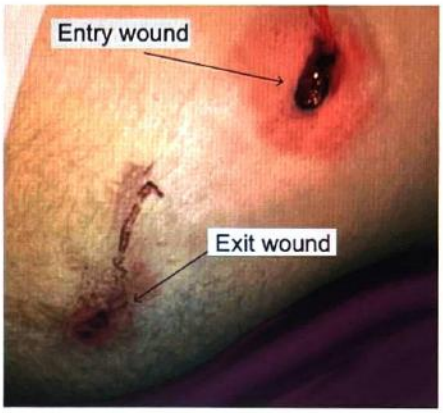
PEA

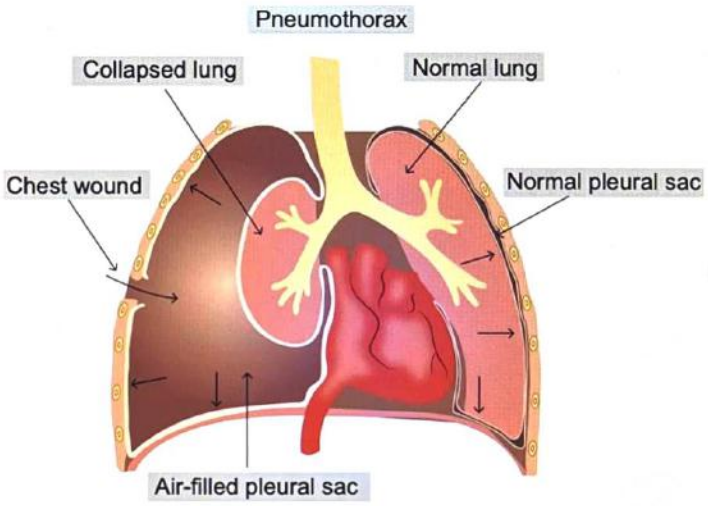
00:05:15

- Causes
  - Empty Heart
  - Hypovolemia
    - → E.g. Bullet injury to the abdomen causing ripping of aorta superior mesenteric artery/ inferior mesenteric artery
    - → Patient can have hypovolemia → resulting in decompensatory shock → pulselessness here, even though muscle of heart is working fine, there is no blood to be pumped

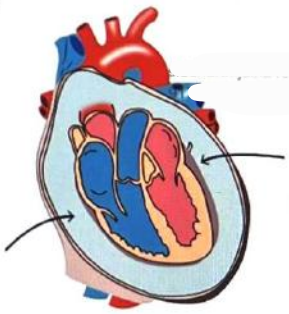
Tension pneumothorax

- E.g. Bullet injury to the chest causing air rush into the chest cavity-increased +ve pressure- lung collapse - kinking of SVC & IVC. → venous return is compromised → cardiac output is compromised → pulselessness



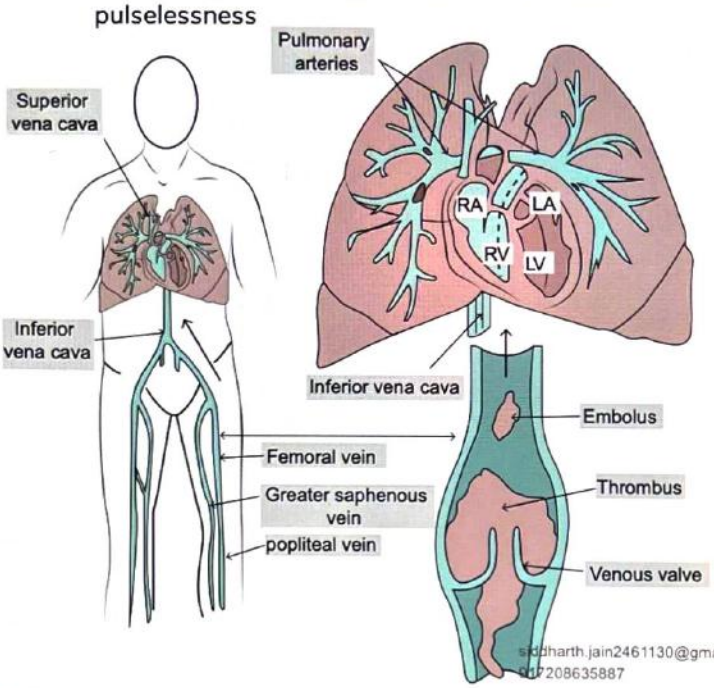


- Cardiac tamponade - fluid in pericardial space which exerts pressure on heart from outside → venous return to heart is compromised → Cardiac output is compromised → pulselessness



- Electromechanical dissociation - Conduction normal
  - E.g. Acute MI: stunned myocardium (hypoxia) (extensive ant. wall MI) (no effective contractions: → CO)

- E.g. Pulmonary embolism Postpartum patient
- Orthopedic surgery E.g. THR, TKR
  → Develop deep vein thrombosis d/t immobilization resulting in pulmonary embolism → which causes severe hypoxia & right sided heart failure → pulselessness



- Echocardiogram → shows dilated right ventricle

TREATABLE CAUSES OF CARDIAC ARREST

00:11:02

$5^{H's}$	$5^{T's}$
• Hypoxia (AMI)	• Toxins
• Hypovolemia (Aortic rupture)	• Tamponade (cardiac)
• Hydrogen ion excess (acidosis)	• Tension pneumothorax
• Hypo / Hyperkalemia	• Thrombosis, Pulmonary
• Hypothermia (Causes BCT < 35°C)	• Thrombosis, Coronary

- Hyperkalemia → K+ 8.0m Eq/L → causes diastolic arrest → pulselessness
- Hypokalemia → causes Diaphragmatic paralysis → resulting in hypoxia → which decreases function of heart
- Hypothermia (T < 35°C)

- Toxins: TCA Toxicity
- Tamponade(cardiac)
- Tension Pneumothorax
- Thrombosis (Pulmonary embolism)
- Thrombosis, (Coronary- Myocardial Infarction)

Revise causes

Heart	Condition	Lung
• Hypovolemia	• Not enough blood	• Hypoxia
• Cardiac Tamponade	• Squeezed	• Tension pneumothorax
• Acute MI	• Killed	• Massive pulmonary • embolism

- **H** - Hypothermia
- **E** - Electrolyte Imbalance (K+/ K+)
- **A** - Acidosis
- **P** - Poisoning

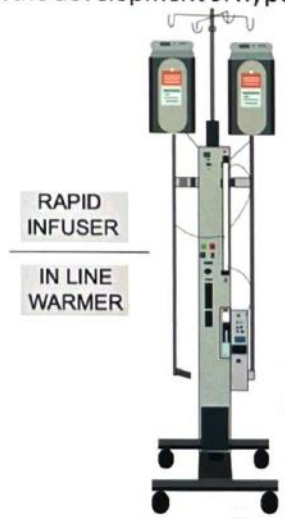
How to remember

- HEAP

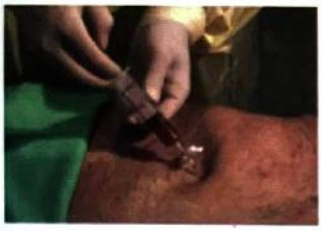
INTERVENTION

00:18:19

- Rapid infuser → having a in line warmer so that it can prevent the development of hypothermia.



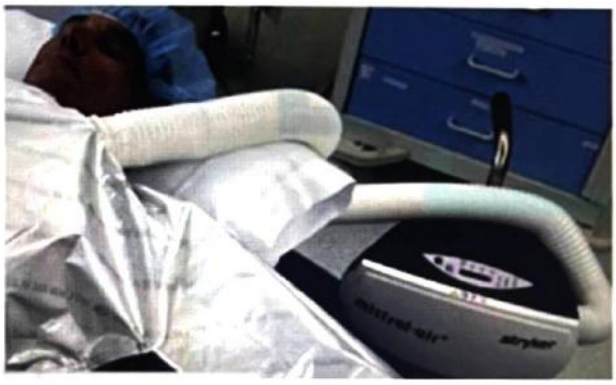
- Echocardiography guided Pericardiocentesis managing cardiac tamponade



- Wide bore needle in 2nd intercostal space managing tension pneumothorax

- If the soldier with tension pneumothorax has bullet proof vest in place/males with big pectoral muscles then, needle is placed in 5th intercostal space, in mid-axillary line

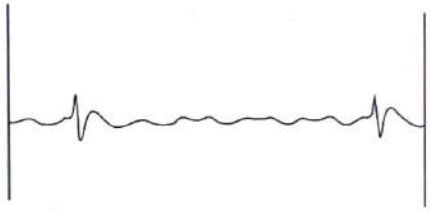
Air Blanket → Treat Hypothermia



5 ASYSTOLE AND POST RESUSCITATION CARE

Asystole 00:01:24

- No pulse & no rhythm
- Flat line on ECG (not necessary to be seen always)
- Final rhythm to other rhythm
- Very poor prognosis



- True Asystole (rule out other causes of an isoelectric ECG)
- Ensure that no another rhythm is masquerading as flat line
- Operator errors
- Loose leads / not connected
- No power
- ECG signal gain too low

MANAGEMENT OF ASYSTOLE 00:05:10

- High quality CPR being performed through BLS, Primary and secondary assessments.
- Secure I.V / I.O access
- Administer emergency drug – epinephrine 1 mg IV/IO every 3 to 5 minutes
- Consider advanced airway and capnography
- Check rhythm after 2 minutes of CPR

If electrical activity is present with good pulse	Begin post cardiac arrest care.
If no electrical activity and pulse	Continue CPR

- Asystole is non-shockable rhythm
- Definitive management – to treat the cause

TERMINATING RESUSCITATION IS DONE IN THE FOLLOWING CASES

00:10:23

- Time stretch of collapse of patient to CPR is too long
- Time between collapse to first defibrillation attempt.
- Multiple co-morbidities
- Pre-arrest state of patient
- Initial arrest rhythm
- VF & V_T: Good prognosis
- PEA & asystole: Poor prognosis
- No response to CPR
- $ETCO_2$ <10 mm Hg even after 20-30 minutes of CPR
- Out of hospital resuscitation done
- If Return of spontaneous circulation (ROSC) achieved already
- If care of patient is transferred to Advanced cardiac life support (ACLS)
- Presence of reliable criteria indicating irreversible death
- A valid DNAR (Do not attempt resuscitation)
- If resuscitating person is exhausted
- If scene becomes unsafe for patient and the rescuer

Prognosis & Terminal rhythm

- Asystole is identified as a terminal rhythm in a resuscitation attempt that started with another rhythm i.e. VF / pulseless V_T leading to asystole
- First rhythm to be identified in a patient with unwitnessed arrest or prolonged arrest
- Poor prognosis unless the patient is witnessed during arrest or exact cause is known.

POST RESUSCITATION CARE 00:21:58

- Optimization of hemodynamics
- Ventilation effort
- Initiate targeted temperature management to prevent neurological damage.
- Provide immediate coronary reperfusion (PCI)
- Provide neurological care and prognostic for patients of acute stroke who progressed to cardiac arrest.

RETURN SPONTANEOUS CIRCULATION

- Adequate ventilation & circulation
- Assess airway and breathing
- An unconscious patient will require an advanced airway.
- So, definitive airway is inserted into the patient and attached to capnography machine for continuous wave form capnography
- Lowest inspired oxygen concentration that will maintain arterial oxygen level above 94% (Target SPO2 in post resuscitation care- 94%)
- Avoid excessive ventilation (Target PET CO_2 – 35 to 40 mm Hg)

- Treat hypotension (Target systolic BP above 90 mm Hg)
- I.V care
- I.V bolus 1-2 L of normal saline
- Ionotropes should be started
- Norepinephrine -0.1-0.5 mcg /kg per minute
- Epinephrine – 0.1– 0.5 mcg / kg per minute
- Dopamine – 5-10 mg per minute I.V infusion
- Targeted temperature maintenance (TTM)
- Patients who remain comatose Protect their vital organs
- Protect vital organs –maintain constant target temperature between 32-36 degree Celsius for at least 24 hours. (It will decrease the metabolic activity of body)
- Maintenance of constant temperature can be done by rapid infusion of ice cold isotonic non-glucose fluid (30 ml /kg)
- Assess prognosis 72 hours after return of spontaneous circulation
- In hypothermic patients, the prognosis assessment is delayed for >72 hours after attaining the core body temperature to normal.
- A good post resuscitation care gives a good outcome in cardiac arrest patients.

6 BRADYCARDIA / BRADYARRHYTHMIAS

DEFINITION

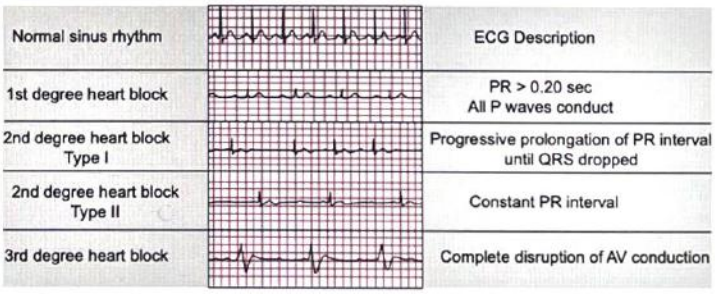
00:00:30

Bradycardia

- Heart rate less than 60 beats/min
- Heart rate less than 50 beats/min (AHA)

Rhythms of Bradycardia

- Sinus Bradycardia
- 1st degree AV block – PR interval > 0.2 sec
- 2nd degree AV block
  - Type 1 (Wenckebach / Mobitz I) – progressive increase in PR interval
  - Type 2 (Mobitz II) – Few non conducting P impulse
- 3rd degree AV block / complete AV block



Cornerstone of Management

- Recognize sign & symptoms caused d/t slow heart rate
- Correctly diagnose the presence & type of AV block
- Use of atropine
- Decision regarding – TCA / Epinephrine / Dopamine
- Expert consultation

Important Information

- **Bradycardia can be normally present**
- **It can be abnormal but chronic – patient asymptomatic**

SYMPTOMATIC BRADYCARDIA

00:10:13

- Heart rate is slow
- Patient has symptoms d/t slow heart rate

Symptoms

- Hypotension
- Chest Discomfort
- Decreased Mentation
- Weakness, Fatigue, Light Headedness
- Acute Heart failure

Management

- Step 1 – Identify the condition
- Step 2 – primary assessment
  - Maintain patient airway
  - Supplement oxygen
  - Cardiac monitor
  - IV access
  - 12 lead ECG

ASYMPTOMATIC BRADYCARDIA

00:12:37

- No signal / symptoms
- Observe the patient

Management of patient with symptoms

- 1st line of management – atropine
- Dose: 0.5 mg IV (repeat till max dose of 3 mg)
- Avoid relying on atropine for Type II 2nd degree block or 3rd degree block or other infranodal block
- Use transcutaneous pacing / Beta adrenergic support

ATROPINE

- 1st line of treatment in symptomatic bradycardia
- Reverse the cholinergic action
- Should be used in condition in cases of acute coronary ischemia / MI

TRANSCUTANEOUS PACING (TCP)

- It delivers pacing impulse to the heart through the skin by using cutaneous electrodes

Indications of TCP

- Hemodynamically unstable bradycardia
- Unstable clinical condition likely d/t bradycardia
- Bradycardia with vascular escape rhythms

Precautions of using TCP

- Contraindicated in secure hypothermia
- Not recommended for asystole
- Carotid pulse cannot be assessed

- Requires analgesia

Steps of TCP
- Place pacing electrodes on the chest
- Turn paces on & set demand rate to approx 60/min
- Set the current milliamperes

Important Information

- Beta adrenergic agonist
  - Rate accelerating effect
  - Not 1st line of management
  - Used in overdose of ß-blocker & calcium channel blocker
- Commonly used
  - Epinephrine: Infusion dose – 2 to 10 mg/min
  - Dopamine: Infusion dose – 2 to 20 mg/kg/min

BRADYCARDIA WITH ESCAPE RHYTHM

00:24:21

- Always proceed with TCP

Standby pacing
- ACS – Severe bradycardia rhythms
- Symptomatic
- Asymptomatic mobitz Type II
- Asymptomatic 3rd degree heart block
- Newly acquired LBBB or RBBB

7 TACHYCARDIA

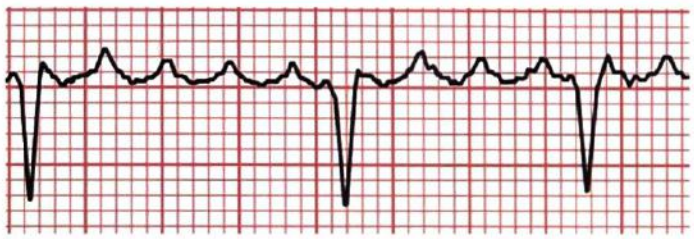
DEFINITION

00:00:30

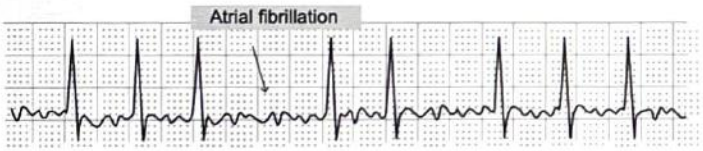
- Patient with HR > 100 /min
- Always physiologic – compensatory to compromise stroke volume or to increase cardiac output
- Treat the primary condition in patients with Tachycardia

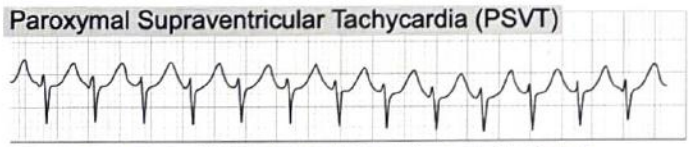
Classification of Tachycardia

- On the basis of
  - QRS complex – narrow or wide
  - Heart rate
  - Rhythm – Regular or Irregular
- Narrow QRS complex (QRS less than 0.12 seconds)
  - Most of them are atrial in origin
  - Sinus Tachycardia
  - Atrial fibrillation – Fibrillating P waves
  - Atrial flutter – Saw toothed P waves
  - AV nodal reentry
  - PSVT



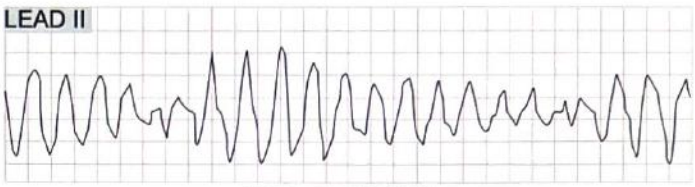
Atrial flutter



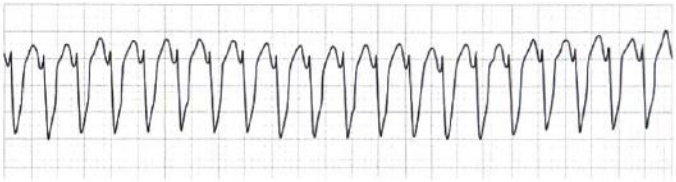


- P waves may not be visible and often hidden in the preceding T wave
- Atrial rate is 120-200

- Wide QRS complex (QRS 0.12 seconds or more)
  - Monomorphic VT
  - Polymorphic VT
  - SVT with aberrancy



Polymorphic VT



Monomorphic VT

SINUS TACHYCARDIA

00:08:45

- HR greater than 100/min & is generated by sinus node
- Gradual onset & gradual termination
- Mostly d/t external influence on heart e.g. fever/ Anemia / Hypotension/ Blood loss / exercise
- Cardioversion is contraindicated
- Goal is to treat underlying cause

Management

- Narrow QRS complex Tachycardia
  - Attempt vagal maneuver – carotid sinus massage
  - Adenosine – 1st dose – 6mg IV push
    → Not responsive
    → 2nd dose – 12 mg IV push
  - Dose reduced in patient with transplanted heart / central line administration / patient on dipyridamole or carbamazepine
  - Contraindicated in Bronchial Asthma / COPD
- Vagal maneuver alone terminate 25% of SVTs
- Wide QRS complex Tachycardia
  - Most wide complex Tachycardia are Ventricular in origin
  - SVT aberrancy
    → Rate regular & monomorphic
    → Adenosine
- Rhythm not clear – Start IV antiarrhythmic
  - Procainamide
  - Amiodarone
  - Sotalol
- Irregular wide complex – Expert consultation

UNSTABLE TACHYCARDIA

00:15:11

- **Present with**
  - Hypotension
  - Acutely altered mental status
  - Signs of shock
  - Ischemic chest discomfort
  - Acute heart failure
- **Management**
  - Give oxygen
  - ECG
  - Evaluate blood pressure
  - Establish IV access
  - Identify & treat reversible cause

TREATMENT

00:17:17

- Based on type of tachycardia
- Pulseless – treat according to cardiac arrest rhythm
- Unstable with pulse regular uniform wide complex UT
  - Monomorphic
    - → Synchronized cardio version with 100J
  - Polymorphic
    - → High energy unsynchronized shock
- Unstable Tachycardia with narrow QRS complex
  - Synchronized Shock
    - → Unstable AF – 120 – 200 J
    - → Unstable flutter / SVT – 50 - 100 J

CARDIOVERSION

00:20:45

- Modern defibrillators / cardioverters deliver 2 types of shock
  - Unsynchronized shock (Defibrillators)
    - → Ventricular fibrillation
    - → Pulseless ventricular tachycardia
    - → Periarrest
    - → Unstable polymorphic Tachycardia
    - → Not clear polymorphic or monomorphic
  - Synchronized shock (cardioversion)
    - → Unstable Atrial fibrillation
    - → Unstable Atrial flutter
    - → Unstable SVT
    - → Unstable regular monomorphic Tachycardia with pulse

Unsynchronized shock	Synchronized shock
• Electric shock delivered when operative pushes shock button • A high energy required	• Uses a sensor to deliver a shock that is synchronized with peak of QRS complex • A low energy required

Important Information

- Unstable atrial fibrillation – 120 -200 J
- Unstable SUT / Atrial flutter – 50 – 100 J
- Unstable regular monomorphic tachycardia with pulses - 100 J
  - Polymorphic UT – Defibrillation Dose

8 ACUTE CORONARY SYNDROME

ACUTE CORONARY SYNDROME 00:01:30

- It is common presentation in ED
- Mean age - 68 years (55-72)
- Male preponderance 3: 2

Non-ST Segment Elevation

- It is present on 70 % of patients.
- It is not massive acute myocardial infarction
- Patients have more subtle findings or non-finding on their initial ECG.

DEFINITION 00:03:30

- Any new conditions that leads to inadequate myocardial perfusion relative to demand.
- Coronary atherosclerosis may develop a spectrum of clinical syndromes
- It representing varying degrees of coronary artery occlusion
  1. Unstable angina
  2. Non ST segment elevated MI } Presentation
     spectrum varies
  3. ST elevation MI

1. Unstable Angina 00:04:54

- Patient has H/o impaired exercise tolerance
- No ECG findings/ No elevated troponins

2. Non ST elevation MI 00:06:10

- Ischemic pain in any setting at exertion or rest.
- ECG changes
  - ST depression/ T wave inversion
  - Troponin elevated

3. ST elevated MI 00:07:18

- It is classical acute emergency
- Diagnosis & management should start early for saving life.
- **Time is muscles**
  - Survival depends on out hospital response
  - Ventricular fibrillation or pulseless tachycardia - precipitating rhythm in most of these case.
  - M / c / c of ventricular fibrillation or pulseless tachycardia - ACS (STEMI)

COMMUNITIES SHOULD DEVELOP PROGRAM TO RESPONSE TO OHCA 00:10:42

1. Recognizing symptoms of ACS
2. Activating the EMS
3. Providing early CPR
4. Early defibrillation
5. Coordinated system or care among the EMS, ED & cardiology

Goals

1. Identifications of patients with STEMI & triage for early reperfusion
2. Relief of Ischemic chest discomfort
3. Prevention of MACE (major adverse cardio vascular event)
4. Treatment of acute life threatening complications

MANAGEMENT 00:14:48

- Based on AHA algorithm

STEP-1

- Recognize symptoms suggestive of Ischemia
- Most common symptoms: Retro sternal chest discomfort
  - Uncomfortable pressure, fullness, squeezing or pain in the chest (lasting several minutes)
  - Chest discomfort

1. Spreading to shoulders, neck, one or both arms or jaw
2. Spreading into back in b/w shoulder blade
3. With light headedness, nausea, vomiting
   - Shortness of breath.

- EMS should immediately start assessment & action
  - Monitor & support ABCs
  - Oxygen
  - Aspirin
  - Nitroglycerine - (sublingual)
  - Morphine
  - 12-lead ECG.
- Monitor & support of ABC's includes

1. Monitoring vital signs 7 cardiac rhythm
2. Be prepared to provide CPR

3. Defibrillator

- Oxygen
  - High inspired-oxygen tension
    → Maximize arterial oxygen saturation

ASPIRIN

- Action: Inhibits thromboxane A_2 production by inhibiting platelet cyclo-oxygenase (C O X-1)
- Platelet: Earliest & principal participant in thrombus formation.
- Prevents reocclusion
- **Dose**: 160-325 mg [Non enteric coated aspirin to chew]
- **Contraindication**
  - Allergy
  - Recent GI bleeding
- Rectal aspirin suppository: 300 mg (Incase of GI disorders)

NITROGLYCERINE

- It reduces ischemic chest discomfort
- Action-Peripheral arterial & venous dilatation
  - Reduce LV & RV preload
- **Dose**: Sublingual tablet every 3-5 min.
- **Contraindication**
  1. Inferior wall M I & R V infarction
  2. Hypotension, brady/ tachycardia
  3. Recent phosphodiesterase inhibitor use

MORPHINE

- Reduce pain/ chest discomfort
- Not given in non-STEMI
- Sympatholytic effect
- **Action**
  - Venodilation-decrease LV load & oxygen requirement
  - Reduces after load
  - Redistribute blood in acute pulmonary edema

EMERGENCY DEPARTMENT

00:29:10

- Assessment <10 min
- Check vitals & give oxygen
- Establish IV access
- Brief targeted history, PE
- Review/ complete Fibrinolytic checklist, cardiac marker levels, electrolytes Coagulation studies
- Portable X-ray (<30 min)

ECG interpretation

- STEMI-ST elevation
- NSTE-ACS-ST depression or dynamic T-wave inversion
- Low/intermediate risk ACS - Normal ECG.

STEMI

00:34:58

Characteristic

- ST-segment elevation in 2 or more contiguous leads or new LBBB

Threshold values

- J- point elevation greater than 2 mm in leads V_2 & V_3 mm or more in all others leads or New LBBB

ST elevation

- It means complete occlusion of an epicardial coronary artery

Management

- Timely reperfusion: Early fibrinolytic/ PCI
- If fibrinolytic given within hour of onset of symptom 47 % reduction in mortality

4D's

- Door-Data-Decision-Drug
- Time should be minimized

TIME FRAMES FOR REPERFUSION

00:38:57

- For PCI - within 90 minutes after entering E D
- Fibrinolytics - within 30 min
- Ineligible for fibrinolytic - Transfer to PCI within 30 minutes

Management

- Percutaneous intervention (PCI)
  - M.C used form of PCI-coronary Intervention with stent placement
  - Preferred over fibrinolytic
  - Within 90 min from first medical contact
  - Within 120 mins from first medical contact if transfer required
  - Patients who cannot be given fibrinolytic.

- Fibrinolysis
  - Fibrin specific agents
  - Recombinant tissue type plasminogen activator (rtpa)

Inclusion criteria for fibrinolysis

- In absence of contraindication within 12 hours of onset of presentation
- After 12 hours, not recommended
- But in presence of continuous chest pain after 12 hours, then we can give an attempt

Exclusion criteria for fibrinolysis

- Significant head trauma or prior stroke in previous 3 months.
- Arterial puncture at non compressive site in previous 3 months.
- History of previous Intracranial hemorrhage
- Elevated BP (SBP > 185, DBP > 110)
- Active internal bleeding
  - Acute bleeding Diasthesis
  - Platelet count < 1lakh / mm^3
- Heparin received within 48 hrs (prolonged aPTT)
- Current anticoagulants INR > 1.7 or PT > 15 sec
- Blood glucose <50 mg/dl
- CT multilobular infarction

Adjuncts of management 00:47:15

- Heparin
- Bilivalirudin
- IV Nitroglycerine
- Beta-Blockers
- Glycoprotein IIb / IIa inhibitors

9 CONGESTIVE CARDIAC FAILURE

00:00:15

- CHF is one of the most common illness treated in emergency department
- Causes when ventricles fail to maintain blood circulation after when cardiac demand increases
- Chronic congestive heart failure may get precipitated in acute congestive heart failure in case of some precipitating event
- Typical complete of shortness of breath and peripheral edema seen due to venous return of organs affected

PRECIPITATING EVENTS

00:01:26

- Cardiac ischemia or dysrhythmia
- Infection
- Physical or emotional stress
- Noncompliance in medication or diet, volume overload

NYHA CLASSIFICATION OF CHF

00:03:01

- Class I: 'Ordinary activity not limited by symptoms
- Class II: Ordinary activity leads to Dyspnea
- Class III: Marked limitation of ordinary activity
- Class IV: Symptoms at rest or with any physical activity

Systolic dysfunction	Diastolic dysfunction
• Dilated left ventricle with impaired contractility o Systolic function of ventricle is affected because contractility of left ventricle is affected • Typically fit ischemia, infarction, cardiomyopathy	• Ventricle is unable to relax but remains intact and is normal in size which happens in infiltrative cardiomyopathy • Better prognosis than systolic dysfunction • More common than systolic dysfunction • More common than systolic dysfunction

00:06:25

High output failure	Low output failure
• Cardiac output is high or normal but insufficient to supply O_2 demands	• Decrease in cardiac output
• Can be seen in hypothyroidism, beriberi, anemia, pregnancy, AV fistulas, paget's disease	• Secondary to myocardial damage such as ischemia, dilated cardiomyopathy, valvular disease or chronic hypertension

00:07:20

Right sided Failure	Left sided failure
• Can lead to congestion of pressure and fluid into right ventricle • Results in hepatic enlargement, increased SVP and dependent edema	• Congestion of pressure and fluid into left ventricle • Results in pulmonary congestion

Differential diagnosis of CCF

- COPD
- Pulmonary Embolism
- Pneumothorax
- Anaphylaxis
- Asthma
- Pneumonia
- Foreign body obstruction
- Acute coronary syndrome

INITIAL ACTIONS AND PRIMARY SURVEY

00:08:56

- Airway, breathing, circulation - to be stabilized first
- Attach cardiac monitor, pulse oximetry, IV access with two large bore IV cannula
- IC patient hypoxic-Give 0_2 via 100% non-re-breathing face mask
- If not able to maintain oxygenation → Invasive methods like CPAP, BiPAP or intubate if needed
- Elevate the head of the bed and may required to dangle the leg of patient on side of stretcher to reduce venous return and decrease pre-load
- IC chest pain → Obtain ECG and medicate with NTG
- Order a chest X-ray
- Draw labs for CRC, Troponin T, PT /PTT, SNP. May be include lactate and blood cultures to rule out any infections.

PRESENTATION 00:13:17

- History is important
- Classical presentation of CHF → Dyspnea on exertion (sensitivity of 100%a specificity of 17%)
- Absence of exertional dyspnea → Patient is not a case of congestive cardiac failure
- Orthopnea (sensitivity of 88% and specificity of 50%)
- Paroxysmal nocturnal dyspnea (sensitivity 39% and specificity of 80%) → Increase likelihood of CHF
- Cough with productive sputum which is pink and frothy
- Swelling of lower extremities

ON AUSCULTATION 00:15:34

- Pales secondary to alveolar edema. Absence of Rates decreases the likelihood of CHF (sensitivity 29% a specificity 77%)
- Wheezing may or may not be present
- Tachycardia with S_3 gallop(sensitivity 24% and specificity 99%)-Presence of S_3- 'Increase likelihood of CHF

Chest X-ray

- Common findings Cardiomegaly and surrounding effusion around heart
- X-ray finding may lag behind 12hrs from onset of symptom and persist for several days after dissolution of the symptoms
- In chest X-ray
  - Cardiomegaly: Cardio-thoracic ratio >50% diameter. → Patient with diastolic Failure may have normal heart
  - Peri bronchial cuffing: thickened bronchial walls secondary to edema
  - Perihilar congestion: large hilar with indistinct margin suggest pulmonary vasculature edema
  - Cephalization: redistribution a b/f to upper lobes. Only seen on upright films
  - Pleural effusion: Meniscus at the angle of the diaphragm
  - Kerley B lines: Dilated lymphatic channels. Typically 2 cm in length and horizontal, peripherally located perpendicular to pleura

BNP (B-TYPE NATRIURETIC PEPTIDE & N-TERMINAL-PRO-BNP) 00:18:27

- Produced by heart muscles is a biomarker of CHF
- As the value of BNP and N-terminal - Pro-BNP increases—very much suggests congestive heart Failure
- BNP- sensitivity 90% a specificity 76%
- Release is stimulated by high ventricular filling pressures
- Has diuretic effect and anti-hypertensive effect by increasing amount of sodium in urine
- <100 BNP: Suggest that CHF is unlikely
- 100-500: Potentially CHF, but could also be pulmonary embolism, pulmonary HTN, ESRD, cirrhosis or HRT
- >500 Most likely CHF

ECG

- Shows any underlying cardiac ischemia, dysarrhythmia, LVH or heart block
- A normal ECG has a high negative predictive value for systolic dysfunction

Echocardiogram with ejection fraction

- Normal ejection fraction→ .55-75%.
- Patients with severe CHF may have EF <207.
- Echocardiogram can also be used to visualize ventricular size, any wall abnormalities, valvular pathology, pericardial thickening, tamponade, constrictive pericarditis etc

EMERGENCY DEPARTMENT RX 00:22:20

- After initial management (IV, O_2, monitor etc.) have been successfully completed, medication need to be started as per requirements
- **For normotensive patients**
  - Give nitrates to vasodilate (sub lingual, IV, transdermal patch, nitropaste)
  - Morphine can be given if chest pain is present and to vasodilate as well
  - Diuretics such as lasix are given so that it increases urine output
- **For hypertensive patients**
  - IV nitroprusside drip for severe persistent HTN and nitrates can be given as a supplementation along with it
- **For hypotensive patients**
  - Avoid nitrates and morphine as they further decrease the blood pressure
  - Use inodilators like amrinone or milrinone (m/c used), dopamine, dobutamine and nor epinephrine
- Severe or chronic low output CHF-Use ACE inhibitors to increase hemodynamic stability and exercise capability
- Diastolic CHF-CCB can be used but avoided in case of depressed LVF patients as this may increase the mortality and recurrence of CHF

Surgical therapies

- As patients CHF becomes advanced, the patient may need an automatic internal cardiac defibrillator (AICD), left ventricular assistance device (LVAD) or heart transplant ultimately.

Disposition

- Cardiac care unit for further management

10 APPROACH TO SHOCK

00:01:05

Definition

- **Shock**: patho-physiologic state in which the oxygen supply to body tissue inadequately meets metabolic demands, resulting in dysfunction of end organs.

Shock may be due to

- Impaired delivery of oxygen to tissues
- Impaired utilization of oxygen by tissues
- Increased oxygen consumption by tissues, or a combination of these processes.
- Early recognition of shock and institution of empiric treatment is of utmost importance.
- Shock presents with an identifiable etiology (i.e. gunshot wound to the abdomen) or may be undifferentiated
- A rapid and accurate assessment of the airway, breathing and circulation followed by a focused histology and physical examination
- Examination of
    - Neck veins (+ Jugular venous distention)
    - Cardiopulmonary examination
    - Abdominal examination
    - Extremity examination (+ signs of hypoperfusion or abnormal vasodilation)
    - A rectal examination to assess for melena or hematochezia
- Robust I.V access (i.e. two large bore IV catheter, intraosseous access, or central venous catheter) should be established
- The patient should be placed on continuous telemetry, non-invasive blood pressure monitoring and pulse oximetry.
- An ECG and chest radiograph should be obtained.
- Bedside ultrasonography (i.e. FAST examination)- to narrow the differential diagnosis.
- Initiation of intravenous fluid resuscitation is usually indicated

11 HYPOVOLEMIC SHOCK

00:10:04

- ↓ intravascular volume
- Resulting in
  - ↓ preload
  - ↓ stroke volume
  - ↓ cardiac output
  - ↓ perfusion

Hypovolemic shock	
Hemorrhagic Shock	**Non – Hemorrhagic Shock**
(Major trauma, gastrointestinal bleeding, ruptured ectopic pregnancy, etc). ↓ If not, resuscitation may lead to ↓ V. Fibrillation, PEA (pulseless Electrical activity)	(Vomiting, diarrhea, burns, etc.)

CARDIOGENIC SHOCK

00:11:53

- Impaired cardiac output secondary to cardiac pump function, rhythm aberrancy or structural defect.
- Arrhythmogenic (brady or tachy-dysrhythmia)
- Cardiomyopathic
- Myocardial infarction,
- Heart failure
- Cardiotoxicity from medication
- Structural defect
- Valvular insufficiency
- VSD

DISTRIBUTIVE SHOCK

00:13:20

- Pathologic vasodilatation leading to relative intravascular volume depletion
- Anaphylactic (IgE mediated allergic reaction)
- Septic
- Pneumonia,
- Pyelonephritis,
- Neurogenic (spinal cord injury)
- Toxicologic (vasodilatory medication overdose)

OBSTRUCTIVE SHOCK

00:15:00

Impaired cardiac output due to

- Pulmonary vascular obstruction
- Pulmonary embolism,
- Venous air embolism,
- Mechanical obstruction
- Tension pneumothorax,
- Pericardial tamponade,
- Left ventricular outflow tract obstruction

MIXED/ OTHER SHOCK

- Cellular poisons
- Carbon monoxide,
- Cyanide
- Endocrinologic
- Adrenal insufficiency,
- Myxedema coma
- Environmental (hypothermia)
- Toxicologic etiology
- Cardiac and
- Vasoplegic manifestation (calcium channel blocker, etc.)

HISTORY

00:18:30

- C/o lethargy or weakness, or altered mental status (i.e. CNS- end organ damage)
  - Nonspecific symptoms (chest pain or dyspnea) may be due to the primary insult (i.e. ACS, IE) or a result of end organ tissue ischemia secondary to shock.
- Hypovolemic shock due to hemorrhage is suggested from trauma bleeding from:
  - Melena or hematemesis from a gastrointestinal source,
  - Vaginal bleeding from a gynecologic source
- Non – hemorrhagic hypovolemic shock
  - Decreased oral intake,
  - Diarrhea,
  - Vomiting or
  - High ostomy output.
- Cardiogenic or obstructive shock
  - chest pain,
  - shortness of breath,

- recent leg swelling or
- syncope.

- Anaphylactic shock
  - sudden onset of pruritus,
  - hives or
  - swelling following exposure to an inciting trigger.
- Septic shock associated with signs of infection
  - Fever,
  - Cough,
  - Dysuria,
  - Abdominal or prank pain or rigors.
  - Non-focal, vague complaints
  - Weakness,
  - Altered mental status or
  - Malaise may be the only presenting symptoms of shock.

PHYSICAL EXAM

00:23:00

- Hypotension alone should not be used as the sole determinant of shock, Early shock may present with normal or even elevated blood pressure and normal heart rate.
- Without prompt treatment of the patient in early shock, hypotension and tachycardia frequently follows.
  - Altered mental status
  - GPE- Thready pulse, tachycardia and tachypnea
  - Skin and mucosa -cool, pale or ashen skin
  - Decreased capillary refill and
  - Dry mucous membranes;
- In distributive shock- extremities are warm.
- In cardiogenic shock,
  - Arrhythmias,
  - Dependent edema, or
  - New murmurs
  - Jugular venous distention is seen in both cardiogenic and obstructive shock
- Focused bedside ultrasonography to assess for
- Intraperitoneal hemorrhage,
- Pneumothorax, or
- Pericardial tamponade
- Global cardiac function
- Intravascular volume status.
- Abdominal aortic aneurysm with rupture may also be identified

E.g.

- Pump refers to cardiac function, measured by cardiac output in term of cardiac contractility and heart rate (HR).
- Tank refers to the amount the preload present measured by central venous pressure (CVP),
- Pipes are assessed by systemic vascular resistance (SVR).

Category	HR	CVP	Contractility	SVP
Hypovolemic	↑	↓	±↑	↑
Cardiogenic	↑ or ↓ (tachy or brady)	↑	±↓	↑
Distributive (sepsis)	↑	↓	±↑	↓
Obstructive	↑	↑	±↑	↑

- Serum lactate
  - A marker of as aerobic metabolism the degree of tissue, organ supply and demand imbalance, is particularly useful in the initial assessment and assessing response to therapeutic interventions.
  - No isolated diagnostic test is sensitive or specific for shock.

Investigations

- CBC with differential
- Electrolytes,
- BUN, creatinine,
- Glucose
- Lactate
- Coagulation studies
- Hepatic function
- Calcium
- Urinalysis
- ECG
- Chest radiograph
- Pregnancy test (blood or urine)
- Blood gas

If a particular etiology of shock is suspected, further studies may be indicated.

- Hemorrhagic etiology – type & screen
- Infectious etiology
  - Blood and urine cultures;
  - CSF studies;
- Focused CT
- Ultrasound.
- Cardiogenic
- Cardiac enzymes (ACS, myocarditis)
- Echocardiogram (heart failure or structural etiology)

Obstructive

- CT (PE)
- Echocardiogram (pericardial tamponade)
- Often deferring image in favor of empiric management in the ED and critical care setting is advisable until the patient's condition has stabilized.
- Timely empiric treatment for shock pt is crucial to minimize morbidity and mortality
- Critical findings involve airway, breathing, circulation (I.e "the ABCs") to be emergently addressed
- Ensuring proper oxygenation is critical for all types of shocks
- Arterial oxygen saturation to be maximized.

Airway and breathing

- Intubation should be strongly considered for:
  - Airway protection,
  - Optimization of oxygenation and ventilation
  - ↓ the metabolic load increased from work of breathing.
  - Some induction and sedative medication used to facilitate intubation, as well as positive pressure ventilation itself can reduce preload and have negative hemodynamic effects.
  - Important in patients in which preload in impaired,
- Obstructive or distributive shock states.
- Interventions to optimize hemodynamics prior to intubation may be indicated before intubation i.e.
  - Needle decompression in tension pneumothorax;
  - Rapid fluid bolus in septic shock

Circulation

- Two large-bore intravenous cannulas allow to rapid volume infusion and mediation delivery.
- If peripheral IV access is unable to be established, intraosseous access or a percutaneous introducer catheter may be inserted.
- Volume expansion with crystalloid fluids (normal saline or Ringer's lactate) should be administered as rapid bolus infusion.
- In cardiogenic shock- Fluid administration worsens the hemodynamic state and precipitates pulmonary edema.

Treatment 00:38:48

- In hemorrhagic shock
  - Blood products may be indicated
  - Crystalloid infusion should be minimized to reduce clotting factor dilution and hyperchloremic acidemia.
  - If volume resuscitation does not improve patient's hemodynamic state,
  - Initiation of vasopressor medications e.g.: norepinephrine or epinephrine
- Hypovolemic shock
  - Due to hemorrhage: Source control via surgical intervention or radiographically guided percutaneous intervention (IR)
- Distributive shock
  - Due to sepsis to be treated with
  - Goal directed therapy
  - Lactate clearance,
  - Accurate hemodynamic monitoring
  - Appropriate antibiotic treatment
  - Infection source control
  - If indicated vasopressor such as norepinephrine may be required.
- Due to anaphylaxis should be treated with
  - Intramuscular epinephrine.
- Cardiogenic shock may necessitate
  - Synchronized cardioversion,
  - Cardiac pacing,
  - Emergent angiography, or
  - Surgical procedures (i.e. valve repair).
  - Intra-Aortic Balloon Pump (IABP) or Extra Corporeal Membrane Oxygenation (ECMO) may be beneficial.
- Obstructive shock
  - Due to pulmonary embolism may require systemic thrombolysis,
- Cardiac Tamponade requires
  - Pericardiocentesis
- Tension pneumothorax requires
  - Pleural decompression
  - Needle thoracostomy followed by thoracostomy tube
- Adequate resuscitation of a shock state is not indicated by any specific end point or parameter.

Indication of appropriate resuscitation and resolution of a shock state includes

- Normalization of hemodynamic parameters (i.e. BP, HR and urine output)
- Lactate clearance with reduction in lactate concentration below 2 mmol/L.
- Volume status restored
- Resolution of acidosis and return to normal metabolic parameters.

- Patients in shock usually require admission to a critical case setting under the case of an Intensivist and other consulting services as needed.

HYPOVOLEMIC SHOCK

- Hypovolemic shock is due to → Significant blood/ fluid loss [i.e. intravascular volume is ↓]

Causes of hypovolemic shock 00:01:13

- Depending upon history, causes are of 2 types.

1. Non-Haemorrhagic Shock

Digestive losses	Renal losses	Skin losses	3rd space losses	Hypothermia
• Diarrhea, • Vomiting, • Nasogastric suction, • Biliary loss, • Digestive fistula	• D.M, • Polyuria • Diuretics overdos, • osmotic substance, • polyureic phase of acute renal failure	• intense physical effort • exertional heat stroke • burns	• Peritonitis • Intestinal occlusion, • Pancreatitis, • Ascites, • Pleural effusions	

2. Haemorrhagic Shock

- Due to loss of blood volume mostly due to

External blood loss	Exteriorization of internal bleeding	Internal bleeding	Traumatic shock
• Wounds	• Hematemesis, • Melena, • Epistaxis, • Hemoptysis	• Hemothorax, • Hemoperitoneum, • Splenic rupture	

ATLS [ADVANCED TRAUMA LIFE SUPPORT] CLASSIFICATION OF HEMORRHAGIC SHOCK

00:02:37

	Class I	Class II	Class III	Class IV
Blood loss (ml) %	• <750 ml • 15%	• 750 - 1500 ml • 15 - 30%	• 1500-2000 ml • 30-40%	• >2000 ml • >40%
HR	• <100	• >100, <120	• >120	• >140
BP	• Normal	• Normal	• Decrease	• Decrease
PP	• Normal	• Decrease	• Decrease	• Decrease
RR	• 14-20	• 20-30	• 30-40	• >35
Urine Output	• >30ml/hr	• 20-30ml/hr	• 5-15ml/hr	• Negligible
CNS	• Slightly anxious	• Mildly anxious	• Anxious • confused	• Confused • lethargic

- Management & urgency depends upon the class

PATHOPHYSIOLOGY OF HYPOVOLEMIC SHOCK

00:05:35

Hypovolemia
↓
↓ venous → return → ↓ Preload → ↓ CO
↓
Hypotension
↓
Perfusion failure & Tissue hypoxia
↓
Organ dysfunction
↓
Multi-organ failure

- (CO= SV X HR)
- (BP = CO X TPR)

- So, stop at the level of ↓ C.O
- Resuscitate, so that preload, C.O. can be maintained & hypotension, perfusion failure can be prevented

ASSESSMENT

Clinical Signs & Symptoms

- Anxiety
- Dizziness
- Altered mental status [C.N.S dysfunction]
- Thirst
- Pale, cool & clammy skin (due to inadequate perfusion)
- Delayed capillary refill time
- Tachycardia,
- Tachypnoea
- Weak thready & rapid pulse.
- Wide pulse pressure
- Hypotension
- ↓sed urine output
- All these are due to end-organs dysfunction → which further deteriorate the patient

ACTION

00:08:35

- **A - Airway** → if patient is comatosed → secure airway → to prevent aspiration
- **B - Breathing** → if patient is tachypneic → support ventilation of patient
- **C - Circulation** → Assess the circulation & give appropriate intervention.

How to remember

- ABC

- Maintain airway & breathing, give oxygen & keep the patient warm
- Make the person lie flat with feet elevated → if no contraindication to do so.
- Secure 2 wide bore I.v cannula (16/18 G)
  - Start giving warm crystalloid fluids
- Initially give 1 L of RL/NS → 20ml/kg body wt to assess the response
- If intravascular volume can't be maintained through crystalloid fluids, then give blood transfusion
- Blood & blood products must be infused at earliest possible time, in a bleeding patient→ in transient /non –responding patient.
- Activate Massive Transfusion Protocol (MTP) if indicated
  - Transfusion of > 10 units of blood in 24 hrs/ > 4 units in 1hr
- Early resuscitation with blood & blood products in low ratios
  - Recommended in patients with evidence of class III/IV hemorrhage
- Patients with severe shock → resulting from trauma can develop coagulopathy → From blood loss/ dilution from large volume crystalloid fluid resuscitation /Hypothermia → resulting in further blood loss
- Thus → warm blood & blood products should be given immediately to stop the blood loss d/t coagulopathy

MASSIVE TRANSFUSION PROTOCOL (MTP)

00:13:15

- Profusely bleeding patient in class 3/class 4 shock with expected blood loss >30% blood volume may require massive blood transfusion
- MTP → should be activated by treating doctor & steps should be taken for early definitive control of on-going hemorrhage
- To ensure rapid & timely availability of blood & blood components → Blood bank is alerted
- Early administration of packed R.B.C, plasma, platelets & minimizing aggressive crystalloid administration → may result in improved survival.
- Large prospective study demonstrated → ↓ mortality →
  - When tranexamic acid is given within 3 hrs of injury/ trauma
- 1 gm dose is given in pre-hospital setting & repeat dose is administrated in emergency department.
- Early monitoring of coagulation & replacement of clotting factors minimize transfusion needs, who are taking anticoagulant medications.
- Thrombo-elastography, Rotational thrombo-elastometry→to pinpoint the precise coagulation deficiency
- Resuscitation with fluids & blood → not a substitute for control of continued blood loss
- Definitive management - to stop the cause of bleeding

Bleeding

- External
  - Best managed by direct pressure to the site/ suturing (scalp wounds)
  - Splinting of long bones fractures and use of pelvic binder in pelvic fracture is useful in reducing blood loss
- Internal
  - Identify it fast & bleeding has to be stopped immediately
  - Injury to thorax, abdomen, including retroperitoneum, pelvis & long bones
  - Definitive management is to stop the cause of bleeding.

12 SEPTIC SHOCK

Definition 00:00:16

- Sepsis is defined as life threatening organ dysfunction caused by a dysregulated host response to infection.
- Organ dysfunction can be identified as an acute change in total SOFA score ≥ 2 points consequent to the infection.
- SOFA: Sequential organ function Assessment score
  - Baseline SOFA Score can be assumed to be 0 in patients with no known organ dysfunction
  - A SOFA Score > 2 – high mortality
  - Increased mortality can be identified by quick SOFA alteration in mental status
- SBP < 100 mm Hg
- RR > 22/min
- Septic shock is a subset of sepsis in which underlying circulatory and cellular/metabolic abnormality profound enough to increased mortality
  - Paramount in the management of patients with sepsis is the concept
  - Septic shock is a medical emergency.
  - As with polytrauma, acute myocardial infarction and stroke,
  - Early identification and appropriate immediate management in the initial hours after development of sepsis improves outcome.

GUIDELINES 00:06:42

- Urgent assessment and treatment initial fluid resuscitation to prevent end-organ dysfunction and control the source of infection.
- Laboratory results
- Attaining more precise measurement of hemodynamic status.

2018: Bundle care

- Time zero or time of presentation is defined as the time of triage in the emergency department

Hour 1 Bundle

- Measure lactate level remeasure if initial lactate > 2 mmole
- Obtain blood cultures prior to administration of antibiotic
- Administer broad- spectrum antibiotics
- Begin rapid administration of 30ml/kg crystalloid for hypotension or lactate > 4 mmol/l
- Apply vasopressor if patient is in hypotension during or after fluid resuscitation, in order to maintain MBP > 65 mmHg (In septic shock, peripheral vasodilatation → systemic vascular resistance ↓ ses → preload
- To maintain perfusion → more crystalloid should be given
- Measure lactate level
  - Serum lactate-not a direct measure of tissue perfusion,
  - Serves as a surrogate, ↑ lactate tissue hypoxia, accelerated aerobic glycolysis driven by excess beta-adrenergic stimulation.
- If initial lactate is elevated (>2mmol/L) it should be remeasured within 2-4 hours to guide resuscitation to normalize lactate.
- Broad spectrum antibiotic should be started with one or more intravenous antimicrobials to cover all likely pathogens should be started immediately. It should be narrowed once pathogen identification and sensitivity are established.
- Discontinue if no pathogen is found

Choice of fluid

- Start with crystalloid
- No evidence of colloid being superior
- No evidence of blood products superior

Vasopressor

- Start with Noradrenaline
- Dopamine can be added if blood pressure is not increasing.
- No evidence of vasopressin being better than other vasopressor.

13 EMERGENCY MANAGEMENT IN ACUTE STROKE

DEFINITION

00:00:30

- It refers to acute neurologic impairment that follows interruption in blood supply to a specific area of the brain

Types of Stroke

- Ischemic stroke: MC (87%)
- Hemorrhagic stroke: 13%
  - Intracerebral: 10%
  - Subarachnoid: 3%

Goal

- To minimize brain injury
- To minimize patients recovery

8 "D" OF STROKE CASE

00:03:13

- Detection
- Dispatch
- Delivery
- Door
- Data
- Decision
- Drug / Device
- Disposition

NATIONAL INSTITUTE OF NEUROLOGICAL DYSFUNCTION & STROKE

- Immediate general assessment – 10 min
- Immediate neurological assessment – 25 min
- Acquisition of CT of the head – 25 min
- Interpretation of the CT scan – 45 min
- Administration of fibrinolytic therapy – 60 min

Important Information

The above mentioned time guidelines are when the patient entered in emergency department

- Administration of fibrinolytic therapy timed from onset of symptoms: 3 hrs/ 4.5 hrs
- Administration of Endovascular therapy timed from onset of symptoms: 6 hrs

Admission to a monitored bed: 3hrs

Signs & symptoms of stroke

- Sudden weakness on one side of the body
- Sudden confusion
- Trouble speaking or understanding / seeing / walking
- Dizziness or loss of balance
- Sudden severe headache

EMS should be activated

- Identifying possible stroke patients
- Providing high priority dispatch
- Instructing by standers in life saving CPR skills
- Pre arrival notification to hospital

CINCINNATI PREHOSPITAL STROKE SCALE

00:12:00

1. Facial droop
2. Arm drift
3. Abnormal speech

1. Facial Droop

- Ask patient show teeth or smile
- Normal: Both sides of face move equally
- Abnormal: One side of face does not move as well as the other

2. Arm drift

- Patient closes eye end extends both arm straight out, with palms up for 10 seconds
- Normal: Both arms move the same
- Abnormal: One arm does not move or one arm moves down compared with the other.

3. Abnormal speech

- Patient slurs words

Cincinnati prehospital stroke scale

- Sensitivity: 59%
- Specificity: 89%

Interpretation

- If any 1 of these 3 sign is present, then its abnormal

In emergency department

- Assess the ABC's & evaluate baseline vitals
- Provide oxygen & establish IV access
- Check glucose
- Perform Neurological assessment
- Activate stroke team
- Order CT brain
- Obtain 12-lead ECG

NEUROLOGIC ASSESSMENT

00:16:25

- Time is brain
- Goal – to do within 25 minutes
- National institute of health stroke scale uses 15 items to assess the responsiveness of stroke patients

Decision

- Hemorrhage
  - Not a candidate of fibrinolytics
  - Give aspirin

FIBRINOLYTIC THERAPY

00:17:30

- Excellent outcome
- Should be give before 3hrs/ 4.5 hrs

Indication

- Diagnosis of ischemic stroke
- Onset < 3 hrs
- Age ≥ 18

Contraindication

- Significant head trauma or prior stroke within 3 months
- Arterial puncture at non-compressive site within 3 months
- History of previous intracranial Hemorrhage
- Elevated blood pressure (SBP > 185, DBP > 110)
- Active internal bleeding
- Acute bleeding diasthesis
  - Platelet count < 1 lakh / mm^3
  - Heparin received within 48 hrs (prolonged aPTT)
  - Current anticoagulants INR > 1.7 or PT > 15 sec
- Blood glucose < 50 mg/dl
- CT multilobular infarction

Important Information

- Above contraindicated patients can undergo endovascular therapy

14 COMA

00:00:33

1. Approach in emergency department
2. Stabilizing in emergency department

- Altered state of consciousness
- Coma is a common reason for visits to emergency room.
- One of the most difficult condition to manage because complex presentation multiple potential etiologies
- So, a Systematic approach is needed to manage coma patients
- The phenomenon of consciousness requires two intact & interdependent physiological and anatomical components
- Arousal & its underlying neural substrate → depends on Ascending Reticular Activating System (ARAS) & Diencephalon
- Awareness: requires the functioning of Cerebral Cortex.
- Any problem in ARAS/ Diencephalon/cerebral cortex →results in Coma
- E.g.: drugs, trauma etc can affect these systems resulting in coma

DEFINITION OF COMA

00:03:30

- Coma is characterized by an uninterrupted loss of capacity for arousal.
- Eyes: Closed/open
- Sleep/wake cycles: Disappear
- On vigorous stimulation, only a reflex response is elicited instead of conscious response.
- Acute and potentially life-threatening emergency.
- Evaluation of a comatose patient demands a systemic approach for appropriate diagnostic & therapeutic endeavors.
- Urgent steps: To prevent/ minimize permanent brain damage from reversible cause.

ETIOLOGY

00:05:48

Refer Table 14.1

GLASGOW COMA SCALE

00:10:54

- Determines prognosis of Coma Patient
- Glasgow coma scale (GCS) is widely used as a standard method to describe neurological function in a quick & concise manner.

Behavior of the patient		Score
Eye opening	Spontaneously	4
	To speech	3
	To pain	2
	None	1
Verbal response	Oriented	5
	Confused	4
	Inappropriate	3
	Incomprehensible	2
	None	1
Motor response	Obeys commands	6
	Localizes to pain	5
	Withdraws from pain	4
	Flexion to pain (Decorticate)	3
	Extension to pain (Decerebrate)	2
	None	1
Minimum Score		3
Maximum Score		15

- If GCS is <9, airway should be secured with intubation & protected from Aspiration.

APPROACH TO COMA IN EMERGENCY DEPARTMENT

00:18:05

A – Airway

- Open by Head tilt chin lift / Jaw thrust
- If still closed, use oral or nasal airway
- If the airway is still closed, then use advanced airway
  - Supraglottic airway device/ endotracheal tube

B – Breathing

- Support 100% oxygen
- If oxygenation is still not maintained, give Bag & mask ventilation

C – Circulation

- Assessed by looking at pulse, capillary refill time, feeling temperature of the pt, B.P maintained by giving Ringer's lactate/ Normal saline
- Trauma patient, only Jaw thrust is done: head tilt, chin lift is never done as it may damage cervical spine

In coma, measures to be taken

- Both hyper & hypotension are deleterious but aggressive control of B.P is not required as it may effect brain perfusion.
- So, Mean Arterial Pressure (MAP) should be > 100 mmHg
- Hypertension -Treated only if MAP is >160mm Hg
- Hypotensive agents that do not increase ICP by their vasodilating effect like Esmolol are to be preferred.
- Targeted Urine output should be → 0.5 ml/kg/hr
- After ABC, brief neurological examination is mandatory to distinguish b/w true coma & malingering.
- Hand drop from the head → if pt tries to control his/her hand, then the pt may be Malingering
- Papillary size and its response to light
- Abnormal eye movement
- Grimacing / withdrawal from noxious stimuli
- Plantar response

MANAGEMENT

00:25:10

Initial Management

- Hypoglycemia is most imp reversible cause of coma.
- So, Quick blood sugar is done
- Dextrose -to treat hypoglycemia
- Naloxone - for opioid toxicity
- Coma Cocktail: When we get a patient in coma, initial management after stabilizing ABC is to give coma cocktail in which Glucose, Thiamine & Naloxone is given
- Supportive care and sedation are given for agitated withdrawal states.
- Intravenous Fluids for
  - Dehydration
  - Hypovolemia,
  - Hypotension
  - Hyperosmolar states such as HHNS (or) Hypernatremia
- Empirical antibiotics are given for suspected
  - Meningitis urosepsis,
  - Pneumonia
- Rewarming or Aggressive cooling is done for extreme temperature
- Antidotes for specific toxins -Fomepizole, pyridoxine, digoxin-fab fragments
- Controlled reduction Of Blood Pressure
  - Nitroprusside
  - Labetalol (or)
  - Fenoldopam

  } for Hypertensive Encephalopathy.
- For profound Hyponatremia with seizures (or) Altered Mental Status-Hypertonic Saline
- For metastatic CNS lesions with vasogenic edema: Glucocorticoids
- For Wernicke's encephalopathy: Thiamine

DISPOSITION

00:31:24

Decision to admit the patient to an ICU setting / to hospital ward is based on

- Hemodynamic stability,
- Etiology of the Altered Mental Status,
- Expected course,
- Need for close monitoring,
- Airway management issues
- Institutional resources.

Table 14.1

Structural Brain Lesions	Metabolic & Systemic Disorders	Infection/ Inflammatory Pathology	Drugs & Toxins	Others:
• Any space occupying lesion/injury/trauma in the brain → can lead to coma		• Any infection/ inflammation of brain → lead to coma		
• SAH, ICH, • Ischemic infarction, • Global cerebral hypoperfusion, • Cerebral venous sinus thrombosis, • Traumatic brain injury, • Hydrocephalus, • Basilar artery occlusion, • Central pontine myelinolysis, • Large hemisphere masses, • Pituitary Apoplexy, • Cerebral abscess.	• Anoxia/hypoxia (most imp), • Hypercapnia (asthma / CO_2 narcosis) • Hypotension, • Hypoglycemia, • Hyperglycemia, • Diabetic ketoacidosis, • Hyper / Hyponatremia, • Hyper/ Hypocalcemia, • Hypo/ Hyperthermia, • Wernicke's Encephalopathy, • Hepatic Failure, -Uremia.	• Bacterial, Viral or Fungal meningitis/ • Meningoencephalitis, • Acute disseminated encephalomyelitis, • Syphilis, • Sepsis, • Malaria, -Waterhouse • Friderichsen syndrome, • Typhoid Fever, • Systemic infections	• Opioid, • Alcohol, • Sedative agents, • Carbon monoxide poisoning, • Arsenic poisoning	• Psychogenic Coma, • Heat stroke d/t • Hypovolemia • Hypoperfusion

15 HEADACHE- APPROACH IN EMERGENCY DEPARTMENT

HEADACHE

00:01:10

- Only symptom present
- Or a part of constellation of symptoms

ETIOLOGY

00:01:40

Non emergent etiology	Emergent Etiology
• Tension	• SAH
• Migraine headache	• Epidural Hemorrhage
• Fever associated headache	• Subdural Hemorrhage
• Sinusitis	• Intracranial Hemorrhage
• TMJ disease	• Stroke (although ischemic stroke uncommonly presents with headache)
• Cluster Headache	
• Trigeminal Neurologic	

- CNS infection (Meningitis / Encephalitis / Abscess)
- CNS mass / increased ICP
- Pseudotumor cerebrii (Idiopathic intracranial hypertension)
- Venous thrombosis
- CO poisoning
- Acute angle closure glaucoma
- Temporal Arteritis

APPROACH TO THE PATIENT

00:04:55

- ABC: 1st priority
- Majority will not require immediate intervention

Primary survey

- Brief assessment of gross neurological deficit & assessment of mental status
- Headache+altered mental status-immediate intervention
- Signs & symptoms of CNS infection
- Headache following Trauma – Full trauma assessment with cervical spine immobilization

HISTORY

00:08:51

- Help in establishing diagnosis
- Onset – sudden or gradual
- Thunderclap headache
- Associated symptoms
- Aggravating or alleviating factors
- Prior history of headache
- Inquire about treatments attempted
- Inquire if others in the home have similar symptoms

PHYSICAL EXAMINATION

00:14:50

- Neurological exam
- Includes testing of motor & sensory function, cranial nerves, reflexes, pronator drift, rapid alternating movements, finger to nose & heel to skin testing, Romerg's Test, Gait assessment, MMSE.
- Pupillary & Fundoscopic exam
  - Acute angle closure glaucoma – minimally reactive mid dilated pupils with ciliary flush
  - Papilledema or loss of spontaneous venous pulsations – increased ICP
- Tenderness in the temporal arm – Temporal arteritis

DISPOSITION

- According to the suspected radiology

16 INTRACEREBRAL HEMORRHAGE

ICH

- 10% -15% of all strokes
- It is rupture of blood vessel into the brain parenchyma

Presentation

- Headache
- Altered level of consciousness
- Focal neurologic symptoms

ETIOLOGY

00:01:48

- Hypertension - M/C
- Trauma
- Cerebral amyloid angiopathy
- Vascular malformation
- ICH- more commonly seen in older patients
- Long standing hypertension will damage the perforating arteries deep within brain structures (Thalamus, basal ganglia & Pons)
- ICH can also happen deep in ventricles
- m/c location For ICH are the basal ganglia, thalamus, pons & cerebellum
- ICH doesn't have specific features
- So it can be differentiated from ischemic stroke and other neurologic disorder by neuroimaging
- ICH at least must be considered in any case of sudden onset of acute neurologic symptoms

INITIAL SURVEY & PRIMARY MANAGEMENT

00:05:18

- **ICH**
  - present with coma / seizure
  - In such cases priority is ABCD's.
- Secure the airway
  - IF there are concerns about oxygenation, ventilation, airway protection, prolonged seizure or rapidly deteriorating clinical status
  - A Neuroprotective rapid sequence intubation protocol is preferred
- Brief assessment of Breathing, Circulation, Disability

Documentation

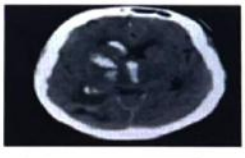
- Documentation of a pre intubation Neurological exam
  - Helpful in determining prognosis
- Documentation include:
  I. Glasgow coma score;
  II. Pupillary size 4 reactivity
  III. Motor strength in the 4 limbs
  IV. Sensation & reflexes can be included (if time permits)
- Check a finger stick glucose before intubation
- **Cushing's triad**
  - It describes the physiologic response to rapidly increasing ICP a imminent brain herniation
  - Its feature include
    → Hypertension
    → Bradycardia
    → Abnormal respiratory patients
  - Other sign of imminent herniation are:
    → Lack of Pupillary reaction &/or
    → Pupillary asymmetry
- Sign of imminent herniation necessitate emergent intervention

DIAGNOSIS

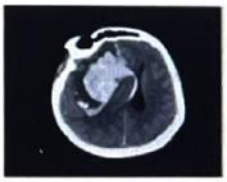
00:12:25

- Head CT: Mainstay of diagnosis in ICH
- On CT
  - Acute bleeding- hyperdense (whiter)
  - Sub acute phase (b/w days 3 & 14) isodense
  - After about 2 weeks – hypodense (darker)
- On sub- acute phase- easy to miss intracranial bleeding as the blood and the brain may appear the same shade
- Radiological Signs
  - Midline shift
  - Ipsilateral compression of the ventricle (with or without contralateral ventricular enlargement)
  - Obliteration of the sulci & gyri
  - Blurring of the grey & white junction

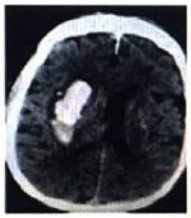
- Suspected SAH with negative CT
  - Lumbar puncture is often the second diagnostic step



Mid line shift



Huge hemorrhage (obliterate the ventricle)



Huge hemorrhage (present in the ventricle)

- Two CSF features are most important
  - Absence or clearing of blood
  - Xanthochromia

MANAGEMENT

00:15:10

- Assess and reassess the ABCD's
- Discontinue / reverse anticoagulation
- Prevent hypotension & hypoxemia
- Control ICP
- Prevent Seizure-prophylaxis may be necessary depends type extent of bleeding
- Treat fever & infection aggressively
- Control blood glucose (target 140-1 85 mg/gL)

ICP control can be managed by

- Monitoring / lowering blood pressure
- Elevating the head of the bed to 30°
- Providing adequate sedation & Analgesia
- Considering mannitol or mild hyper ventilation
- Ifs Sign or rapidly rising ICP or herniation (target CO_2 around 30 mmHg)
- Require close observation in ICU or Neurosurgical Ward

17 ANEURYSMAL SUBARACHNOID HEMORRHAGE

SUB ARACHNOID HEMORRHAGE

- Sub arachnoid hemorrhage – bleeding into the sub arachnoid space surrounding brain & spinal cord.
- M/C etiology: Aneurysmal rupture (10-20% cryptogenic)
- It has significant mortality (20-30% within 30 days) & persistent Neurologic morbidity

RISK FACTORS

00:02:12

- Hypertension
- Cigarette smoking
- Alcohol
- History of Aneurysmal/ SAH in first degree relative

CLINICAL PRESENTATION

- Sudden severe headache (that reach maximum intensity within seconds i.e., Thunder clap headache)
- Syncope and persistent alteration in the level of consciousness
- Meningismus

DIAGNOSIS

- NCCT: 95% sensitivity of done within 24 hrs
- Lumbar puncture: Xanthochromia (it is done if the NCCT is negative)

MANAGEMENT

00:04:20

A - Airway protection
B - Breathing
C - Circulation

- Both profound hypotension & hypertension can worsen secondary injury
- So physician should avoid any other medication which would further cause hypotension
- To date, there is no clear blood pressure goal
- It is often determined by premorbid blood pressure
- American stroke association's 2012 guidelines recommend a systolic blood pressure less than 160 mm Hg
- However, over aggressive lowering of BP can lead to under perfusion & resultant ischemia
- Therefore, close monitoring is necessary (with neurospecialists)
- Placement of an arterial line can help drive real time management of BP

DISABILITY

- Neurological assessment done
- GCS/ AVPU scoring
  - C - Institute anticonvulsant prophylaxis, laxative, pain & anxiety Control
  - D - Order cerebral angiography
  - E - External ventricular drain (if patient's ICP is ↑ ed)

EXPOSURE

- Fast assessment for any injury

RISK ASSESSMENT

00:11:10

WFNS grading system

WFNS grades	GCS score	Motor deficit
I	15	Absent
II	14-13	Absent
III	14-13	Present
IV	12-7	Present / Absent
V	6-3	Present / absent

- If the WFNS grade increases chances of mortality in the patient increase

Hunt and Hess classification

Grade	Criteria	Index of perioperative mortality (%)
0	Aneurysm is not ruptured	0-5
I	Asymptomatic or with minimal headache and slight nuchal rigidity	0-5

II	Moderate to severe headache, nuchal rigidity, but no neurologic deficit other than cranial nerve palsy	2-10
III	Somnolence, confusion, medium focal deficits	10-15
IV	Stupor, hemiparesis medium or severe, possible early decerebrate rigidity, vegetative disturbances	60-70
V	Deep coma, decerebrate rigidity, moribund appearance	70-100

DEFINITIVE MANAGEMENT

00:15:50

- Clipping/ coiling of the lesion (best at high volume tertiary care centers)
- Thus, early identification of SAH by ED physician should prompt inter hospital transfer (for better management)
- Number of studies showed, that if the clipping of aneurysm is done within < 4 hrs of onset of symptom → Chances of survival is maximum
- Patients with delayed presentation (days) → Early treatment after consultation with Neurospecialists
- Nimodipine modulate vascular flow (by preventing the influx of calcium into smooth muscle cell) and is associated with improved outcomes
- Routine use of Nimodipine currently is recommended with the level 1A evidence by the American stroke association
- Regimen include either oral or NG dosing
- Seizures are seen in one-fourth of patients presenting with SAH
- Routine use of anti epileptic medication has questionable benefit
- Neither phenytoin nor levetiracetam has been shown to reduce seizures significantly in SAH patients when administered prophylactically
- Patients who seize while in the ED should be managed acutely as any patient with seizure
- Ensure the ABCs are maintained, start with administration of intravenous benzodiazepine and consider loading with an anti epileptic in consultation with Neurosurgeon or intensivists

RISK OF SAH

- Re-bleeding
- Ventricular arrhythmia

18 MENINGITIS & ENCEPHALITIS

DEFINITION

00:00:33

- Meningitis
  - Meningitis is an inflammatory process of the membranes that surround the brain & spinal cord
- Encephalitis
  - It is an infection of the brain parenchyma causing inflammation within the CNS & is often viral in origin
  - Most treatable cause – HSV encephalitis

> **Important Information**
>
> - It can be difficult to distinguish encephalitis from severe cases of bacterial meningitis since both have similar signs & symptoms

INITIAL EVALUATION & PRIMARY SURVEY

00:01:33

- ILL appearing patient with signs of infection, decreased level of consciousness should be promptly evaluated & critical case interventions should be completed immediately

Management

- Stabilization of ABC
- Broad spectrum antibiotics should be started after prompt lumbar puncture or blood cultures drawn
- Encephalitis
  - CNS infection + altered mental status or a focal neurologic deficit
  - Antiviral therapy is recommended

CLINICAL FEATURES

00:06:00

- Classic triad: Fever, neck stiffness & altered mental status
- Non specific early symptoms
- As the disease progress, patient experience altered mental status or seizures
- Patients at the extremes of age & immuno-compromised presents with vague symptoms
- In infant – present with irritability, lethargy, poor feeding, rash or bulging fontanelle
- Seizures – major presenting symptoms in pediatric patients
- Altered mental status – major presenting symptom in adults
- Encephalitis – classic triad + altered mental status or neurologic symptoms
- Petechial & purpura – Meningococcal meningitis

Clinical finding

- Nuchal rigidity
- Kernig's sign
- Brudzinski's sign

LUMBAR PUNCTURE

00:12:17

- Preferred diagnostic procedure
- CT scan should be considered before LP in following conditions
  - Altered mental status
  - New onset seizures
  - Immuno-compromised state
  - Focal neurologic signs or papilledema
- 4 tubes of CSF with each containing 1 ml
  - 1st tube: Cell count & differential count
  - 2nd tube: Protein & glucose levels
  - 3rd tube: Gram's stain
  - 4th tube: Bacterial culture
- Other CSF studies
  - HSV or enterovirus PCR
  - Bacterial antigen testing
  - Specialized fungal testing
- Complete routine lab investigations including blood culture should be done even though they are non-specific

TREATMENT

00:17:55

- Suspected bacterial meningitis – empiric IV antibiotic therapy & hospital admission
- Severe disease – require ICV case
- Viral encephalitis – supportive case (mostly)
- HSV encephalitis – intravenous acyclovir
- Adjunctive corticosteroid before or concurrently with the 1st dose mat decrease mortality & neurologic sequelae
  - IV dexamethasone every 6 hrs for 4 days in adults & children 3 months & older

DISPOSITION

00:21:05

- Admission & Empiric IV antibiotics & further evaluation
- Disposition depends on the condition of patient

19 SEIZURE / STATUS EPILEPTICUS

00:00:16

- Seizure result from abnormal, excessive activity of the CNS

Generalized	Focal (Partial)
• Involves both hemispheres of the brain with loss of consciousness • Simple Partial Seizures • Cognition is not impaired	• Involves only one hemisphere • Complex Partial Seizures • Cognition is impaired
• Partial Seizure with Secondary Generalization • Focal seizures may generalize to involve both cerebral hemispheres	

Presentation 00:02:40

- Brought to the ED after a witnessed seizure
- Most seizures lasts for 1 – 2 minutes, but duration varies
- Postictal State - When the patient awakens, witnesses or EMS will report that they were confused for several minutes before they returned to baseline
- Seizure may also be unwitnessed presenting to ED stating that they have been waking up confused or on the floor
- Rarely, patients who have had seizures will present to the ED having been "found down" without a clear cause of loss of consciousness

Physical examination 00:05:10

- Post-ictal confusion that resolves while in the ED
- Tongue trauma from biting
- Urinary/Bowel incontinence
- Minor head trauma may be present but does not help to distinguish between seizures and other etiologies
- A focal neurologic deficit mimicking a stroke, referred to as Todds paralysis,
- O/E
  - Tachycardia
  - Diaphoresis
  - Anxiety
- These findings may suggest alcohol withdrawal, drug use, or hypoglycemia as possible causes of seizure.

PRIMARY SEIZURE 00:07:40

- Medical noncompliance (most common cause of recurrent seizure)
- Sleep deprivation
- Emotional or physical stress

Etiologies of secondary, or reactive seizures 00:08:44

- Hypoglycemia
- Hyponatremia
- Alcohol withdrawal
- Trauma
- Drugs/ toxins
- Tumor
- Infection (Eg- Meningitis, encephalitis, CNS abscess)
- Eclampsia
- Diagnosis that mimic seizure
- Pseudoseizure
- Syncope

Lab investigations

- For new onset, first- time seizure, the only lab values routinely recommended are chemistry panel (for sodium and glucose) and a pregnancy test
- In Recurrent Seizures
  - Medications levels- checked rapidly in the emergency department (phenytoin, carbamazepine, phenobarbital)
  - Further testing- guided by the history and physical exam but can include urine analysis and pregnancy test.
- In Status Epilepticus
  - Complete laboratory profile including LP (Lumbar puncture) to identify underlying causes
  - CT scan

Indication in patient with recurrent seizures

- Change in their seizure pattern
- New type of seizure
- Increased frequency of seizures
- Significant trauma,
- Fever
- Prolonged postictal time
- New neurological deficit
- Concerning symptoms
- All patients in status epilepticus should undergo Head CT once stabilized.

- Lumbar puncture (LP
- Indications
- Patient with status epilepticus
- Severe headache
- Fever
- Persistent altered mental status
- Immunocompromised state (HIV)
- Head CT scan to be done before LP to rule out an intracranial lesion that may cause herniation during LP

ALCOHOL WITHDRAWAL

00:14:04

- Delirium Tremens
- Anxiety
- Tremulousness
- Altered mental status.
- Abnormal vital signs
- Tachycardia,
- Hypertension,
- Hyperthermia
- Tachypnea.
- Predominantly a clinical diagnosis

ECLAMPSIA

00:15:07

- Life threatening disorder
- Must be treated immediately
- All female patients presenting with seizure must be assessed for pregnancy.
- Pregnant women with an underlying seizure disorder may experience
- ↑ seizure frequency during pregnancy, therefore, must be suspected of having eclampsia

Diagnosis

- Clues include
- Vision complaints
- Edema of the face, hands and feet
- Proteinuria on urine analysis
- Hypertension

Drug History

- Many drugs have the potential to lower the seizure threshold,
- But several drugs cause seizures more commonly.
- For TB treated patients, suspect isoniazid-induced seizures.
- Patients with a history of depression may have overdose of tricyclic antidepressants
- On ECG
  - Widened QRS
  - Prominent terminal R wave in aVR

Intracranial hemorrhage

- Intracranial hemorrhage or brain injury should be suspected in patients with any signs or symptoms of head injury.

PSEUDOSEIZURE

00:17:58

- Pseudoseizure is a difficult diagnosis to make.
- 25% of patients initially thought to have Pseudoseizure are eventually diagnosed with a true seizure disorder.

Diagnosis

- Clues include
  - A rhythmic, controlled shaking activity,
  - Ability to talk or follow commands during the seizure,
  - Recall of a seizure that involves both sides of the body, or
  - Lack of a postictal period.
  - EEG monitoring is helpful in assessing for Pseudoseizure

STATUS EPILEPTICUS

00:19:54

- In Status epilepticus there is no return to baseline between seizures

Diagnosis

- Non-convulsive seizures, are a difficult diagnosis to make without immediate EEG monitoring.
- Physicians must have a low threshold to treat patients aggressively even if they cannot confirm the diagnosis immediately.

Treatment

General treatment principles

- ABC's
- Supplemental oxygen
- Scene safety
  - Keep the patient safe from falling or other injuries
  - Remove restrictive clothing
  - Do not place anything in the patient's mouth except a bite block or oropharyngeal airway to protect the tongue

First line	Second line	Third line
• Benzodiazepines • usually lorazepam	• Fosphenytoin • Phenobarbital • Valproic acid	• Pentobarbital • Propofol infusions

- Preferred route: Intravenous administration
- If I.V line can't be obtained, then

- Lorazepam (2mg)
- Midazolam (2mg)
- Diazepam (5mg)

} I.m. route

- Rectal diazepam also available.
- C/I: Oral administration of medications untill mental status is normal

DOSE

00:25:29

- Lorazepam/midazolam: 2mg PO/IM/IV every 2-5 min as needed
- Diazepam: 5mg PO/IM/IV every 2-5 min as needed (also available PR)
- Phenytoin: 15-20 mg/kg PO/IV
- Fosphenytoin: 15-20 phenytoin equivalents/kg IV
- Phenobarbital: 20 mg/kg IV (use single dose of 60-120 mg PO for oral load)
- Valproic acid: 15-45 mg/kg IV

Specific treatment

Etiology	Treatment
• Eclampsia	• Magnesium Sulfate
• Hyponatremia	• Hypertonic Saline
• Isoniazid	• Pyroxidine
• Hypoglycaemia	• Dextrose

- Intubation - to control the airway
- Outpatient follow-up, usually with a neurologist.
- First-time seizure patients require further work-up for seizure
  - MRI
  - EEG.
- Warned to avoid engaging in activities where they are at risk of another seizure
  - Swimming or bathing alone,
  - Cooking with open fire,
  - Driving until they have been cleared to return to these activities.
- Many states require physician to report any patient with a diagnosis of seizure and have restrictions on driving privileges.

20 ACUTE RESPIRATORY DISTRESS SYNDROME

00:00:20

- Cardiogenic Pulmonary Edema → ↑ left atrial pressure secondary to LVF → edematous fluid is Transudate
- Acute Respiratory Distress syndrome is due to Non-Cardiogenic Pulmonary Edema
- E.g.: ARDS occurs secondary to swine flu/bird flu. Because swine flu virus ultimately damages Pneumocytes. That will end up in ARDS.

↓

Collapse of alveoli

↓

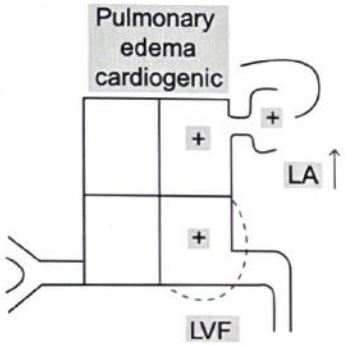
Hypoxia

↓

Damage of Endothelium (damage to gap junctions)

↓

Leakage of fluids from pulmonary capillary Into alveoli



ARDS occurs in 10% of ICU patients

- Sudden onset Respiratory distress
  - CXR B/L infiltrates (no cardiac cause found)
  - ↓PO_2 (demonstrable hypoxia)
  - Normal left atrial pressure (LAP)

TRIGGERS FOR ARDS

00:05:20

Direct (M/C)	Indirect
• Pneumonia (H1N1) • Mendelson syndrome • Toxic gas inhalation • Pulmonary contusion • Near drowning	• Sepsis • Trauma (multiple bone fracture) • Flail chest • Head injury • Burns • Multiple blood transfusion /TRALI • Acute pancreatitis • Post cardio pulmonary bypass

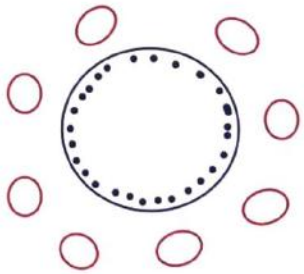
- Mendelson syndrome: Aspiration of stomach acid (Chemical Pneumonitis)

Important Information

- Both pneumonia > sepsis are the leading causes of ARDS
- Leading cause of death after blood transfusion – TRALI
- All of the followings are the causes of ARDS except:
  - Status asthmaticus (disease of airway)
  - There would be no CXR findings in fat embolism

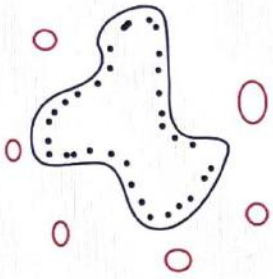
Clinical Presentation

- Exudative Phase (0-7 days)
- Triggers ⟶ Symptoms begin
  In 12-36 hrs
- **Type 1 pneumocytes:** Covers 90% of surface area - (injured during adult ARDS)
- **Type 2 pneumocytes:** Produce surfactant (most abundant cell of alveoli)
- **In ARDS**
  - Type 1 pneumocytes are affected
  - Type 2 pneumocytes are not affected



- Normal surfactant with reduced surface area of alveoli for gas exchange
  - Ventilation and perfusion imbalance (perfusion is secondary to hypoxia component)
- (Hypoxia causes dilatation of all blood vessels in body except vessels of pulmonary circuit)
- Most vulnerable cells/most damaged cells in ARDS – endothelial cells of alveoli - d/t Hypoxia

- ↓ Endothelial cells become Leaky Resulting in
- Exudative, high protein pulmonary edema
- ↓ forming a layer resulting in
- Further ↓ of gas exchange
- In heart failure → low protein pulmonary edema



FEATURES: (SUDDEN ONSET RESPIRATORY DISTRESS)

00:19:59

Exudative phase	Proliferative phase	Fibrotic phase of lungs
(0-7 days) • Triggers 12-36 hrs	(7-21 days) • Able to wean off the ventilator • Proliferation of type 2 pneumocytes • Some differentiate into type- 1 pneumocytes [recovery is possible]	(>21 days) • Require supplemental oxygen for rest of their life [requirement varies from case to case] • It results in pulmonary artery hypertension

Intrapulmonary Shunting
- Blood is getting wasted in the lungs→ d/t alveolar collapse/endothelial injury
- There is sudden onset of distress,

- ↑ work of breathing: ↓ CO_2
- ↓ PO_2, ↑ PCO_2 (Refractory hypoxia)
- Dead space ↑ acidosis

} Type 2 Respiratory failure / Respiratory

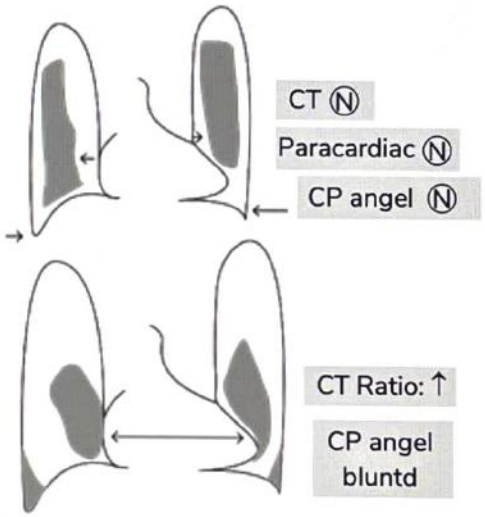
- **In initial phase of ARDS**: There will be Respiratory alkalosis (CO_2 washout)
- **In acute asthma**: type-1 respiratory failure & respiratory alkalosis → d/t CO_2 washout
- **In status asthamaticus**: Type-2 respiratory failure & Respiratory acidosis d/t CO_2 overproduction in lungs
- When PcO2 = 60mm of Hg, then the compensatory mechanisms begin to fail.

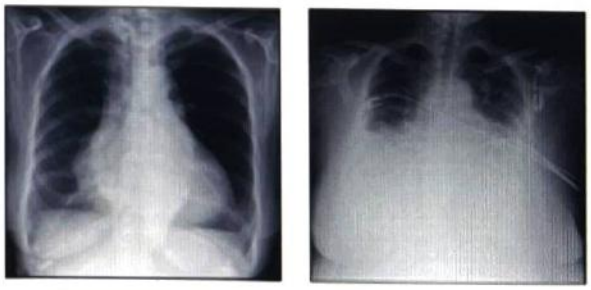
Important Information

- Bulla/blebs along with fibrosis and (PAH) pulmonary artery hypertension are seen in ARDS.

Work-up
- CXR – B/L extensive infiltrates
- Cardiothoracic ratio – normal
- Para cardiac area sparing
- CP angle – normal

In cardiogenic pulmonary edema
- Cardio thoracic ratio – increased
- Bat wing edema
- CP angle blunted





CP angle Ⓝ
CT Ratio ↑
Echo = EF = 65%
LA Ⓝ

CP angle blunting
Echo = EF
LA pressure ⊕

- Echo- ejection fraction is normal, LA pressure – normal ARDS CPE
- In cardiogenic pulmonary edema, echo shows
- Decrease ejection fraction

LA pressure increased

- ABG - ↓ PO_2, ↑ PCO_2 [ventilation, perfusion imbalance]

KEY WORDS: ESSENTIALS OF DIAGNOSIS

00:38:59

1. Sudden onset resp. distress

After multiple Bone fractures/ Massive blood transfusion ↓ ARDS	central line insertion ↓ Pneumothorax	S. Aureus pneumonia ↓ receiving i.v Vancomycin for 2 day ↓ Pneumatocele rupture resulting in Pneumothorax

2. CXR: B/L pulmonary infiltrates
3. $PaO_2/FiO_2 < 300$: Most imp diagnostic criteria for ARDS
4. Absence of LA Hypertension

- Above mentioned keywords with case of H1N1 / multiple blood transfusion / acute pancreatitis – confirm diagnosis of ARDS.

Grading

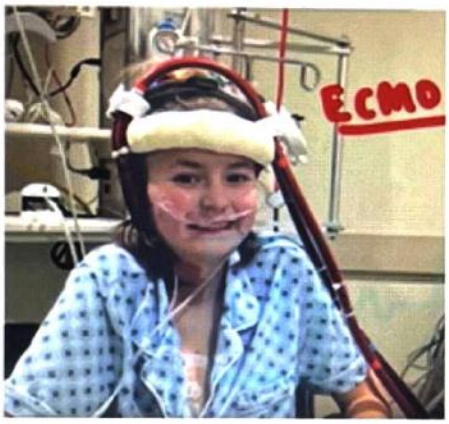
ARDS	PaO_2 / FiO_2
Mild	<300
Moderate	<200
Severe	<100

- Berlin Criteria: 1+2+3+4

RX OF VOLUTRAUMA

00:42:14

- Seen in high/normal volume ventilation resulting in Pneumothorax
- Low volume ventilation = 6 ml/kg → to minimize Barotrauma to chest. (Normal tidal volume = 12 ml/kg - Maintained in diaphragmatic paralysis Etc)
- Plateau pressure of ventilator = <30 cm H_2O
- Respiratory rate of ventilator = <35/min
- Prone position ventilation
- Risk of extubation, CVP line lost, orthopedic injuries
- Extracorporeal membrane oxygenation (ECMO): Pt. In which heart and lung are too weak for ventilation
- Limited fluids & diuretics – To maintain normal LA pressure
- Ensure neuromuscular paralysis: Cisatracurium (for effective ventilation)
- Steroids, surfactant therapy & high frequency jet ventilation (HFJV) have no role in treatment
- M/c cause of death in ARDS: Sepsis (non-pulmonary causes)



Congestive heart failure	ARDS
• Transudative pulmonary edema	• Exudative pulmonary edema
• Hydrostatic pressure increases	• Hydrostatic pressure normal
• LA pressure increased	• LA pressure normal
• Low protein pulmonary edema	• High protein pulmonary edema
• BNP increased	• BNP normal

21 CHRONIC OBSTRUCTIVE LUNG DISEASE

COPD IS[illegible]ON IN INDIA 00:00:16

- **Clinical presentation**
  - Wheezing
  - Productive cough
  - Dyspnea on exertion
  - Hypoxia
  - Tachycardia
- **History**
  - Increase use in routine inhalers
  - Change in sputum (color & amount)
  - Dyspnea when sleeping in supine position
  - Differential diagnosis is to be considered
  - History and atypical physical examination is important
  - Definitive diagnosis is not possible in emergency department

TYPICAL SYMPTOMS OF COPD 00:02:35

- Cough
- Wheezing
- Chest congestion
- Fatigue
- Sputum change in color or quantity
- Fever / chills
- Spectrum of symptoms may vary from mild to severe
- Management depends on the severity of the symptoms
  - Moderate: Bronchodilators
  - Severe: Intubation

CAUSES OF ACUTE DECOMPENSATION OF COPD PATIENT 00:04:02

- Superimposed infection
- Continued smoking
- Non-compliance to routine drugs
- Lack of usual medication or oxygen therapy
- Spontaneous pneumothorax

PHYSICAL EXAMINATION (DETERMINES THE SEVERITY) 00:05:37

- Work of breathing
- Look for use of accessory muscles
- Look for retractions and respiratory rate
- Look for pursed lip breathing and cyanosis
- Severely affected patients may have finger clubbing

On Auscultation

- Variety of lung sounds like wheezing, rhonchi or even rales
- Severe bronchospasm has absent breath sound bilaterally
- Inadequate oxygenation (hypoxia and hypercapnia) causes impaired mental status (depressed GCS)

INVESTIGATIONS 00:08:15

- Investigations are also guided by physical findings on patient
- Patients with long standing COPD may also develops CHF, right heart failure
- One examination, other conditions like elevated JVP, peripheral edema, hepatomegaly may be seen which shows the severity

MANAGEMENT 00:09:07

- Begin with assessing airway, breathing and circulation
- Obtain vital signs including oximetry reading
- Start with oxygen therapy where initially low flow devices are used like venture mask or nasal cannula
- Low flow devices are used initially because in COPD, respiratory drives are dependent on hypercapnia state.
- Central respiratory drive can be obtunded if high flow devices are used initially. However, it is used if the inspiratory flow of patient is very low and patient is not responding to flow device.
- Management and reassessment is important
- Non-invasive ventilation with PPV is done first, but if the patient is deteriorating, then invasive ventilation is done
- A quick decisive therapy based on initial clinical assessment will yield the best outcome on patient

Diagnosis

- Confirmatory is spirometry (pulmonary function test) which is not possible in acute exacerbated state of COPD in emergency department
- Based on clinical symptoms and physical examination, a probable case of exacerbated COPD is diagnosed
- Generally cases are older patients with history of smoking, multiple co morbidities with the possibility of broad differential diagnosis. So, multiple investigations are needed to rule out other possible conditions.

DIFFERENTIAL DIAGNOSIS

00:14:25

- CHF
- Acute coronary syndrome
- Pulmonary embolus
- Pneumothorax
- Pericardial effusion
- Pneumonia

Investigations that can be done to rule out these conditions

- Chest x- ray
- Electrocardiogram
- BNP
- ABG
- Cardiac markers

Chest x-ray (m/c study in evaluating COPD patient)

Typical findings

- Increased AP diameter
- Flattening of the diaphragm
- Decreased lung markings
- Other abnormality like pneumothorax, pulmonary edema or infiltrate can also be rule out
- Significant abnormalities such as pneumonia, pulmonary edema or pneumothorax will require change in therapy
- ECG in significant but can help in ruling out other causes which has similar presentation like STEMI, ACS
- Typical findings in ECG of COPD patient is multifocal atrial tachycardia

Common ECG findings

- Low voltage, right axis deviation and right rightward axis deviation
- P pulmonale – peaked P waves in II, III, aVF
- Right atrial hypertrophy
- Tachycardia
- Multifocal atrial tachycardia (rare, but specific to COPD)
- Obtaining ABG is very important and essential because it helps in evaluating the degree of hypoxia, acidosis & hypercapnia which in turn helps in deciding the therapy
- Serial ABG may be required and management may vary accordingly

Rx

00:19:27

- **Bronchodilators**
  - First line therapy with SABA like albuterol which is preferred beta-agonist whicyh provide rapid response
  - After nebulization of albuterol with multiple doses, eventually clinical response is observed
- **Side effects**
  - In older patient, it produces tachycardia which can result in cardiac ischemia
  - Use of anti-cholinergic bronchodilators such as Ipratropium bromide can also be used as a first line management. However, it is not given continuously but it is given every 4 hourly.
  - Use of both beta agonist and Ipratropium bromide together are also seen but studies have not found any benefit of combining the two drugs.

STEROIDS

00:22:32

- Steroids are very good in acute exacerbation of COPD. It is started with IV methyl prednisolone for 10-14 days and tapered
- Corticosteroids have shown to reduce treatment failures

Side effects

- Impaired blood sugar control
- Gastritis
- Hypertension getting worse
- Steroid psychosis

Antibiotics

- Empiric antibiotics are started because infections are one of most important causes for exacerbation of COPD
- Despite of all the medications, if the condition of the patient is deteriorating into respiratory decompensation with more CO_2 retention, then the next Mx is NPPV (endotracheal intubation)
- No guideline as to start ventilation at a specific PCO_2. It is based on exacerbation of clinical symptoms and response of patient to drug management
- Next step in therapy for patient not responding to standard treatment is NPPV.
- General ventilator guidelines include
  - Correcting acidosis
  - Correcting hypoxia
  - Avoiding high peak and plateau airway pressure by ongoing use of bronchodilators and aggressive suctioning of secretions
- Along with NPVV medical management is also continued

DISPOSITION

00:27:30

- Based on assessment of work of breathing, pulse oximetry and social support
- Patient who desaturate significantly or too dyspneic to complete ambulation cannot be discharged
- Patients with mild to moderate exacerbation of COPD after initial stabilization in ED are disposed towards for further management.
- Patients with severe exacerbation of COPD who required NPVV or endotracheal intubation are disposed to ICU for further management

22 THORACIC AORTIC DISSECTION

00:00:16

- Approach in emergency department
- Any patient coming to ED with chest pain → apart from Acute Myocardial infarction, there are few emergency conditions [high mortality] which we need to assess to manage & dispose the pts appropriately
- One of the conditions among those is Thoracic Aortic Dissection.
- Thoracic aortic dissection should be considered for every patient presenting to emergency department with chest pain, particularly if accompanied by neurologic signs or symptoms
- This condition is uncommon, difficult to diagnose & is associated with serious, often lethal complications.
- If prompt diagnosis is made, emergent surgical consultation should be taken → only then there is possibility of favorable outcome. So, time is crucial to manage this condition.

Epidemiology

- Half of patients with Type A dissection die within 24-48 hours without appropriate treatment.
- Even with Surgical management – mortality is high [may exceed 25%]
- Overall, in-hospital mortality rates for treated patients in a large multicenter series – 33% for type A dissection and 13% for type B dissection
- Incidence: 3 in 1 lac persons/ year
- 2-3 fold more common in males > females
- Prevalence ↑ with age

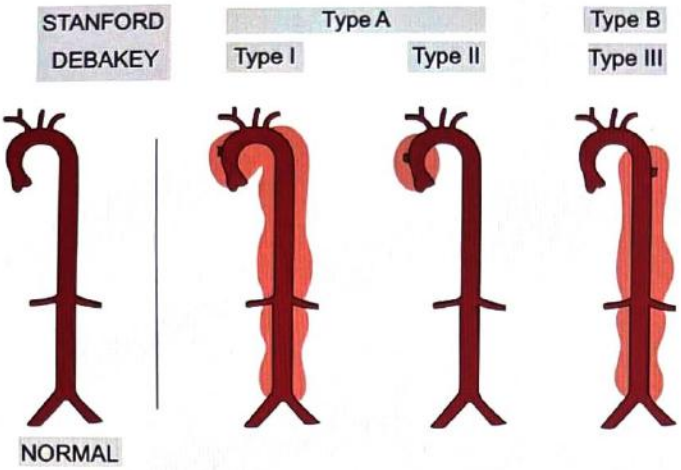
RISK FACTORS

00:05:06

- Genetic syndromes
- Marfan syndrome,
- Loeys-Dietz syndrome,
- Turner syndrome,
- Ehlers-Danlos syndrome (vascular type)
- Acute and chronic cardiovascular conditions.
- Family history of aortic dissection or aneurysm [vascular malformation]
- Personal history of :
- Aortic aneurysm or
- Coarctation of aorta,
- Chronic hypertension,
- Acute hypertension (as in stimulant abuse),
- Polycystic kidney disease
- Inflammatory vasculitis (eg: Giant cell arteritis)
- Pre-existing aortic valvular disease (eg: bicuspid aortic valve),
- Recent aortic manipulation (open or endovascular)
- Pregnancy elevates the risk for women with chronic connective tissue disorders.
- Long-term exposure to corticosteroids or other immunosuppressive drugs
- With multiple risk factors in a pt, then investigations should be done to rule out/ diagnose this condition as fast as possible.
- Two principal classification schemes exist to describe the portion of the aorta involved in dissection.

00:08:15

Stanford classification	Debakey classification
• Most often used in emergency medicine practice	
• Stanford type A → Ascending aorta • Stanford type B dissections → Ascending aorta is not involved.	• Type-I affects → both Ascending and Descending aorta & aortic arch. • Type II affects → only the Ascending aorta. • Type III affects → only the Descending aorta
• Type A dissections may also affect descending aorta/ arch of aorta, but it has to affect ascending aorta	



- Stanford type A corresponds to DeBakey types I and II
- Stanford type B dissections are DeBakey type III
- Approximately two-thirds of aortic dissections are classified as type A

DIFFERENTIAL DIAGNOSIS

00:10:54

- All the conditions which causes chest pain are differential diagnosis for thoracic aortic dissection
- Acute coronary syndrome
- Aortic aneurysm
- Cardiac tamponade (from another cause)
- Esophageal rupture (Boerhaave's syndrome)
- Pneumonia, pneumothorax [Pleuritic chest pain], pulmonary embolism
- Stroke / Transient Ischemic Attack.

UNSTABLE PATIENT

00:11:51

- ABCs
- 2 large-bore I.V cannula
- Uncross matched blood to the bedside
- Cardiorespiratory monitoring
- 12-lead ECG
- Portable CXR
- Bedside cardiac ultrasound to rule out pericardial effusion/ cardiac tamponade and systolic dysfunction etc
- Labs- CBC, Creatinine Muscle Phosphate, PT / PTT, blood group type and cross match, troponin, lactate.
- If we are suspecting thoracic aortic dissection in the pt, then it is always a rule to inform Cardiothoracic surgeon.
- In Thoracic Aortic Dissection, there are few clues like:
  - Auscultation of an Aortic Insufficiency Murmur → should similarly raise suspicion for type A dissection, as distortion or dilatation of the aortic root can also lead to this murmur.
- If Patient is complaining of chest pain, then do
  - 12-lead electrocardiogram
  - Presence of ST-Elevation MI → doesn't rule out thoracic aortic dissection
- They may co-exist in many cases.
- Portable chest x-ray: Not a diagnostic tool of this condition but shows the presence of
  - Widened mediastinum or aorta
  - Apical pleural cap / displaced intimal calcification
- Advanced diagnostic imaging → is must in stable patient with these classical presentations.

PRESENTATION

00:17:13

- **Classic Presentation**
  - Typically a male patient in his 60s with a h/o chronic hypertension who presents with "sharp" sudden onset, severe chest pain that radiates to the back. [like Acute Coronary Syndrome]
  - 3/4 th of patients with acute thoracic aortic dissection will complain of chest pain, and greater than 95% will complain of pain- always at same location i.e they are very consistent with the area of pain [In acute coronary syndrome location of pain may not be consistent]
- **Classical features**
  - Pain is sudden-onset
  - Radiate to the interscapular area of the back (between the shoulder blades) or to the abdomen and low back, depending on the location of the dissection.
  - Patient experiences very severe pain, often described as "sharp", "ripping", or "tearing" in quality.
  - Approximately 1/6 patients will exhibit neurologic symptoms.
  - Syncope is reported in roughly 10% of cases and may be due to several causes (eg, cardiogenic shock from tamponade or myocardial ischemia, hemorrhagic shock from rupture, bilateral carotid occlusion)
- There isn't any pathognomonic feature to clearly establish the diagnosis.
- Patients with type A dissection often present slightly differently than those with type B dissection

Type A dissection	Type B dissection
• Complains of chest pain or syncope	• Complains of back or abdominal pain
• Hypotension.	• Hypertension (SBP >150 mmHg),
• Rx: Emergent surgical repair	• Rx: medically in the acute phase

DIAGNOSIS

00:21:36

- None of the clinical features are pathognomonic.
- So, diagnosis is established by following ways:

1. Chest x-ray

- Non-specific findings that should further raise concern for thoracic aortic dissection include the following:
- Widened mediastinum (>8cm at aortic knob)
- Abnormal aortic or cardiac contour
- Displaced intimal calcification
- Widened right paratracheal stripe (≥ 5mm)
- Tracheal deviation (usually rightward)
- Opacified aorto-pulmonary window
- Pleural effusion (usually left)
- These doesn't establish the diagnosis but raise the suspicion to establish diagnosis.

2. CT angiography (CTA)

- It has excellent sensitivity and specificity, each approaching 100% with newer machines and techniques
- It can diagnosis thoracic aortic dissection & can also rule out number of differential diagnosis of thoracic aortic dissection.
- Because of this condition, if end organ perfusion is compromised→ this can also be assessed by CT angiography
- S/E: Dye-induced anaphylactic shock

3. MRI

- Even though MRI has excellent test characteristics for diagnosis or exclusion of this condition,
- Never an option in emergencies as it takes much time.

Treatment

- Based on Stanford classification
- Type A dissections require emergent surgical repair
- Type B dissections are largely managed medically in the acute phase
- "Complicated" type B dissections which occur in 20 – 30% of patients→ if medical management is not appropriate, can go for open surgical or endovascular techniques by carefully weighing the risk factors in the patient.

EMERGENCY DEPARTMENT MANAGEMENT

00:26:04

- For both type A and type B dissections is to keep
- Both heart rate and blood pressure normal
- One shouldn't ↓ it as end-organ perfusion is affected
- One shouldn't ↑ as it results in deterioration
- 2010 ACC/AHA guidelines for the diagnosis and management of patients with thoracic aortic disease recommend the use of Beta-adrenergic blockers (or calcium channel blockers if contraindications to beta blockers exist) to reduce the heart rate to 60 beats per minute or less.
- If a systolic blood pressure greater than 120 mmHg persists, they direct the addition of intravenous vasodilators to lower the blood pressure further.
- Again, end-organ perfusion should not be compromised in the interest of targeting a specific number.
- Thus, in emergency department the main goal is to stabilize the patient's ABC to keep the B.P & H.R controlled till one dispose the pt to
- CTVS for surgical repair
- Cardiology department for medical management

23 DEEP VEIN THROMBOSIS OF PULMONARY EMBOLISM

- Deep vein thrombosis (DVT) of pulmonary embolism (PE) Venous thromboembolism (VTE)
- VTE may often be deadly

DEEP VENOUS SYSTEM (LOWER EXTREMITIES)

00:01:32

- Calf veins (anterior tibial, posterior tibial or peroneal veins)
- Popliteal vein
- Femoral vein or external iliac veins
- Superficial femoral vein
- Difficult to diagnosis
- Atypical symptoms & presentation
- High index of suspicion
- Initial ABC evaluation
- PE may present as mild shortness of breath, chest pain, fatigue
- Massive pulmonary embolism: Cardiac arrest with PEA (Pulseless electrical activity)
  - Intubation initially for airway support
- Patient with chest pain or shortness of breath
  - Cardiac monitor, IV access, supplemental oxygen
  - Initial EKG of CXR should be obtained
- Initial DVT symptoms may be subtle & non – specific
- Complaints general leg pain or cramping sensation, fullness in calf, swelling
- Differentials: Musculoskeletal strain or tear, cellulitis, superficial thrombophlebitis, venous insufficiency, lymphedema or popliteal (Baker) cyst.

CLASSICAL FINDING ON PHYSICAL EXAMINATION

00:07:35

- Unilateral swelling or edema
- Tenderness to palpation
- Palpable venous "cord"
- Homan's sign (neither specific nor sensitive of DVT)

History of primary survey

00:08:26

- Pertinent information
- Risk factors
- Calculated pretest probability
- Well's score

WELL'S SCORE

Active cancer treatment within previous 6 months	1 point
Paralysis, paresis or recent immobilization	1 point
Recent bed ridden >3 days or major surgery within 12 weeks requiring general anesthesia	1 point
Localized tenderness along the distribution of the deep venous system	1 point
Entire leg swollen	1 point
Calf swelling at least 3 cm greater than other side	1 point
Pitting edema confined to symptomatic leg	1 point
Collateral superficial veins (non-varicose)	1 point
Previously documented DVT	1 point
Alternative diagnosis at least as likely as DVT	(-) 2 points

Total Score

- 0 : Low probability
- 1 -2 : Moderate
- >3 : High

Classical presentation of PE includes shortness of breath or chest pain

- Syncope (Vague complaints: General malaise, functional deterioration)
  Pleuritic component to the chest pain
- Unilateral leg symptoms (signs of right sided heart failure jugular vein distension, peripheral edema)
- M/C vital sign abnormality: tachycardia (in normal pulse oxygenation)

Pulmonary Thromboembolism DX

Clinical symptoms of DVT	3 points
Other diagnosis likely than pulmonary embolism	3 points
Heart rate >100	1.5 point
Immobilization (3 days) or surgery in the past 4 weeks	1.5 point
Previous DVT /PE	1.5 point
Hemoptysis	1 point
Malignancy	1 point

Clinical Probability

- Low probability than 2,
- Moderate: 2-6
- High: more than 6

DIAGNOSTICS

00:16:21

- Chest X ray useful to rule out other diagnosis like
  - Pneumothorax
  - Congestive heart failure
  - Pneumonia
- Unique CXR signs for pulmonary embolism
  - Hampton's hump (unilateral atelectasis)
  - Westermark's sign (unilateral lung oligemia)
- X ray of lower extremity : useful in workup of DVT
- EKG: usually non specific
  (RBBB) : (S1Q3T3) pattern may be seen

D-DIMER TEST

00:19:20

- Directly relates to pretest probability
- D-Dimer: protein derived enzymatic breakdown of cross-linked fibrin
- ↑ level: presence of clot formation
- Very non- specific (can be increased in malignancy infection, MI, strokes, inflammation, advanced age & pregnancy)
- Different lab techniques: may affect the sensitivity of the test
- Current : diagnostic test of choice
  - Venous duplex ultrasonography : sensitivity & specificity – 95%
  - Moderate or high pretest probability : CT pulmonary angiography (CTPA) or V/Q scan
  - CTPA: accepted study of diagnosis
  - CT can also show other possible etiologies

TREATMENT

00:23:13

- Confirmed PE or DVT : anticoagulation (unfractioned Heparin or low – molecular weight heparin) (e.g. enoxaparin)
- Contraindication for anticoagulation – active bleed (cerebral or GI)
  - Contraindicated patients can go for IVC (inferior vena cava) filter.
- Thrombolytic therapy in PE : Controversial (indicated in massive PE of significant cardiopulmonary compromise)
- Patient with isolated DVT without PE : anticoagulants at home.

24 ABDOMINAL PAIN

How to approach a patient with abdominal pain in E.D?

- Abdominal pain – Most common reason for a visit to E.D
- Incidence of non-traumatic abdominal pain – Increasing systematic approach is required for focusing the primary diagnosis of patient & life of the patient can be saved
  - **A - A**irway
  - **B - B**reathing → should be assessed first
  - **C - C**irculation

- Triaging is important to get an idea of how to approach an etiology
- Make a list of etiologies for complaints a patient is coming in E.D (abdominal pain, chest pain etc), & has to be triaged in the form of:-
  - Benign causes
  - Serious causes
  - Life threatening causes

ABDOMINAL PAIN

00:03:10

Benign causes	Serious causes	Life threatening causes
• GERD (reflex pain) (patient gives long history of → off & on pain) • Viral illness (patient will have pain along with fever, vomiting etc. but patient is stable with no problem with circulation, breathing, etc.)	• Appendicitis • Intestinal obstruction • Diverticulitis • Pancreatitis • Kidney stone	• Abdominal aortic aneurysm • Ruptured A.A.A (Even if it is diagnosed & with urgent surgical intervention mortality is very high) • Ruptured ectopic pregnancy • Perforation

Extra-Abdominal causes of abdominal pain

- Diabetic ketoacidosis
- Pneumonia
- Herpes zoster
- Henoch-Schonlein purpura (HSP)
- Malignancy
- Testicular /ovarian torsion

ASSESSMENT OF PAIN

00:08:35

- Depending upon character of pain we can assess whether the pain is appearing from visceral structures / parietal structures.

Visceral pain

- Originate in hollow organs & capsules of solid organs
- Dull /aching pain - Patient cannot pinpoint the particular area of pain (diffuse pain)
- Pain corresponds to embryonic development of that particular organ.
- Depending upon structure's embryonic origin, the area where pain is occurring depends
- All organs forming from
  - Foregut, pain is felt at epigastrium area
  - Midgut, pain is felt at peri-umbilical area
  - Hindgut, pain is felt at supra-pubic area
  - Retroperitoneal organs like
  - Abdominal aorta } Flank pain/ Back pain etc.
  - Kidney etc
  - Gaseous distension of organ } Causes visceral pain
  - Ischemia of the organ
  - Stretching of the organ
  - Torsion of the organ

Referred Pain

- Pain is referred to those structures which have similar innervation to that organ
- Diaphragmatic irritation → shoulder pain

Parietal Pain

- Due to stretching /irritation of parietal peritoneum
- More pinpointed → stabbing pain

HISTORY

00:12:20

- Onset
- Sudden: A.A.A, ruptured ectopic pregnancy
- Gradual – peritonitis, obstruction (mostly)
- **P – P**osition /Provocative /Palliative factor
  - Provocative factor – E.g.- Patient was very active and

precipitated to pain.(Eg. A.A.A, M.I etc)
  - Palliative factors- maximum pain on palpation
  - Certain things Certain food / positioning reduces pain
  - Help in differential diagnosis of abdominal pain
- **Q – Quality of pain**
  - Parietal pain – sharp pain /stabbing pain
  - Visceral pain – dull /achy pain
  - Referred pain

- **R – Radiation**- To back, chest etc
  - Region /referred – fluid collected under diaphragm → referred to shoulder
- **S – Site /Symptoms associated**
  - Nausea & vomiting + Pain → Abdominal pathology
  - Back /chest pain + abdominal pain → Cardiac pathology
- **T – Timing**
  - When did it start? (like after dinner /after doing some activity etc)
  - How did it start?
  - Whether pain is constant /intermittent? (less emergency)
  - Patient with increasing pain – emergency situations

How to remember

- PQRST

PHYSICAL EXAMINATION

00:17:32

- Inspection
  - Look for surgical scar, skin changes
  - Patient with traumatic abdominal pain, there is bluish discoloration under flanks etc.- Points towards traumatic abdominal pain
  - Hemorrhage
  - Grey turner sign
  - Cullen sign

  } Intra-abdominal organs
- Auscultation: Limited utility
- Palpation
  - Localization of tenderness
  - Patient with abdominal pain, it is better to come to the area where he is complaining the pain at last, from side, i.e. coming from non-tender area to tender area
  - Helps in localizing pain
- Identification of peritonitis
  - Enlargement of any viscera (like enlarged liver / spleen etc) → Test for peritoneal irritation→ Rebound tenderness /guarding

Special Exam Maneuver

Carnett sign	• Abdominal wall tenderness
Murphy's sign	• Gall bladder pain (Acute cholecystitis)
Rovsing sign	• Appendicitis

Extra abdominal findings
- Helps in establishing diagnosis

Investigations 00:21:20
- Lab Testing
- CBC
- LFT/RFT

Establishment of diagnosis: by imaging
- Plain Radiograph
  - Has limited utility in evaluation
  - Helpful in
  - Free intraperitoneal air,
  - Calcified aortic aneurysm,
  - Air fluid levels on obstruction
  - It is not necessary that patient with perforation will always show free air. So, this imaging modality have limited capability
  - So, it's not possible to rule out perforation in plain abdominal radiograph

Ultrasound
- In E.D, bedside ultrasound has become like a stethoscope (big help) for emergency physician
  - Abdominal aortic aneurysm diagnosis,
  - Gall bladder
  - Genitourinary pathology diagnosis
- CT scan
  - Good for most intra-abdominal procedures
  - Very good for vascular lesions with contrast

DISPOSITION

00:25:15

- In abdominal pains, we should always have high index of suspicion
- If we are suspecting a patient with emergent cause, we always have to:
- Admit the patient,
- Get all investigations done &
- Rule out Emergent causes → disposition of patient accordingly.
- Especially in elderly & compromised patients

Abdominal Pain		
Patient is stable & have mild/benign cause	**Urgent cause**	**Emergent/urgent life-threating cause**
• Discharged & follow up in OPD	• Patient is again admitted • Disposed to surgical department • Make final diagnosis	• Fast action is important & patient has to be disposed accordingly,eg: Ruptured A.A.A • Immediately shift to operating room • Immediate surgical management

- **Disposition**
- Whether patient is disposed to ward /O.R. /discharged → It depends on
  - History
  - Examination
  - Investigations

25 GASTROINTESTINAL BLEEDING

00:00:30

- GI bleed: Commonest cause of visit in Emergency department
- GI bleed categorized into upper & lower GI bleeds
- The ligament of Treitz crosses the small intestine at the duodeno–jejunal junction
- This is anatomical dividing line b/w upper & lower GI bleed
- Bleeding
  - Above this ligament Upper GI bleed
  - Below can also be categorized as
- GI bleed can also be categorized as

1. Acute: (need more urgent intervention)
2. Subacute

PRIMARY SURVEY & INITIAL STABILIZATION

00:25:53

- Adequacy of the airway, breathing & circulation must be initial concern for any patient with an acute GI bleed and/or hemodynamic instability

AIRWAY & BREATHING

00:03:26

- Some patients, particularly those with hematemesis, may require intubation for airway protection
- Patient may have altered mental status d/t blood loos (hypovolemia)
- Severally ill patient may be necessary to facilitate urgent endoscopy in the emergency department

CIRCULATION

00:04:51

- Intravenous access should be obtained during your initial evaluation of the patient
- Minimum of a large bore IVs should be placed
- If patient is acutely unstable, consider transfusing uncrossmatched blood & IV fluids while a type & IV fluids while a type & cross is being performed
- If your hospital has a massive transfusion protocol & you think it may be needed d/t large volume blood loss, activation of this protocol after the initial evaluation should be considered as well
- Signs & symptoms of massive blood loos on arrival are the same as in a acutely injured trauma patient or any other acute diagnosis of blood loss
- Tachycardia, hypotension or active bleeding indicates the need for IV fluid & consideration of a blood transfusion if vital signs remain abnormal despite administration of isotonic saline

LAB TEST

00:09:23

- Patient's blood count
  - Renal function
  - Coagulation profile
  - Generally, a CBC, chemistry (including BUN, creatinine) PT, PTT & type and cross should be sent
  - The lab studies often serve as a baseline for future comparison. In the setting of hemodynamic instability, blood should be cross – matched & held until the patient bleeding issues resolved.

BLOOD TRANSFUSION

00:10:26

- Massive upper or lower GI bleed

e.g. 1000 ml maroon colored thin liquid stool every 20-30 min NG tube with a constant output of blood

- Hemoglobin dropping at rate of 3g/dl over 2-4 hrs
- Hemoglobin less than 9 (active bleed)
- Anemia induced end organ injury

HISTORY

00:11:57

- Helps in indentifying the source of bleed
  - Hematemesis or coffee ground emesis → upper GI bleed
  - Melena → 70% of UGIB; 30% LGIB
  - Hematochezia – LGIB or an UGIB with significant bleeding

MANAGEMENT

00:13:41

- PPI's – first line for acid suppression in UGIB
  - Low risk patient – empiric IV PPI (40 mg IV BID)
  - High risk (more severe active bleed or co-morbidities) → IV bolus, followed by 8mg/hr drip (PUD)
- H2 histamine blockers – 2nd line
  - Used to reduce acid production
- If PPI is contraindicated, H2 blockers seldom used
  - Mostly in OP setting (GERD, gastritis)
- Suspected variceal bleeding → octreotide (synthetic somatostatin)
  - ↓ Secretion of gastric acid
  - Vasoconstriction of splanchic blood flow
- If GI blood with cirrhosis → antibiotics are administered
  - Ceftriaxone – 1st line

- (Ciprofloxacin 2nd line, if ceftriaxone is contraindicated)
- Mild GI blood → dispositioned to home
- (if (+) stool guaiac or blood streaked emesis – prompt follow up)
- More severe or acute GI bleed require admission
  - Unstable vital signs
  - Rate of bleeding
  - Need for blood transfusion
  - Potential for decomposition
  - Co morbidities
  - Need for procedures
- Many patients → ICU setting → Emergent endoscopy
- Massive LGIB → operating room → partial small bowel resection

26 ABDOMINAL AORTIC ANEURYSM

ABDOMINAL AORTIC ANEURYSM (AAA) 00:01:05

- Abdominal Aortic aneurysm can present in 2 states

1. Ruptured
2. Unruptured

- Unruptured aortic aneurysm → Life threatening
- Abdominal aortic aneurysm can be found in autopsy – in up to 4% of the patients in the age of 50 years
- It ↑ses as the age gradually ↑ses (increases up to 10% in elderly age > 65 yrs)
- Up to 15% in men with peripheral vascular disease
- Patient with ruptured aortic aneurysm → presents to emergency department with chief complaint of sudden pain, ↑ intensity in abdomen, back/flank
- If an elderly patient /patient with history of P.V.D presents to E.D with similar complaints then DD of abdominal aortic aneurysm should always strike the mind

MISDIAGNOSED 00:03:00

- Renal colic
- Pancreatitis
- Bowel ischemia
- Diverticulitis
- Cholecystitis
- Bowel obstruction
- Myocardial infarction
- Back pain
- Ruptured abdominal aortic aneurysm is fatal, unless treated surgically

CLASSICAL TRIAD OF ABDOMINAL AORTIC ANEURYSM (AAA) 00:03:50

Unruptured A.A.A 00:04:35

- Incidental diagnosis
- Patient may complain of
- Abdominal, back/flank pain
- Abdominal mass, fullness, sensations of abdominal pulsations

Ruptured AAA

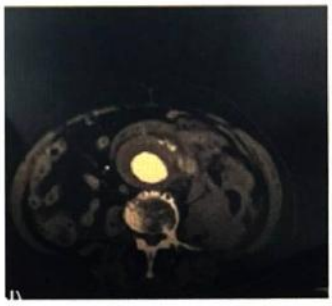
- How to approach a patient with ruptured A.A.A in emergency department:

Case study

- A 65-year old male with hypotension, presented with intense, sudden abdominal pain & on palpation, pulsatile abdominal mass was found
- The patient showed a classical triad of ruptured AAA
- When a patient has known/suspected rupture AAA, the patient should be considered unstable
- As a primary survey; we should see ABC of the patient (Airway, Breathing, Circulation)
- Generally, circulation is initially compromised
- Hemodynamically unstable patient with known / suspected ruptured AAA should be transferred to operating room as soon as possible & the patient should be resuscitated by:
- Put a large bore i.v cannula & start fluids & maintain perfusion of patient.
- Defibrillator should be kept nearby as ruptured AAA causes hypovolemia → Ventricular fibrillation → cardiac arrest

Investigations

- Ultrasound is the ideal study for detection of AAA
- Sensitive
- Done bedside
- 99% cases of ruptured AAA can be diagnosed
- C.T scan: Extremely accurate



Ruptured Aortic Aneurysm

X-ray

- Eggshell pattern of calcification

Investigation of choice for diagnosis & planning repair of aortic aneurysm in patients of renal failure

- MRI

00:06:35

TREATMENT

- Resuscitation followed by emergent surgical intervention.

Indications of surgery in AAA

- Diameter 5.5 cm in males / 5cm in females
- Symptomatic aneurysm
- Rate of expansion >1cm/year
- Atypical aneurysm (pseudoaneurysm, dissecting aneurysm, saccular)

Important onliners

- Mc cause of AAA: Atherosclerosis
- Mc site: Infrarenal aorta
- Mc site of atherosclerosis: Infrarenal aorta
- Most important predictor for rupture: Width / Diameter
- Mc symptom in AAA: Chronic vague abdominal pain / back pain (because location of aneurysm is retroperitoneal)
- Mc & lethal complication of aneurysm: Rupture
- Mc site of rupture: Left retroperitoneum
- Mc cause of death after AAA repair: MI
- Mc complication after AAA repair: Non fatal MI > Renal failure

27 ANAPHYLAXIS

00:00:20

Anaphylaxis	Anaphylactoid reactions

• Life-threatening clinical manifestation
• IgE mediated
• Immediate hypersensitivity reaction involving
  ○ Mast cell and basophil degranulation
  ○ With release of
  → Histamine
  → Tryptase
  → Prostaglandin
  → Leukotrienes
	• Clinically indistinguishable from anaphylaxis
	• Not IgE mediated
	• Direct mast cell degranulation

• Causes of Anaphylaxis
  ○ Food (nuts, eggs, fish milk)
  ○ Antibiotics
  ○ Vaccines
  ○ Anesthetics
  ○ Insulin and other hormones
  ○ Antitoxins
  ○ Blood and blood products
  ○ Insect bite / snake bites
  ○ Latex
  ○ Allergy immunotherapy
	• Causes of Anaphylactoid reaction
	  ○ NSAID
	  ○ Opiates
	  ○ Sulphite
	  ○ Radio contrast media
	  ○ Neuromuscular blockingzagent
	  ○ Gamma globulin
	  ○ Antisera
	  ○ Exercise

00:03:30

Presentation of Anaphylaxis / Anaphylactoid reaction

- Can present within minutes of exposure, usually < 1 hour after exposure
- Rapid reaction on parenteral route
- 1st symptom: Flushing, pruritus and sense of doom.

CLINICAL MANIFESTATION

00:04:20

- Eye: Pruritus, lacrimation, conjunctival erythema, periorbital edema.
- Cardiovascular: Hypotension, tachycardia, arrhythmias, cardiac arrest
- Gastrointestinal: Nausea / vomiting, diarrhea, abdominal pain
- Skin: Pruritus, flushing, urticaria, angioedema
- Respiratory: Dyspnea, stridor, difficult swallowing, pulmonary edema
- Neurologic: anxiety, sense of doom, seizure
  - Immediate treatment is indicated for all patients with significant respiratory, cardiac or gastrointestinal symptoms
  - No absolute contraindications to epinephrine

MANAGEMENT

00:05:50

- Airway: Early intubation, cricothyroidotomy
- Breathing: Oxygen
- Circulation
  - 2 large i.v bore cannula,
  - Epinephrine given as fast as possible, cardiac monitor to be attached.
  - Epinephrine is the drug of choice
    - → Epinephrine I.M.: 0.3-0.5 mg 1 : 1000 to anterior or lateral thigh
    - → Epinephrine I.V.: 0.1-0.2 mg (1 ml) 1:000 in 10 ml 0.9 % Nacl (0.1 mg/ml) every 1-2 min

If patient response to epinephrine

- Treat all patients with histamine 1 and 2 receptor (H1, H2) blockers
- Diphenhydramine (H1) 25-50 mg I.V
- Ranitidine (H2) 50 mg I.V

If no response to epinephrine

- Glucagon 1-2 mg I.V/I.M every 5 min to effect
- For continued hypotension
  - Continuous I.V epinephrine infusion at 0.1 microgram / kg/min titrated to effect
  - Continuous aggressive fluid resuscitation
- In anaphylaxis, I.V steroid has no role in acute phase but has role in preventing phase 2 reaction
- Once patient has stabilized and there is no known cause, then refer the patient to an allergist for testing and monitoring

28 HYPOTHERMIA

HYPOTHERMIA

00:00:18

- Is defined as a core body temperature < 35° C

1. Cold injuries: Include
   A. Frost Bites
   B. Trench foot.
- People without the resources or ability to protect themselves from exposure to cold temperatures will often present (or be brought) to ED for treatment
- Patients suffering from the Hypothermia and cold injury range from
  - Outdoor adventurists to intoxicated individuals,
  - Undomiciled, the mentally or physically ill, the elderly, children, and the poor.
- Environment exposure is obvious,
- Atypical Cases,
  - Wet indoor patients are more subtle, patients with altered mentation may not be able to communicate a history of cold exposure.
  - Bystanders may report a change in personality, paradoxical undressing, rocking, dysarthria, ataxia or frank confusions.

A. Frost bite

00:04:16

- Cold injury: Damage to exposed extremities
- Made worse by poor circulation to the extremities because of constrictive clothing
- Hypothermia: Reduces blood flow to the periphery.
- Patient's first experience pain followed by numbness in affected area may mistaken the disappearance of pain for improvement.
- Tissues: Pale & Firm,
- Poor capillary refill and sensation.
- Severe Frost Bite appears purple due to blood Sludging.

B. Trench foot

00:05:13

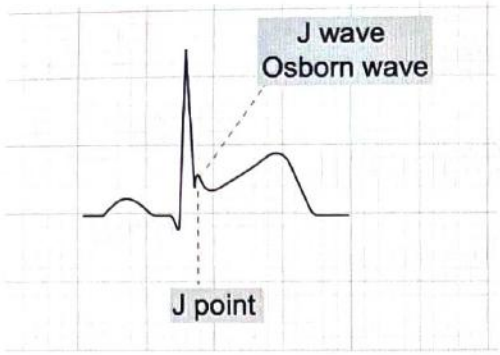
- In lower extremities with prolonged exposure to wet and cold conditions develop tissue damage,
- Skin - red swollen at first,
- Progressing to blisters and bullae and finally
- Hemorrhage in the skin and deeper tissues
- Presents as
  - Paresthesia's,
  - Pain,
  - Numbness

Refer Table 28.1

INITIAL STABILIZATION AND PRIMARY SURVEY

00:12:06

- Minimize jerky movements of the severely hypothermic patients as movement or exercise may precipitate ventricular fibrillation.
- Stabilize Airway- Endotracheal intubation for unresponsive patients.
- Stabilize Breathing- Administer warm humidified oxygen, Endotracheal intubation for respiratory failure.
- Stabilize circulation- Monitor, ECG, intravenous access, warm IV fluids
- Remove cold wet clothing, Dry patient,
  - Cover with warm dry covering or
  - Warming blankets (Bear hugger).
  - Initiate active rewarming measures
- Look for signs of accompanying cold injury, trauma, or underlying illness (2° Hypothermia)
- Best method for measuring and monitoring core Body Temperature - low reading temperature probe in the esophagus or rectum
- Rectal temperature probes (not thermometers)
  - Should be inserted five inches into the Rectum;
  - Lodging the probe in feces will give a falsely low reading.
- Rectal temperature also lags true core temperature during rewarming.
- Laboratory testing may reveal
  - Hemoconcentration,
  - Hypo/hyperkalemia,
  - Hypo/hyperglycemia. or
  - Abnorm[illegible]gulation,
  - Tests in which blood is warmed may give false positive or false negative results, e.g.: Arterial blood gas, coagulation studies.
- A plain chest film may reveal pulmonary edema.
- ECG
  - J (Osborne) wave
  - In moderate: severe cases
    → Prolonged intervals (PR, QRS, QT)
    → Arrhythmias (Atrial or ventricular)



- Frost Bite and Trench foot are diagnosed by history & physical examination

FINAL MANAGEMENT

00:16:43

- All wet clothing should be removed,
- Patient's skin should be dried.
- The invasiveness of rewarming methods correlates to the severity of the hypothermia,
- Rapid rewarming is the goal for all patients.

Severity of hypothermia	Methods of rewarming	Examples
• Mild	• Passive external rewarming	• Blankets (remove cold wet clothes first) • Oral hydration with sugared drinks
• Moderate	• Passive external rewarming + Active external rewarming	• Electric or forced warm air blankets + • often add noninvasive internal; • warm intravenous fluids, -warm humidified oxygen
• Severe	• Active internal rewarming	• Warm intravenous fluids, -warm humidified oxygen, -warm bladder and gastric, o • Sometimes thoracic or peritoneal lavage (rarely done anymore) • Central arteriovenous or venovenous rewarming, -cardiopulmonary bypass, -dialysis.

- In most Arrhythmias
  - Provide supportive therapy,
  - Never anti-arrhythmics.
  - Cardiac pacing and atropine are often ineffective for bradycardia

- Ventricular Fibrillation
  - Preform CPR and Defibrillation per ACLS protocols,
  - Except that anti Arrhythmics and repeat Defibrillations should be deferred until the patients is warmed to above 30°c
- Resuscitated patients in cardiac arrest while rewarming to a core temperature of 32° should not be terminated unless the patient's has frozen ice or there is ice filling the airway.
- The patient's temperature may decrease during rewarming called as Afterdrop as peripheral vessel dilate and increase return of cool blood from extremities to the core.
- Rewarming shock (hypotension) may occur as rewarming can also drop pH, electrolyte imbalance and coagulopathy

- **Frost Bite management**
  - Immediately immerse the affected area in warm water (37° C-39° C)
  - Remove constricting clothes & jewelry
  - Consult surgical services for exploration and debridement,
  - Amputation is usually delayed until the gangrenous tissue has clearly demarcated.

Treatment

- Ibuprofen as an anti-inflammatory agent
- Tetanus toxoids,
- Elevation of the affected area
- Narcotic analgesia
- New therapy-tissue plasminogen activator
  - Improve perfusion
  - Decrease the incidence of amputation
- Observe for the development of compartment syndrome
- Hypothermia should be treated before frostbite.
- **Trench Foot management**
  - Keep feet warm, clean & dry
  - Elevate the extremities.

DISPOSITION

00:25:16

- Patients with hypothermia & cold injuries must be sent to warm places and not to be discharged back to the cold environment.
- Moderate to severe hypothermia requires care in a monitored or intensive care setting for
  - Close monitoring of core temperature

- Cardiopulmonary support
- Correction of electrolyte imbalance
- Care for comatose or altered patients
- Observation for the developments of DIC, rhabdomyolysis, pancreatitis, seizures, and other complications.

Table 28.1

Degree of Hypothermia	Mild (32-35 C)	Moderate (28-32 C)	Severe (<28C)
• Vital signs	• Tachypnea • Tachycardia without hemodynamic instability	• Bradycardia • Bradypnea	• Bradycardia • Hypotension • Pulselessness • Bradypnea/apnea
• Muscular	• Shivering	• Cessation of shivering	• Rigidity
• Reparatory	• Hyperventilation	• Hypoventilation	• Pulmonary edema
• Cardiac		• Arrhythmias, cardiac output	• Ventricular fibrillation • severely ↓ cardiac output
• Neurologic	• Dysarthria, ataxia, amnesia altered judgment, apathy, fine and gross motor impairment; "Mumbles, grumbles, stumbles, fumbles, tumbles"	• Paradoxical, undressing • Decreased responsiveness, Hyporeflexia, Dilated and sluggish • pupils, • Dysarthria ataxia	• Coma/ unresponsiveness • Areflexia • Fixed pupils
• Urine output	• Polyuria (cold diuresis)		• Oliguria

29 HYPERTHERMIA

HYPERTHERMIA

00:00:16

- Heat related illness is a continuum of conditions that results from body's inability to deal with heat stress and dissipate excessive heat.
- It includes minor problems such as
  - Heat cramps
  - Heat edema
  - Prickly heat
  - Heat syncope
  - Heat exhaustion.
- Classical heat stroke & exertional heat stroke represent the
  - Most severe manifestations of heat related illness
  - Associated with neurologic dysfunction and multi-system organ damage.

CLINICAL FEATURES

00:02:17

- Typically, temperature exceeds 40°C
- Tachycardia
- Normotensive, but may be hypotensive
- Severe CNS dysfunction
- Pupils may be fixed, dilated, pinpoint or normal
- Nystagmus or oculogyric episodes present.
- Hyperdynamic cardiovascular state.
- Hypodynamic state signifies impending cardiovascular collapse
- Tachypnea & Hyperventilation.
- Gastrointestinal hemorrhage
- Jaundice & elevated liver enzymes
- Muscle tenderness & cramping
- Rhabdomyolysis & ARF are common complications of exertional heat stroke
- Fulminant hepatic failure and DIC rarely occur.

HEAT STROKE

00:04:04

- Heat stroke is defined as hyperthermia usually > 40°C associated with severe CNS dysfunction and anhidrosis
- Mortality rate of up to 70%
- The temperature is considered a relative criterion since some patients may be cooled prior to temperature measurement
- Diagnosed clinically by assessing a constellation of symptoms & physical findings, not by an absolute definition.

00:05:48

Refer Table 29.1

Clinical features

- Abdominal cramping
- Nausea
- Vomiting
- Myalgia
- Diarrnea
- Headache
- Dizziness
- Dyspnea
- Weakness
- Syncope typically precede exertional heat stroke

Risk factors

- Obesity
- Poor physical fitness,
- Precedent illness
- Lack of acclimatization

Heat cramps

00:12:36

- Brief, intermittent, severe cramps in muscles fatigued by excessive exercise
- Occur at a time of rest.
- Related to salt deficiency
- Occur in first days of excessive work in a hot humid environment.

Heat edema

- Edema of feet & ankles in unacclimated individuals exposed to warm tropical or subtropical climates.
- Result of ↑ hydrostatic pressure & vasodilation resulting in vascular leak & edema.

Heat syncope

- Temporary loss of consciousness caused by intravascular shunting to cutaneous circulation,
- Pooling in the lower extremities due to prolonged standing & volume depletion.
- Elderly are susceptible

HEAT EXHAUSTION

00:15:10

- Vague malaise, fatigue, nausea, vomiting,
- Weakness & headache in the setting of heat stress.
- Core temperature is frequently normal (or) just mildly elevated.
- Patient is frequently water depleted from inadequate hydration but may be salt depleted from rehydration with hypotonic solutions.
- Patients with true heat exhaustion have normal mental status, but may be tachycardiac

Important Information

- **Clinically heat exhaustion & heat stroke may be difficult to differentiate & if the diagnosis is unclear, cooling should be immediately initiated.**

Primary survey and management

- Obtain fasting blood sugar in all patients with altered mental status as it should be an initial step
- Rapidly obtain rectal temperature.
- Initiate rapid cooling simultaneously with any basic resuscitative measures.
- No laboratory changes are pathognomonic of heat stroke
- Patient can be hypernatremic/hyponatremic depending upon patient's hydration status
- Hypokalemia is common in early heatstroke,
- Hyperkalemia predominates in the setting of muscle damage

Refer Table 29.2

TREATMENT

00:20:43

- Aim at administration of simultaneous basic resuscitative measures and immediate & aggressive cooling measures.
- Consider intubation of any patient not able to protect his airway.
- Two large bore I.V cannulas should be established for resuscitation and the patient should be placed on continuous cardiac monitoring & pulse oximetry
- A thermistor probe should be placed in rectum of esophagus to continuously monitor core temperature.
- Immediate cooling is the cornerstone of management of heat related illness.
- Extent of hyperthermia is primary determinant of outcome.
- Core temperature should be rapidly lowered to 38-39°C
- Clothing should be removed.
- Special cooling blankets are now routinely employed to rapidly lower body temperature.
- In the absence of these specialized blankets, patient may be sprayed with water or may be covered with water-soaked sheets.
- Blowing air with fans over the patient aids in evaporative cooling. Ice packs may be placed in groin and axilla.
- Ice water submersion is extremely effective but is now avoided since it may induce shivering which can generate increase heat and leads to vasoconstriction of cutaneous vasculature that reduces heat transfer.
- Other cooling methods - peritoneal, bladder, and gastric lavage with ice water, cold IV Fluids & cool oxygen.
- Antipyretics should never be given to patients with heat related illness since they have no efficacy in reducing the temperature and may exacerbate the hepatic, renal and coagulopathic abnormalities associated with heat stroke.

Dantrolene

- Mechanism
  - Muscle relaxant that attenuates the amount of calcium released from the sarcoplasmic reticulum in skeletal muscles to the cytosol.
  - Decreased intracellular calcium levels lead to reduced muscle metabolic activity and heat production.
- Use
  - Malignant Hyperthermia
  - Neuroleptic Malignant Syndrome
- Dantrolene is not suggested to use as a first line treatment for heat stroke,
- May be considered in patients unresponsive to conventional cooling methods or patients with excessive shivering.

Methods to cool the patient

- Evaporative techniques
- Wetting body surface with continuous fanning
- External (non- invasive) conduction techniques
- Tap water immersion
- Ice water immersion
- Application of cold packs
- Cooling blanket
- Internal (invasive) conduction techniques
- Gastric, peritoneal or bladder lavage.
- Cold IV fluids
- Pharmacologic techniques
- Dantrolene

Shivering leads to increased heat production

- Controlled with benzodiazepines (Also the drug of choice for controlling seizures)

- Barbiturates are the 2nd line of therapy (since phenytoin is not effective in this setting)
- Hemodynamic instability requiring vasoactive support is associated with a significantly higher mortality rate and increase in neurologic disability.
- Fluid resuscitation should be the initial treatment iterated to volume status and hemodynamic parameters.
- **Additional treatments**
  - Based on the individual's underlying medical status
  - Typically, heatstroke mimics a sepsis like distributive shock and similar treatment modality may be employed.
- Rhabdomyolysis is a relatively common complication of exertional heat stroke caused by release of large amounts of Myoglobin from damaged muscle that precipitates in the kidneys and can cause acute renal failure.
- Dark tea colored urine and tender muscles are classic findings.
- **Treatment** includes infusion of large amount of IV fluids and alkalinisation of urine with IV bicarbonate infusion to prevent myoglobin precipitation in the renal tubules.
- Hemodialysis may be necessary for patients with acute renal failure (ARF).

POOR PROGNOSIS

00:27:31

- Initial temperature >41°C
- Persistent elevation in temperature despite cooling
- GCS <12
- Severe pulmonary edema
- Delayed or prolonged Hypotension
- Lactic acidosis in patients with classic heat stroke
- ARF & Hyperkalemia
- Aminotransferase levels > 1000 in the first 24 hours.
- Elderly
- Pre-existing cancer, CAD (or) treatment with diuretics
- Transportation to hospital by ambulance.

Table 29.1

Types	Classical heat stroke	Exertional heat stroke
Definition	• Body is unable to dissipate heat during environmental heat waves	• Body is unable to dispel heat due to endogenous production
Affected age group	• Very young • Elderly & chronic • Mentally ill	• Young healthy individual • Athletes & military recruits
Presentation	• Hyperthermia, CNS symptoms like irritability, delusions, hallucinations, Altered sensorium, seizures/coma.	• Hyperthermia, Diaphoresis & an Altered sensorium
Sweating	• Ceases In majority of cases	• Maintain the ability to sweat, so, have lower core temperature
Precipitating factors	• Diurectis, antihypertenives, anticholinergics and neuroleptics	• Extreme physical exercise in a hot, humid environment
Others	• Advanced age, hypotension, coagulopathy and endotracheal intubation are poor prognostic indicators	• Cocaine or amphetamines or prolonged seizure activity can also cause due to increased motor activity
Complications: Rhabdomyolysis, Acute Renal Failure(ARF), coagulopathy, lactic acidosis & hypoglycemia	↑↑	↑↑↑↑

Table 29.2

LFT	• Elevated as a sign of Hepatic Failure
Muscle Function Test	• Indicate muscle damage and necrosis and may rise to ≥100,000 in patients with EHS
CBC	• An elevation in WBC & thrombocytopenia
RFT	• Critically important to evaluate since patients with muscle necrosis release myoglobin which is nephrotoxic
Urine analysis	• Evaluate for hematuria • A microscopic urinalysis to evaluate for the presence of RBCs and urine myoglobin • Hematuria on dip stick in the absence of RBCs on • microscopic exam suggests Rhabdomyolysis
Chest X-ray	• Demonstrate pulmonary infarction, edema or atelectasis
Head CT or Lumbar Puncture	• Rule out CNS abnormalities in patients with altered mental status
EKG	• Sinus tachycardia with non-specific ST-T wave changes • Conduction abnormalities may be present (RBBB or prolonged QT)

30 SUICIDAL EMERGENCY MANAGEMENT

SUICIDAL EMERGENCY MANAGEMENT

00:00:15

- Emergency providers recognize and provide care for patients who attempt to harm themselves or to commit suicide
- Patient presenting with non-psychiatric complaints have depression and up to 10% may have suicidal thoughts.
- Stressors include
  - Relationship concerns
  - Socio-economic difficulties
  - Loneliness
  - Mental illness

Initial Survey 00:02:35

- The primary goal -to assure safety of the patient and staff
- Evaluate ABCs with specific attention to
  - toxidromes
  - injuries (cervical spine protection)
- In the presence of any abnormal vital signs, a full medical exam is indicated

PRESENTATION

00:04:15

- Victims of overdose or injury either by private vehicle / in the custody of police / rescue personnel
- Examined for signs of injury including
  - Burns,
  - Ligature marks,
  - Gunshot wounds,
  - Lacerations
  - Fractures depending on the history available
- Conside
  - Intoxication
  - Acute psychosis
  - Acute medical condition precipitating the presentation
- Overtly suicidal or already attempted self-harm are often quite apparent.
- Each patient with depression should be assessed for risk of suicide
- Consider suicide as a possibility in:
  - Single vehicle road traffic collisions,
  - Pedestrians struck by automobiles,
  - Falls
  - Shootings
  - Stabbings
- No routine panel for "medical clearance"
- All workups should depend upon the history & presentation of the patient. [no fixed routine workup panel in these pts]
- Pregnancy testing for female patients in childbearing age
- If mental status is altered, then blood glucose level should also be assessed.
- Test to aid the management of poisonings - EKG for tricycle antidepressant overdose
- Chemistry to assess
  - Acid-base disorders or
  - Acetaminophen level,
- X-rays to identify
  - Foreign bodies
  - Fractures in hanging attempts
  - Patients who have jumped from a height

MANAGEMENT

00:09:51

- Should focus on ABCs and identifying immediate threat to life
- Any poisonings or injuries should be treated appropriately
- Kept in safe environment, shouldn't be left alone and should not be permitted to leave the ED before treatment, until the risk assessment & psychiatric evaluation is completed.
- Document completely and comply with local legal requirements whenever a patient is held for psychiatric evaluation

FOCUSED PSYCHIATRIC ASSESSMENT

00:11:41

- To see how much risk is present in these patients / whether this pt needs further psychiatric evaluation or not.
- **M** - Memory → long and short term
- **O** - Orientation to time,place and person,
- **M** -Mood (a symptom), "How do you feel?" "Happy," "Mad", "Sad?"
- **M** - Mentation ask about hallucinations, delusions, paranoid thoughts
- Affect (a sign), how does the patient act? Whether he has eye contact, how is his speech, mannerism and demeanor?
- **S** - Speech → is it organized and logical or disorganized

and tangential?

- **S** - Suicidality. Is there a plan, intent, objective, preparation and /or rehearsal etc?

How to remember

- MOMMAS2

RISK FACTORS

00:15:05

- Prior attempts,
- Previous psychiatric history,
- Family history of mental illness or suicide,
- Signs of depression or substance abuse,
- How much access he has to different methods for committing suicide.
- How serious was his method of self-harm when he committed suicide?
- For serious suicide attempt, he needs to be further evaluated for any psychiatric illness.

THE MODIFIED SAD PERSONS SCORE

00:16:23

- Can be utilized to assess suicidal risk.
- Guideline useful for determining patients who are at high risk for committing suicide.
- Should not be the only factor to determine which patients can be discharged.
  - **S** - Sex (male → chances of re-attempt can be more)
  - **A** - Age (<19 or 45)
  - **D** - Depressive symptoms and hopelessness
  - **P** - Previous suicide attempt or Psychiatric illness
  - **E** - Excessive alcohol or drug use
  - **R** - Rational thinking loss
  - **S** - Single, Separated, divorced or widowed
  - **O** - Organized or serious suicide attempt
  - **N** - No social support
  - **S** - Stated future intent

How to remember

- SAD PERSONS

- Give 2 points for each positive answer marked with a
- All other positives score 1 point
- Score
  - ≤ 5 -Low risk, consider potential discharge
  - 6-8- Moderate risk, consider psychiatric consultation
  - ≥ 9- High risk, need to be admitted, as re-attempt is more common in these pts
- If suicide risk is recognised, evaluated & managed properly , then the chances of re-suicide can be prevented
- If the pt is stable after suicide attempt, he shouldn't be discharged until & unless the risk is assessed
- Disposal of these patients can be done by taking
  - Precautions
  - Admission
  - Discharge