BASIC LIFE SUPPORT

Definition:

→ 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 Bystander CPR

→ Survival Rate ≡ 20%

Cardiac Arrest

1. Unresponsive person [As there is no cerebral Blood flow]
2. Pulseless [Carotid pulse is checked as it is closest to heart & last one to go in case of cardiac arrest]
3. Apnea/ Gasping Respiration/ Agonal Rhythm

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Pulseless conditions

→ Ventricular Fibrillation

→ Ventricular Tachycardia

→ Asystole

→ Pulseless Electrical Activity [PEA]

Shockable Rhythms

Ventricular Fibrillation and Ventricular Tachycardia

→ Treatment is Cardioversion

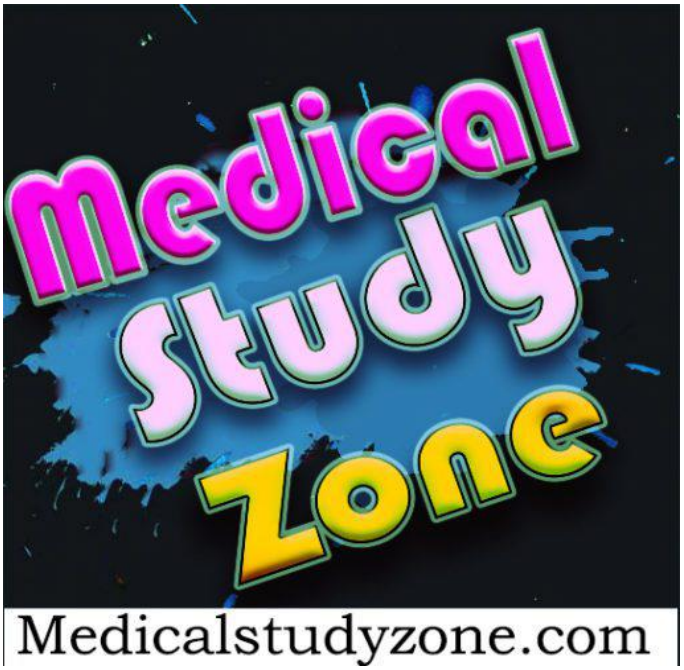
→ Survival chances are higher

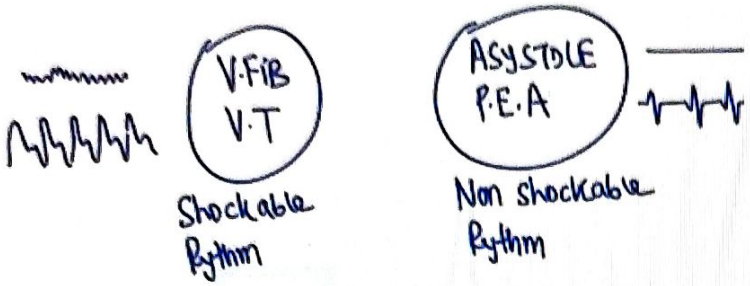
Non – shockable Rhythms

Asystole and Pulseless Electrical Activity

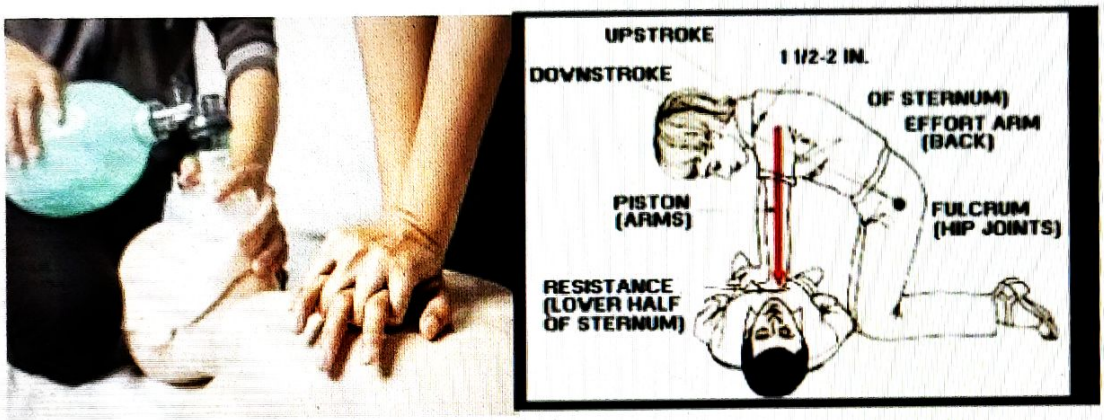
→ Survival chances are low

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





Cardio Pulmonary Resuscitation (CPR)



Q. 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)

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**

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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

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

Management

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

If Shockable Rhythm

↓

Press button of AED

AED will give a DC shock of 200 J

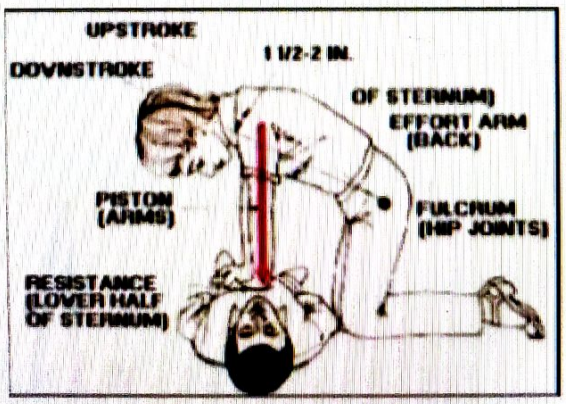
↓

After shock is delivered, check for return of Pulse & Respiration

If Non-shockable

↓

Continue CPR till Medical health team arrives.



Ratio of CPR administration

Adult	→ 30 : 2	1 or 2 Rescuer
Paediatrics (Child)	→ 30 : 2	1 Rescuer
	→ 15 : 2	2 Rescuer
Neonate	→ 3 : 1	2 Rescuer mandatory

Sternal Depression

Adults → 5cm

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)

-By AMBU Bag

-Ensure → Head tilt

→ Chin lift

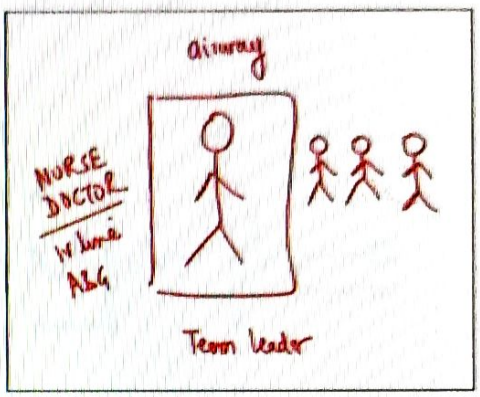
Head tilt & chin lift ensure patency of airways so that air can enter r into lungs during BMV

- **Position of patient** → **Sniffing position**

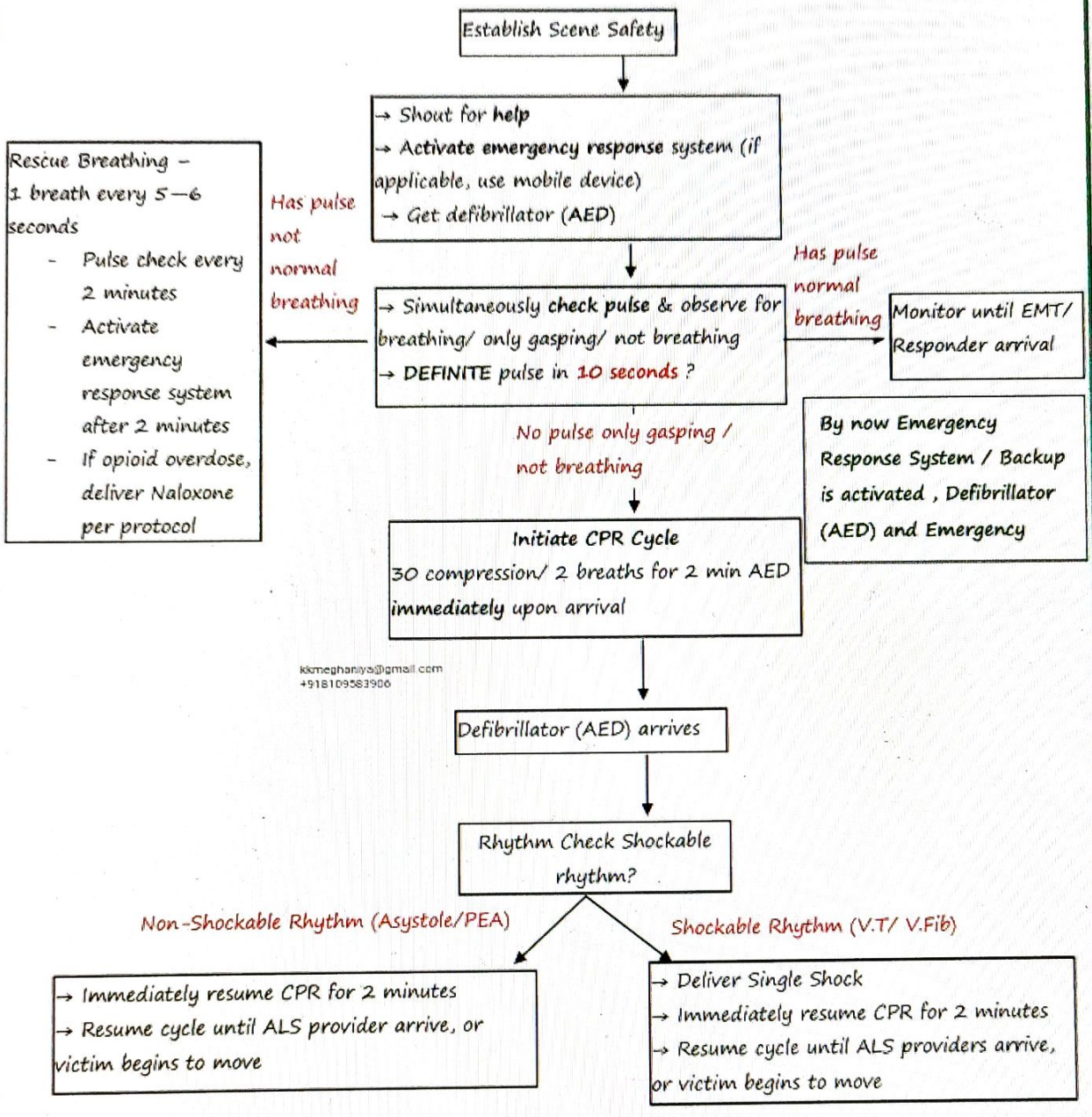
→ **Correct application of mask** -The Mask should be held b/w **thumb & an index finger of a left hand** in a '**C**' **shaped manner** so that air does not leak.

-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



→ **AED** is used immediately upon arrival. We don't wait for CPR cycle to get completed. We attach AED electrode immediately.

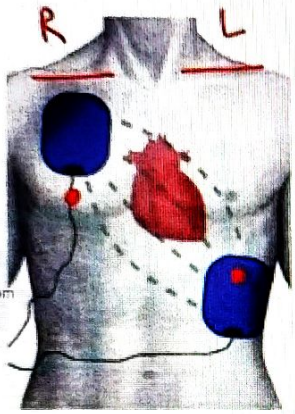
AED → Electrode placement

→ 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



200J
biphasic

Etiology of cardiac Arrest

1. Coronary Artery Disease (CAD)→ Main/ Leading cause

Other causes

2. Cardiomyopathy e.g. → Hypertrophic obstructive cardiomyopathy (HOCM)

→ TakoTsubo cardiomyopathy (TTCM)

3. Valvular Lesions like- Aortic Stenosis,

-Mitral Stenosis, Mitral Regurgitation. → Left Atrial dilation→ A. Fib→ Stroke

4. Bundle Branch Block

BRUGADA Syndrome (Channelopathy)

→ Na^+ channel defect

→ Predisposes to development of **Torsades D pointes** or Ventricular Tachycardia

→ Gene involved → SCN5A

→ TDP

5. Electrolyte Abnormalities

→ Hyperkalemia

6. Toxins

→ e.g. TCA overdose

→ Organophosphate poisoning (OPC poisoning)

→ Cocaine/ Alcohol overdosage

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

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ADVANCED CARDIAC LIFE SUPPORT

Q. How ACLS is different from BLS ?

A. 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

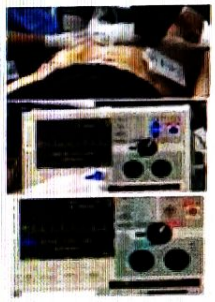
-in **BLS** the main aim is to maintain brain perfusion by giving chest compressions

D/D of Pulselessness

1. **Ventricular Tachycardia**
2. **Asystole**
3. **Pulseless Electrical Activity**
   a. Hypovolemia
   b. 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



CARDIOVERSION
Syn. DC SHOCK ↓ ← Peak of R wave

PSVT
A. FIB
A. FLUTTER

Q. Difference b/w **Defibrillation** & **Cardioversion**

A. 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

↓

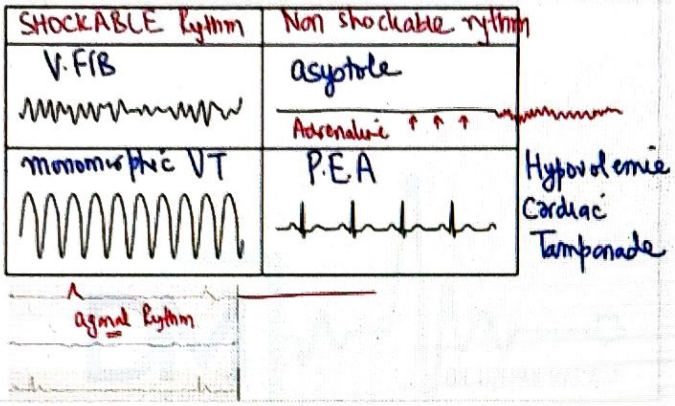
Return of spontaneous circulation

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 VT



- 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

-5 H

&

-5 T

Hypovolemia	Hypoxia	Hydrogen Ion (acidosis)	Hypo/Hyperkalemia	Hypothermia
Loss of fluid volume in the circulatory system. Look for obvious blood loss. 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	Both high and low K+ can cause cardiac arrest. Signs of high K+ include taller, peaked T-waves, and widening of the QRS complex. Sings of low K+ include flattened T – waves, prominent U-waves ,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 > 86 F and 30℃ The patient may not respond to drug or electrical therapy while hypothermic.

Toxins	Tamponade	Tension Pneumothroax	Thrombosis (Heart: acute, massive MI)	Thrombosis (Lungs: Massive PE)
Accidental overdose: tricyclics, digoxin, betablockers, and calcium channel blockers **Cocaine** is the most common street drug that increases incidence of pulseless arrest. **Physical signs** include bradycardia, pupil symptoms, 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 QRS complex, rapid heart rate. Physical signs: Jugular vein distention (JVD), no pulse or difficulty palpating a pulse, and muffled heart sounds. Perform: Pericardiocentesis to reverse.	Tension pneumothorax shifts in the intrathroacic structure and can rapidly lead to cardiovascular collapse and death. ECG signs: Narrow QRS complexes ,slow heart rate. Physical signs: JVD, tracheal deviation, unequal breath sounds, difficulty with ventilation, 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, chest pain / pressure. Treatment: Use of fibrinolytic therapy, PCI (Percutaneous coronary intervention). PCI (coronary angioplasty with or without stent)	Can rapidly lead to respiratory collapse and sudden death. ECG signs of PE: Narrow QRS complex, 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.

- **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

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

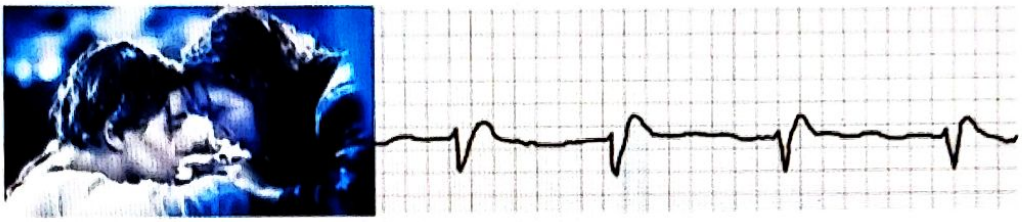
Epinephrine can be repeated every 3-5 minutes

Amiodarone 3 doses can be given

1st dose = 300 mg
2nd dose = 150 mg after 3-5 mins
Amiodarone is useful for VT as well as shock refractory VT
ROSC (Return of Spontaneous Circulation)
↓
TTM (Target Temperature Management) or
Therapeutic Hypothermia (limit the progress but can't reverse damage)
-Cardioprotective
-Neuroprotective
Prognosis of these patients isn't good

PULSELESS ELECTRICAL ACTIVITY (PEA)

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 VT	65-70% (Pulseless)	shockable - rhythm
V-Fib	25-40%	
PEA	11%	non shockable rhythm
Asystole	0.2%	

Rx -cause specific Rx.

PEA

Causes:

1)Empty Heart a) Hypovolemia

↓

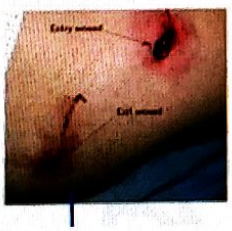
eg Bullet injury to the abdomen causing ripping of aorta / superior mesenteric artery/ inferior mesenteric artery

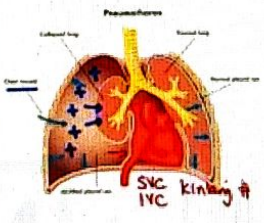
↓

Patient can have hypovolemia → resulting in decompensatory shock → pulselessness

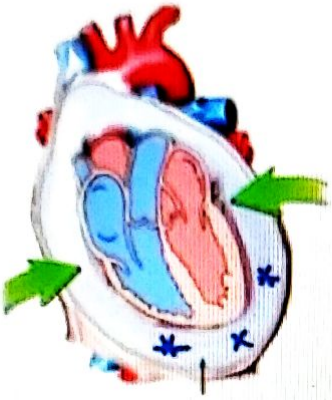
- here, even though muscle of heart is working fine, there is no blood to be pumped

(b) **Tension pneumothorax** - Eg 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





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

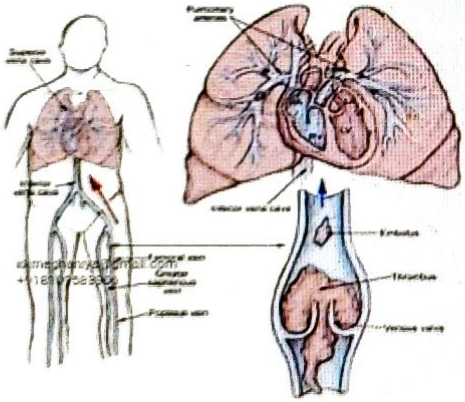


2) Electromechanical dissociation - Conduction normal

Eg. Acute MI → stunned myocardium (hypoxia) (extensive ant. wall MI)

(no effective contractions - ↓ CO)

Eg. Pulmonary embolism
Postpartum patient
orthopedic surgery eg. 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

5^H^	5^T^
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

Note: Way to remember the causes Reversible of Cardiac arrest

H	Hypothermia
E	Electrolyte Imbalance (↑ K+ / ↓ K+)
A	acidosis
P	Poisoning

Intervention

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

Echocardiography guided Pericardiocentesis
managing cardiac tamponade

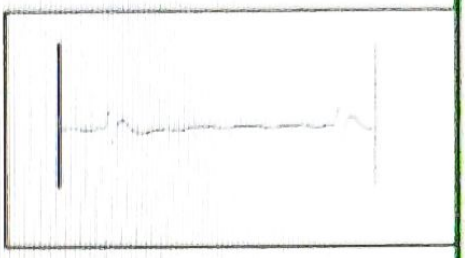
wide bore needle in 2nd intercostal space
managing tension pneumothorax

→ 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

ASYSTOLE AND POST RESUSCITATION CARE

Asystole



- → 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

- → 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: -

- → 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 & VT – Good prognosis
  - PEA & asystole – Poor prognosis
- → No response to CPR
- → ETCo2 <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 VT 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

- → 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.

1. **Return spontaneous circulation**

2. **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 PETCO2 – 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

3. **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.

Approach to shock

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

1. Impaired **delivery of oxygen** to tissues
2. Impaired **utilization of oxygen** by tissues
3. **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:
     1. Neck veins (± Jugular venous distention)
     2. Cardiopulmonary examination
     3. Abdominal examination
     4. 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

1. Hypovolemic shock

- ↓ intravascular volume

- resulting in
  1. ↓ preload
  2. ↓ stroke volume
  3. ↓ cardiac output
  4. ↓ perfusion

Hypovolemic shock

Hemorrhagic Shock (Major trauma, gastrointestinal bleeding, ruptured ectopic pregnancy, etc).

Non – Hemorrhagic Shock (Vomiting, diarrhea, burns, etc.)

If not, resuscitation may lead to

V. Fibrillation, PEA (pulseless Electrical activity)

2. Cardiogenic shock

→ 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

3.Distributive shock

→ Pathologic vasodilatation leading to relative intravascular volume depletion

- Anaphylactic (IgE mediated allergic reaction)
- Septic
  - pneumonia,
  - pyelonephritis,
- Neurogenic (spinal cord injury)
- Toxicologic
  - (vasodilatory medication overdose)

4.Obstructive shock

→ Impaired cardiac output due to:

- **Pulmonary vascular obstruction**
  - pulmonary embolism,
  - venous air embolism,
- **Mechanical obstruction**
  - tension pneumothorax,
  - pericardial tamponade,
  - left ventricular outflow tract obstruction

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5 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

→ 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

→ 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

Eg

- 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

1) CBC with differential
2) Electrolytes,
3) BUN, creatinine,
4) glucose
5) Lactate
6) Coagulation studies
7) Hepatic function
8) Calcium
9) Urinalysis
10) ECG
11) Chest radiograph
12) Pregnancy test (blood or urine)
13) 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.

The underlying etiology of shock should be aggressively treated

→ 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 eg - 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
  - (i.e. 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:

➔ Depending upon history, causes are of **2 types**.

i) 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 overdose, -osmotic substances, -polyureic phase of acute renal failure	-intense physical effort - exertional heat stroke -burns	- Peritonitis - Intestinal occlusion, - Pancreatitis, - Ascites, -Pleural effusions	

ii) HAEMORRHAGIC SHOCK

→ Due to loss of blood volume mostly due to

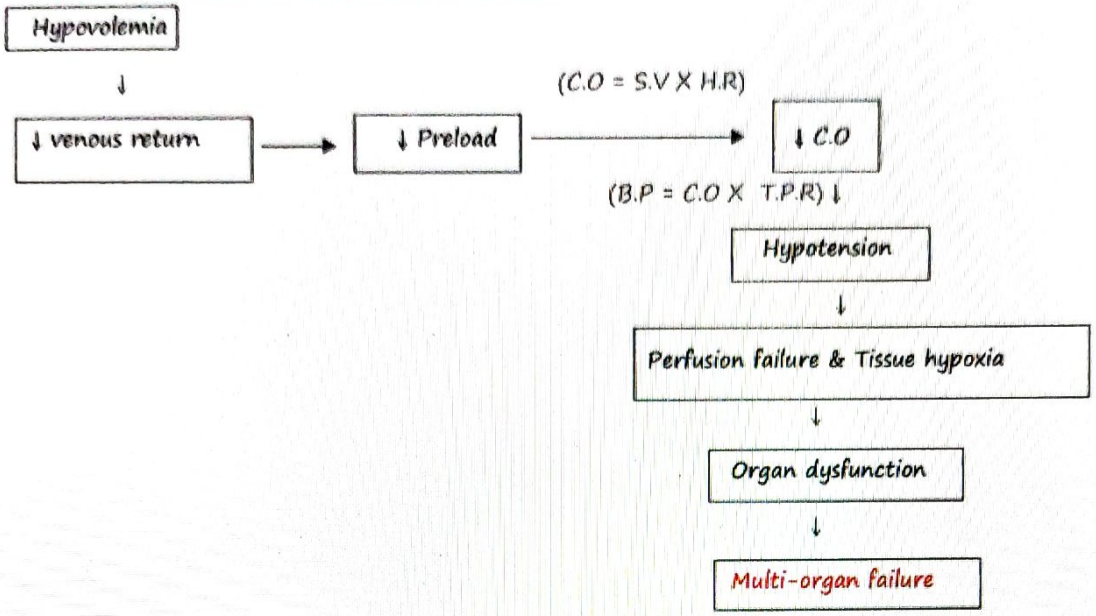
External blood loss	Exteriorization of internal bleeding	Internal bleeding	Traumatic shock
wounds	-hematemesis, - melena, -epistaxis, - hemoptysis	-hemothorax, - hemoperitoneum, - splenic rupture	

ATLS [Advanced Trauma Life Support] CLASSIFICATION OF HEMORRHAGIC SHOCK:

	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:



→ So, stop at the level of ↓ C.O

→ Resuscitate , so that preload, C.O can be maintained & hypotension, perfusion failure can be prevented

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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:

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.

→ 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)

→ **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

*1gm 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.**

SEPTIC SHOCK

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Definition

→ 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

1. **Urgent assessment and treatment** –
   a. initial fluid resuscitation to prevent end-organ dysfunction and
   b. control the source of infection.
2. **Laboratory results**
3. 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.

COMA- APPROACH IN EMERGENCY DEPARTMENT

COMA – 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

1. AROUSAL & its underlying neural substrate → depends on **Ascending Reticular Activating System (ARAS) & Diencephalon**
2. AWARENESS → requires the functioning of **Cerebral Cortex.**

- Any problem in ARAS/ Diencephalon/cerebral cortex → results in Coma
  Eg- drugs, trauma etc can affect these systems resulting in coma

DEFINITION OF COMA:

→ 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:

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 /CO2 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

GLASGOW COMA SCALE:

➔ **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:

- 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 like
    - -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:

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:

- 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.

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Seizure / Status Epilepticus- Approach in Emergency Department

Seizure

→ **Seizure** result from abnormal, excessive activity of the CNS

Generalized	Focal (Partial)
-Involves both hemispheres of the brain with loss of consciousness	-Involves only one hemisphere

Simple Partial Seizures	Complex Partial Seizures
-Cognition is not impaired	-Cognition is impaired

Partial Seizure with Secondary Generalization
-Focal seizures may generalize to involve both cerebral hemispheres

Presentation

→ 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

→ 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

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

Etiologies of secondary, or reactive seizures:

- → 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

- → **Delirium Tremens**
  - Anxiety
  - Tremulousness
  - Altered mental status.
- → **Abnormal vital signs**
  - tachycardia,
  - hypertension,
  - hyperthermia
  - tachypnea.
- → Predominantly a clinical diagnosis

Eclampsia

- → 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

- → 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

- → 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

→ **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.

ACUTE RESPIRATORY DISTRESS SYNDROME

CARDIOGENIC PULMONARY EDEMA → ↑ left atrial pressure secondary to LVF → edematous fluid is TRANSUDATE

Acute Respiratory Distress syndrome is due to NON-CARDIOGENIC PULMONARY EDEMA

NON-CARDIOGENIC PULMONARY EDEMA

Eg: ARDS occurs secondary to swine flu/bird flu. Because swine flu virus ultimately damages Pneumocytes. That will end up in ARDS.

↓

Collapse of alveoli

↓

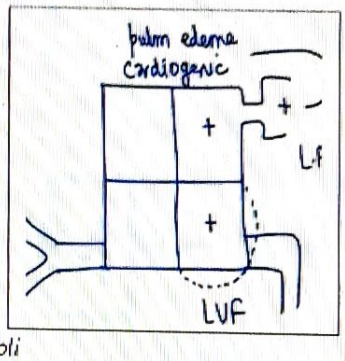
Hypoxia

↓

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)
- \+ **↓ pO2** (demonstrable hypoxia)
- \+ **Normal** left atrial pressure (LAP)

Triggers for ARDS:

Direct (M/C)

- Pneumonia (H1N1)
- **Mendelson** syndrome
- Toxic gas inhalation
- Pulmonary contusion
- Near drowning

Indirect

- **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 one liners:

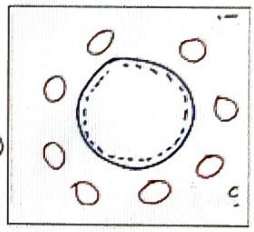
- Both **pneumonia** > **sepsis** are the leading causes of ARDS
- Leading cause of death after blood transfusion – **TRALI**
- All of the followings are the causes of ARDS **except**:
  - **Status asthmaticus** (disease of airway)
- There would be **no CXR findings** in fat embolism

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**)

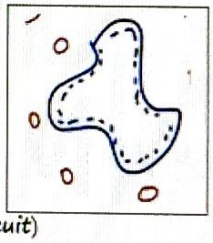
IN ARDS,

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

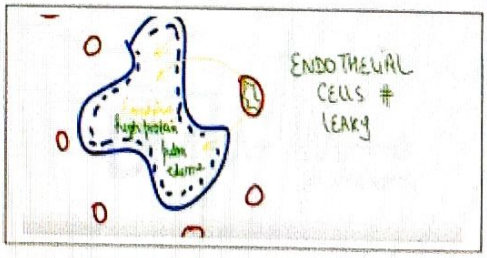
↓ Endothelial cells become LEAKY RESULTING IN

Exudative, high protein pulmonary edema

↓ forming a layer resulting in

Further ↓ of gas exchange

- In heart failure → **low protein** pulmonary edema



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FEATURES: (Sudden onset Respiratory Distress)

EXUDATIVE PHASE → (0-7 days)
↓
12-36 hrs
Triggers
↓

PROLIFERATIVE PHASE → (7-21 days)
[able to wean off the ventilator]
↓
proliferation of
type 2 pneumocytes
↓
Some differentiate into
type- 1 pneumocytes
[recovery is possible]

FIBROTIC PHASE OF LUNGS (>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: ↓ CO2
- ↓ pO2, ↑ pCO2 (Refractory hypoxia)
- Dead space ↑

} Type 2 Respiratory failure / Respiratory acidosis

→ In initial phase of ARDS → There will be Respiratory alkalosis (CO2 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 CO2 overproduction in lungs

→ When Pco_2 = 60mm of Hg, then the compensatory mechanisms begin to fail.

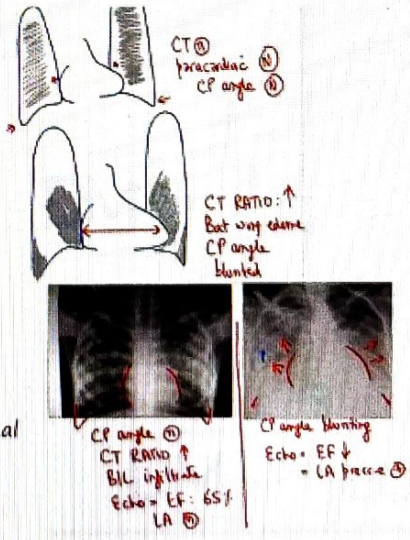
Note :

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

Work -up:

1. 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
2. Echo- ejection fraction is normal, LA pressure - normal



In cardiogenic pulmonary edema, echo shows

- Decrease ejection fraction
- LA pressure increased

3. ABG - ↓ pO2, ↑ pCo2 [ventilation, perfusion imbalance]

Key words: - essentials of diagnosis:

1. Sudden onset resp. distress

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

2. CXR: B/L pulmonary infiltrates
3. Pao2 / 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	Pao2 / Fio2
- Mild	<300
- Moderate	<200
- Severe	<100

*BERLIN Criteria – 1+2+3+4

Rx of VOLUTRAUMA – seen in high/normal volume ventilation resulting in PNEUMOTHORAX

→ 1. 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 H2o
- Respiratory rate of ventilator = <35/min

2. **Prone** position ventilation
   - Risk of **extubation**, CVP line lost, **orthopedic injuries**
3. Extracorporeal membrane oxygenation **(ECMO)**- Pt. in which heart and lung are too weak for ventilation
4. **Limited fluids** & diuretics → To maintain normal LA pressure
5. **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
1.**Transudative** pulmonary edema	**Exudative** pulmonary edema
2.Hydrostatic pressure **increases**	Hydrostatic pressure **normal**
3.LA pressure **increased**	LA pressure normal
4.Low protein pulmonary edema	**High** protein pulmonary edema
5. BNP **increased**	BNP normal

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THORACIC AORTIC DISSECTION-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:

- 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 of 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:

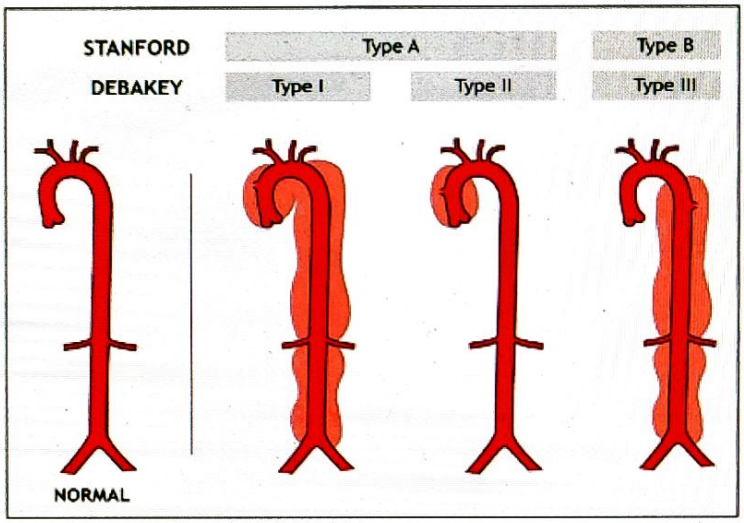
- Genetic syndromes –
  - Marfan syndrome,
  - Loeys-Dietz syndrome,
  - Turner syndrome,
  - Ehlers-Danlos syndrome (vascular type)
- Acute and chronic cardiovascular conditions.
- Family history of aortic dissection or aneurysm [vascular malformation]
- Personal history of :
  - Aortic aneurysm or
  - 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.

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:

➔ 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:

→ 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:

→ **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:

- ➔ 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:

→ 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

ABDOMINAL PAIN – APPROACH IN EMERGENCY DEPARTMENT

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 – Airway

B – Breathing → should be assessed first

C- Circulation

→ 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

E.g. Abdominal pain

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

→ 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
  - Kidney etc

} Flank pain/ Back pain etc.

→ Gaseous distension of organ
→ Ischemia of the organ
→ Stretching of the organ
→ Torsion of the organ

} Causes visceral pain

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→ 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

→ Onset
  - **Sudden** - A.A.A, ruptured ectopic pregnancy
  - **Gradual** – peritonitis, obstruction(mostly)

→ P – Position /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
- Eg: 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)
- Eg- patient with increasing pain – emergency situations

Physical examination

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 etc

Extra abdominal findings

→ helps in establishing diagnosis

Investigations

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
→ For :
  - Abdominal aortic aneurysm diagnosis,
  - Gall bladder &
  - Genitourinary pathology diagnosis

CT scan:

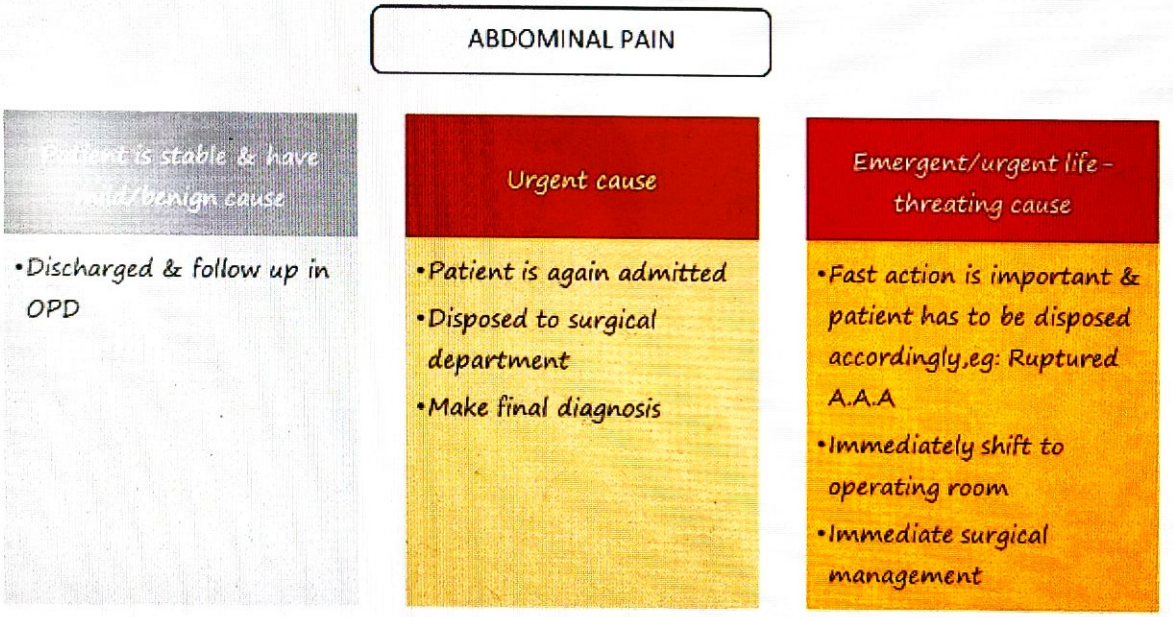
→ Good for most intra-abdominal procedures

→ Very good for vascular lesions with contrast

Disposition

→ 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



→ Disposition – whether patient is disposed to ward /O.R. /discharged → It depends on

- History
- Examination
- Investigations

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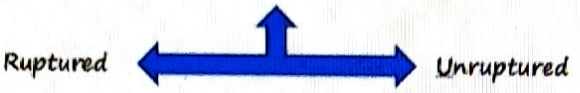
Abdominal Aortic Aneurysm- Approach in Emergency Department

Abdominal Aortic Aneurysm (AAA)

- Abdominal Aortic aneurysm can present in 2 states



→ 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

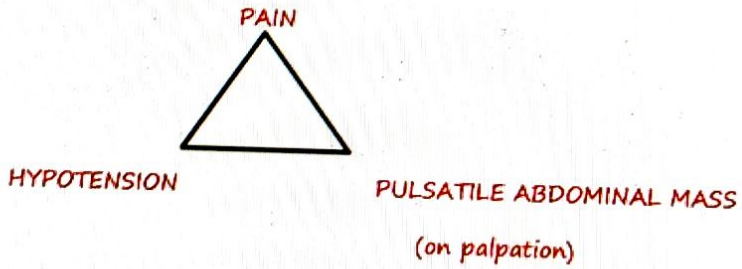
→ If an elderly patient /patient with history of P.V.D presents to E.D with similar complaints then DD of **abdominal aortic aneurysm** should always strike the mind

→ Misdiagnosed as :

- 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)



Unruptured A.A.A

→ **Incidental** diagnosis

→ Patient may complain of

- Abdominal, back/flank pain
- Abdominal mass, fullness, sensations of abdominal pulsations

Ruptured A.A.A

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

Case study

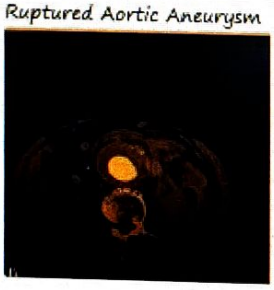
- A 65-year old male with hypotension, presented with intense, sudden abdominal pain & on palpation, pulsatile abdominal mass was found
- The patient showed a classical triad of ruptured A.A.A
- When a patient has known/suspected rupture A.A.A, the patient should be considered unstable
- As a primary survey; we should see A.B.C of the patient (Airway, Breathing, Circulation)
- Generally, **circulation is initially compromised**
- Hemodynamically unstable patient with **known / suspected ruptured A.A.A** 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 A.A.A causes hypovolemia → Ventricular fibrillation → cardiac arrest

Investigations

→ **Ultrasound** is the **ideal study** for detection of A.A.A
  - Sensitive
  - Done bedside
  - 99% cases of ruptured A.A.A 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**

→ **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 oneliners:

- → 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

ANAPHYLAXIS- Approach in Emergency Department

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 blocking agent → Gamma globulin → Antisera → Exercise

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

→ **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

→ **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

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Hypothermia – Approach in Emergency Department

Hypothermia

- 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

   → 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.

B. Trench foot

   → In lower extremities with prolonged exposure to wet and cold conditions develop tissue damage,
   → Presents as
      - paresthesia's,
      - pain,
      - numbness.

Degree of Hypothermia	Mild (32-35° C)	Moderate (28-32° C)	Severe (<28°C)
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

A. Frost Bite-

- Tissues - pale & Firm,
- poor capillary refill and sensation.
- Severe Frost Bite appears purple due to blood Sludging.

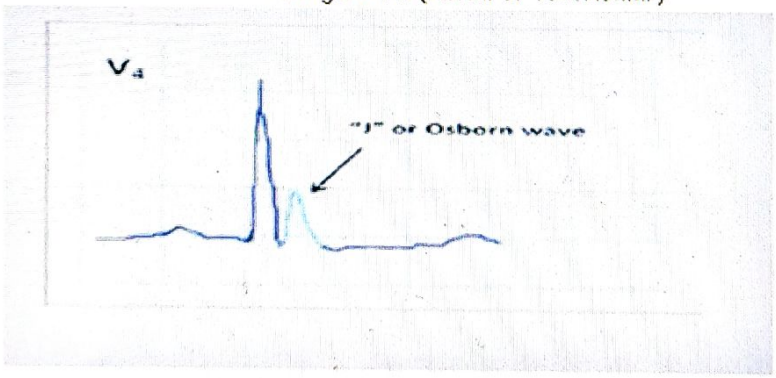
B. Trench Foot-

- Skin - red swollen at first,
- progressing to blisters and bullae and finally
- Hemorrhage in the skin and deeper tissues

Initial Stabilization and Primary survey

→ 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
  - Abnormal Coagulation,
  - 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

- → 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 °c 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

→ 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.

HYPERTHERMIA – APPROACH IN EMERGENCY DEPARTMENT

Hyperthermia

- → 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
  - **most severe** manifestations of heat related illness
  - Associated with neurologic dysfunction and multi-system organ damage.

Clinical features

- → 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

- → 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.

Types	Classical heat stroke	Exertional heat stroke
Defination	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	↑↑	↑↑↑↑

Clinical features

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

Risk factors

- → Obesity
- → Poor physical fitness.
- → Precedent illness
- → Lack of acclimatization

- **Heat cramps**
  - 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

- → 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

- 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**.

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)

Treatment

→ 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

→ 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.

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SUICIDAL EMERGENCY MANAGEMENT

SUICIDAL EMERGENCY MANAGEMENT

- Emergency providers recognize and provide care for patients who attempt to harm themselves or to commit suicide
- → The risk of suicide increases 600 % after a single presentation for self-harm
- → 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:

- → 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:

- → 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
- → Consider
  - 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:

- → 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:

- to see how much risk is present in these patients / whether this pt needs further psychiatric evaluation or not.

MOMMAS2

- 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
- A- 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?

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→RISK FACTORS:

- → 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**
  - 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*
- → 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.