STEM-S

ESSENTIAL NOTES

Surgical Group

PRESENTED BY
Stem-S

- Definition: any vessel size increased by >50%.
- Normal vessel size - 2 cm
- If vessel size > 3 cm - aneurysm
- 2-3 cm - ectasia
- Most common site of aneurysm is infra renal abdominal aorta.
- Reason: the elastic lamella in the wall is very less. More chance of dissection or aneurysm.

Term	Definition / Key Features
Ectatic vessel	Abnormal dilation of a blood vessel **not meeting aneurysm criteria**.
Arteriomegaly	• Large, smooth, diffusely dilated, tortuous arteries **without focal aneurysms**. • **36% have family history**. • Higher risk of aneurysm formation.
True aneurysm	Dilation of **all three arterial layers** (intima, media, adventitia) to **>1.5 × normal diameter**.
False aneurysm / Pseudoaneurysm	• **Disruption of all three arterial wall layers** → blood contained by surrounding tissue. • Usually iatrogenic or traumatic. • **High rupture risk**.
Fusiform aneurysm	**Symmetric, circumferential** dilation of the entire artery diameter.
Saccular aneurysm	**Localized, asymmetric, eccentric** outpouching; usually focal weakness.

• Classification of Aneurysm

Based on etiology	Based on shape of aneurysm	Based on the wall	Based on presentation
o Degenerative aneurysm o Infection o Inflammatory o Trauma	- Fusiform - Saccular - - concentric or eccentric aneurysm	- All 3 layers are involved - True aneurysm - Does not have all layers - Pseudo aneurysm	- Asymptomatic - Symptomatic aneurysm: - It can be either complicated (embolism, rupture or fistula) - Uncomplicated

- **Risk Factors of Etiology:**

Abdominal aortic aneurysms	Thoracoabdominal aneurysm
- Atherosclerosis - Inflammation - Infection - Trauma - Connective tissue disorder	- Aortic dissection - Connective tissue disorder. - Infection - Inflammation - Atherosclerosis (Co- existing condition; not an etiology)

DM is considered protective for AAA

- Weakening of the arterial wall and increased local hemodynamic forces - MMP and proteolytic enzymes in wall
- "Staccato" pattern of growth
- 3-4 mm/yr.
- larger diameter- faster growth

- Marfan syndrome, familial thoracic aortic aneurysm and dissection, and vascular-type Ehlers-Danlos
- degradation of extracellular matrix
- reduction of elastin concentration
- role of matrix metalloproteinases and deficits of antiproteolytic enzymes
- role of the immune response and hormone milieu
- a degenerative complication after AD.

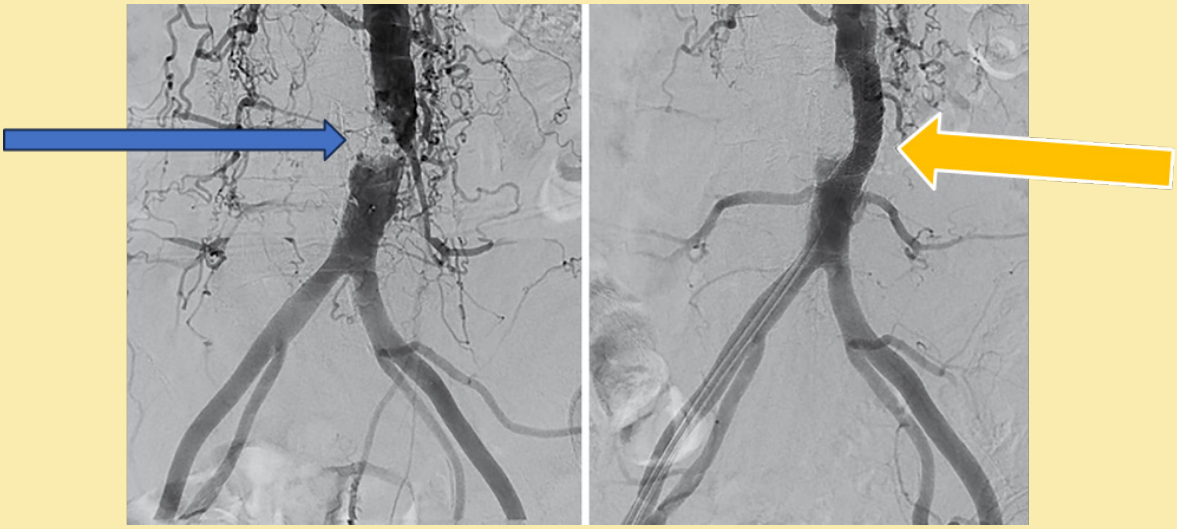
- **RISK FACTORS for AAA:**

AAA Development	AAA Expansion	AAA Rupture
· Tobacco use · Hypercholesterolemia · Hypertension · Male gender · Family history (male predominance)	· Advanced age · Severe cardiac disease · Previous stroke · Tobacco use · Cardiac or renal transplantation	· Female gender · ↓ FEV_1 · Larger initial abdominal aortic diameter · Higher mean blood pressure · Current tobacco use (duration > amount) · Cardiac or renal transplantation · Critical wall stress–wall strength relationship

VASCULAR SURGERY

Category	Key Points
Epidemiology	• Account for **5% of intra-abdominal aneurysms** • Typically present in the **6th decade of life**
Etiologic Background	**Historically:** syphilis **Currently:** mostly atherosclerotic or secondary to medial degeneration (e.g., FMD) Associated with: • Collagen vascular diseases • Inflammatory conditions (e.g., arteritis) • Rare inherited disorders (e.g., **EDS, NF-1**)
Clinical Presentation	• Often **asymptomatic**, detected incidentally • May present with **life-threatening rupture** causing abdominal pain + hemorrhagic shock
Rupture Risk	**Historical:** 25–70% **Contemporary:** 10–15% (due to endovascular techniques + better perioperative care)
Diagnosis	• **CTA**, **MRA**, **DSA**
Rx	• Surgical repair has remained the mainstay of treatment, endovascular techniques are increasingly applied for treatment of both elective and ruptured cases. • Endovascular therapy alternatives include • Treatment of both the aneurysm and pathologic vessel (i.e., the isolation technique) • Aneurysm exclusion with parent vessel flow preservation (i.e., the exclusion technique) • Direct percutaneous puncture and embolization with thrombin, glue, or ethylene vinyl alcohol copolymer (EVOH)

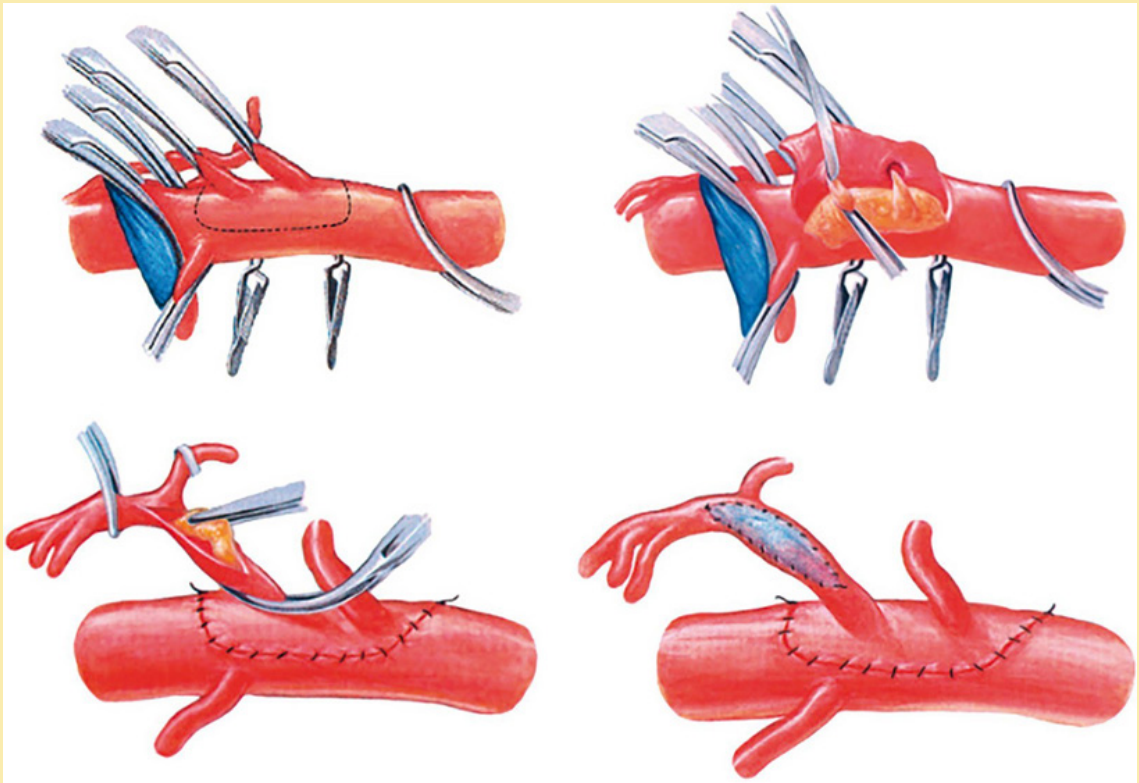
Feature	Splenic Artery Aneurysm (SAA)	Hepatic Artery Aneurysm (HAA)	Superior Mesenteric Artery Aneurysm (SMAA)
Epidemiology	• Most common splanchnic aneurysm (60%) • 3rd most common intra-abdominal aneurysm • 2–4× more common in females • Strong association with multiparity	• 20% of splanchnic aneurysms • Male : Female = 2 : 1 • Mostly extrahepatic, solitary • Common & right hepatic arteries most involved • <30% intrahepatic	• 8% of splanchnic aneurysms • More common in males
Etiology / Risk Factors	• Arterial dysplasia • Portal HTN • Local inflammation • Hormonal & hemodynamic stress • Hypertension (50%) • Up to 20% synchronous aneurysms • Dissection • PAN, SLE	• Atherosclerosis (most common) • PAN, cystic medial necrosis • Portal HTN, FMD • Other acquired arteriopathies • Iatrogenic pseudoaneurysm (~50%)	• Atherosclerosis • Infection, dissection • Medial degeneration • FMD, vasculitis • Connective tissue disorders • Trauma • Pseudoaneurysm: pancreatitis, PUD
Clinical Features / Course	• Rupture into lesser sac → temporary containment • Second rupture into peritoneum → double rupture phenomenon → rapid collapse	• RUQ / epigastric pain radiating to back • GI bleed from erosion into stomach/duodenum • Biliary obstruction → hemobilia, jaundice	• Abdominal pain, nausea, emesis, GI bleeding • Fever in mycotic aneurysm • Pulsatile mass in ~⅓
Natural History / Risks	• High rupture risk in pregnancy & portal HTN	• Risk of rupture, GI bleed, biliary obstruction	• Progressive expansion & rupture • Thromboembolism → acute mesenteric ischemia
Operative Indications	• All ruptured aneurysms • All pseudoaneurysms • Any size in women of childbearing age • >3 cm, enlarging, or symptomatic • Portal HTN (esp. transplant candidates) • Non-atherosclerotic aneurysms • Rapid growth	• All pseudoaneurysms • All symptomatic aneurysms • All vasculopathy/vasculitis-related aneurysms • True aneurysm >2 cm (low risk) • >5 cm if open repair in high-risk patients	• Repair all true and false aneurysms regardless of size • Exception: dissection-related aneurysms → observe unless refractory
Surveillance	• Annual CT/US for: – <3 cm – Stable, asymptomatic true aneurysms – High comorbidity / limited life expectancy	–	–
Vaccination	• If splenectomy → vaccinate ≥ POD 14 to reduce OPSI	–	–
Preferred Treatment	• Endovascular therapy = treatment of choice	Depends on anatomy & hepatic perfusion	Depends on anatomy & bowel perfusion
Endovascular Techniques	• Isolation technique preferred • Coil embolization: efferent first → sac + afferent • Coils oversized by 10–20%	• Covered stents preferred (>4 mm vessels, 1–2 cm seal) • Embolize sac branches to prevent reperfusion • Intrahepatic → arterial embolization	• Stent graft + embolization increasingly used • Limited by thromboembolism & endoleak
Open Surgical Management	–	• Revascularization with autologous vein • Coil embolization discouraged if high ischemic risk • May require liver lobe resection	• Aortomesenteric bypass • Aneurysmorrhaphy or branch ligation if collaterals adequate



ENDARTERCTOMY:

- Infra renal aorta – Transperitoneal – midline laparotomy – easier app
- Visceral segments involvement(supra renal) – Thoraco-laparotomy – RP exposure

Trapdoor Aortotomy - facilitate endarterectomy and complete clearance of the plaque from each of the visceral orifices - may be combined with bypass to the visceral vessels or even replacement of a segment of the aorta if adequate end points cannot be obtained



• ARTERIAL DISEASE AND EVALUATION

Occlusive diseases:

- Pathology: atherosclerosis, vasculitis (TAO) hypercoagulation.
- Systemic illness
- Approach of these patients;

Bedside evaluation	Imaging
· ABPI · TBI · Segmental pressure assessments · Pulse volume recording	· Duplex · CTA · MRA · DSA

ABPI: Ankle Branchial Pressure Index;

- Measures the significance of occlusion
- Assumes patient's UL is free of disease.
- Calculated as

SBP of anterior/posterior tibial artery (highest)

SBP of branchial artery (rt. Or left) highest

- Measured with hand doppler or BP cuff
- Normal ABPI is 0.9 to 1.1

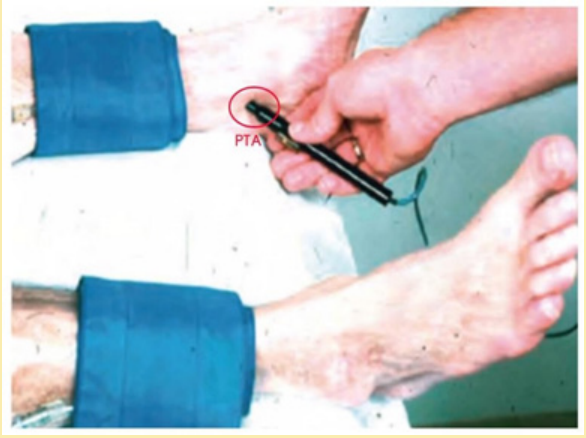
– > 1.1 - Calcified artery

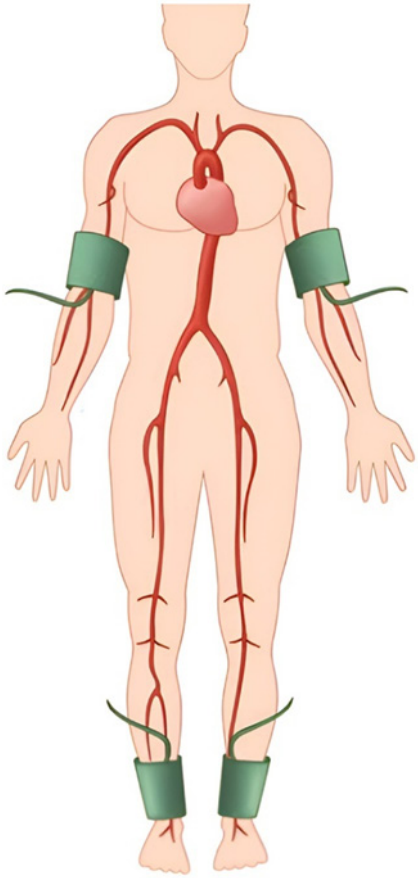
– < 0.9 >0.7 Mild disease

– < 0.7 > 0.4 Moderate disease

– < 0.4 severe disease

- Underlying CAD - ABPI <0.9





Right ABI = ratio of
Higher of the right ankle systolic pressures (posterior tibial or dorsalis pedis)
Higher arm systolic pressure (left or right arm)
Left ABI = ratio of
Higher of the left ankle systolic pressures (posterior tibial or dorsalis pedis)
Higher arm systolic pressure (left or right arm)

pediatric surgery

Term	Definition
Preterm	< 37 completed weeks of gestation
Full term	Between 37 and 42 completed weeks of gestation
Neonate	Newborn baby up to 28 days after the estimated date of delivery (EDD)
Infant	Up to 1 year of age
Child	All ages up to 16 years (minimum school leaving age), divided into preschool child (usually < 5 years), child, and adolescent (puberty up to 16 years)
Young person	A popular term referring to anyone under 18 years

Aspect	Infants (vs Older Children) – Key Points
Facts	· Wide abdomen • Broad costal margin • Shallow pelvis • Liver edge extends below the costal margin • Bladder is partly intra-abdominal • Ribs are more horizontal and flexible • Umbilicus is relatively low-lying
Implications	• Transverse supra-umbilical incisions provide better access than vertical midline incisions • Abdominal trauma (including surgical incisions) can easily damage the liver or bladder • Rib geometry necessitates greater diaphragmatic movement for ventilation compared to adults • Lower abdominal stomas must be carefully sited so that the stoma bag does not interfere with the umbilicus

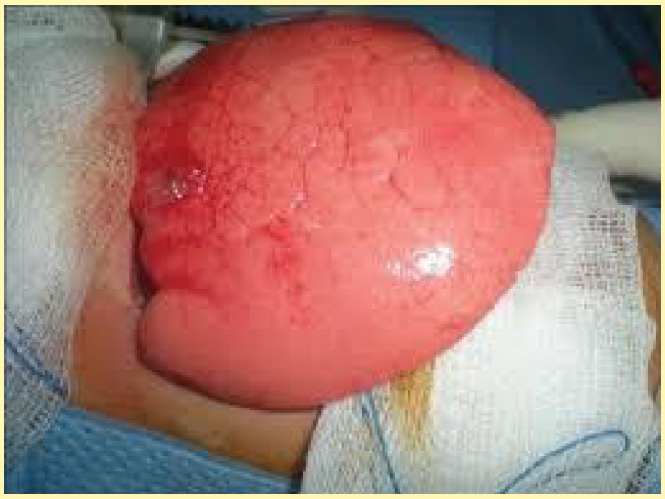
EPIDERMOID CYST	DERMOID CYST
Slow growing benign lesions in scalp and skull	
Contents: Epidermal tissue & Keratin debris Rx: Excision	Commonly occurs @ Anterior fontanelle Forehead Lateral corners of eyebrow Posterior Auricular area Rx: R/O Intracranial extension f/b Excision

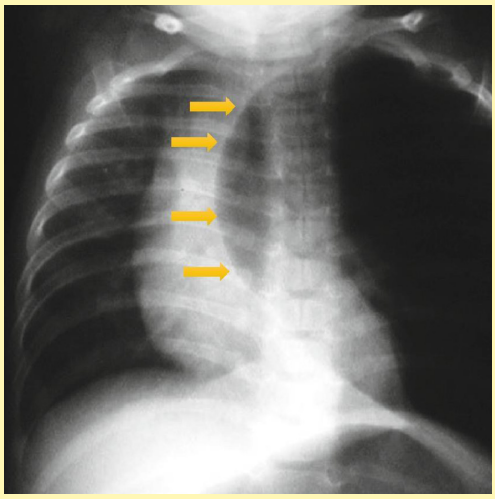
The neonatal lung has fewer type II pneumocytes, which produce surfactant, a lipoprotein mixture of phospholipid, protein, and neutral fats.

- Surfactant regulates alveolar surface tension, thereby increasing functional residual capacity.
- **Lecithin, the most predominant phospholipid, can be measured in amniotic fluid, and the lecithin- to-sphingomyelin ratio is used to determine fetal lung maturity.**
- Hence, premature infants are at greater risk for alveolar collapse, hyaline membrane formation, and barotrauma from mechanical ventilatory support.
- Exogenous surfactant therapy has had a major impact on the management of premature infants.
- This has resulted in improved survival and decreased incidence of bronchopulmonary dysplasia, a condition characterized by oxygen dependence, radiologic abnormality, and chronic respiratory symptoms beyond the first 28 days of life.

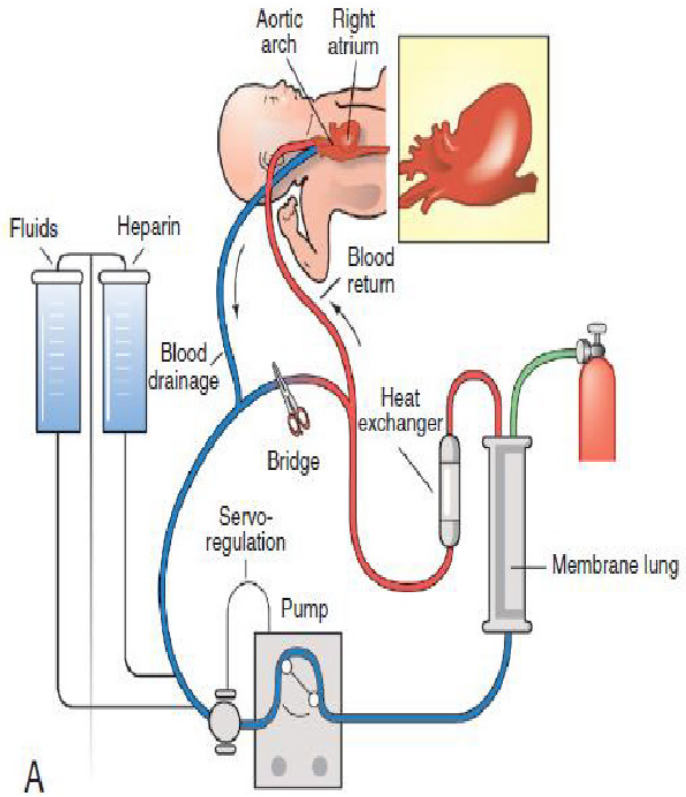
NUTRITION:

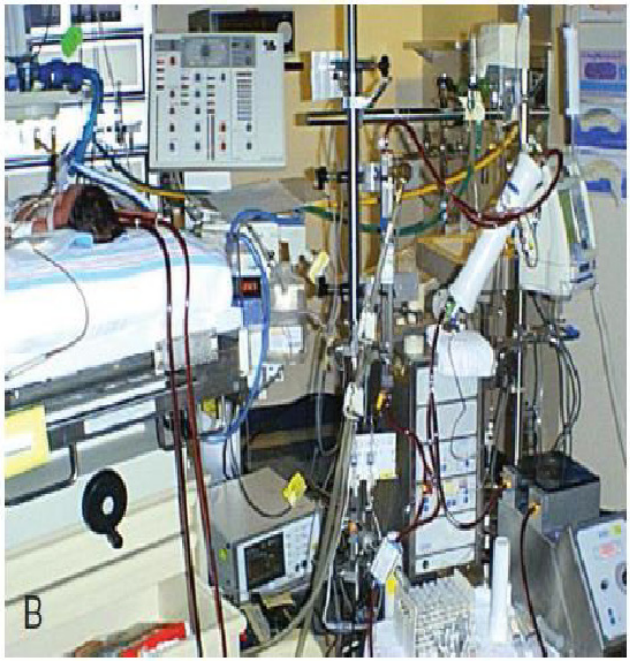
- Infants only have energy reserve to withstand short periods of starvation(2-3 days)
- **infant's need for parenteral nutrition addressed promptly**
- total TPN **infusion rate kept at steady-state** to meet daily fluid requirements
- **concentration of nutrients gradually increased daily until goals are met**
- **TPN associated cholestasis:**
  - seen in infants with surgical conditions
  - due to by prolonged TPN support
  - rule out other causes causes
  - Sequence of level elevation- serum bile acid → direct bilirubin → liver enzymes
  - Ideal treatment - **restoring enteral feeding**
  - Prevention - **omega-3 fat emulsion (Omegaven)**





CDH	EP	C/F	Mx
Failure of closure of pleuroperitoneal canal in the developing fetus. Left side > right	Pleuroperitoneal cavities become separated by the developing membrane during weeks 8 to 10 of gestation Process fails, the pleuroperitoneal canal does not close, and a posterolateral diaphragmatic defect. Posterolateral - Bochdalek hernia (most common) Anteromedial - Morgagni hernia (Space of Larry) ; m/c organ=Transverse Colon Ipsilateral lung is affected more severely, however both lungs are affected by pulmonary hypoplasia. Unfavorable outcome -Factors: Earlier detection(Antenatal); Late onset CDH is small in size usually Liver/Stomach on top(Inside abdominal cavity) Larger defect Rt side Polyhydramnios LHR <1 ; Associated anomalies	LHR: Lung area to Head circumference ratio Sonographic predictor of prognosis LHR= Lung Head Ratio o/e LHR: % of expected mean for gestational age of the Observed/Expected LHR **36%-45%** — **Mild** 26-35% — Moderate 15-25% — Severe <15% — Extreme LHR less than 1 and abnormal liver position at 24 weeks – unfavourable outcomes. Pulmonary vasculature increased thickness of arteriolar smooth muscle.(Muscularization of pulmonary vasculature) Severity of pulmonary hypoplasia and pulmonary hypertension significantly affect the overall morbidity and mortality in CDH infants. Honeymoon period. The most reliable prenatal predictor of postnatal survival is absence of liver herniation.	





Two guidelines for predictors of survival without ECMO:

1 Alveolar–arterial difference in partial pressure of oxygen (PAO_2 – PaO_2)$AaDO_2$

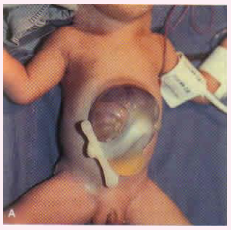
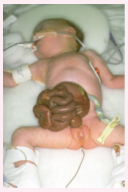
Criteria for ECMO:

1 $AaDO_2$ > 610 for longer than 8–12 hours
2 $AaDO_2$ > 620 for 6 hours
3 Associated with extensive barotrauma
4 Severe hypotension requiring inotropic support

2 Oxygen Index (OI)

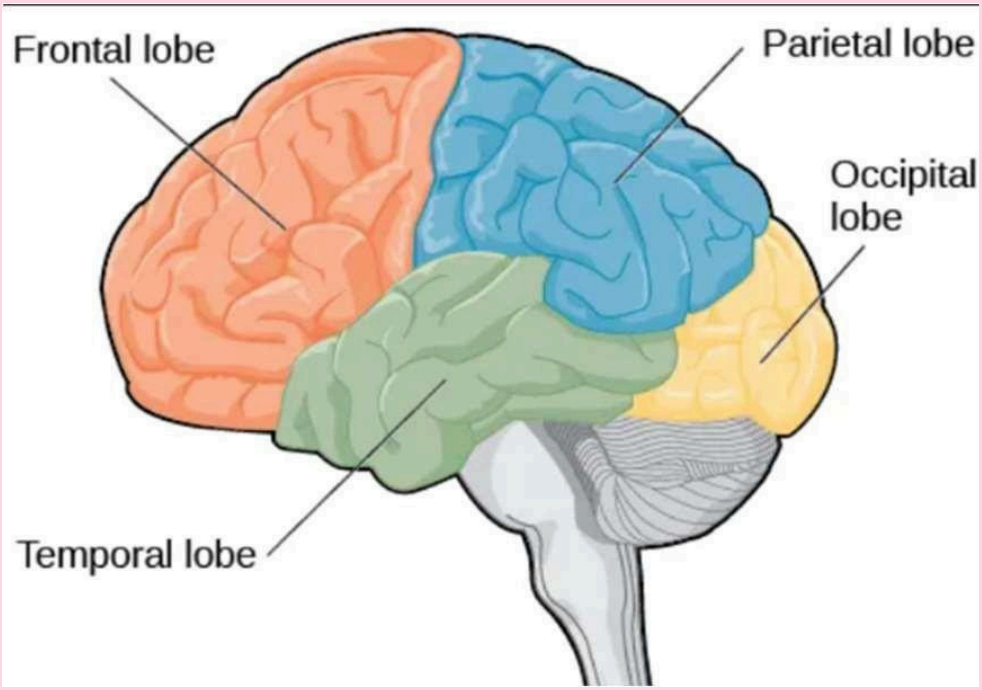
1 Fraction of inspired oxygen (usually 1.0) × mean airway pressure × 100, divided by PaO_2
2 Oxygen Index > 40 → ~80% mortality

ABDOMINAL WALL CONDITIONS

Feature	Exomphalos / Omphalocele	Gastroschisis
Basic definition	Central defect of abdominal wall with herniation of viscera	Bowel faces the sky; no covering sac
Site of defect	Central, in continuity with umbilical cord 	Right of umbilical cord (obliterated right umbilical vein) 
Size of defect	Usually > 4 cm Defects < 4 cm → hernia of the cord	Fascial defect ~ 4 cm
Covering sac	Present – three-layered membrane	Absent
Composition of sac	Peritoneum + Wharton's jelly + Amnion	—
Effect of amniotic fluid	Protected by sac	Direct exposure → bowel thickening, edema, inflammation
Associated anomalies	~ 50% associated anomalies	Much fewer associated anomalies
Chromosomal associations	Trisomies 13, 15, 18, 21	Not mentioned
Syndromic association	Beckwith–Wiedemann syndrome	—
Intestinal atresia	—	15% of cases ↑ risk of short bowel syndrome
Timing of atresia repair	—	Addressed after 6–8 weeks (after inflammation subsides)
NEC (late occurrence)	—	20% of patients
Subtypes	Exomphalos minor: < 5 cm, liver not involved Exomphalos major: liver commonly involved	—
Initial management (sac care)	Sac dressing with topical antibacterial agents (manuka honey, silver sulfadiazine) → epithelialisation	—
Primary surgical management	Primary closure for small–medium defects	Primary reduction successful in 50–80%
When primary closure not possible	Prosthetic patch (Gore-Tex), skin flap, or silo	Silo (silastic bag with ringed edge) / sutureless closure
Giant defect management	Topical escharotic agents (povidone-iodine, merbromin, silver nitrate) → gradual epithelialisation	Serial reduction of bowel over 5–7 days
Long-term outcome	Results in ventral hernia, repaired electively at 3–4 years	—

Neurosurgery

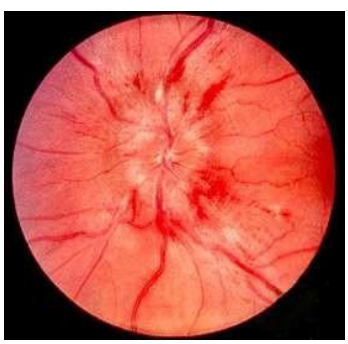
LOBE	AREAS & FUNCTIONS		SYMPTOMS
FRONTAL	Primary motor area Broca's area (Posterior Inferior) Executive function Decision making Emotional control		C/L Hemiplegia/hemiparesis Broca's aphasia (Words output is decreased; words have meaning)
PARIETAL	Primary Sensory area Spatial relationship(self & environment) Angular gyrus Supramarginal gyrus	Calculation Writing Right -Left coordination Recognition of body parts	GERSTMANN SYNDROME Acalculia Agraphia Right-left incoordination Finger agnosia
OCCIPITAL	Primary Visual cortex		C/L Hemianopia
TEMPORAL	Wernicke's aphasia(Postero superior) Primary auditory cortex Inferior Optic radiation Memory circuit		Wernicke's aphasia (Word salad; words don't have any meaning) B/L Cortical deafness Pie on sky Memory disturbances



VENTRICULAR SYSTEM

- At any point of time, 150 ml of CSF
- Total volume of CSF produced in a day: 500- 550 ml –18-20 ml/hr CSF is produced
- CSF turnover rate :3.4 times in a day.

Presentation of raised ICP in a patient

Adult	Child
Early morning headache Vomiting (It relieves headache) Why so ? During sleep (lying supine) ↓ Reduced venous drainage from brain + Hypoventilation during sleep ↓ ↑ $PaCO_2$ ↓ Cerebral vasodilatation ↓ ↑ Cerebral blood volume ↓ ↑ Intracranial pressure (ICP) ↓ Early morning headache Raised ICP ↓ Stimulation of vomiting center (medulla) ↓ Projectile vomiting ↓ Sudden fall in ICP ↓ Temporary relief of headache Visual disturbances(Papilledema) Lateral Rectus palsy	Incessant/ Irresistible cry/Refusal to feeds O/E :  Head circumference increased Bulging scalp veins Anterior fontanelle – Tense/ Bulging Upgaze paslsy Sunset sign- due to DORSAL MID BRAIN COMPRESSION Ddx : Sunset Sign : RICP Hydrocephalus (mcc= Aqueductal stenosis) FUNDOSCOPY : Papilledema – It is a LATE finding Margins blurred Hemorrhage 

Tier 1	Tier 2	Tier 3
Adequate anaesthesia and analgesia,often mainly in form of short acting continuous infusions that are paused intermittently A Ventriculostomy may be placed to drain CSF	Hyperosmolar therapy with Saline or Mannitol, creating a gradient to reduce edema in regions of brain with intact BBB Neuromuscular paralysis may be added at this time, with consideration of repeat CT imaging.	Rescue/Salvage therapies Interventions to decrease brain metabolism with barbiturate class medications and mild hypothermia, neither of which have demonstrated outcome benefit.

HEAD INJURY

Magnitude: 5% Incidence

-Most common cause of death (42%)
Causes: MCC of Head Trauma: RTA (Bailey) ; Falls (Sabiston)
Principles in Mx: The main aim is to reduce ICP ; to prevent secondary head injury.

Primary brain injuries	Secondary brain injuries
Skull Fractures ▪Intracranial Hematomas ▪Contusion ▪Diffuse axonal injury ▪Arterial dissection	Hypoxia Hypoperfusion Hypotension Increased ICP

Mechanism of head injury:

-Blunt
-Penetrating
-Specific foci or Diffuse injuries

PRIMARY	SECONDARY
At the time of trauma	Raised ICP • Hypoxia • Cerebral ischemia • Oedema

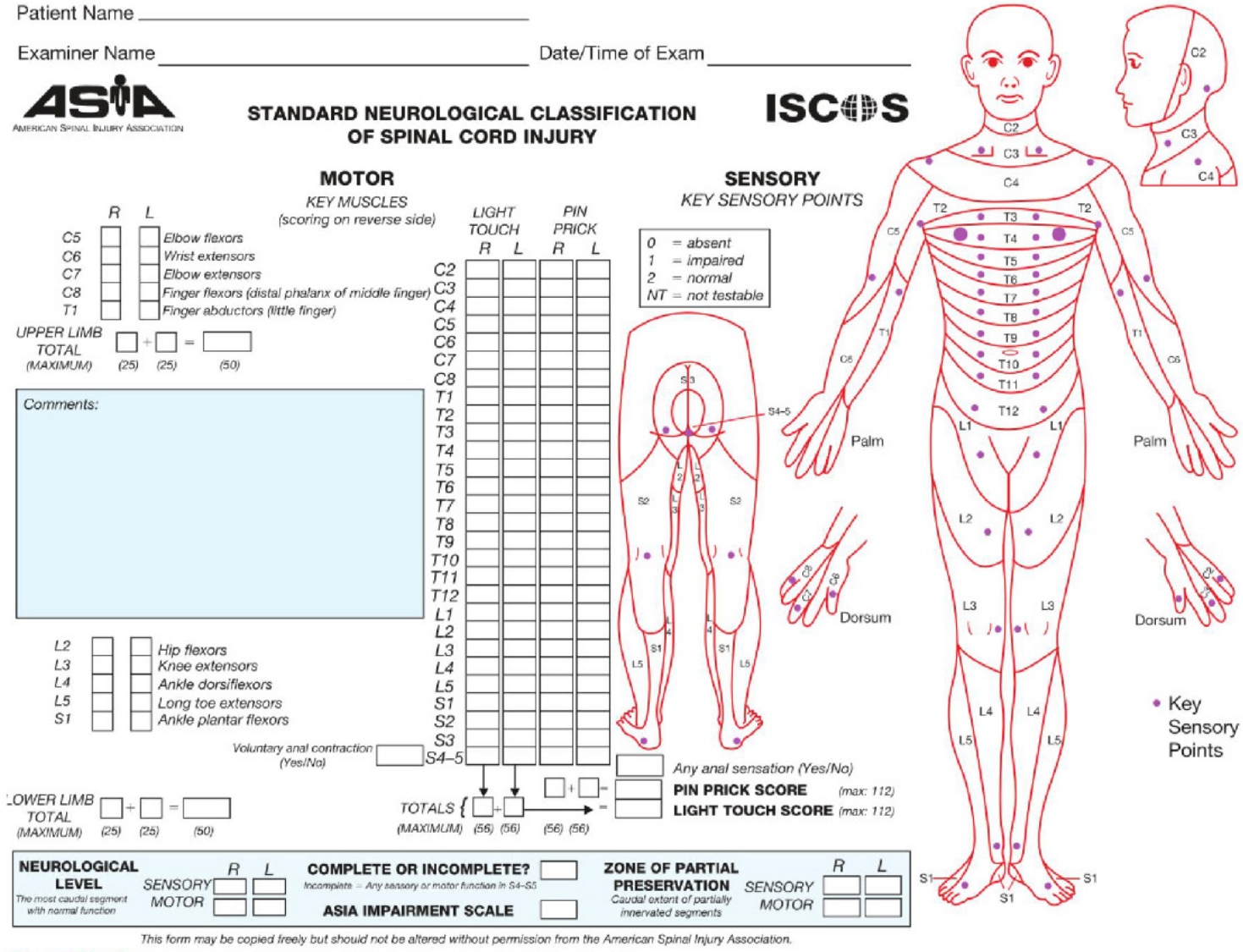
Types of head injury:
B/o Impact

EDH	Acute SDH	Chronic SDH
Low energy mechanism is sufficient	High energy mech	Even Minor trauma
Rupture of : Middle meningeal vessels	Cortical vessels	Cerebral atrophy in elderly people stretch bridging veins which rupture, bleed and then tamponade by itself.
Lucid interval seen	No lucid interval	No lucid interval
CT: Biconvex lesion	Diffuse concavo-convex (Crescent)	Diffuse concavo-convex (Crescent)
Indications of craniotomy: Clot volume > 30 cc Clot thickness > 1.5 cm Midline shift > 5mm		
Overall mortality is 10-20%	Mortality is 50%	

World federation of neurological surgeons grading for **Subarachnoid Hemorrhage**:

Grade	Glasgow coma scale	Focal deficits
I	15	-
II	13-14	-
III	13-14	+
IV	7-12	+/-
V	3-9	+/-

Focal deficit = dysplasia or limb weakness



This form may be copied freely but should not be altered without permission from the American Spinal Injury Association.

Figure 25.13 American Spinal Injury Association neurological evaluation.

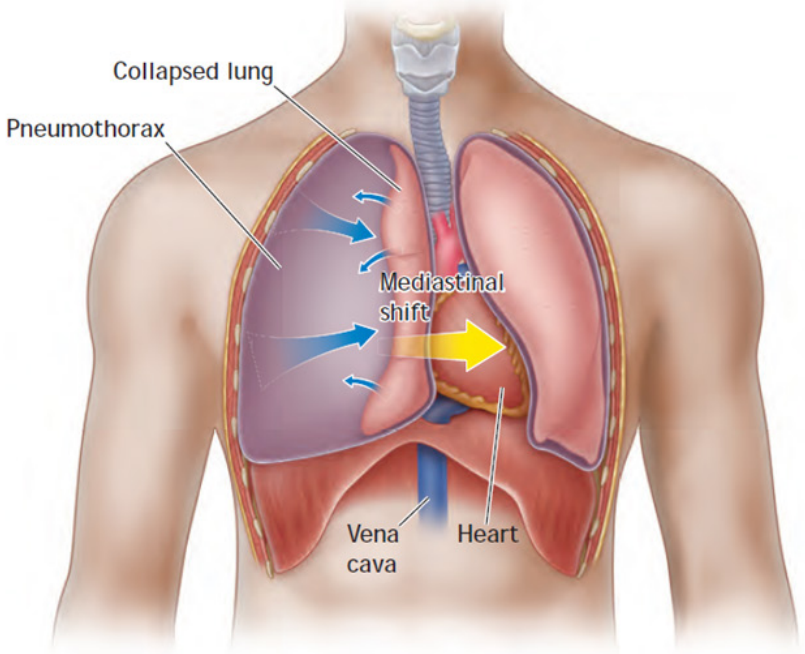
Evaluation : Gold standard = CT

MRI : when neurological deficit +/ when ligamentous injury is suspected

Burst #	Involves injury to anterior & middle column
Compression #	Only Anterior column
Mechanism of cervical spine injury	Direct drama to neck itself Motion of head relative to Axial skeleton (WHIPLASH INJURY or Severe flexion-extension injuries) Direct axial load imparted on Cranium (Fall from height causing burst fractures)
Thoracolumbar injury	Compression or burst Rotation or translation Distraction mechanisms

BREATHING & VENTILATION

Topic	Details
Airway vs Ventilation	Airway patency alone does not ensure adequate ventilation. Adequate gas exchange is required for oxygenation and elimination of carbon dioxide.
Requirements for Effective Ventilation	Requires adequate function of: • Lungs • Chest wall • Diaphragm
Immediate Life-Threatening Injuries Affecting Ventilation **(as per ATLS 11th edition)**	• Tension pneumothorax • Massive hemothorax • Open pneumothorax • Pulmonary contusion • Tracheal or bronchial injuries
Timing of Identification	These injuries should be identified during the **primary survey** and often require **immediate attention** to ensure effective ventilation.
Injuries Affecting Both Breathing and Circulation	Tension pneumothorax compromises ventilation and impairs venous return to the right heart, resulting in hypotension, respiratory distress, and decreased breath sounds on the affected side.
Emergency Intervention	Chest decompression is required as an emergency procedure to restore both ventilatory and circulatory function.
Oxygen Therapy	Every injured patient should initially receive supplemental oxygen, preferably via a mask-reservoir device to achieve optimal oxygenation.
Monitoring	Pulse oximetry is used to monitor adequacy of hemoglobin oxygen saturation; frequent reassessment is necessary.
Important Clinical Caution	A simple pneumothorax can convert to a tension pneumothorax after intubation and application of positive pressure ventilation.



■ FIGURE 4-1 Tension Pneumothorax. A "one-way valve" air leak occurs from the lung or through the chest wall, and air is forced into the thoracic cavity, eventually collapsing the affected lung.

The name **TENSION** indicates patient is **hemodynamically unstable (**It's a surgical emergency)

Treatment: Decompression in triangle of safety followed by ICD @ same site.

EMERGENCY RESUSCITATIVE THORACOTOMY:

INDICATIONS:

1. Salvageable post injury cardiac arrest:
2. Persistent severe post injury HypoTN (SBP<60 mmHg) seen in – Cardiac tamponade, Air embolism, Intrathoracic /intraabdominal Hemorrhage

What do you do?

1. Open pericardium longitudinally to relieve cardiac tamponade
2. Perform internal cardiac massage
3. Cross clamp the distal thoracic aorta
4. Manage intrathoracic bleeding.
5. Control of massive air leak

CRITERIA FOR SELECTION:

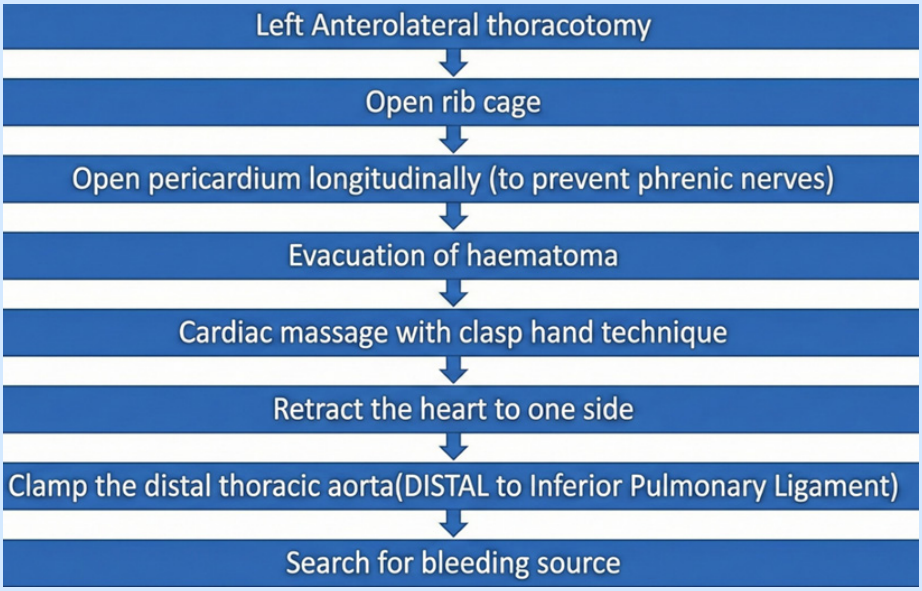
Patients with penetrating thoracic trauma who have signs of life reaching ER, with survival rates …. %

Blunt trauma patients usually have poor outcomes with survival rates as low as … %

C/I of ER Thoracotomy:

- CPR for more than 15 minutes (despite endotracheal intubation) in the presence of penetrating thoracic trauma;
- CPR for more than 10 minutes (despite endotracheal intubation) in the presence of blunt thoracic trauma;
- Blunt trauma when there have been no signs of life at the scene.

Emergency resuscitative Thoracotomy:



TRAUMA SCORING SYSTEMS

ANATOMICAL	PHYSIOLOGICAL
• AIS (not used now) • ISS • NISS(New ISS) : Sum of squares of 3 worst injuries irrespective of region	• GCS • RTS • TRISS= RTS

- **Scoring Systems:**

RTS score	TRISS Score	MESS Score
· R- Respiratory rate · T- Tie and see BP (Blood pressure) · S- Scale (Glassgow Coma scale)	TRISS includes · R- RTS · I- Injury Severity score · S- Seen Age · S- Specific Mechanism (Blunt or Penetrating)	· S- Shock · I-Ischemia of extremity · M- Main energy that caused injury · A – Age

INJURY SEVERITY SCORE(ISS) =

Calculated by sum of squares of 3 most severely injured body regions

ISS > 36 : DCS

ISS	Injury
<9	Minor
9-15	Moderate
16-25	Serious
>25	Severe

- **ABBREVIATED INJURY SCALE (AIS):**

Has a Seven Digit Code and represented as 123456.7

Pre dot Code for Body region (1st digit) is as follows

1	Head
2	Face
3	Neck
4	Thorax
5	Abdomen & Pelvis
6	Spine
7	Upper Extremities
8	Lower Extremities
9	Burns and other

1	Anatomic Body Region
2	Type of anatomic structure
3/4	Specific anatomic structure
5/6	Level of injury
Post dot 7	Grade of injury

ABC(Assessment of Blood Consumption) score to predict need for MTP

It is to predict need for massive transmission protocol(MTP)

Parameters: FAST SPM

FAST +ve

S: SBP <90

P: PR >/=120

Mechanism : Penetrating trauma

SCORE: 0 – 4

Scores <2, less likely to require MTP

Scores >2, more likely to require MTP (SS 75%-90%; SP 86%)

Delaying every minute associated with 5% increase in mortality

DCS – 1st described by Rotondo et al

a.k.a Abbreviated Laparotomy

In complex trauma patients with combined vascular and visceral injuries

Restore physiology over anatomy

Other Fixation strategies:

ETC : Doing definitive management within 36 hours of injury

PRISM

Damage Control Surgery: (Hint : Remember LETHAL TRIAD for DCS)

Criteria for DCS	Criteria for ETC
Hypothermia: **<34°C**	Stable hemodynamics
Acidosis: **pH <7.2**	No need for vasopressor/inotropic stimulation
Serum lactate >5 mmol/L	No hypoxemia, no hypercapnia
Coagulopathy	Serum lactate **<2 mmol/L**
Blood pressure <70 mmHg	Normal coagulation
Transfusion approaching 15 units	Normothermia
Injury Severity Score >36	Urinary output **>1 mL/kg/h**

• Mechanism of head injury:

-Blunt
-Penetrating
-Specific foci or Diffuse injuries

• Types of head injury:

B/o Impact

PRIMARY Impact to brain the time of trauma					SECONDARY
Hematoma	Skull #	Contusion	DAI	Arterial dissection	· Raised ICP · Hypoxia · Cerebral ischemia · Oedema All of these will lead to oedema or ischemia
EDH SDH SAH ICH					

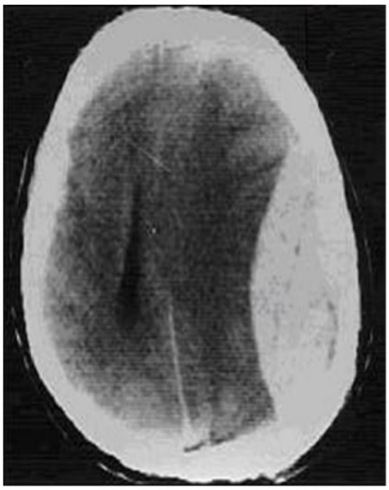
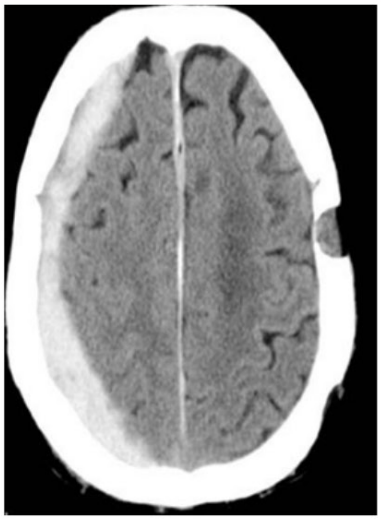
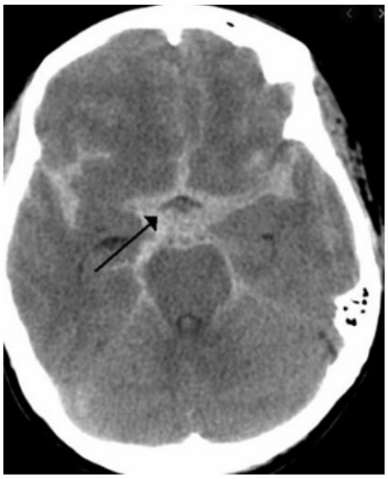
• Severity of head injury: As per GCS

GCS	SEVERITY
15 without LOC	Minor Head injury
15 with LOC / 14	Mild
9 to 13	Moderate
3 to 8	Severe

• Glassgow Coma Scale:

Eye opening:	**Verbal**	**Motor**
• Spontaneous – 4 • To loud voice- 3 • To pain stimuli- 2 • Do not open- 1	• Normal Oriented- 5 • Confused- 4 • Inappropriate words- 3 • Sounds only- 2 • No sounds- 1	• Obeys commands- 6 • Localizes to pain- 5 • Withdrawal flexion- 4 • Abnormal flexion- 3 (decorticate) • Extension- 2 (Decerebrate) • No motor response-1

• INTRACRANIAL HEMATOMAS

EDH	SDH	SAH
		
Biconvex Appearance At the site of impact Usually No underlying brain parenchymal damage	Concavo-Convex appearance Cou-Contre coup injury Underlying brain parenchymal damage occurs	MC type of haemorrhage- Intracerebral haemorrhage due to hypertension in **PUTAMEN.** Above pic : SAH with Blood in Subarachnoid Cisterns
· M/c in <u>young male</u> patients · Always associated with <u>skull fracture</u> · Injured vessel- **middle meningeal artery**** · M/c site of injury is **temporal bone at pterion (most thinnest part of skull)** which overlies the middle meningeal artery · The hematoma is located between bone and Dura mater. · Lucid Interval is seen	· Accumulates in space between dura and arachnoid · Associated with Bridging vein disruption and brain laceration* · Associated with primary brain injury · **C/F-** Impaired consciousness from the impact time itself. · Acute SDH- follows trauma · Chronic SDH- people on Anticoagulants*	· MC cause of SAH nowadays is Trauma · Other cause is Sudden rupture of Berry Aneurysm Note : Traumatic SAH is not a/w vasospasm (as seen with aneurysmal SAH)

WOUND HEALING

HEMOSTASIS

PHASE	DURATION	PREDOMINANT CELLS	Points
INFLAMMATORY PHASE	Begins immediately following wound Lasts for 2-3 days	Platelets (main cells of the phase), PMNs. Leukocytes Macrophages (24-48 hours): They remove the devitalized tissue and microbes. Regulate fibroblastic activity in proliferative phase. The initial framework for structural support is provided by FIBRIN produced by fibrinogen.	Platelets: Release ADP (it causes thrombocytic aggregates to fill the wound) They secrete cytokines (PDGF, IGF-1, TGF-Beta) from alpha granules which attract PMNs and Macrophages. Platelets and injured tissues release amines (They all increase vascular permeability) Histamine Serotonin PGs
PROLIFERATIVE PHASE	3rd day to 3rd week	Fibroblasts Fibroblasts require Vitamin C to produce collagen	Production of collagen and ground substance (GAGs and Proteoglycans) The wound tissue formed in the early part of this phase is called Granulation tissue In the latter part of this phase, there is an increase in tensile strength of the wound due to increased collagen Collagen at first deposited in a random fashion and consists of type III collagen Growth of new blood vessels as capillary loops (Angio neogenesis) Re-epithelialization of the wound surface
REMODELLING/ MATURATION PHASE	After 3 weeks; Maximal at 12th week	Fibroblasts & Myofibroblasts	Maturation of collagen Type 3 collagen converts to Type 1 until Type 3 :1 = 1:4 Realignment of collagen along the lines of tension. Decreased wound vascularity Wound contraction d/t fibroblasts and myofibroblasts activity

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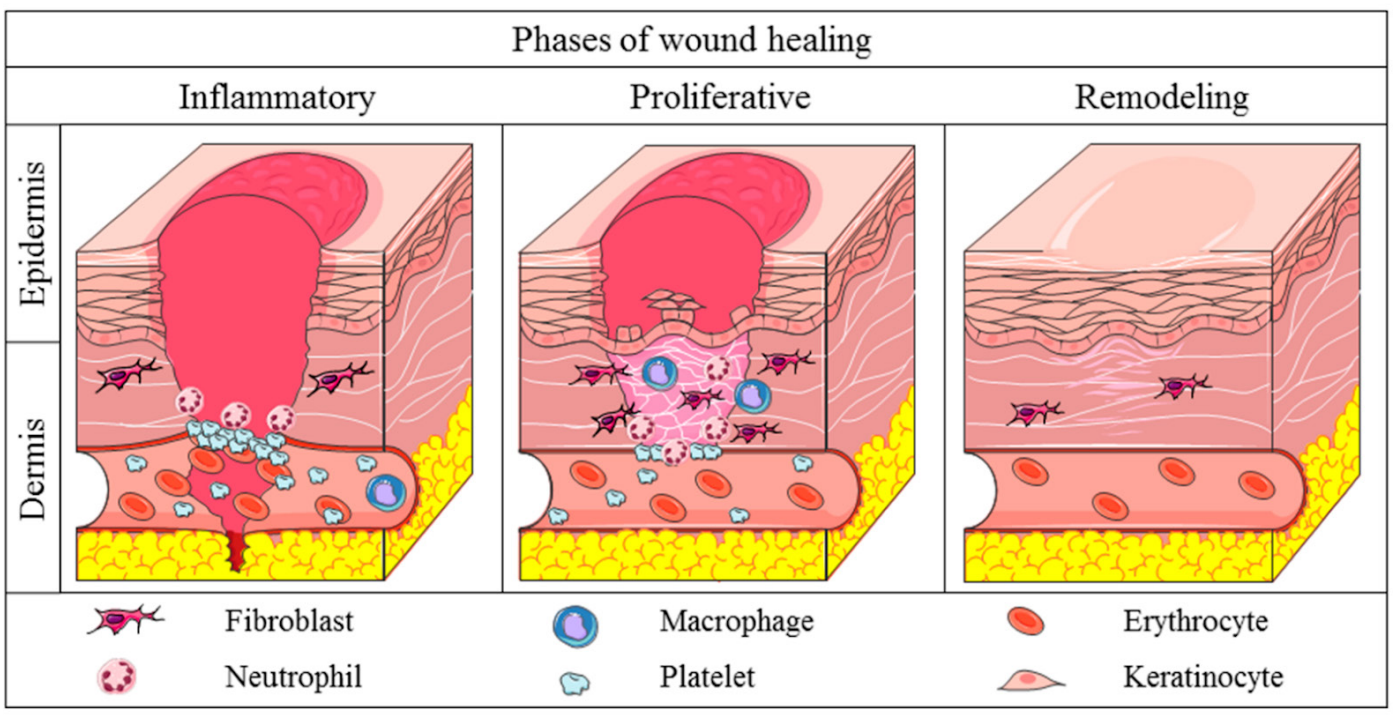

HEALING IN OTHER TISSUES

BONE	NERVE	TENDON
Periosteal and endosteal proliferation- Callus formation Callus is immature bone consisting of osteoid Mineralized by Calcium Hydroxyapatite and laid down by Osteoblasts	Proximal: Traumatic degeneration as far as the last node of Ranvier Distal: Wallerian degeneration	No tensile strength for the first 3-6 weeks due to random nature of the collagen. **INTRINSIC SUPPLY**: Vincular blood flow and synovial diffusion **EXTRINSIC SUPPLY**: Depends on formation of fibrous adhesions between the tendon and the tendon sheath.

Classification of wound closure and healing

- Primary
  - Wound edges apposed
  - Normal healing
  - Minimal scar
- Secondary
  - Wound left open
  - Heals by granulation, contraction and re-epithelialisation
  - Increased inflammation and proliferation
  - Poor scar
- Tertiary (delayed primary)
  - Wound initially left open
  - Edges apposed later when healing conditions favourable

Bleeding wound:

- Should be elevated and a pressure pad applied
- Clamps should not be put on vessels blindly as nerve damage is likely and Vascular Anastomosis is rendered impossible
- Devitalized tissues must be excised until bleeding occurs
- Exception is nerves, vessels and tendons
- Muscle viability is judged by the color, bleeding pattern and contractility

TIDY WOUND

- Repair of all damaged structures
- Repair of nerves under magnification (Loupes or microscope) using 8/0 or 10/0 monofilament nylon
- Vessels such as the radial or ulnar artery may be repaired using 8/0 or 10/0 monofilament nylon
- Tendon repairs particularly those in the hand, non-absorbable 3/0 or 4/0
- Skin cover by flap or graft

HAEMATOMA:

- It may also calcify and therefore requires surgical exploration
- Indication for release by incision or aspiration: Large size, Painful or causing neurological deficit
- In the gluteal or thigh region, there may be associated fat fracture.

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DEGLOVING INJURIES:

OPEN degloving injury	CLOSED degloving injury (Physiological Degloving Injury)
e.g., Ring avulsion injury with loss of finger skin	Injury extends far further than expected, much of the limb skin may be non-viable

COMPARTMENT SYNDROME

- Typically occurs in closed lower limb injuries
- They can occur in an open injury if the wound does not extend into the affected compartment.

Diagnosis:

- Mainly clinical
- Compartment pressure can be measured by using a pressure monitor and a catheter placed in the muscle compartment

Fasciotomy is indicated if:

- Pressure is constantly >30 mmHg
- Obvious clinical signs present

Rx:

- Radical excision of all non-bleeding skin, judged by bleeding dermis.
- Fluorescein perfusion assessment: Under UV light, a viable (perfused) skin will show up as a fluorescent yellowish green color.
- Best method is serial excision until punctate dermal bleeding is obvious
- STSG can be harvested from the degloved non-viable skin and meshed
- STSG can be used to cover the raw areas resulting from debridement.

CRUSH INJURIES that present late

- A late fasciotomy is dangerous
- Dead muscle produces Myoglobinuria and renal failure
- Safer to amputate the limb once viable and non-viable tissues have been demarcated
- **Primary amputation is TOC** in such cases (Compartment syndrome in crush injury presentation more than 6 to 8 hours)

CHRONIC WOUNDS

- Wounds > 3 months (Schwartz: >4-6 weeks)

Surgical Rx is indicated

- Optimized by VAC
- If Non-operative Rx has failed or
- If the patient suffers from intractable PAIN

Meshed skin grafts are more successful than sheet grafts.

Plastic Surgery

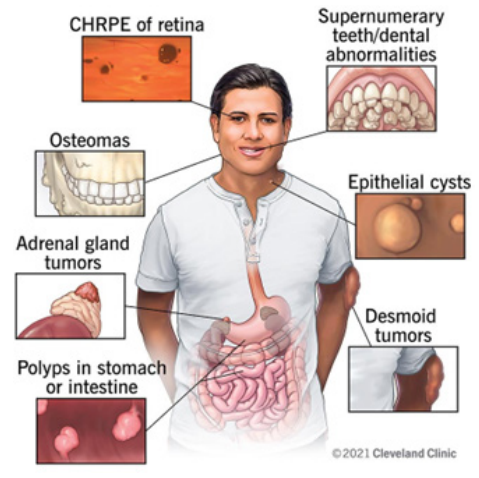
9

SKIN AND SUBCUTANEOUS TISSUE

<table>
<tr><th>EPIDERMIS</th><th>MELANOCYTES</th><th>SWEAT GLANDS</th><th>ANGIOSOMES</th></tr>
<tr>
<td>5% of skin
5 layers:
Stratum corneum(superficial)
S. lucidum
S. granulosum
S. spinosum
S. basale(deep)
Keratinized stratified squamous epithelium
Keratinocytes grow and are replaced by mitosis in s. granulosum
S. lucidum , granulosum and spinosum are thick in glabrous skin and absent in eyelid skin.

Epidermis is thickest on palms, soles, back and buttocks and

Thinnest on Eyelids.</td>
<td>Dendritic / NCC origin
Located in basal dermis
Melanin- transferred via membrane processes
Transferred to keratinocytes in s.granulosum and spinosum
Ethnic-difference in color is due to distribution of melanin but not by number of melanocytes.</td>
<td>
<table>
<tr><th>Eccrine gland</th><th>Apocrine glands</th></tr>
<tr><td>Distributed throughout the entire body surface-except in lips
Secrete sweat in response to emotion or during thermoregulation</td><td>Secretion in response to emotions and hormones-become active at puberty
Found in axilla and groins
Secretion: Malodourous after bacterial degradation</td></tr>
</table>
</td>
<td>3D segment of tissue with arterial and venous blood supply
Blood flows between neighboring angiosomes by choke vessels
Choke vessel: situated within the muscle
Blood supply to the skin anastomoses in
Subfascial
Fascial
Subdermal
Dermal
Subepidermal plexi
Epidermis has no blood vessel; cells nourish by diffusion
Venous drainage is via both valved and unvalved veins
Unvalved veins allow oscillating flow in the subdermal plexus.</td>
</tr>
</table>

	EP	C/F	Rx
NF : Schwann cell form tumors	70%: AD / 30% Sporadic Chr 17 gene mutations		GARDNER'S SYNDROME
Gardner'sSyndrome: AD	Variant of FAP Abn. Gene on Chr 5	Multiple epidermoid cysts and lipomata	
NEVOID BASAL CELL Ca(GORLIN'S SYNDROME)	AD Abnormal tumor suppressor gene on chromosome 9q 22-31 PTCH gene	Multiple BCC , Medulloblastoma Head: Over developed supraorbital ridges; broad nasal roots; Hypertelorism; Molar odontogenic cysts Trunk: Bifid ribs; Scoliosis Hand: Brachymetacarpalism ;Palmar pits	
HYPERHIDROSIS:		Excessive eccrine sweating in palms , soles of the feet , axillae and groins.	Anti-perspirants Local injections with Botulinum toxin A Surgery: Lap Cervical Sympathectomy
LIPODYSTROPHY:	A complication of long-term administration of insulin	A localized or generalized loss of fatty tissue	Protease Inhibitors in HIV and Transplant recipients Normally, Rxed by Autologous fat grafting/Injections of Poly-L-Lactic acid Free tissue transfer

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	EP	C/F	Mx
HIDRADENITIS SUPPURATIVA F: M = 4:1	Apocrine glands Genetic predisposition with variable penetrance A/W Obesity and smoking	M/C in axillae & groins but also seen in scalp, breast, chest and perineum	Stop smoking Weight loss Antiseptic soaps, Tea tree oil Non compressive and aerated underwear Antibiotics Anti-androgen drugs Requires radical excision and reconstruction.
PYODERMA GANGRENOSUSM	Cutaneous ulceration with purple undermined edges Secondary to IBD, RA, NHL or Wegener's granulomatosis		Responds to steroids Surgery may exacerbate the condition.
NF Fournier's and Meleney's are variants	Synergistic, Polymicrobial infection Streptococcal species (Grp A Beta Hemolytic)	Oedema beyond skin erythema Cannot distinguish fascial plain and muscle Disproportionate pain Skin vesicles and soft tissues crepitus Woody hard texture of subcutaneous tissues Lymphangitis: Absent	Xray: Air in subcutaneous tissues Rx: Antibiotics Debridement VAC Early skin grafting Mortality: 30-50%

	EP	C/F and Rx
Milia	Hard Keratin retention cysts M/C in Babies and in chronic sun exposure , in the elderly	
EPIDERMAL CYSTS/SEBACEOUS CYSTS	Stratified squamous epithelium From the hair follicle infundubuli	Fix it to the skin+ Central Punctum Rx: Excision if uninfected
MEIBOMIAN CYSTS	Epidermal cysts on edge of eyelid	
TRICHOLEMMAL CYSTS	From epidermis external root sheath of hair follicle	

SKIN TUMORS- BENIGN

Basal Cell Papilloma (Seborrheic Keratosis , Senile Keratosis, Verruca Senilis)	Papillary Wart(Verruca vulgaris)
Warty lesions Pigmented and Hyperkeratotic From basal cell layer Contains Melanocytes	Infection with human papilloma virus (HPV) Also cause plantar warts and condylomata acuminata

Nevus of Ota	Nevus of Ito
Most common in Oriental and African races M:F =1:4 Dermal , Melanocytic Hamartoma Blue or Grey Macule M/C site : Face (Trigeminal V1 and V2 dermatomes)	Dermal Melanocytosis M/C in shoulder Occurs simultaneously with nevus of ota

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PRIMARY WOUND CLOSURE

GOOD SUTURING TECHNIQUE:

- Incision with the scalpel at right angles to the skin
- Careful handling of tissues to avoid devitalizing the skin margins
- Debridement of skin edges if needed
- Eversion of the wound margin(Subdermal tissues will be in good approximation)
- Precise approximation without tension

How to prevent adverse scarring?

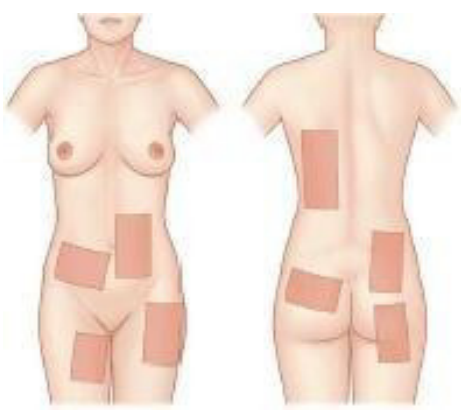
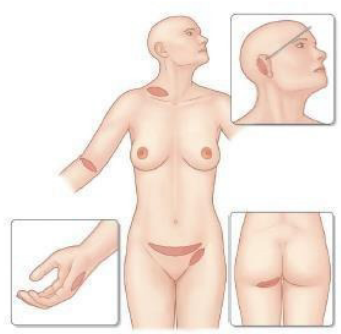
- Post surgical taping of the wound for 3 months
- Moisturizing lotions and moisture-retentive dressings (silicone sheets and gels)
- Pressure garments should be applied as soon as the wound is closed and the patient can tolerate the pressure
- Avoid sun exposure and use of SPF 50+ sunscreens for 1 year post operatively reduces scar hyperpigmentation

GRAFT AND FLAP

C/I: Group A β-haemolytic Streptococcus (absolute contraindication for skin grafting)

- Destroy grafts even in much fewer numbers.
- Secretion of proteases, streptokinase and hyaluronidase
- Prevent adhesion

GRAFTABLE BEDS	POOR GRAFT SURFACES
Healthy soft tissues Periosteum perichondrium, Paratenon Bone surface that is perforated to encourage granulation tissue growth	Exposed bone Exposed cartilage Exposed tendon Fibrotic chronic granulation tissue(Varicose ulcer/Diabetic ulcers/Pressure sores/Radiation ulcers)

STSG	FTSG
0.006 to 0.024 inch Epidermis and a portion of dermis Harvested by dermatome or Humby knife Meshed and expanded into 1: 1.5 or 1: 2 ratio Disadvantages are Contracture over time Abnormal pigmentation Poor durability if subject to trauma	Contains epidermis& entire dermis, with parts of the sweat glands, sebaceous glands, & hair follicles. FTSG can grow hair and secrete sebum to lubricate the skin, The colour and texture of normal skin Has the potential for growth. Graft take is not as predictable because more tissue must be revascularized
Donor site Thigh(Large amount available) Scalp(Faster re-epithelization) Arm Back Leg Gluteal region 	Donor site Upper eyelids, Postauricular crease, Supraclavicular area(Good color & texture) Hypogastrium Hairless groin, or Hypothenar eminence Elbow crease 

Transplant

TRANSPLANT HISTORY

Organ	Year	Surgeon	Location
Kidney	1954	Joseph E. Murray	Boston, MA
Liver	1963	Thomas E. Starzl	Denver, CO
Lung	1963	James D. Hardy	Jackson, MS
Pancreas	1966	Richard C. Lillehei	Minneapolis, MN
Heart	1967	Christiaan N. Barnard	Cape Town, South Africa
Small intestine	1967	Richard C. Lillehei	Minneapolis, MN
Heart–lung	1981	Bruce Reitz	Stanford, CA
Multivisceral	1989	Thomas E. Starzl	Pittsburgh, PA

TRANSPLANT

- Replacing a non-functional organ
- Types

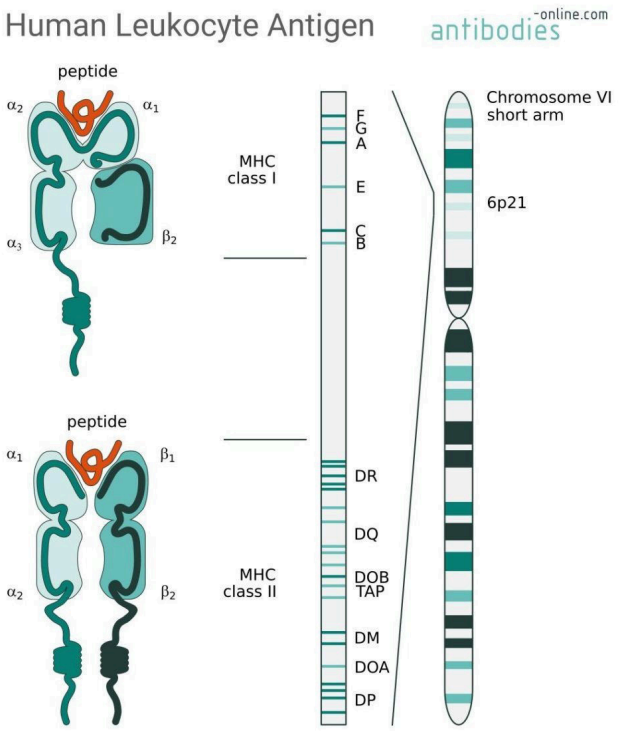
AUTO TRANSPLANT	ISO TRANSPLANT	ALLO TRANSPLANT	XENO TRANSPLANT
Organ is taken from same patient and re-implanted at a different site e.g., Severe irreparable ureteric inury—Renal auto transplant Advantage : No chance of immune suppression	Identical twins	Organs harvested from different individual of same species. Chance of rejection +++ Can be managed with strong immune suppressants.	From different species. E.g., Dog to man Mostly incompatibile.

Transplant

TRANSPLANT IMMUNOLOGY

- Immunity is mainly to protect our body from external infection
- Any outside organ/cell –detected by our immune system as foreign –mount a strong response and kills foreign cells.

HLA : Produced by MHC gene on chromosome 6p



2 types

HLA I	HLA II
All nucleated cells	On Lymphocytes & APCs
HLA A , B, C	HLA DP, DQ,DR

	Donor	Recipient
HLA A	0/0	0/0
HAL B	0/0	0/0
HLA DR	x/0	x/0

Most common cause of graft failure: HLA mismatch

- HLA Matching Order HLA A , B, DR
- HLA Matching: DNA sequencing testing

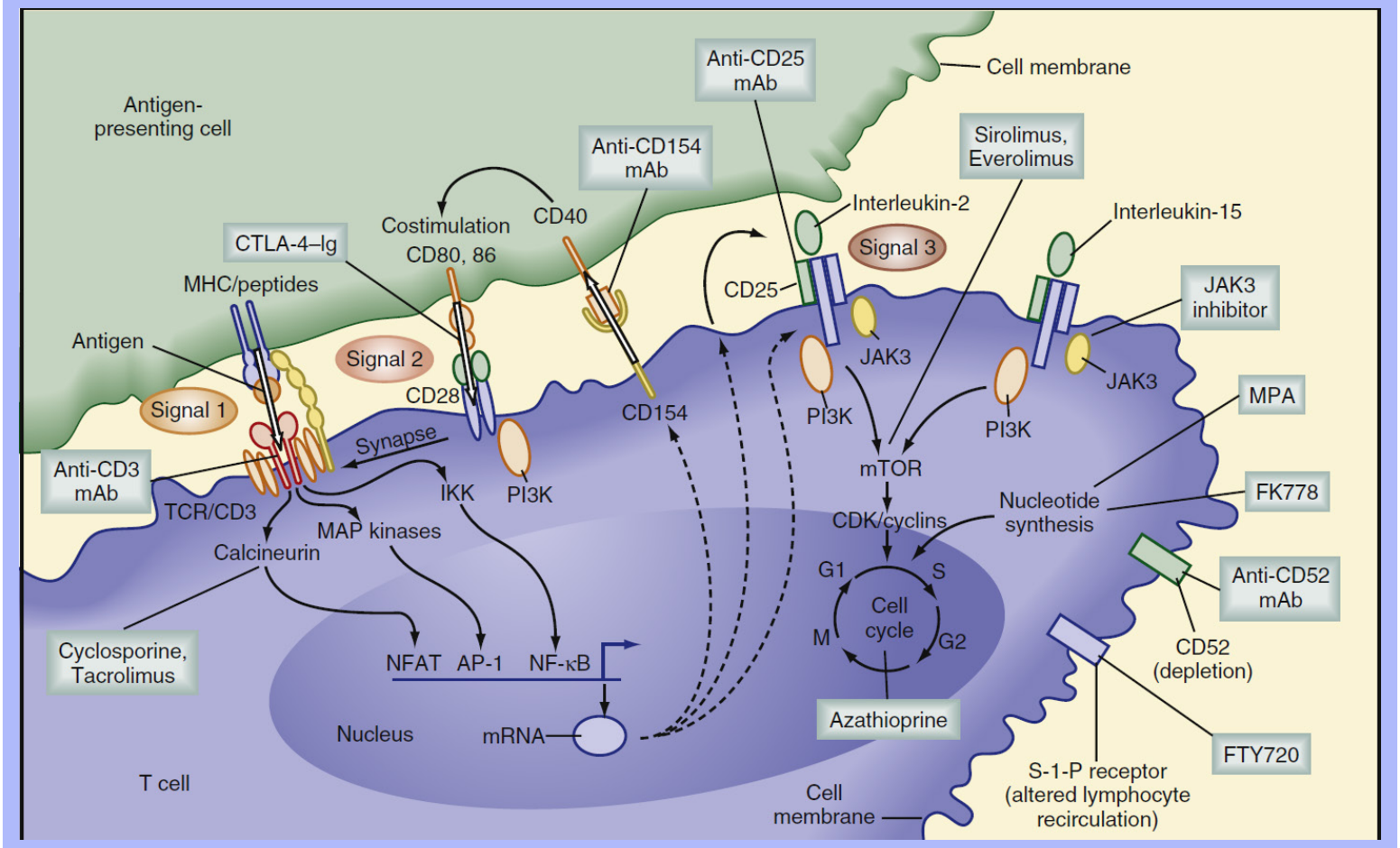
Donor and recipients samples taken

If DR mismatch is +, More chance of rejection.

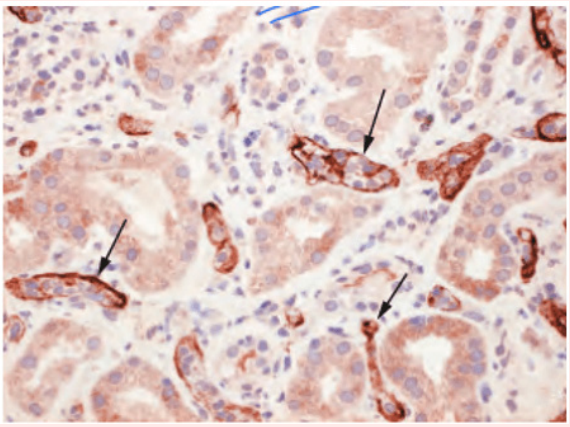
- 0/6 mismatch: Best chance of acceptance and least possibility of rejection
- 0/0 mismatch: Rejection most likely to be positive

Antigen Presentation :

Direct	Indirect
Donor APC	Time consuming Host APC---Internalize to HLA---Process it and presents it to T cells
No need of processing of HLA	Time consuming
It will cause immediate immune identification	Delayed Immune response
Early T cell activation	Sustained immune response
Hyperacute Cell Mediated Response	Chronic rejection+
6-12 weeks (Donor APC die out)	



REJECTION-TYPES

Hyperacute	Acute	Chronic
Minutes or Hours after TxMediated by Class I MHC	Peak incidence: First 3 months(Sabiston :First 6 months) Incidence: 10-20%	Occurs during a period of years.(After first 6 months) Most common cause of graft failure (Most common type of rejection)
Etio : ABO incompatibility or clerical errors in blood grouping test OR Preformed circulating donor specific HLA antibodies(DSA) or ABO antibodies.	Largely mediated by direct antigen presentation. T cells constitute the core element responsible for acute rejection, often termed T-cell mediated rejection. AMR : Antibody Mediated Rejection Antibody mediated damage may also be present as evidenced by deposition of Complement component c4d within graft vasculature.	Mechanism is less well understood. It's an immune based process derived from repeated or indolent T cell–mediated rejection or AMR, but the clinical phenotype of chronic graft fibrosis and deterioration is often secondary to a combination of both immune and nonimmune effects
Widespread intravascular thrombosis and interstitial Hemorrhage	Characterized by :Mononuclear cell infiltration(T cells, B cells , NK cells & activated macrophages) of the graft 	Parenchymal replacement by fibrous tissue with a relatively sparse lymphocytic infiltrate but may contain macrophages or dendritic cells. Why the fibrosis ?Response to alloantigen as well as the ischemia-reperfusion injury associated with the actual transfer of the organ itself. Increased TGF beta causes remodelling of the parenchyma and ensuing fibrous replacement
Untreatable and inevitably results in graft loss	Rx: High dose Pulsed IV Steroids (Methyl prednisone 0.5 g IV for 3 days)Severe or Steroid resistant rejection is Rxed with: Lymphocyte -depleting IV ATG Out of all three types of rejection, only acute rejection can be successfully reversed once it is established.	Terminologies :

Organ Donation & Preservation , Kidney Transplant

Indications:

- CKD
- ESRD-Dependent on dialysis for survival

- Median waiting period for renal Transplant : 3 years
- Severe shortage of organs
- Demand>>>>Availability

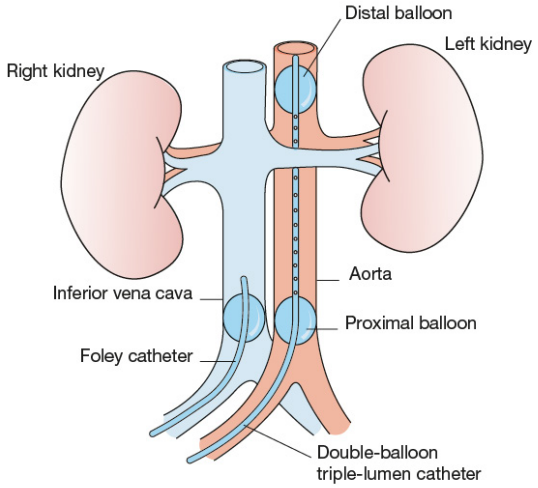


Figure 88.1 *In situ* perfusion of kidneys in a non-heart-beating donor (donation after circulatory death [DCD]). A double-balloon aortic catheter is introduced through a groin incision and 10–15 litres of chilled preservation solution is administered. The perfusate is vented through a Foley catheter introduced into the femoral vein.

Donors

Live Donor	Deceased Donor	
A single kidney is sufficient to have normal life As per NOTA act :Only related living donations are possible— Grand parents, Parents, Children, Siblings, Between Husband&wife Unrelated donors-Illegal	DBD	DCD
	Donation after Brain Death Severe RTA with head injury Irreversible Brain damage Brain stem— RAS : Consciousness Medulla oblangata : Respiratory center Brainstem injury: Permanent loss of consciousness and Spontaneous breathing Maintained on ventilator Heart is functioning well and rest of the organs are well perfused. HEART BEATING DONOR Internal organs are preserved till harvesting.	Donation after Cardiac/Circulatory Death Modified Maastricht Classification: Uncontrolled Donor means there is no time for preparation Insitu cooling Only Kidneys can be harvested Controlled patients are better suited for DCD donors. Happens at ICU Explained to attenders Consent Withdraw the support No- touch period for 5 minutes Shift the patient to OT immediately Canulate the Aorta Warm ischemic Time < 10 minutes Organs can be harvested

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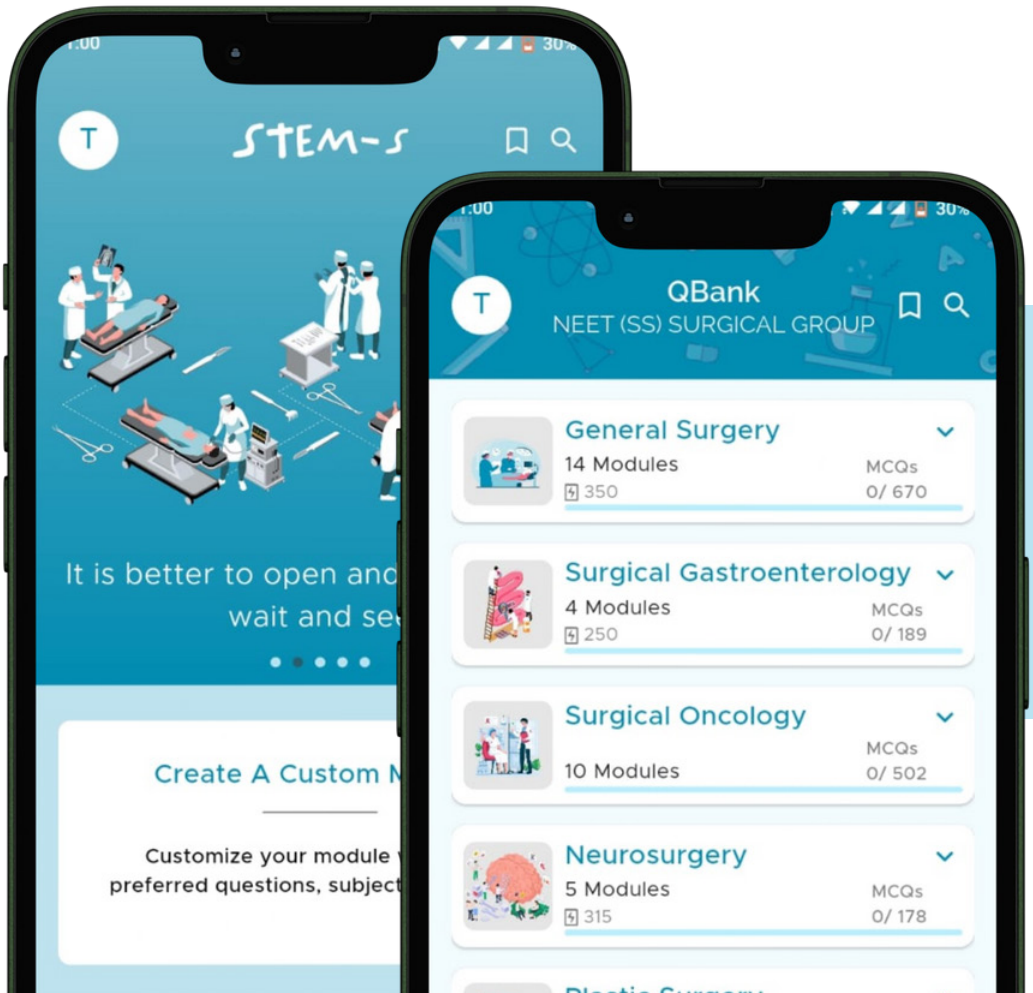

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