AIRWAY

Maneuvers to open up airway - HEAD TILT
CHIN LIFT
JAW THRUST
→ then do → Bag & Mask ventilation = AMBU

Oropharyngeal (Gueded) airway - to prevent tongue from falling back & obstructing airway
- Does not prevent aspiration
- in unconscious / Subconscious patient who cannot guard their airway
- Allows suction of secretions

SUPRAGLOTTIC AIRWAY

- LARYNGEAL MASK AIRWAY → has inflatable cuff
  - → CLASSIC LMA
  - → FLEXIBLE LMA
  - → REINFORCED LMA / INTUBATING LMA - aids ETT insertion
- PROSEAL
- iGEL

} 2nd generation supraglottic airways - have esophageal drain tube

ENDOTRACHEAL INTUBATION

- OROTRACHEAL
- NASOTRACHEAL

RSI - Rapid Sequence Intubation

IV anaesthetic + Rapidly acting Muscle Relaxant

↓

to secure airway quickly

- MACINTOSH LARYNGOSCOPE
- FIBEROPTIC INTUBATING BRONCHOSCOPE

} can be used to introduce an endotracheal tube → DEFINITIVE AIRWAY - secure

Double lumen tubes
Endobronchial tubes

} Allow deflation / collapse of 1 lung + Selective ventilation of the other lung → in Thoracic Sx / Esophageal Sx

INVASIVE AIRWAY

AIRWAY ASSESSMENT

- Mouth opening (3-4 finger) • Frontal & profile view
- Thyromental distance (3-4 finger) = 6-8cm
- Neck mobility
- Tongue size
  - MALLAMPATI

CLASS - I - Soft palate
Fauces
Uvula
Tonsillar pillars

Class II
Soft palate
Fauces
Uvula

Class III
Soft palate
Fauces

Class IV
Soft palate barely visible

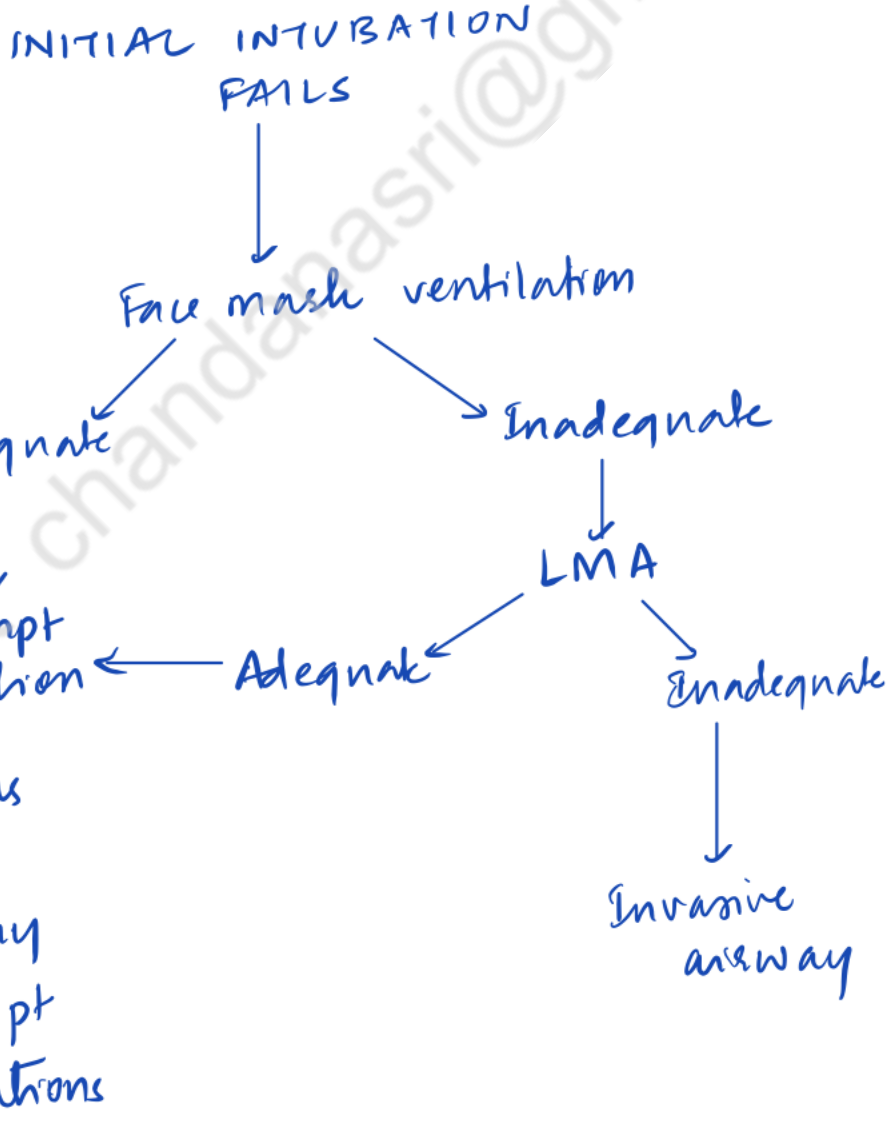
DIFFICULT AIRWAY

Decide primary strategy

Awake intubation (vs) intubation after induction

Non invasive airway (vs) invasive airway

Spontaneous ventilation (vs) mechanical ventilation



AIR EMBOLISM

Air embolism is an event occuring as a consequence of the entry of air into the vasculature

CAUSES OF AIR EMBOLISM

- **SURGICAL PROCEDURES**
  - Cardiothoracic Surgery - CPB, Lung resections, Needle biopsy of lung, CABG
  - Ob/Gyn procedures - hysteroscopy / Tubal insufflation
  - Orthopedic Surgery - arthroscopy
  - Neurosurgery - Craniotomy, shunt placement
  - Neck Surgery
- **CATHETERISATION** → apheresis
  - IV lines - Blood transfusion, infusion
  - Central lines → insertion / removal
  - Hemodialysis, ECMO
  - Angiography / plasty
  - Pacemaker / defibrillator placement
- **TRAUMA**
  - Head and neck injuries
  - Penetrating & blunt chest trauma
  - Blunt abdominal trauma
- **POSITIVE PRESSURE VENTILATION** - Barotrauma
- **DECOMPRESSION SICKNESS** - Rapid ascent in Scuba divers

PATHOPHYSIOLOGY → direct communication between a source of air & vasculature

↓ Pressure gradient

2 types

Venous air embolism

Air enters systemic venous circulation

↓

RV → Pulmonary circulation

- Larger bubbles
  - ↓ Pulmonary outflow tract obstruction
    - ↓ COP
  - Air embolism to coronaries
- Smaller bubbles
  - ↓ Pulmonary vasoconstriction
    - PHTN → Acute RVF
  - Acute V/Q mismatch

Arterial air embolism

- can be d/t - paradoxical - patent FO / ASD
- direct introduction
- incomplete filtration of bubbles by pulm circulation

↓ ischemia in any organ upstream - esp if there is poor / no collateral supply

The effects of the air embolism depends on total volume of air injected, rate of injection, final location

↓
100 mL/s

General adult fatal volume
~3-5 ml/kg
↓
closer to heart – smaller the lethal volume

Cerebral circulation
↓
2 ml fatal

Coronary circulation
↓
0.5 ml fatal

Clinical features

- Chest pain, dyspnea, tachypnea, tachycardia, Altered mental status
- Arrhythmias
- hypotension & CVS collapse
- Mill wheel murmur

Arterial – END, delayed recovery from anaesthesia
– End organ dysfunction - ACS, spinal ischemia

EVALUATION

Sudden ↓ in $ETCO_2$, fall in SPO_2, ABG - hypoxemia, hypercarbia
↑ CVP
Echo → Bubbles

CxR - Pulmonary edema

Management

- O_2, Mechanical ventilation
- Hemodynamic support
- To reduce size of embolus
  - Trendlengberg - Head low → encourage air to move to leg veins
  - Left lateral position - air moves up, allowing RV to empty
  - 100% O_2 → prevents nitrogenation (? Hyperbaric) and therefore halts bubble expansion
  - Large VGE due to central line in RA → aspirate air

± Anticoagulants, Steroids } Questionable

ARDS - Acute Respiratory Distress Syndrome

Non Cardiogenic B/L Pulmonary edema due to LUNG INJURY:

1) Respiratory symptoms developing within 1 week of known insult (or worsening)

2) B/L Opacities consistent with Pulmonary edema on imaging

3) No cardiac failure / fluid overload

4) Impairment of oxygenation

PaO_2/FiO_2 - ↓ (N) PaO_2/FiO_2 - >300

Mild ARDS - 200-300
Moderate ARDS - 100-200
Severe ARDS - <100

Consequences

Impaired gas exchange
↓ lung compliance
Pulmonary HTN

Causes

Sepsis
Aspiration
Pneumonia
Severe trauma / burns
TRALI - Blood transfusion
Stem cell transplant
Drugs
Drowning

Management

Address Underlying cause
Supportive care - O_2, Mechanical ventilation
↑ PEEP, FiO_2
Fluid management
↳ targetted CVP <4 mmHg
Prone positioning
Steroids (within 14d on onset only)

CENTRAL VENOUS PRESSURE

- Pressure in the vena cavae
- Reflects (R) atrial pressure

↖ Depends on venous return

∴ CVP → surrogate for preload

↓

used to measure volume responsiveness

(N) CVP – 3-8 mmHg

↓

measured by connecting CVC to special infusion set

↑ CVP

- Cardiac tamponade
- Heart failure
- Forced exhalation / Mechanical ventilation ∴ ↑ PEEP
- Hypervolemia
- Pleural effusion / Pneumothorax
- Pulmonary embolism

↓ CVP

Distributive shock
Hypovolemia

PULMONARY CAPILLARY WEDGE PRESSURE

↓

measured by wedging a pulmonary catheter (Swan Ganz catheter) with an inflated balloon in a small pulmonary artery branch

Reflects (L) Atrial pressure

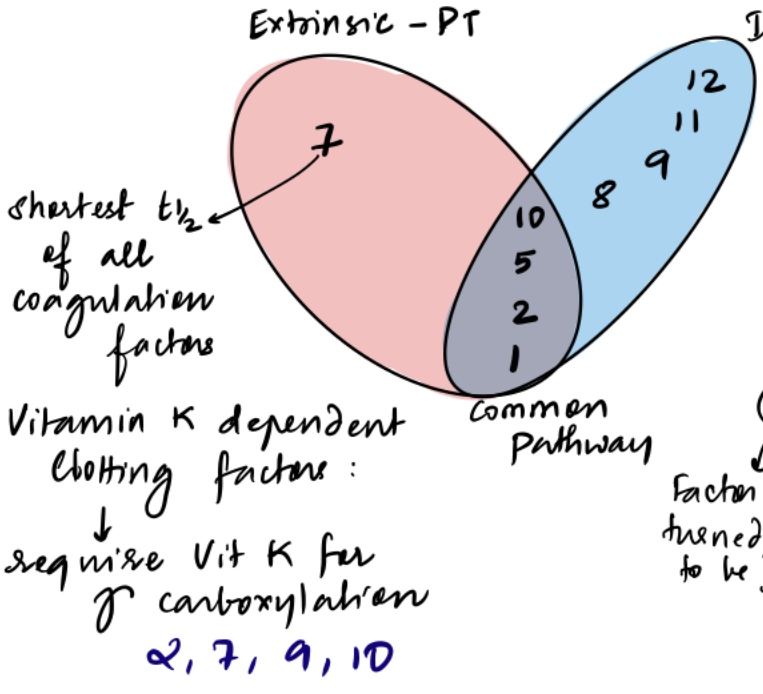
(N) - 6-12 mmHg

PCWP is (N) ARDS

↑ in Acute Pulmonary Edema (Cardiogenic)
Left heart failure
Mitral stenosis

COAGULATION & SURGICAL BLEEDING

COAGULATION CASCADE



(L) = Synthesized in liver

Clotting Factors

1	Fibrinogen	(L)
2	Prothrombin	(L)
3	Thromboplastin	
4	Calcium	
5	Labile factor	(L)
7	Proconvertin	(L)
8	Antihemophilia factor	
9	Christmas factor	(L)
10	Stuart Prower	(L)
11	PTA	(L)
12	Hageman factor	(L)
13	Fibrin stabilising factor	(L)

TESTS OF HEMOSTASIS AND BLOOD COAGULATION

A. Platelet function

1) Bleeding time : Evaluates platelet function upto formation of temporary platelet plug (capillary bleeding)

~2-7 min

Prolonged in – 1) Aspirin & NSAID use - inhibits platelet TXA_2 synthesis
2) Renal failure - platelet aggregation defect
3) Scurvy - Vascular defect d/t defective collagen cross-linking
4) Thrombocytopenia
5) Platelet disorders - Bernard Soulier, Glanzmann's
6) vW disease - vWF deficiency

2) Platelet count : <1,00,000 ⇒ Thrombocytopenia

3) Platelet aggregation tests

4) vWF tests – Ristocetin co-factor assay
vWF antigen assay

B. Tests of Coagulation Cascade

1) PROTHROMBIN TIME: measures function of 1, 2, 5, 7, 10 factors

→ PT reagent (Thromboplastin, Ca^{2+}) + Plasma

↓

Fibrin clot

(N) - 11-15s

INR - to account for variations in thromboplastin activity from lab to lab

→ test of abnormal coag d/t Vitamin K deficiency driven failure

used to monitor Warfarin Rx

2) aPTT - activated partial thromboplastin time

measures activity of 1, 2, 5, 8, 10, 11, 12 (Intrinsic pathway)

aPTT reagent + Plasma

phospholipid substitute
Activator
Ca^{2+}

↓ aPTT = 25-40s

Fibrin clot

Monitor Heparin therapy
Ther. range 1.5-2.5x control

3) Viscoelastic testing

↓

→ better indicator of in-vivo hemostatic function

- Thromboelastogram (TEG)
- Rotational thromboelastometry (ROTEM)

4) FIBRINOLYTIC SYSTEM TESTS - FDPs, D-Dimers

→ for PTE, thrombolytic therapy, DIC

EVALUATION OF EXCESSIVE INTRAOP & POST-OP BLEEDING

CAUSES

- Patients on antiplatelets / anticoagulants
- Patients ↓ Vit K deficiency / Liver / Kidney failure

- Excessive bleeding from the operative field unassociated with bleeding from other sites ⇒ inadequate mechanical hemostasis by surgeon
- Intraoperative HTN → can interfere ↓ hemostasis
- Massive blood transfusion during surgery → Ineffective hemostasis d/t
  - Hypothermia
  - Dilutional coagulopathy
  - Platelet dysfunction
  - Thrombocytopenia
  - Hypofibrinogenemia
- DIC - d/t systemic activation of coagulation system
- Undetected hemostatic defects / Clotting factor deficiencies

CONTROLLING INTRA-OP BLEEDING

1) Local hemostasis - identify & ligate the bleeding vessel(s)
2) Mechanical procedures
   - Local digital pressure - at / proximal to bleeding site
   - Tourniquet use
   - Pringle maneuver for liver
   - Packing - pads / gauze - for diffuse ooze
   - Bonewax for marrow bleeding
3) Thermal agents - heat → denaturation of proteins - vessel seal
   - Electrocautery
   - Harmonic devices
4) Topical hemostatic agents - hemocoagulase, Gelatin foams, Oxidised cellulose, microfibrillar collagen
5) Systemic agents - Tranexamic acid | FFP, Cryoppt, Platelets
   Vit K

FLUID THERAPY

Total body water - 50-60 % of total body weight

♀ ♂

- Higher % of adipose tissue
- Lower % of muscle mass

Newborns - 80%

Infants - 65 %

TBW (60 %)

- Intracellular fluid (40%) — 2/3
- Plasma (5%) — 1/3 (20%)
- Interstitial fluid (15%) — 1/3 (20%)

Plasma Osmolality = $2[Na^+] + \left(\frac{[Glucose]}{18}\right) + \left(\frac{[BUN]}{2.8}\right)$

→ 290 - 310 mOsm/L

(N) Daily water intake — 2L

- 75% water drinking
- 25% from solid foods

Daily water losses

- 800 - 1200 mL Urine
- 250 mL Stool
- 600 mL Insensible losses
  - Skin - 75%
  - Lungs - 25%

↑ by hypermetabolism, fever, hyperventilation

Sensible losses → variable

- Sweat
- Diarrhea
- Vomiting

Daily dietary salt - 3-5 g/d

SHOCK & HEMORRHAGE

Shock - a state of circulatory failure characterised by inadequate oxygen & nutrient delivery to meet cellular metabolic needs and oxygen consumption, producing cellular and tissue hypoxia

TYPES OF SHOCK
(Based on the cause for circulatory failure)

CARDIOGENIC SHOCK
INTRACARDIAC

Cardiac pump failure
↓
Reduced cardiac output
↓
Shock

Causes
- Cardiomyopathy
- Arrhythmia
- Mechanical
  - acute valvular defects

HYPOVOLEMIC SHOCK
Reduced intravascular volume
↓
↓ Cardiac output
↓
Shock

Hemorrhagic
- trauma
- Surgery
- GI bleed
- Hemorr. Pancreatitis
- Ruptured aneurysm

Non Hemorrhagic
- GI loss
- Skin loss
- Renal loss
- Third space loss

DISTRIBUTIVE SHOCK
Severe peripheral vasodilatation
↓
↓ PVR
↓
Shock

- Septic shock
- Anaphylactic shock
- Endocrine shock
- Neurogenic shock

OBSTRUCTIVE SHOCK
Extracardiac causes of cardiac pump failure

- Pulmonary embolism
- Tension pneumothorax
- Cardiac tamponade
- Constrictive pericarditis

COMBINED
Combination of one or more of the other mechanisms

Eg Trauma
↓
Hemorrhagic + Distributive shock

PATHOPHYSIOLOGY

Main determinant of tissue perfusion - Mean Arterial pressure

$BP = COP \times SVR$

COP = Stroke volume × Heart rate
↓
Determined by
- Preload
- Myocardial contractility
- Afterload

SVR → Determined by
- Vessel length
- Blood viscosity
- Vessel tone

Biological processes affecting any of the above determinants → shock

CLASSES OF HEMORRHAGIC SHOCK - ATLS

	CLASS - I	CLASS - II	CLASS - III	CLASS IV
Blood loss (%)	0-15%	15 - 30%	30 - 40%	>40%
Pulse rate	<100	>100	>120	>140
Blood pressure	Normal	Normal	↓	↓
Pulse pressure	Normal	↓	↓	↓
Respiratory rate	14-20/min	20-30/min	>30/min	>35/min
Urine output	>30mL/hr	20-30mL/hr	5-15mL/hr	Negligible
CNS status	Normal	Anxious	Confused	Obtunded
Base deficit	0 to -2 mEq/L	-2 to -6 mEq/L	-6 to -10 mEq/L	> -10 mEq/L
Need for Blood products	MONITOR	POSSIBLE	YES	MASSIVE BLOOD TRANSFUSION PROTOCOL

SHOCK INDEX = Heart rate / Systolic BP } shown to be better at assessing severity of shock than HR / BP alone

>0.9 → predictor of mortality

'Hemodynamic stability indicates'

MODIFIED SHOCK INDEX = Heart Rate / Mean arterial pressure

LETHAL TRIAD

- ACIDOSIS : inadequate tissue perfusion ⟶ lactate ⟶ Acidosis
- HYPOTHERMIA : ↓ substrate & O_2 delivery ⟶ ↓ ATP generation ⟶ hypothermia
- COAGULOPATHY : Dilutional & Consumption coagulopathy

Acidosis ⟶ affects coagulation cascade (pH sensitive enzymes)

↳ Correction: Sodium bicarbonate

THAM - Tromethamine Hydroxymethyl Amino Methane

↳ biologically inert amino alcohol ; low toxicity buffers CO_2 & acids

SEVERITY OF SHOCK

	Compensated shock	DECOMPENSATED SHOCK MILD	MODERATE	SEVERE
Lactic acidosis	±	++	+++	++++
Urine output	Normal	Normal	Reduced	Anuric
CNS	(N)	Anxious	Drowsy	Comatose
RR	(N)	↑	↑↑	labored
PR	(N)	↑	↑	↑
BP	(N)	N/↓	↓	↓↓

Classic cardiovascular responses may not be seen with all patients

RESUSCITATION

FLUID THERAPY

In all cases of shock, regardless of classification, hypovolemia & inadequate preload must be addressed first

IV access - short, wide bore catheters that allow rapid infusion of fluids

(Long, narrow lines such as central venous catheters may have too high a resistance to allow rapid infusion)

TYPE OF FLUIDS

Crystalloids vs Colloids

Crystalloids
- RL > NS
- Hypotonic solutions (Dextrose) are poor volume expanders
  - avoided unless there is need to address free water deficit in conditions such as DI

Colloids
- supposed benefit over crystalloids
  - lesser volume → greater improvement in BP ('Volume benefit')
  - Now known to not be that substantial
  - Only 1.3x times more crystalloid required to bring about same response as colloid

Oxygen carrying capacity of both colloids & crystalloids = ZERO

Dynamic fluid response

FLUID BOLUS - 250-500mL over 5-10min

RESPONDERS
- sustained improvement in cardiovascular status
- Not actively losing fluid, but require filling to a normal volume status

TRANSIENT RESPONDERS
- show improvement
- Revert to previous state over the next 10-20 min
- moderate ONGOING fluid losses (hemorrhage / fluid shift)

NON-RESPONDERS
- Severely volume depleted
- major ongoing loss of intravascular volume
- NEED FOR IMMEDIATE DEFINITIVE INTERVENTION

VASOPRESSOR / INOTROPIC SUPPORT

- Indicated in distributive shock states where there is peripheral vasodilatation
- Indicated in cardiogenic shock / when myocardial depression has complicated a shock state

Noradrenaline, Dopamine, Dobutamine, Vasopressin

The most effective method of restoring adequate cardiac output, end organ perfusion and tissue oxygenation is to restore venous return to normal by locating and stopping the source of bleeding

VOLUME REPLETION will allow recovery from shock only after the bleeding has stopped

PERSISTENT INFUSION OF LARGE VOLUMES OF FLUID & BLOOD is not a substitute for definitive control of bleeding

CONTROLLED / BALANCED / HYPOTENSIVE RESUSCITATION / PERMISSIVE HYPOTENSION

BALANCING

the goal of organ perfusion & tissue oxygenation

WITH

avoiding rebleeding

by accepting a lower-than-normal blood pressure

End points of Resuscitation

- Adequate urine output – Adults – >0.5mL/kg/hr
  Kids – >1ml/kg/hr
  Infants – >2ml/kg/hr
  } traditional; does not detect occult hypoperfusion
- Base deficit: is the amount of base in millimoles required to titrate 1L of whole blood to a pH of 7.4 with the sample fully saturated with O_2 at 37°C & pCO_2 of 40mm Hg
  - Measured by ABG
  - >10–15mEq/L → severe
- Lactate – indirect measure of O_2 debt
- Mixed venous oxygenation

- Near infrared spectroscopy
- Gut mucosal pH
- Laser doppler flowmetry
- Sublingual capnography

SEPSIS AND SEPTIC SHOCK

SIRS - ≥2 of

1) Temperature >38°C or <36°C
2) HR > 90/min
3) RR > 24/min
4) TLC > 12,000/μL or < 4,000/μL or >10% bands on peripheral smear

SIRS criteria → overly sensitive, not specific

Sepsis = SIRS + a proven/ suspected microbial etiology

Severe Sepsis = Sepsis + signs of hypoperfusion/ organ dysfunction

Septic Shock = Severe Sepsis + Systemic hypotension

CURRENT DEFINITION OF SEPSIS - 3rd International Consensus of Sepsis & Septic Shock -

" Sepsis is defined as life-threatening organ dysfunction caused by dysregulated host response to infection

ORGAN DYSFUNCTION - acute change in SOFA score ≥2 points consequent to infection

Severe sepsis - removed from definition

SOFA score - Sequential/Sepsis-related Organ Failure Assessment score

		0	1	2	3	4
RS :	PaO_2/FiO_2	≥400	<400	<300	<200	<100
					↓ resp support	
Coag :	Platelet count	≥150×10³/μL	<150	<100	<50	<20
Liver	Bilirubin	<1.2	1.2-1.9	2-6	6-12	>12
CVS	MAP	≥70mmHg	<70mmHg	DA<5 DBA or	DA 5-15 E/NE-<0.1	DA>15 E/NE>0.1
CNS	GCS	15	13-14	10-12	6-9	<6
Renal	S.Creat	<1.2	1.2-1.9	2-3.4	3.5-5	>5

PATHOPHYSIOLOGY OF SEPTIC SHOCK

Infection (LPS)

↓

Interaction of of TLR & Bacterial products

↓

NF-KB

↓

Cytokines, chemokines, APRs

→ Fever, SIRS

↑ magnitude of infectious insult

↓

Activation of NOS → ↑ NO → Loss of vascular resistance

↓

Distributive shock

SURVIVING SEPSIS CAMPAIGN – 'Bundles of Care' 2012

within 3 hrs

- Measure lactate levels
- Obtain blood culture prior to administration of Abx
- Empirical Broad spectrum Abx
- Fluid – 30ml/kg crystalloid for hypotension / lactate >4mmol/L

within 6 hrs

- Vasopressors if Hypotension does not respond to fluids – to maintain MAP ≥ 65 mmHg
- Measure CVP, $CVSO_2$ if persistent hypotension or Lactate >4mmol/L
- Remeasure lactate

Early Goal directed therapy

- ID ↑ risk pts

→ Source control

- Cultures, Abx
- CVC – CVP – 8-12
- MAP ≥ 65 – Inotropes, addition of vasopressin – upto 0.03 U/min ↓ Mortality
- $ScVO_2$ ≥ 70% within first 6 hours of resuscitation
- HCT ≥ 30%
- Transfusion trigger ≤ 7.0 g/dL
- Blood Glucose – 80–110 mg/dL – achieve via intensive insulin therapy

COMPARTMENT SYNDROME

Surgical emergency characterised by IMPAIRED TISSUE PERFUSION due to an INCREASE in the INTRACOMPARTMENTAL PRESSURE within an unyielding fascial envelope.

PATHOPHYSIOLOGY

* Critical closing pressure: the compartmental pressure above which capillaries collapse from transmural pressure and blood flow is arrested

CAUSES OF COMPARTMENT SYNDROME

Vascular Causes

1. ISCHEMIA-REPERFUSION INJURY

(Acute limb ischemia → reperfusion)

Reperfusion of ischemic tissue
↓
ROS-induced damage
↓
microvascular permeability
↓
tissue & interstitial edema (progressive)

2. Vascular trauma

Arterial → Distal ischemia → Ischemia Reperfusion phenomenon

Venous → Venous outflow obstruction

↑ Tissue & interstitial edema

3. DVT- phlegmasia cerulea dolens

4. Deep venous harvest

5. Intracompartmental hemorrhage
↓
Rapid ↑ in compartment pressure

Non vascular Causes

1. FRACTURE
↓
Injury to surrounding soft tissue
Hemorrhage
↓
Edema
↓
↑ Compartment pressure

(FURTHER EXACERBATED BY TIGHT CAST IMMOBILISATION)

2. CRUSH INJURY
↓
Muscle injury

3. SNAKE BITE
Hematotoxic & cytotoxic venom
↓
Tissue inflammation and edema (within closed compartment)

4. IATROGENIC

- Extravasation of fluid during interventional procedures

CLINICAL FEATURES

High index of suspicion needed for diagnosis

Pain - out of proportion to magnitude of injury
pain on passive stretch

Paresthesia in the distal extremity

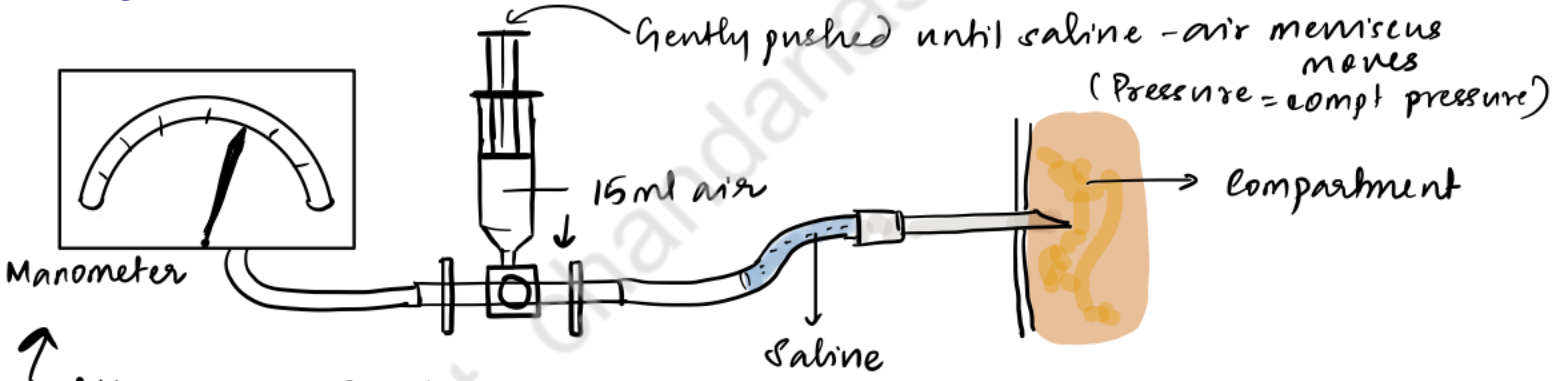
Tense edema

↓ Capillary refill, ↓/absent pulse → late signs

CONTRIBUTORY – ↑ CPK – muscle damage

MEASUREMENT OF COMPARTMENT PRESSURES

→ Not necessary for dx of Compartment Syndrome
may be required when dx is equivocal



↑ WHITESIDE METHOD

(N) Compartment pressure ≤ 10–12 mm Hg

Compartment syndrome occurs when
MAP – ICP is < 40 mm Hg
DBP – ICP is < 10 mm Hg

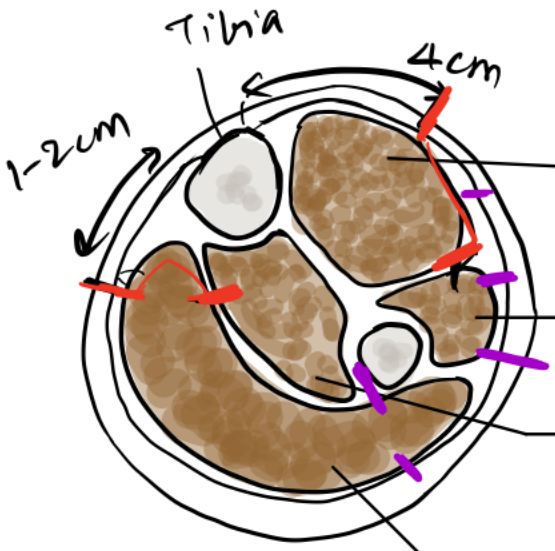
Other methods
- Near infrared spectroscopy
- Laser Doppler flowmetry

SEQUELAE OF COMPARTMENT SYNDROME

Myonecrosis → Renal failure
→ Hyperkalemia → Cardiac arrest

Rx – FASCIOTOMY

LEG



Anterior compartment - Tibialis anterior
EDL, EHL
Peroneus tertius

Lateral compartment - Peroneus longus
Peroneus brevis

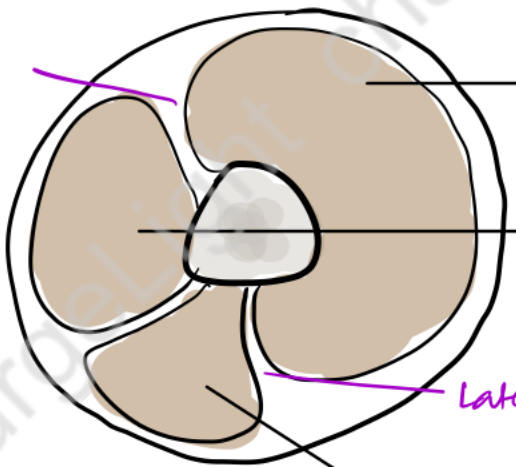
Deep posterior compartment
Tibialis posterior, Popliteus
FHL, FDL

Superficial posterior compartment
Gastrocnemius
Soleus
Plantaris

- Double incision technique

- Single incision technique
(over fibula - 3cm from fibular neck to 4cm from lateral malleolus

THIGH



Anterior compartment
Sartorius
Quadriceps femoris

Medial compartment
Gracilis
Adductus
Pectineus
Obturator externus

Posterior compartment
Hamstrings

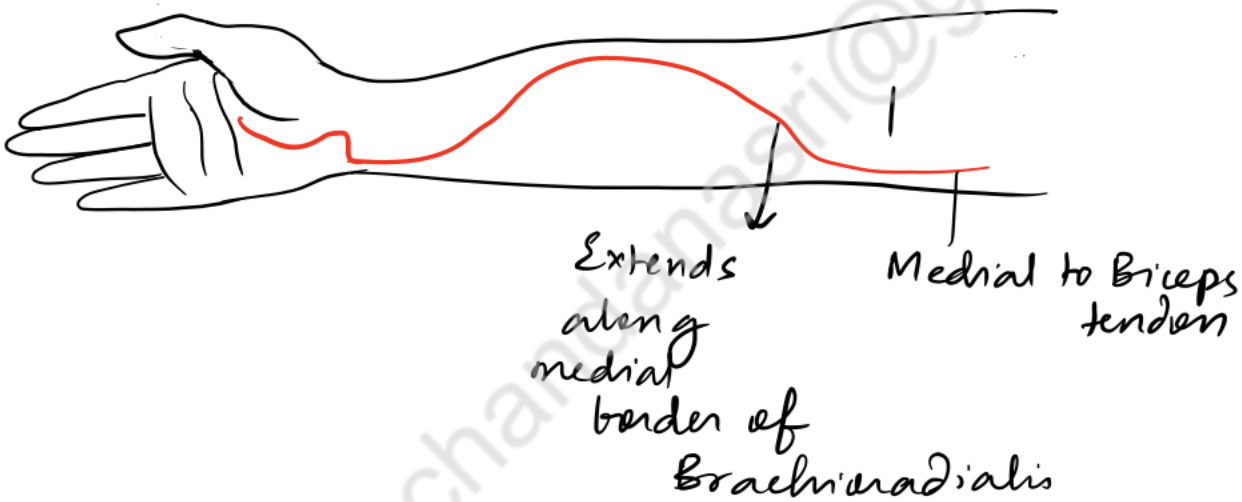
Incision - from intertrochanteric line to lateral epicondyle
incise iliotibial band - Anterior release
Retract Vastus lateralis → expose & incise Lateral IMS

Medial compartment release is rarely necessary

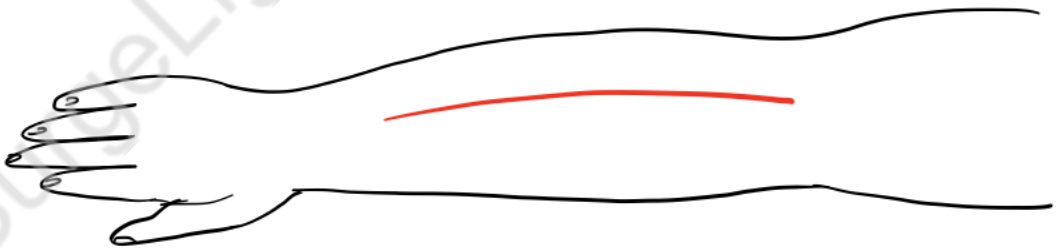
UPPER LIMB

- Extensor
  - Superficial - EDC, ECU, EDM
  - Deep - APL, EPB, EPL, EI, Supinator
- Lateral - Brachioradialis, ECRB, ECRL
- Volar
  - Superficial - PT, PL, FDS, FCR, FCU
  - Deep - FDP, FPL, PQ

Volar compartment → most frequently requires release



Extensor release



Generally indicated for - Volkmann's Ischemia

ABDOMINAL COMPARTMENT SYNDROME

Intra-abdominal hypertension: Sustained/ repeated pathological elevation of IAP ≥ 12mmHg
(Ⓝ - 5-7mmHg)

GRADES — IAP
- I - 12-15
- II - 15-20
- III - 20-25
- IV - >25 mmHg

IAP - measured by detecting pressure changes following instillation of 25ml sterile saline in end expiration, supine position after ensuring absence of abdominal wall contractions

Abdominal Perfusion Pressure - MAP − IAP

ABDOMINAL COMPARTMENT SYNDROME: Sustained IAP > 20mmHg ± Abdominal Perfusion Pressure < 60mmHg a/i new organ dysfunction/failure

- **Primary** → a/i injury/disease in abdominopelvic region → Requires prompt intervention
- **Secondary** → d/t conditions that do not originate in abdominopelvic region

CAUSES OF ↑ IAP

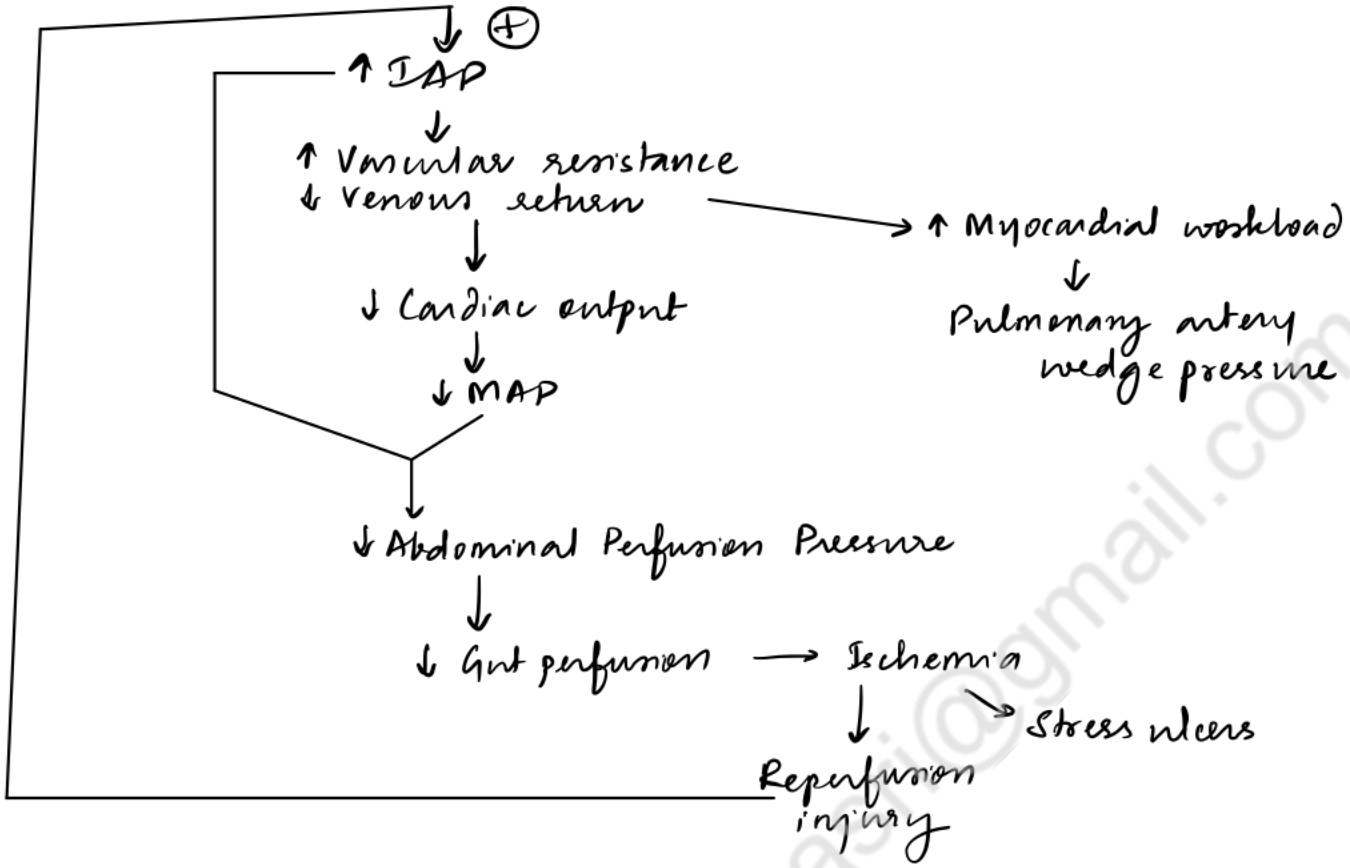
RETROPERITONEAL
- Pancreatitis
- Pelvic/Retroperitoneal bleeding
- AAA rupture
- Abscess

INTRAPERITONEAL
- Hemoperitoneum
- AAA rupture
- Acute Gastric dilatation
- Bowel obstruction
- Mesenteric venous obstruction
- Pneumoperitoneum
- Abscess
- Visceral edema

PARIETAL **
- Burns eschar
- Repair of gastroschisis or omphalocele
- Reduction of large hernias
- Laparotomy closure under extreme tension
- Mechanical ventilation

CHRONIC
- Central obesity
- Ascites
- Large intra-abd tumors
- CAPD
- Pregnancy

PATHOPHYSIOLOGY



↑ IAP → Pushes up diaphragm
- Respiratory restriction
- Atelectasis
→ Hypoxia

↑ IAP → ↓ Renal blood flow → Renal ischemia
⊕ RAAS
→ Fluid & water retention

↑ IAP → ↑ Intrathoracic pressure → ↑ IVP → ↓ Cerebral venous return
→ ↑ ICP → Impaired cerebral perfusion

↑ IAP → ↓ Peripheral venous return
↑ likelihood of DVT

Secondary ACS

Related to Capillary leak & fluid resuscitation

Acidosis → pH ≤ 7.2
Hypothermia
Coagulopathy

Massive blood transfusion > 10 units / 24 h
Severe sepsis
Shock
Massive fluid resuscitation
> 5 L colloid
> 10 L crystalloid / 24 h
in presence of capillary leak & +ve fluid balance

Major burns

MANAGEMENT OF IAH (IAP > 12 mmHg)

Intraluminal decompression	Evacuate Intra-abd SOL	Improve abd wall compliance	Optimise perfusion
• NG decompression • Flatus tube • Prokinetics • ↓/- Enteral nutrition • Enemas • Colonoscopic decompression	Percutaneous drainage Surgical evacuation	Sedation & analgesia • Muscle relaxation • Escharotomy	Goal directed resuscitation

- Refractory (Q4h measurements)
- IAP > 20 mmHg + New organ dysfunction

↓

Surgical abdominal decompression

DAMAGE CONTROL SURGERY

The purpose of damage control surgery is TO LIMIT THE OPERATIVE TIME so that attention may be given to PHYSIOLOGICAL RESTORATION and breaking the LETHAL TRIAD of COAGULOPATHY, HYPOTHERMIA and ACIDOSIS

TEMPORIZATION AND PRIORITIZATION OF PHYSIOLOGICAL RECOVERY OVER ANATOMIC REPAIR

LETHAL TRIAD

1) Coagulopathy - Dilutional

SHOCK / Severe hypovolemia
↓
excessive intravenous colloid/ crystalloid infusion
↓
Hemodilution
↓
Dilution of clotting factors
↓
Coagulopathy

RAAS

Immunoactivation
↓
Activation / Consumption of Complement system

2) HYPOTHERMIA - from evaporative and conductive heat loss during surgical exposure and resuscitation

3) METABOLIC ACIDOSIS -

Shock
↓
anaerobic metabolism
↓
acidosis

exacerbated by aortic clamping
administration of vasopressors
massive RBC transfusions
impaired myocardial performance

Once the cycle starts → each component exacerbates the other

Goals of Damage Control Surgery: HEMOSTASIS, CONTAMINATION CONTROL

Decision to choose DCS over Definitive Surgery is INTRA-OPERATIVE

INDICATIONS FOR DAMAGE CONTROL SURGERY

CRITICAL PHYSIOLOGICAL PARAMETERS

1) Hypothermia
Temp < 35°C

2) Acidosis
pH < 7.2
Base deficit > 15mmol/L
S. Lactate > 5mmol/L

3) Coagulopathy
PT > 16s
aPTT > 60s

+

Prohibitive operative
- time (> 60 - 90min) for definitive surgery
- Hemodynamic instability

> 10 units blood transfused
SBP < 90mmHg for > 60min

INJURY COMPLEXES a/c LOSS OF PHYSIOLOGIC RESERVE

1) High energy blunt torso trauma

2) Multiple penetrating torso injuries

3) Combined visceral injury ± major vascular trauma

4) Injuries across MULTIPLE BODY CAVITIES ± competing treatment priorities
↓ eg
- Closed head injury
+
- Major vascular injury
+
- Pelvic trauma

OTHER CONSIDERATIONS IN TRAUMA PATIENTS

- Injuries better treated with a NON-SURGICAL ADJUNCT rather than a DEFINITIVE SURGICAL REPAIR
↓
eg: Angiographic embolization in
  - Liver trauma
  - Pelvic injuries

- Variable physiologic reserve
  - Elderly
  - Multiple co-morbidities

Damage control surgery forms an important complementary strategy to DAMAGE CONTROL RESUSCITATION (DCR)

DCR

1) Permissive hypotension until definitive surgical control of hemorrhage
2) Minimize Crystalloid usage (to mitigate dilutional coagulopathy)
3) Initial use of 5% Hypertonic saline (to ↓ crystalloid requirements)
4) Early use of blood products - PRBCs, FFP, Platelets, Cryoprecipitates
5) Consider drugs to treat coagulopathy - TXA, prothrombin conc, ± F VII a

STAGES OF DAMAGE CONTROL SURGERY

1. Patient selection (see indications ↑)
2. CONTROL OF HEMORRHAGE AND CONTROL OF CONTAMINATION
3. TEMPORARY ABDOMINAL CLOSURE
4. CONTINUED RESUSCITATION
5. DEFINITIVE SURGERY

HEMORRHAGE CONTROL

Control bleeding + Prevent/reduce ischemia

Exposure - vertical midline - Xiphisternum → Pubis - optimal

1) Large clots removed manually
2) All 4 quadrants packed
3) Cell salvage suction → Autologous blood capture for transfusion

4) Assessment for degree & location of significant injuries
   - Continues to bleed briskly
   - Feasible → Definitive surgery

5) Aortic cross clamping at diaphragmatic hiatus
   - Augments myocardial & cerebral perfusion
   - ↓ abdominal exsanguination
   - Caution: time at clamping should be recorded
     - if timely unclamping is not done → visceral ischemia

6) MAJOR VASCULAR INJURIES -
   - Critical artery → temporary intravascular shunt
   - Non-critical artery → ligated

Avoid definitive reconstruction in unstable patients

6) SOLID ORGAN INJURIES - Avoid prolonged repair.
   - pack
   - Partial / total resection

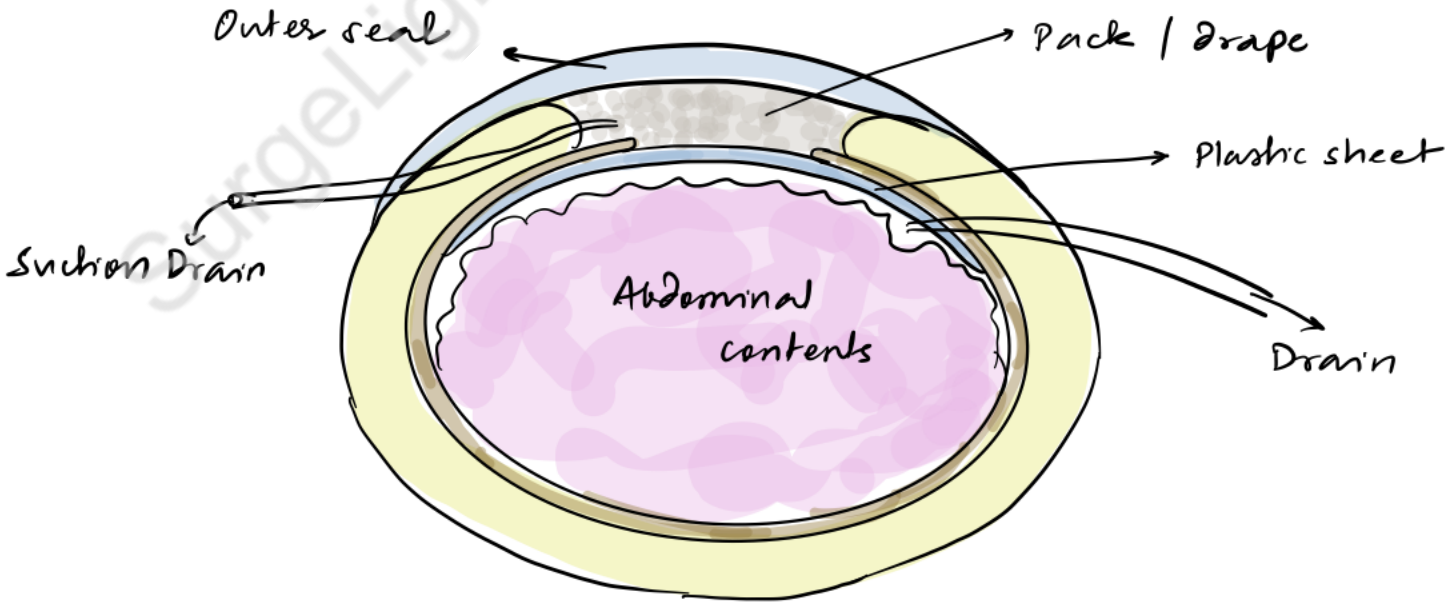
CONTAMINATION CONTROL

- Control spillage of gastrointestinal contents / urine d/t hollow viscus injuries
- Simple bowel perforations → primary repair
- Extensive bowel injury → resect
  anastomosis & even stoma creation avoided in very unstable patients
- Doubtful viability → second look surgery
- Biliary & pancreatic injuries – simple drainage
- Bladder – repair & catheterize
- Ureters – stent / ligate / exteriorise

TEMPORARY ABDOMINAL CLOSURE

Goals →
- Prevent visceral spillage & additional contamination
- Control & quantify effluents
- Prevent fusion of visceral block to the anterior abdominal wall
- Prevent abdominal compartment syndrome, tension
- Facilitate re-exploration
- Facilitate secondary closure later

TEMPORARY ABDOMINAL CLOSURE STRATEGIES



SEQUENCE OF DEFINITIVE REPAIR

- Careful removal of packs
- Inspection / Identification of all injuries
- Definitive gastrointestinal repair
- Thorough abdominal lavage
- Place drain if necessary
- Abdominal closure

Immediate term

Silo placement (Bogota bag)

VAC dressing

Intermediate term

Sequential fascial, skin closure

Interpositional mesh placement

Long term

↓

Planned ventral hernia

↓

Repair

DIAGNOSTIC PERITONEAL LAVAGE

- method to detect presence of blood / contaminants in the abdominal cavity (in the setting of trauma)

Procedure

- Gastric tube placed to empty the stomach
- Urinary catheter placed to empty the bladder
- CANNULA inserted BELOW THE UMBILICUS - directed caudally and posteriorly
- DPA - cannula aspirated - >10ml blood contaminated fluid } +ve
  vegetable fibre
- DPA → negative → 1000ml of WARMED RL allowed to run into the abdomen
  ↓
  drained out via same route
  ↓

Blunt trauma

RBC	$>10^5/\mu L$	$10^5/\mu L$ RBC ≈ 20mL blood
WBC	$>500/\mu L$	→ +ve DPL
Amylase	>175 IU/L	
ALP	>2 IU/L	
Bilirubin	>0.01mg/dL	

- In penetrating trauma - 1/10th of above → +ve
- Drainage of lavage fluid through chest tube ⇒ diaphragmatic penetration

FAST- Focussed Assessment i Sonography for Trauma

- point-of-care ultrasound examination performed ON A TRAUMA PATIENT AT THE TIME OF PRESENTATION by a CLINICIAN
- aim: to identify intraperitoneal free fluid - assumed to be hemoperitoneum in the context of trauma
- has replaced DPL as the preferred initial method to assess for hemoperitoneum

TECHNIQUE

- Patient in supine position
- 3.5- 5 MHz convex transducer
- REGIONS TO BE SCANNED

① SUBXIPHOID / PERICARDIAL VIEW → B & L includes this in eFAST

- to look for pericardial collection - probe placed in EPIGASTRIUM

② Ⓡ FLANK VIEW / RUQ VIEW

- Hepatorenal pouch / Morison pouch
- Ⓡ paracolic gutter

③ Ⓛ FLANK VIEW / Perisplenic / LUQ view

④ SUPRAPUBIC VIEW / PELVIC VIEW → POD / Rectovesical space

- eFAST / extended FAST

⑤ - anterior pleural view for pneumothorax

Study may be limited by obesity, gas, surgical emphysema

FAST is capable of detecting intraperitoneal fluid > 100 ml

- does not reliably determine source of hemorrhage & grade of solid organ injury
- observer dependent
- Not sensitive for retroperitoneal collections

BALLISTIC INJURIES

BALLISTICS

INTERNAL BALLISTICS
describe the projectile within the weapon WHILE FIRING

EXTERNAL BALLISTICS
describe the projectile in FREE FLIGHT

TERMINAL BALLISTICS
describe the interaction between the PROJECTILE AND TARGET TISSUE

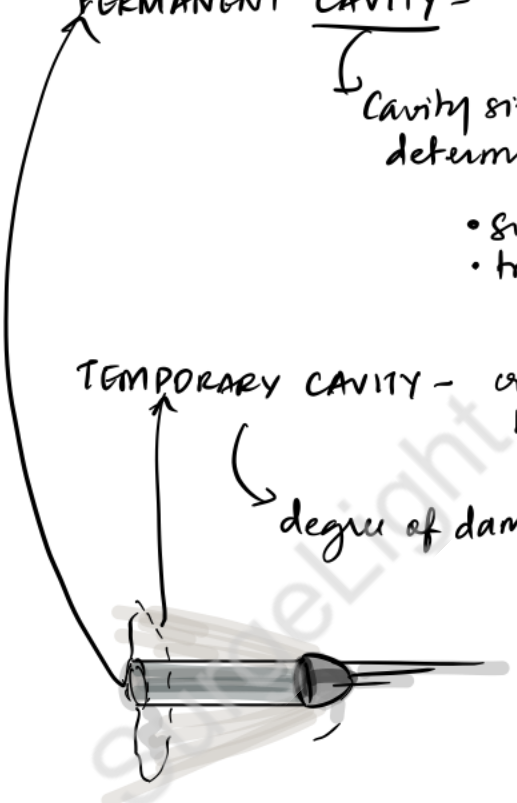
Determinants of Injury
- Weapon
- Type of ammunition
- Range
- Angle
- Clothing / armour

PERMANENT CAVITY – LOCALISED AREA of definitive tissue injury caused by contact with the projectile crushing & lacerating forces

Cavity size is determined by
- Size of projectile
- trajectory of projectile

TEMPORARY CAVITY – created by lateral displacement of tissue that has not been in direct contact with the projectile

degree of damage is determined by
- energy transfered
- material properties of the tissue



GUNSHOT WOUNDS (Bullets)
- High velocity – >2000 ft/sec
- Low velocity – <2000 ft/sec

SHOTGUN WOUNDS (Shots)
- Close range – <20 ft
- Long range – >20ft

BLAST INJURIES

A blast injury is a complex type of physical trauma resulting from a direct or indirect exposure to an explosion

EXPLOSIVE : a substance that can be made to undergo a rapid chemical reaction that will transform a liquid/ solid into gas - liberating a large amount of energy.

DEFLAGRATION : the reaction is propagated by a flame passing through the material at a rate significantly slower than the speed of sound

for 'lower' explosives → COMBUSTIBLE MATERIAL + OXIDANT

Eg: Gunpowder
Gasoline
Pyrotechnics - fireworks, flares

↳ Cause BURNS > BLAST INJURY

DETONATION - 'high' explosives : shock waves pass through the material at SUPERSONIC speeds

- Plastic explosives
- TNT

↓

High pressure expulsion of resultant energy

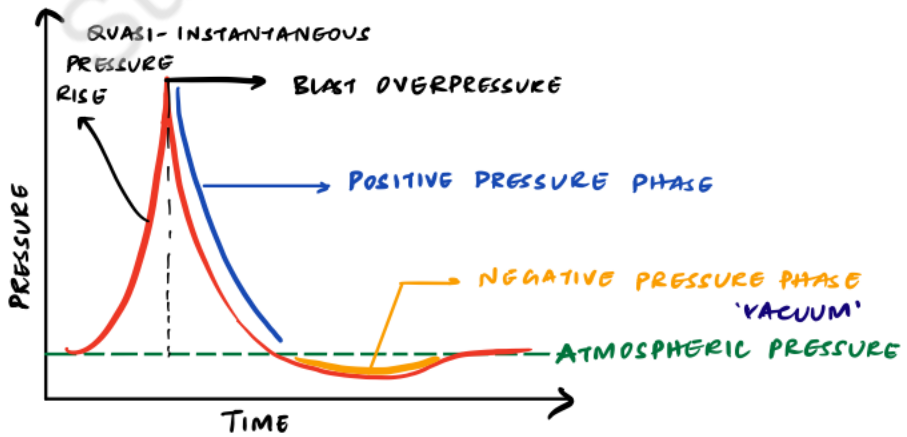
↓

Outward expansion

↓

Wave of detonation - compressed air moves outward at supersonic speeds in a uniform sphere (in a free field)

Peak ↑ of surrounding pressure = BLAST OVERPRESSURE



expulsion of fragments

IED = Improvised Explosive Device

EFP - Explosive formed projectile

BLAST INJURIES

PRIMARY BLAST

- due to BLAST OVERPRESSURE (unique to blast injuries)
- Effect of blast pressure is highest at INTERFACE BETWEEN AIR & TISSUE / LIQUID

SOLID ORGANS relatively resistant to 1° BLAST

INVOLVED TISSUES

- Tympanic membrane injury
  - Otorrhea
  - Hearing loss
- Lung injury - BLAST LUNG

mechanisms: spalling
implosion
rapid acceleration of tissues of diff densities

Immediate	Delayed
Bradycardia	Infiltrates
Apnea	
Alveolar capillary rupture	
Pneumo/hemothorax	
Air embolism	
Pulm edema	

- Intestinal blast injury

CAECUM - most sensitive
Small bowel - mesentery tear
Intra-op - subserosal hemorrhage
Mural hematoma
Tissue necrosis

Bone - 'Brissiance'

SECONDARY BLAST

2/t fragmentation of
- Device casing
- Deliberate fragments in device like nuts, bolts, nails
- Nearby objects

SHRAPNEL

↓

Penetrating injuries

↓

depend on
- range and
- energy of fragment

Unlike ballistic penetrating injuries,

↓

Blast penetrating injuries less predictable

℞ -

Treat like other types of Penetrating injury

BUT

- Thorough search for fragments

Imaging + Intra op

TERTIARY BLAST

↓

2/t gross movement of
- personnel
- objects
- infrastructure

due to BLAST WIND

↓

ANALOGOUS TO CONVENTIONAL BLUNT TRAUMA

Eg:
- Blunt TORSO TRAUMA
  - Chest
  - Abdomen
  - Pelvis
- Head trauma
- Crush injuries
- Traumatic amputation

QUATERNARY BLAST

↓

MISCELLANEOUS injuries

not unique to blast

- Burns 'flash burns'
- Inhalational injury

Prediction of blast lung injury

↓

Indicate proximity to the blast

QUINARY BLAST

↓

EFFECT OF 'DEVICE ADDITIONS'

Eg:
Radiation sickness 2/t Radioactive material
- Infection 2/t Biowarfare

BLOOD & BLOOD SUBSTITUTES

Donor criteria (General)

1) 18-60y
2) Wt > 45Kg
3) Hb ≥ 12.5 g/dL (measured by $CuSO_4$ drop test)
4) No h/o HIV
   HBV
   HCV
   HIN
   CAD
5) No h/o jaundice
6) No h/o Anti HBV Ig / Antirabies Ig } in the last 1 year
7) Not currently pregnant; h/o pregnancy }
8) No h/o - transfusion
   - tattoo
   - skin/nose/ear piercing } in the last 6m
9) No h/o blood donation } in the last 3m (4m-♀ donation)
10) No h/o Malaria }
11) No h/o immunisation in the last 1 month
12) No h/o acute illness in the last 15 days
13) No h/o dental procedures / aspirin intake in the last 3d
14) No h/o antibiotics / Ayurvedic / Siddha drug in the last 2d
15) No h/o intoxicant use over the last 24h

VITALS NORMAL
CURRENTLY NOT MENSTRUATING

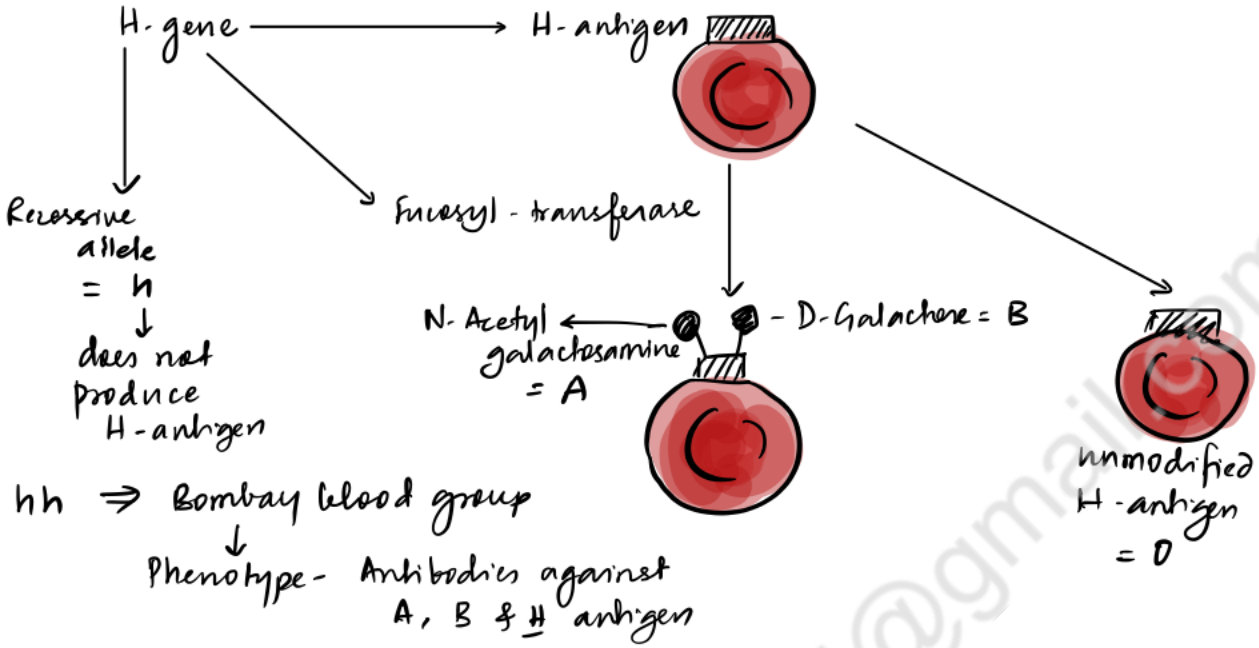
- Whole Blood Donor - upto 450ml
- Apheretic donor → select components taken, rest - reinfused
  Complication: Hypocalcemia (2/t Citrate anticoagulant)
  Eg: SDP

Autologous blood transfusion : Pts undergoing elective surgery predonate their own blood upto 3 weeks before surgery for retransfusion during the procedure

Intra-op blood loss —OR— → collected in cell saver → RBS returned to pt

BLOOD GROUPS

- > 400 BGs
- ABO - most important



INDICATIONS FOR BLOOD TRANSFUSION

① Acute blood loss - to replace circulating volume and maintain O_2 delivery
- Trauma
- Surgery
- Acute loss 2/t massive: GI bleeding, GV bleeding

② Perioperative anemia - to ensure adequate oxygen delivery during peri-operative phase

③ Symptomatic chronic anemia - without ongoing hemorrhage / imminent surgery
- Decompensated

TRANSFUSION TRIGGERS

Traditionally → Hb < 10g/dL, HCT < 30% for peri-op transfusion

CURRENT STRATEGY = RESTRICTIVE TRANSFUSION → Hb > 7-9g/dL
- no adverse effect on mortality

Cardiac surgery - 8g/dL

PRE-TRANSFUSION TESTS -

ABO, Rh - Grouping
Cross match
- Major - Donor RBC, Recipient serum
- Minor - Donor serum, Recipient RBC

Coombs
- Direct: Presensitized RBC + Serum → Complement fixation
- Indirect: Commercial RBC + Ab + Serum

BLOOD AND BLOOD COMPONENTS

Donor

↓

Whole Blood (WB) (450ml)

↓

Components (Separated into components within 8 hr)

- Rarely used in civilian practice
- used in military settings
- Advantages:
  - ↑ HCT, clotting factor activity & platelet count compared to 1:1:1 component therapy esp if fresh

RBC (~200ml)

Stored at 2-6°C

Shelf life - 42d (CPD SAGM) → Adenine, Mannitol; Saline, Glucose

Other preservatives

and ACD, CPD → 21 days

CPDA → Dextrose, Adenine → 35 days

Citrate, Phosphate

FFP (~150ml)

Stored at ≤ -18°C

Shelf life - 1y

Stored Blood - Depletion of factor 8 > 5

FFP → Centrifugation → Cryoprecipitate: fibrinogen, Factor 8, 13, vWF

Cryoreduced plasma → Rx of TTP

Platelets (~ 70ml)

suspended in plasma

Stored at 20-24°C

Shelf life 5d

Dose - 1U/10Kg BW

1 SDPU = 6 RDPU

↓

Raises platelet count by 17,000 to 50,000/cumm

RATE OF TRANSFUSION = 2-4ml/Kg/hr - RBC - within 4h; FFP, Platelets - 30-60min

MASSIVE BLOOD TRANSFUSION

- Defined as transfusion of ≥10PRBCs in 24 hours
- For patients who require >6 units of PRBCs, 6 units of FFP and 1SDPU/6 RDPU must be transfused (1:1:1 ratio)
- If Massive transfusion is anticipated, initiate the 1:1:1 ratio early - ideally within first 2 units.
- After first 6 PRBCs, check fibrinogen levels (first coagulation factor to fall to critically ↓ levels in hemorrhage)

If fibrinogen levels are ≤200mg/dL

↓

give 20 units of cryoprecipitate ~2g FIBRINOGEN

COMPLICATIONS OF TRANSFUSION

1) Non hemolytic Transfusion Reactions - NHTR - fever a/i transfusion
m/c complication
d/t preformed cytokines in donated blood, recepient antibodies
↳ can be reduced by using **LEUCOCYTE-REDUCED BLOOD PRODUCTS** i/c/o Platelet transfusion
↳ ensure storage of < 5d

℞ - PCT

- Bacterial contamination - usually **GRAM -Ve**
  ~℞ - abx
- Allergic reactions - Rash, Hives, itching
  ↳ d/t transfusion of antibodies from hypersensitive donors
  transfusion of antigens to which recepient is hypersensitive
  ↓
  m/c i platelets, FFP

℞ - Antihistamines ⟶ Epinephrine, steroids

2) TACO - Transfusion - associated Circulatory Overload
↳ occurs with rapid infusion of blood, plasma expanders and crystalloids → esp. in older pts i Heart Disease
↓
↑ CVP, Dyspnea, Cough
- PULMONARY EDEMA - Rales

℞ - Diuresis
Slowing the rate of transfusion
minimize fluids during transfusion

3) TRALI - Transfusion Related Acute Lung Injury
↳ Non Cardiogenic Pulmonary edema
<6h

d/t Anti HLA / Anti HNA antibodies in transfused blood
↓
attack circulatory / pulmonary leucocytes
↓
higher levels in female donors (!!)

- Hypoxemia (SpO_2 < 90%)
- Bilateral infiltrates
  ± Tachycardia, Hypotension
- No preexisting cardiaresp illness

℞ - Discontinue transfusion
O_2 → mechanical ventilation

4) Hemolytic reactions

ACUTE

↓

d/t transfusion of ABO incompatible blood

↓

Preformed IgM Ab to ABO Ag

↓ → sudden chest tightness, facial flushing, tachycardia

- Fever
- Hypotension
- DIC
- Hemoglobinemia
- Hemoglobinuria
- Renal insufficiency

↓

℞ - stop transfusion immediately
monitor U/O
Resuscitate

DELAYED (2-10 d of transfusion)

↓

Ig G mediated response to non-ABO antigens
- Low antibody titres at time of transfusion, which rise later (ANAMNESTIC RESPONSE)

↓

Coombs test usually +ve

↓

usually presents as - low grade jaundice
hemoglobinemia
hemoglobinuria

5) CONSEQUENCES OF MASSIVE BLOOD TRANSFUSION

- Hypothermia (prevented by prewarming)
- Hypocalcemia (d/t citrate - chelates Ca^{2+})
- Hyperkalemia - d/t hemolysis esp ↑ stored blood → Arrhythmias, cardiac arrest
- Acidosis
- Metabolic alkalosis + Hypokalemia may also occur
- Dilutional Coagulopathy - prevented by following 1:1:1 protocol
- Chronic transfusions → Iron overload (each PRBC unit ≈ 250mg elemental Fe)

6) TRANSMISSION OF DISEASE

HIV, HBV, HCV → routine screening

CMV

Malaria, Chagas disease, Brucellosis, Syphilis

Prion disease

PLASMA EXPANDERS

CRYSTALLOIDS | COLLOID SOLUTIONS

DEXTRAN - complex branched glucan (Polysaccharide)

- DEXTRAN 150
- DEXTRAN 70 → Molecular weight - 70
- DEXTRAN - 40 → Molecular weight - 40 → LMW Dextran

Owing to size - do not diffuse out of (N) capillaries
Osmotically active
→ **Volume expansion** - used in shock

Negative charge - inhibits vWF mediated platelet adhesion
Decreases blood viscosity
→ used in microsurgery to improve flap survival

- can interfere in blood grouping - esp. Dextran 70

Metabolism
→ 40% excreted by kidneys
rest is slowly metabolised

Complications: - Anaphylaxis, allergic reactions
- Bleeding
- Acute volume overload
- Pulmonary edema
- Cerebral edema
- AKI

Not preferred in septic shock resuscitation
Not used in the first 24 hours following burns

HETASTARCH - HYDROXY ETHYL STARCH

GELATIN - GELOFUSIN

ALBUMIN

More rapid plasma volume expansion since solution remains in vascular space

But,

Alveolar capillaries → highly permeable to albumin

↓

Pulmonary edema

Allergies

BLOOD SUBSTITUTES

Fluids that can carry O_2

The ideal blood substitute
- Delivers O_2
- Requires no compatibility testing
- Has ↓/- side effects
- has prolonged storage capabilities
- is durable in circulation
- is cost effective

TYPES

HEMOGLOBIN BASED

↓

HBOC - Hb Oxygen Carriers

Biomimetic

↓

use the O_2 carrying capacity of Hb

Hb sources - Bovine / Porcine / Human Blood
Transgenic E. Coli

Stroma-free Hemoglobin

AEs: Osmotic diuresis, Nephrotoxicity, Coagulation abnormalities, short $t_{1/2}$, Vasoactive effect 2/t Free radicals

Polymerised Hemoglobin

↓ → to stabilise Hb

encapsulation in liposomes - microspheres

'ARTIFICIAL RBCs' - universally compatible

AEs: Free radical damage
Methemoglobinemia
Immunosuppression

PEG-hemoglobin

PEG-conjugated Hb

NON HEMOGLOBIN BASED

↓

Perfluorocarbons

Abiotic

↓

Synthetic O_2 carriers

Perfluorocarbons → emulsions

↓

can dissolve large volumes of gases

have a LINEAR O_2 dissociation curve

(Not Sigmoid like O_2-Hb)

Disadvantage - for full loading ↑ Partial pressure & ↑ FiO_2 are required

↓

inefficient oxygen delivery

Eg: OXYGENT

AEs:
Thrombocytopenia
ILI

METABOLIC RESPONSE TO INJURY

Concept of 'milen interier' (Claude Bernard)

Homeostasis (Walter Cannon)

'GRADED RESPONSE'

- Degree of response depends on severity of injury
- Minor injury (eg. elective surgery of intermediate severity)

  transient / modest ↑ in
  - temperature
  - HR, RR
  - Energy expenditure
  - TLC
- Major trauma

  SIRS

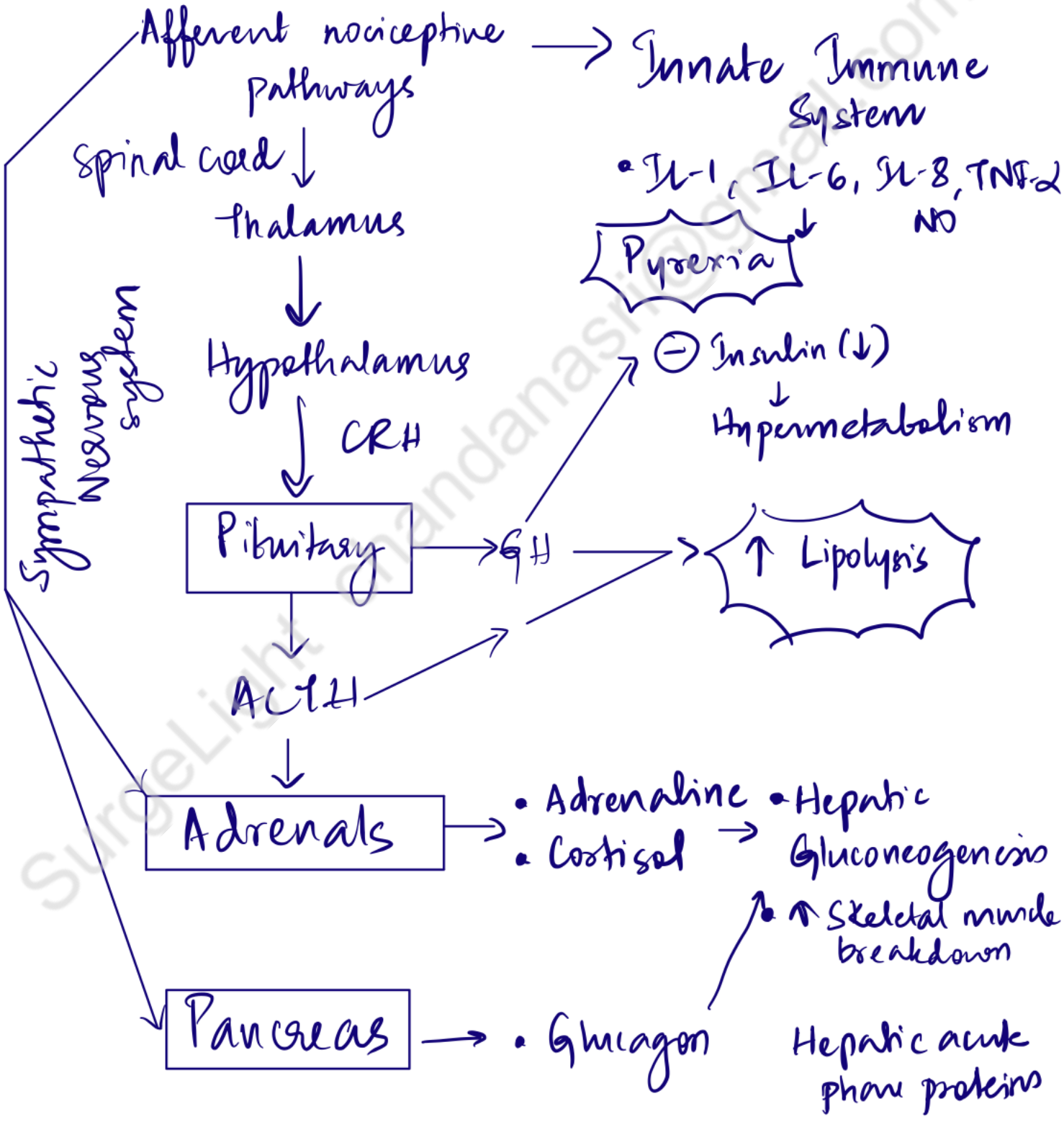
  MODS

EVOLUTION OF METABOLIC RESPONSE

Proinflammatory ⟶ CARS (Compensatory Antiinflammatory Response Syndrome)

MEDIATORS OF RESPONSE

1) Neuroendocrine system
2) Sympathetic Nervous System
3) Immune system



CARS

Prolonged proinflammatory response

↓

endogenous cytokine antagonists (IL-1 Receptor antagonist)

↓ IL-4, 5, -9, 13, TGF β

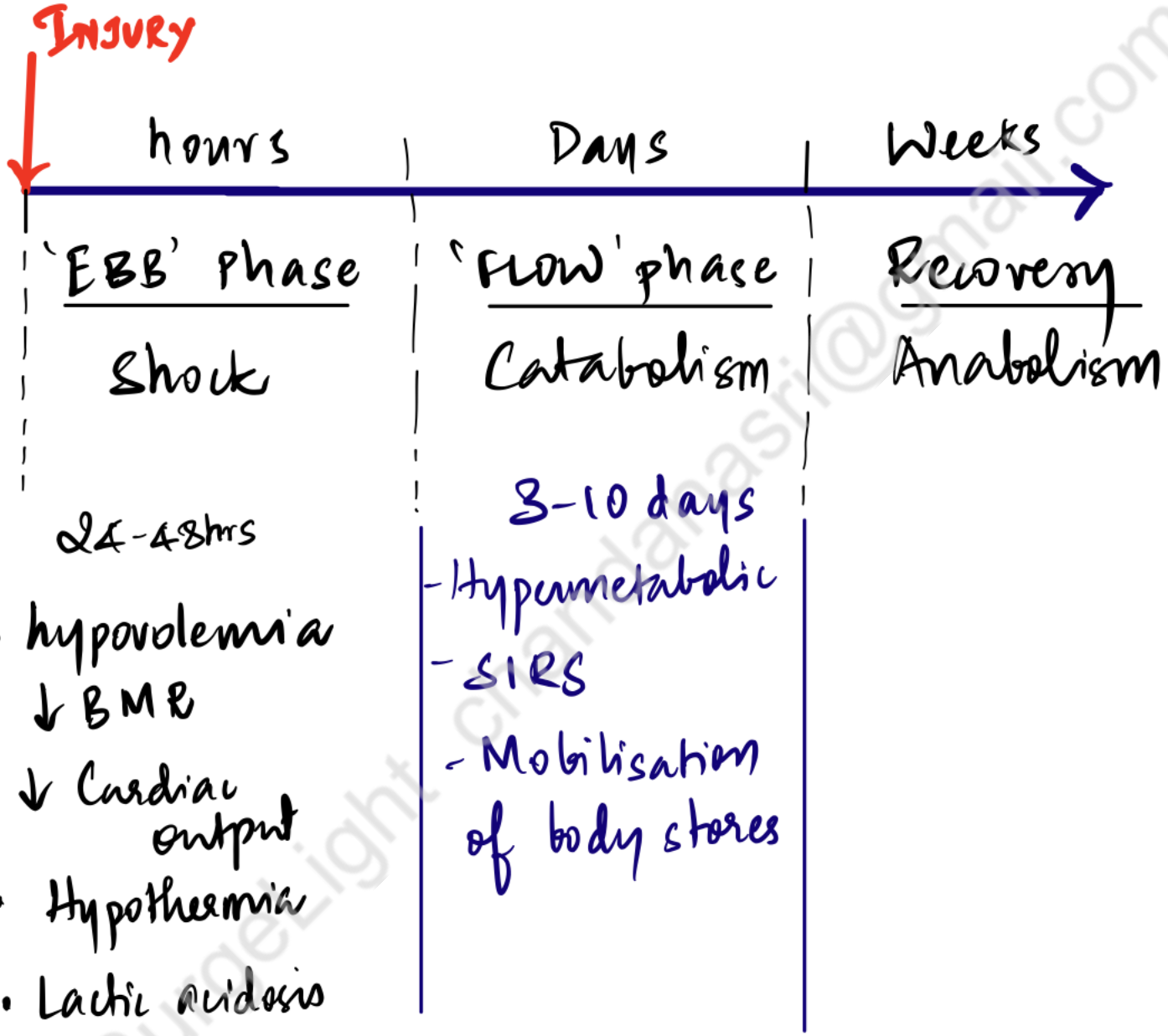
Counterinflammatory Th_2 response

↓

- Immunosuppression
- ↑ Susceptibility to nosocomial infections

EBB & FLOW MODEL

David Cuthbertson



ERAS is based on this metabolic Response

RETAINED FOREIGN OBJECTS

- any surgical item found in a patient after he/she has left the OR (necessitating a 2nd Sx for removal)

m/c - Surgical sponge

Others - needles
instruments

Frequent cause of litigation

Surgical 'Never' event

GOSSYPIBOMA / TEXTILOMA / COTTONOID

- mass of cotton matrix / sponge left behind in a body cavity during surgery

- Abdominal cavity
- Pelvis
- Thoracic cavity
  - Pleural
  - Pericardial

RISK FACTORS

- Emergency procedures
- ↑ bleeding / contamination
  - ↓ obscuring the field
    - ↑ chances that packing is required
- Inaccurate keeping of mop counts
- Changeover of operating / assisting surgical / nursing staff esp. in long procedures

Prevention

Follow strict Mop counts

Surgical safety checklist

PATHOLOGY

ASEPTIC FIBROUS RESPONSE

↓

Adhesions, encapsulation

↓

Granuloma formation

LATE PRESENTATION

EXUDATIVE RESPONSE

↓

Secondary bacterial contamination

↓

Abscess / Fistula

PRESENT IN EARLY POST OP PERIOD

Rare Consequences - Migration into GI tract 2/t erosion of wall
obstruction

Imaging - Spongiform appearance ± gas bubbles

low density mass ± thin enhancing capsule

Calcifications

Rx - Re-operation, removal

TETANUS

Clostridium tetani - terminal spore-bearing, gram positive bacillus

Spores - widespread in soil, manure

traumatic military/ civilian wounds

↓

Infectious form - Enters via wound
- Contaminated wounds
- Crush injuries
- Septic labor
- Contaminated surgical instruments

Incubation Period
- 7-10 days

Prolonged in Latent tetanus

↓

Germination of spores
(anaerobic medium)

↓

Tetanus bacilli

↓

EXOTOXINS

Tetanospasmin

Blood / lymph / Perineurally

Affects:
- Neuromuscular junction
- Motor neurones of anterior horn of spinal cord
- Cranial nerves
- CNS

Tetanolysin → Hemolysis

Acts on presynaptic membrane of inhibitory neurons

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Sustained excitation

Sphincter disturbance

CNS seizures

Respiratory muscle spasm

Spasm - AXIAL MUSCLES
Opisthotonos
Orthotonos
Emprosthotonus
Pleurosthotonus

CRANIAL NERVES
III, IV, VI - Ophthalmoplegia
V - TRISMUS
VII - Risus Sardonicus
VIII - Hyperacusis
IX, X, XII - Dysphagia

Complications

- Musculoskeletal injury
- Aspiration pneumonia, respiratory failure
- Cardiac arrhythmias
- Toxemia

MANAGEMENT

- Isolation
- Source control
  - Abx - Penicillin
    Metronidazole
- Neutralise toxin

  ATG - 3000-6000 IU IM
  (Human)
  → 100x more potent than Equine

- Symptomatic

  Spasmolytics: BZDs
  Barbiturates
  Muscle relaxants

  Autonomic stabilization - Fluids
  α_1 Blockers
  α_2 agonists

  Respiratory support

Prevention

TT - Tetanus toxoid
0.5ml IM