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* wound healing *

• wound :- A cut/break in the continuity of any tissue cause by injury.

• Healing :- It is a body response to an injury an attempt to restore the normal structure & function.

* The process of healing involve 2 distinct process :-

A) Regeneration -

B) Repair

A) Regeneration :- when healing takes place by proliferation of parenchymal cell.

- Depending upon their capacity to divide, the cells of the body can be divided into 3 groups.

1) Labile cells :- These cells continuously dividing cells throughout life. under normal physiologic conditions.
E.g. surface epithelial cells of resp. tract, alimentary tract, vagina, cervix, haematopoietic cell of bone marrow etc.

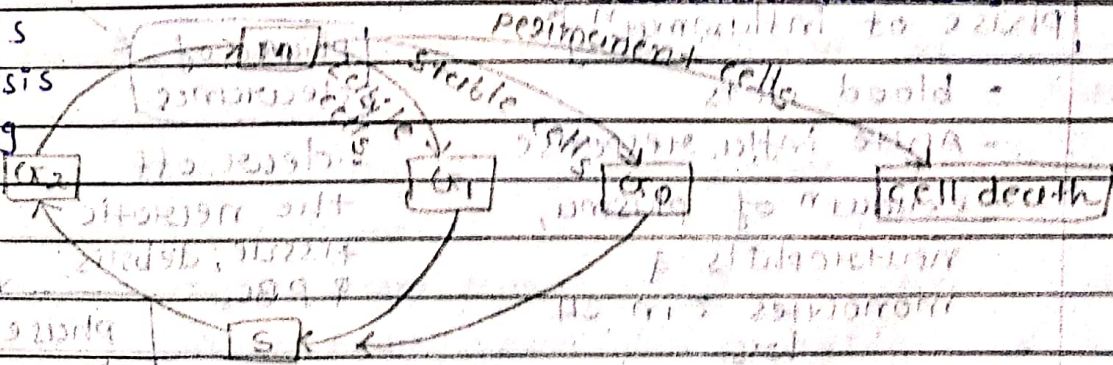
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2) Stable cells :- These cells lose/lose their ability to proliferate after adolescence but maintain the capacity to multiply in response to stimuli throughout adult life.
E.g. parenchymal cell of organ like liver, kidney, pancreas, thyroid.
mesenchymal cell like smooth muscle cells, fibroblast, bone & cartilage cells.

3) Permanent cells :- These cells lose their ability to proliferate around the time of birth.
E.g. neurons of nervous system, skeletal m. cells, cardiac m. cells.

M-mitosis
S-synthesis
G₀-resting phase



cell cycle & Parenchymal cells relation

- mitosis is controlled by genes & encode for release of specific protein molecules that promote/inhibit the process of mitosis at diffⁿ steps.

- mitosis-promoting protein molecules are cyclin A, B & E.

- These cyclins activate cyclin-dependent kinases (CDKs).

B) Repair :- when healing take place by proliferation of connective tissue.

2 processes involved :-

1) granulation tissue formation

2) wound contraction

- Response by mesenchymal cells, endothelial cells, platelets, parenchymal cells.

1) granulation tissue formation :- 3 phases

Phase of inflammation

- blood clots
- Acute infla. response
- exudatⁿ of plasma, neutrophils & monocytes in 24 hrs.

Phase of clearance

- clear off the necrotic tissue, debris & RBCs.

Phase of granulation tissue formation

- Angiogenesis
- fibrogenesis

Angiogenesis :- proliferation of endothelial cells.

↓
- proliferated endothelial cells are solid buds.

↓
- develop lumen in few hrs.

↓
- start carrying blood.

↓
- new blood vessels are leaky

↓
- oedematous appearance of new granulatⁿ tissue.

↓
- new blood vessels differentiates

↓
- muscular arterioles, thin walled venules, & true capillaries formed.

Fibrogenesis :- new blood vessels in amorphous ground subⁿ / matrix.

↓
myofibroblast → intermediate features of s. muscle & fibroblasts.

↓
collagen fibres appear by 6th day.

↓
surface receptors for fibronectin molecules on myofibroblast form bridge betⁿ collagen fibres.

↓

↓
maturation proceed.
↓
more collagen formed.
↓
no. of active fibroblast & new
blood vessels ↓se.
↓
inactive looking scar formed
↓
cicatrization.

* Types of wound :-

- few classification have been given below

1) close & open wound

A. close wound

1. Abrasion

2. contusion (bruise)

3. Hematoma

• Abrasion :- It is all superficial injury of skin due to the friction betn the object & epidermis layer of skin.

↓ resulting.

scraping like structure seen on superficial layer of epidermis of

In abrasion bleeding is slight & healing is rapid.

• contusion :- It is a effusion of blood in subcutaneous tissue due to rupture of capillary caused by applicatⁿ of hard blunt forced

• Haematoma :- "extravascular accumulatⁿ of blood clot"

It may be subcutaneous, intramuscular

B. open wound

1. Incised

2. Lacerated

3. Penetrating

4. Crushed

5. Punctured

6. Perforating

• Incised wound :-

- It is a cut & produced by sharp edge of weapon like knife, blade, glass, katar etc.

- It gives neat & clean scar.

• Lacerated wound :-

- A lacerated wound is a wound in skin, muscle / underlying tissue are torn as a result of application of hard, blunt & rough weapon.

- Caused by hard, blunt, & rough weapon like road traffic accidents (RTA).

• penetrating wound :-

- It is similar to incised but that its depth is more.

- Here wound terminated in body cavity.

i.e. chest, abd. etc.

cause intestine, liver, spleen may damage.

• punctured wound (stab wound)

- It is deep wound produce by any pointed end of weapon / object entering in body.

- caused by - Tip of knife

- bullet

- Point of pen

- Iron Rod.

• crushed wound

- It is occur due to fall down, run over it by vehicle etc. accident.

- These wound is more dangerous because it cause severe haemorrhage.

- may be crushing of blood vessels & tissue

II) Tidy & untidy wound

A. Tidy :- Incised, clean, healthy tissue
- heal by primary union

B. untidy :- crushed, unclean, unhealthy
- Tissue loss

- heal by secondary union

III) Acute & chronic wound

A. Acute :- stab wound

abrasion.

sign of healing in less than 4 weeks.

B. chronic :- leg ulcer.

- prolonged inflammatory phase

* The 5 Basic processes takes place in wound healing are :-

- Inflammation
- wound contraction
- Epithelization.
- granulation tissue formation.
- scar remodelling

A) Inflammation :-

Disruption of tissue integrity

↓

Inflammation starts

↓

Platelet become adherent & clotting factor form a haemostatic plug.

↓

Bleeding stopped



blood vessels undergo transient vasoconstriction



Histamine → Primary mediator of inflammatory vascular responses.



histamine released by platelets, mast cells & granulocytes.



local vasodilatation



↑ permeability of small vessels



protein & plasma leak out of vessels



histamine action is short lasting & local sources are depleted soon



kinins & prostaglandins (PGE₁ & PGE₂) start local inflammatory vascular response.



Kallikrein: enzyme found in plasma & granulocyte



kallikrein release bradykinin & kallidin



In presence of kinins, cells produce a variety of prostaglandins.



prostaglandins → final mediator of acute inflammation

↓
leukotaxine → peptide formed in damaged tissue by enzymatic destruction of albumin

↓
leukotaxine acts as chemotactic agent

↓
attracts actively motile leucocytes into wound

↓
leucocytes starts engulfing & removing cellular debris & injured tissue fragments

↓
At 1st polymorphonuclear leucocytes dominate

↓
Transient phase of white cell migration ends.

↓
granulocyte's shorter life die & release enzymes & hydrolyses into local environment

↓
previous ratio of 1 granulocyte & monocyte was 1:1 & now monocyte proportion ↑

↓
monocytes continue their scavenging activity for weeks

↓
monocytes dominate by 5th day.



they are phagocytic & ingest cellular debris.

inflammation $\rightarrow$ Rubor, tumor, calor, dolor, function laesa.

B) wound contraction

wound contraction seen in open wounds.

tissue loss

Begins after 3 to 4 days of wound

lag period $\rightarrow$ when no contraction seen.

lag period is followed by period of rapid contraction

contraction completed by 14th day

wound reduces to 80% of its original size

This contracting mechanism is located in the margin of wound. $\rightarrow$ (picture frame area)

Histologic area of picture frame area reveals a collection of large, stellate, pale staining cell called myofibroblast

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myofibroblast responsible for moving overlying dermis.



contraction depends on the skin amount available surrounding the wound

- cause :- Removal of fluid
 - contraction of collagen
 - contraction of granulation tissue
- Inhibiting factors :-

- corticosteroid

- Burns

- Skin grafting. Immediate.

- X-irradiation.

- Tetracycline → acts on actin in myofibroblast, smooth m. inhibitor.

- colchicine & vinblastin → inhibitors of microtubule formation in myofibroblast

- cytotoxic agents.

e) Epithelialisation :-

Epidermis adjacent to the wound edge begins thickening on 1st day

marginal cells lose their firm attachment to the underlying dermis



enlarge & begin to migrate into wound



fixed basal cells in a zone near the wound edge undergo rapid mitotic divisions



daughter cells migrate



entire wound surface is re-epithelialized



wound defect bridged



migrating epithelial cells lose their flattened appearance



cells become columnar in shape

layering of epithelium

surface cells keratinize

• epithelial thickening & keratinization may produce marked foreign body reaction & formation of sterile abscess.

• no regeneration of hair follicles, sweat & sebaceous glands in epidermis

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D) Granulation tissue formation :-

- Haematoma in the wound is soon replaced by granulatⁿ tissue, consisting of new capillaries & fibroblast

• phase of traumatic inflammation

• phase of Demolition

- dead tissue cells → autolytic enzymes
- polymorphs & proteolytic enzymes
- mononuclear cells & macrophages digest & remove particulate matters.
- macrophages fuse to form foreign body giant cells

• Granulation tissue formation

A. stage of vascularization.

wound clot invaded by macrophages.

↓
phagocytosis.

↓
particulate matters removed.

↓
solid buds of endothelial cells grow out of existing damaged blood vessels at surface of wound

canalization & anastomosis to neighbours

series of vascular arcades formed.

gaps betⁿ endothelial cells.

basement membrane poorly formed.

new capillary loop leaks protein

Tissue fluid formed acts as medium for fibroblastic growth.

capillary loops differentiate.

Arterioles, venules & capillary formed

direct arteriovenous shunts also formed

fibroblast accompany the capillary loop

become larger to become elongated fibrocytes

pH become alkaline during fibrogenesis.

fibroblast forms collagen

Basic molecules secreted by fibroblast are
trypocollagen.

Tropocollagen condenses in mucopolysaccharide extracellular space

↓
fibrils formed

↓
fibrils group together

↓
reticulin fibres formed

↓
condensation together

↓
collagen fibres formed

- Type-I collagen is found in tendon, ligament, skin & bone
- Type-II collagen is found in cartilage, mainly in articular & costal cartilage
- Type-III → fetal dermis + later replace type-I

Amino acid in collagen - hydroxyproline
hydroxylysine

• Fibroblasts responsible for production of mucopolysaccharide ground subⁿ

B. stage of devascularization

fibroplasia proceeds



some vessels undergo atrophy



endothelitis obliterans → lumens obliterated due to intimal proliferation



granulation tissue looks pale



nerve fibres & lymphatics form from existing ones



mast cells appear & their granules are derived from ground sub.M



mast cells disappear



hyalinization occurs

E) Scar remodelling :-

in 7th day wounds show a delicate fine reticulum of young collagen fibres



physical properties of scar depend on collagen fibres



Scar strength ↑ progressively $\propto$ time



Scar maturation takes period of many months.



Scar slowly & progressively change in bulk & form.



Scar changes their anatomic arrangement.



Oriented fibres seem to become thicker.



non-oriented fibres disappear



Wound edges together by 3 dimensional weave



central scar remodel itself to fill the defect after contraction



gain in tensile strength begins about 5th day & rises rapidly betⁿ 6th - 17th day

→ Tensile strength :-

- Acts as the main safeguard against wound dehiscence.

- Tensile strength of wound corresponds to the rise in amount of collagen +vt.

• factor influence the tensile strength

- Directⁿ of wound
- pull of underlying muscles
- previous wound
- Abdominal binders.

• factor affecting granulation tissue formⁿ

- cortisone administration
- scurvy
- protein starvation

* Types of wound healing

- 1) healing by 1st intention / primary union
- 2) healing by 2nd intention / secondary "

- 1) primary union :-
- clean & uninfected
 - surgically incised
 - Cost much less of cells & tissue
 - edges of wound are approximated by surgical suture
 - primary union

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Events :-

Haemorrhage

↓
inflammatory response

↓
epithelial changes

↓
epithelialisation

↓
suture sticks

2) secondary union - unclean & infected

- irregular margins

- suture not used

- extensive loss of cells & tissue

- healing through exuberant granulatⁿ tissue to fill gap

Events :-

Haemorrhage

↓
inflammatory phase

↓

proinflammatory

epithelial changes



granulation tissue formation



wound contraction



epithelialisation

* complication :- Implantation cysts

painful scar

epithelialisation.

keloid formation

Neoplasia.

* factor Influencing healing

A) General factor

• Age :- fast in youngsters

- Nutrition :- protein deficiency
vit. c deficiency
vit. A deficiency
zinc, calcium, copper & manganese deficiency
- Hormones :- corticosteroid
- deoxy corticosterone acetate
& anabolic steroid like testosterone
- cytotoxic drugs & Radiation.

-> Conditions delay wound healing :-

- Anaemia
- uraemia
- Jaundice

- Diabetes

- Blood dyscrasias

b) Local factors

- position of skin wound
- Blood supply
- Tension
- Infection
- movement
- exposure to ionizing radiation
- foreign bodies
- Adhesion to bony surfaces

- Necrosis

- Lymph drainage

- ultraviolet light

- Faulty technique of wound closure

* wound management

- Admission in the hospital

- monitoring of temperature, pulse & respiration

- Systemic Antibiotics

- Injection tetanus toxoid for prophylaxis against tetanus.

- wound cleaning, dressing & suturing.

Burns

- Defⁿ :- It is defined as thermal injury & causes damage of skin / other part of body occur due to Extreme heat, flame, contact w/ heated object & chemicals.

- Types :-
 1. Thermal injury
 2. electrical injury
 3. chemical injury
 4. mechanical injury
 5. cold injury
 6. ionising radiation
 7. sun burn

1. Thermal injury :-

- caused by dry & moist heat.
- scald, blister, bomb injury, Hot steam, liquid etc

2. electrical injury :-

- low voltage electrical sources produces direct injury.
- majority of electrical burns caused by high-voltage current.

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3. chemical injury

- caused by strong acid & strong base
- severity of burn depends upon concentration

4. mechanical injury

- caused direct contact with heated object.

5. Ionised Radiation

- occur due to exposure of x-ray, / radium

6. cold injury

- caused by exposure of extreme of cold like chilblain.

7. sun burn

- caused by extreme of sun heat.

• classification :- Based on,

- 1) Dupuytren's classification
- 2) Thickness of skin
- 3) severity of burn
- 4) % of burn

I: Dupuytren's classification :-

- classify burn into 6 degree,

• 1° :- only superficial layer of skin involved.
- Red, inflamed & painful.

• 2° :- in the superficial layer.
- Blister formation occur
- painful, Redness.
- Heal to out scar formation.

• 3° :- Epidermis + dermis involved.
- Heal to scar formation.
- extremely painful.

• 4° :- whole thickness of skin involve
- some like 3°.

• 5° :- Include skin + subcutaneous tissue.
- less painful due to total damage of sensitive nerve ending.

• 6° :- Involve skin + deep to subcutaneous tissue.
- less painful.
- Involve muscles + bone + organ also.
- grafting require.

2. Depend on thickness of skin :-

A. partial thickness burn :-

- involve 1° & 2° of burn
- Red, painful & blister

B. full thickness burn :-

- involve 5° & 6° of burn
- skin is charred, insensitive.

3. Depend on percentage of burn :-

- This is based on body surface area (BSA) & calculated by "Rule of 9"

Burn	% of total body surface area
------	------------------------------

A. Head & neck = 9%

B. front of chest = 9%

C. Back of chest = 9%

D. front of Abd. = 9%

E. Back of Abd. = 9%

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- 1. Rt. upper limb = 9%
 - 2. Lt. " " = 9%
 - 3. Front of Lt. lower limb = 9%
 - 4. Back of Lt. " " = 9%
 - 5. Front of Rt. lower limb = 9%
 - 6. Back of Rt. " " = 9%
 - 7. Perineal area = 1%
- 100% of whole body

4. Depend on severity of Burn :-

- A. mild :- partial thickness
 - adult :- 15%
 - child :- 10%
 - full thickness :- 2%

B. moderate :-

- 2° :- 15-25% burn affected area
- 3° :- 2-10% " " "
- Ext involve eyes, ears, face, hand & feet

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C. Severe :-

- 2° :- Adult - $> 25\%$

child - $> 20\%$

- 3° :- $> 10\%$

- involve eyes, ears, face, hand & feet

• Clinical Feature :-

- Redness

- Painful

- Blister

- Anxiety

- fluid loss

- swelling

- Tachycardia

- Tachypnea

- Hypovolaemia

• Management :-

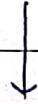
1. Local T/t

2. General T/t

3. Treatment of shock

• pathophysiology

- heat causes coagulatiⁿ necrosis of skin & subcutaneous tissue.



- Release of vaso-active peptide



- Altered capillary permeability



- loss of fluid → hypovolaemic



↓ se cardiac
output



↓ se myocardial
function



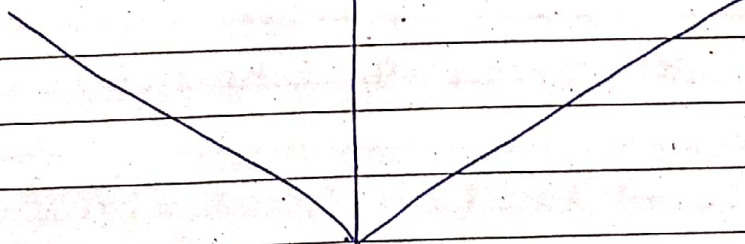
↓ se renal
blood flow



oliguria



altered
pulmonary
resistance
causing
pulmo. edema



Infection



systemic inflammatory response
syndrome



multi organ dys-function
syndrome

I. Local Tx :-

A. Pre-Hospital care :-

- Stop source of burning
- keep away / Remove pt from burning environment
- quickly apply clean cold water
- elevate the limb to reduce swelling
- Check other injury

B. Hospital care :-

- Admit the pt & keep him in clean environment

i) Dressing :-

- 2 method applying for dressing :

• open wound method :-

- wound can be left open to atmosphere after applying silver sulfadiazine cream

• close wound method :-

- After applying silver sulfadiazine, 1% silver nitrate & metenide cotton Rollage is applied to close.

Bandage applied.

ii) skin grafting :-

- Tangential excision followed by grafting is done in 48-72 hrs.
- Burn wound is excised tangentially till fresh bleeding occurs.
- Latex skin grafting is done.

• Advantage :- It prevents secondary infection & septicemia.

- ↓ se hospital stay
- ↓ incidence of hypertrophic scar.

iii) physical Therapy :-

- After burn extremities become stiff.
- To prevent this contracture physical therapy should be started.
- early movement start as early as possible.

- Immobilisation of pt. causes stiffness.

2. General T/t :-

A. Antibiotic :-

- Burns causes ↑ risk of contamination of gram +ve organism.

- Topical Antibiotic play significant role in burns.

- prophylactic Antibiotic are to be avoid.

- penicillin is given.

B. Analgesics :- for relief in pain.

C. Tetanus :-

- Burns causes muscle spasm & boxen attitude.

- 0.5 mL Tetanus Toxoid (TT) Im.

- if Active immunisation is done in

10 yr ⇒ 250 + 500 unit

D. Nutritional support :-

- In all case of major injury metabolism of body twice than normal.

↓

So, exogenous calories of N_2 to prevent excessive catabolism.

- Nutrition always give by nasogastric tube in initial days.
- If diarrhoea occur $\Rightarrow$ IV fluid supply rather than orally.

E. GIT care:-

- Burn causes $\Rightarrow$ Intestinal motility loss
- Curling ulcer in stomach.
- $\uparrow$ se vomiting - Aspiratⁿ of vomit into lungs.
- gastrointestinal bleeding.

- T/t :- Antacid $\Rightarrow$ H_2 receptor blocker

- nasogastric feeding

- IceD saline solⁿ for Hemorrhage

- Ryle's tube for Curling ulcer (acute peptic ulcer)

3. T/t of Shock :-

A. sedation of pt. :-

By IV in minimum dose

- morphin, Barbiturate in children.
- morphin :- 10-12 mg Im.

- Sedation help in recovery of pt. early

B. Fluid resuscitation :-

- In shock there is hypovolumic state create.

• Indication :-

- much blood loss occur
- moderate to severe burn
- more than 15%
- if more than 20% occur $\Rightarrow$ Blood transfusion need.

• Formula for fluid resuscitation :-

$$i) 1^{st} \text{ Ration} = \frac{\% \text{ of burn} \times \text{body weight (kg)}}{2}$$

- 3 Ration in 12 hr
1st 12 hr
" Next 12 hr
" " " "

ex: 40% burn, 60 kg weight.

$$1^{st} \text{ Ration} = \frac{40 \times 60}{2} = \underline{1200 \text{ mL}}$$

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1st 12 hr = 3600 mL fluid
2nd " = 2400 mL fluid
3rd " = 1200 mL fluid
RL
Eringers
(electrolyte)
Esol. n 30% depletion
ment of plasma

ii) 4 mL/kg $\Rightarrow$ 24 hr.

$\frac{1}{2}$ kg $\Rightarrow$ 8 hr.

$\frac{1}{4}$ kg $\Rightarrow$ 8 hr.

C. Airway maintenance

- During shock $\Rightarrow$ Dyspnea, Tachypnea, respi.
coughs, comm.

- T/F $\Rightarrow$ upper airway treat by
Inhalation of cooling air.

- endotracheal tube insertion occur.

- Tracheostomy done.

D. Temperature

BP

pulse rate

Respi. Rate

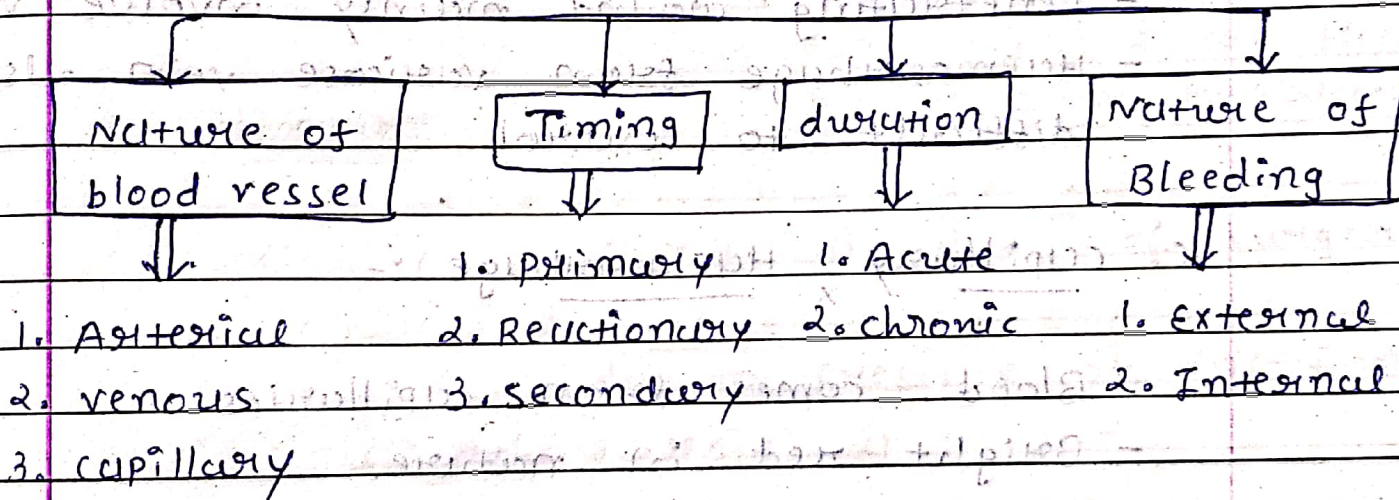
should be
maintain

* Hæmorrhage *

• Def.ⁿ - It is defined as an excess loss of blood due to ruptured blood vessel.

• classification

- It is classify broadly into 4 Types:



1. Based on Nature of blood vessel :-

A. Arterial Hæmorrhage :-

- Hæmorrhage comes from artery
- Bright red in colour
- eject spurts (spray)
- easily controllable

B. venous Hæmorrhage :-

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- Haemorrhage comes from venous s.

- Dark red in colour

- eject steadily (सुत)

- copious Haemorrhage esp. when large vein is affected.

- Not easy to stop.

- penetrating wound mainly involve veins

- Haemorrhage from varicose vein also difficult to control.

c. capillary Haemorrhage :-

- Blood comes from capillaries

- Bright red in nature.

- oozes out rather than flow

- easy to control

2. Based on timing of Haemorrhage :-

A. primary Haemorrhage :-

- occurs at the time of surgery

- cause is injury to vessel

- may be arterial, venous / capillary

- more common in surgery on malignancy.

B. Reactionary Haemorrhage

- Bleeding in 24 hrs of surgery.

- Cause is slipping of ligature / dislodgement of clot.

- Bleed start when there is rise in the arterial / venous pressure.

ex. sup. thyroid A. can bleed few hr. after thyroidectomy.

C. Secondary

- Bleeding occur after 7-14 days of surgery.

↓
- Bleeding occur due to presence of infection on sutured material.

↓ causes

- slough formation on sutured wall.

- common after Haemorrhoid (Piles) surgery.

i.e. CR surgery / amputation

3. Based on duration

A. Acute

- Bleeding occur suddenly

- large quantity of blood loss.

- seen in case of Accident

oesophageal varices

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B. Chronic

- Blood loss occurs over a time

- Haemorrhage either by internal / external.

- i.e. chronic duodenal / peptic ulcer

- TB

- Haemorrhoids

4. Based on Nature of bleeding :-

A. External bleeding

- External / visible bleed

- soft tissue injury

B. Internal bleeding

- Internal / invisible bleed

- blunt / penetrating trauma

- It is concealed (скрыт) Haemorrhage

- due to rupture of liver or spleen

- sometime this concealed Haemorrhage may be externally seen

not Hematemesis, Haematemesis, Haematuria

• pathophysiology

- loss of more than 30-40% blood volume
resulting in falling B.P. leading

"Haemorrhagic shock"

- Haemorrhagic shock can be classified in

1. mild Haemorrhage (class-I) :-

- when blood loss is less than 750 mL
($< 15\%$ blood volume)

clinical features

1. mild Tachycardia

- Thirst normal

- urine output normal

BP normal

2. moderate Haemorrhage (class-II) :-

- when blood loss is 800-1500 mL

($15-30\%$ of total BV)

- powerful vaso-constriction by Adrenalin

Non-adrenalin

C/F :-

↑ Thirst

- Heart rate :- 100 - 130/min

- urine output :- ↓

- BP :- systolic normal
diastolic elevated (↓)

3. Severe Haemorrhage (Class-III)

- when blood loss is 1500-2000ml

(30-40% of total BV)

• C/F :- Heart rate more than 130/min

- Thirst ↑

- urine output ↓

- BP = fall (systolic & diastolic)

- Drowsiness, aggressive & confuse

4. Very severe Haemorrhage (Class-IV)

- when blood loss is more than 2000ml

(40% of total BV)

• C/F :- Heart rate more than 130/min

- Thirst ↑

- urine output ↓

- BP = very low / unrecordable

- They may be renal shut down

- may be multi organ failure

↓
Kidney = Acute renal failure

Heart = myocardial Ischemia

Lung = acute lung injury

• Management :-

Goal for Tx + T

1. prevent Blood loss
2. provide less Blood.

I. Blood loss prevention

A. Rest

- Restlessness causes more blood loss.
- If pt. become restless due to pain, Hemorrhage will be more.
- give sedative => morphine is good sedative.
- morphine => IV/IM.
- It should avoid in children & older age.
- Also when respiratory depression.
- some sedative also give along w sedative i.e. Benzodiazepam

B. position

- some position help to reduce Hemorrhage.

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i.e. elevate head in case of thyroidectomy wound

- elevate foot (Trendelenburg position) in case of varicose vein haemorrhage.

c. pressure & packing from outside :-

i) Apply gauze piece

- In case of wound by stab / external sterile gauze & bandage may be used as pressure bandage

- To reduce external bleeding

- if gauze is not available then clean linen cloth can also use

ii) Tourniquet

I. Indication

- when blood less field is desire during operation therefore

- Reduction of fracture

- Repair of Tendon

- Repair of nerve
- Now a days Tourniquet is only used in operation theater

2. contra-indication

- Pt. with peripheral vascular Dz

3. Types

- Rupture Bandage
- pneumatic cuff or Bandage

4. pre-caution

- Too loose $\Rightarrow$ meaningless to apply / not.
- Too tight $\Rightarrow$ Thrombosis $\Rightarrow$ gangrene
- Too long $\Rightarrow$ causes gangrene

5. complication

- Ischemia
- gangrene
- Tourniquet nerve palsy

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D. operative method

i) In case of Haemorrhage 1st of all detect injured artery



- stopped by artery forceps & clips apply to artery



- Ligate & cut / silk the artery

- In case of small vessel artery is join by courtesy

- In case of large vessel artery is join by silk

ii) In those cases where bleeding vessel can't be detected



- Blood is prevent by gauze pack for 5 min



- After 5 min gauze pack remove & slight bleeding can be identified by long curved artery forcep.

iii) when blood is ooz



- only apply gletine / adrenaline sponge

↓

↳ can stop bleeding.

2. Restore the blood volume

A. Natural

- As soon as haemorrhage take place

↓

- Body start to restore blood

↓

- 1st of all interstitial fluid is absorb into vessel

↓

- causes haemodilution (water res)

- plasma protein replaced by liver

- Red cell take 5 to 6 week for

recovery

B. Blood Transfusion

- when much loss of blood occur

- Blood sample should be sent for grouping & cross matching

↓

- Blood + crystalloid should be given

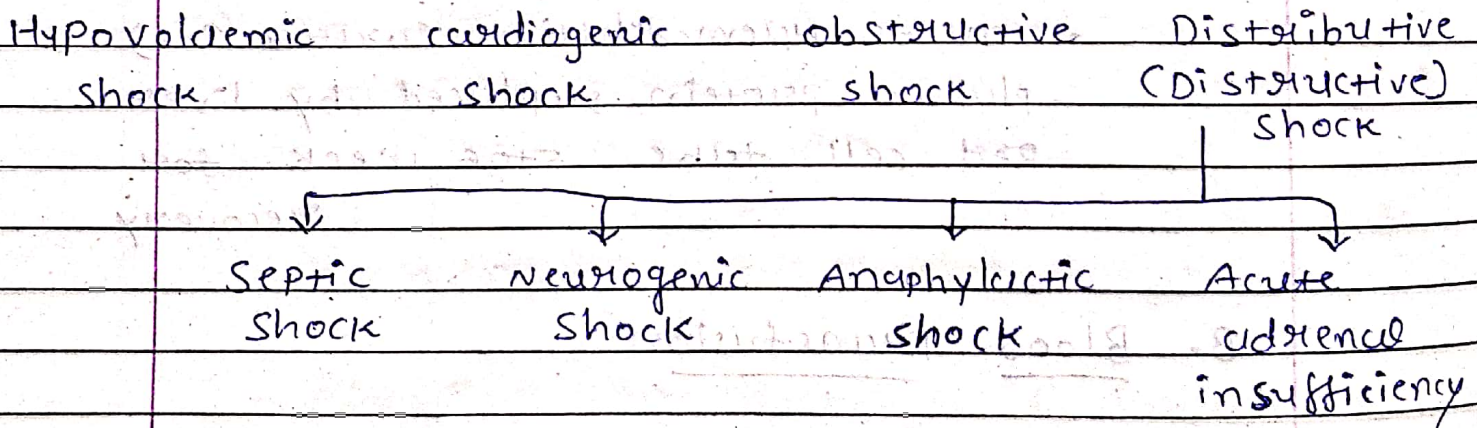
* Shock *

• Defⁿ :- It is clinical state of not enough blood flow to the tissue of body as result of change in circulatory system.

- It is clinical state in which the vital function of body are depressed due to acute & severe reduction in cardiac output & effective circulatory blood volume.

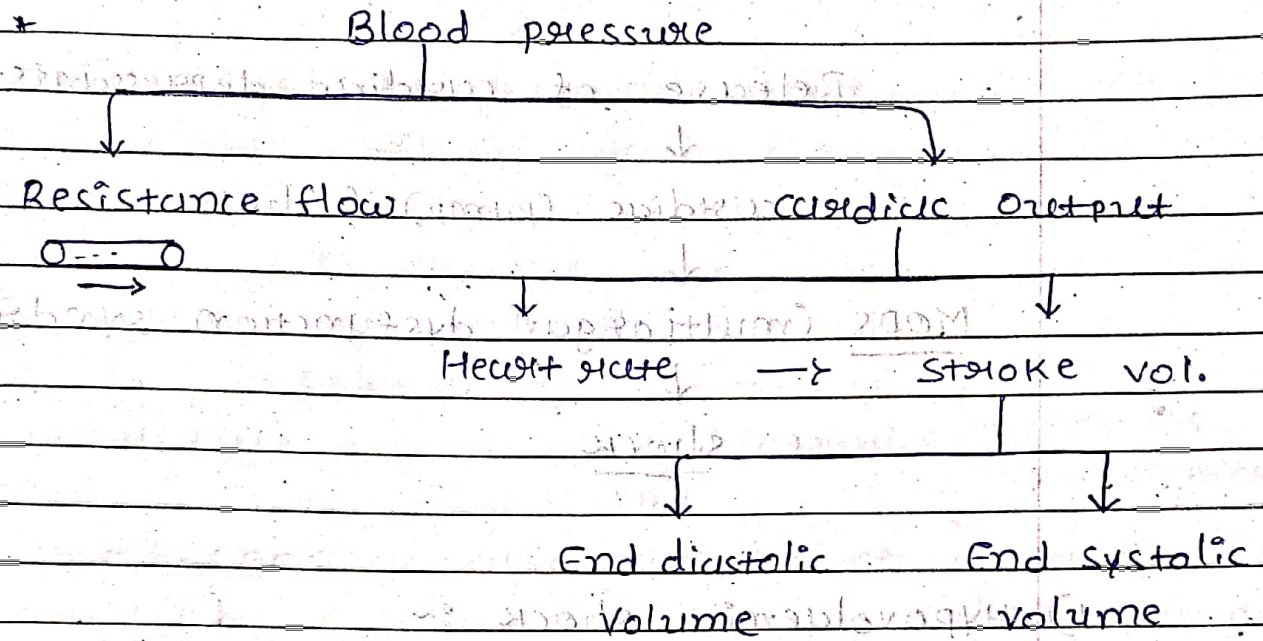
• classification :-

Shock



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stage of shock

1. initial compensated shock (non-progressive, reversible)
2. progressive decompensated shock
3. irreversible decompensated shock

infection, trauma, burns,
haemorrhage, hypovolaemia

↓ causes

Hypoxia & its effects

↓

SIRS (systemic inflammatory response syndrome)
is due to vasodilationⁿ

↓

metabolic acidosis

↓

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Release of circulatory depressants



cardiac (pump) failure



MODS (multiorgan dysfunction syndrome)



shock

1) Hypovolemic shock :-

- reduction in blood volume :- Hypovolemic shock

- loss of blood :- Hemorrhagic shock

- loss of plasma :- Burn shock

- loss of fluid :- Dehydration

- It occurs due to defect in ability of the heart pump the blood

- It occurs due to trauma

Trauma



end diastolic volume ↓



↓ stroke volume



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↓ use in cardiac output
↓
shock.

• compensatory mechanism :-

- Release of Epinephrine

ADH

catecholamine.

Acts as
vasoconstrictor

Blood pressure
↑ se.

• also called :- cold shock

T/t :- Restore blood volume

- tissue reperfusion & oxygenation to
normal as early as possible.

- Blood transfusion.

• C/F :- tachycardia

↓ se bp

↓ se cardiac output

cold & clammy extremities.

2) cardiogenic shock :-

• cause :- occurs due to improper function
of heart due to heart dz.

e.g. myocardial infarction.

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- improper function of heart results in ↓ end systolic volume

Blood pressure ↓

Resistance flow

cardiac output ↓

Heart state

↓ stroke volume

end diastolic vol.

↓ end systolic vol.

- C/F :- ↓ contractibility of heart
 - Lt. ventricular pressure ↑
 - tachycardia
 - ↓ BP
 - ↓ urine output

- T/t :- Improve cardiac muscle function
 - oxygenation
 - improve cardiac muscle contractibility
 - vaso dilators such as nitro glycerine may dilate the coronary artery.

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Intra-aortic balloon pump / ventricular assist devices may be used to augment cardiac output.

3) obstructive shock :-

- It can be due to cardiac tamponade / due to tension pneumothorax
- pathophysiology :-

physical obstruction in blood flow



↓ venous return



↓ cardiac output



↓ cellular & tissue perfusion



obstructive shock

A. cardiac Tamponade

- Features :- In obstructive shock, there is impedance to either inflow / outflow of blood into / out of the heart.

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- In cardiac Tamponade, the pericardium is filled w blood & hampers venous filling as well as outflow.
- The central venous pressure is high & the blood pressure is low.

- T/t :- To maintain preload w fluids / blood as indicated.
- Relief of obstruction, drain the pericardial cavity as early as possible.

B. Tension pneumothorax

- causes :- injury to lung due to trauma
- ventilator - induced barotrauma.
- feature :- profound cyanosis, distended neck veins.
- Tachypnea, dyspnea / respiratory
- Tachycardia, hypotension
- T/t :- pleural effusion.

4) Distributive shock :-

- In distributive shock, the after load is excessively reduced affecting circulation.

A. Septic shock

- This is result from massive sepsis / infect.ⁿ & the release of toxins (vasoactive sub.ⁿ)
- produced by gm +ve / -ve & protozoa
- It may occur severe septicemia, cholangitis, meningitis.

Pathophysiology

- due to infection.

↓
inflammatory process starts.

↓
vasodilation & ↑ capillary permeability.

↓
↓ venous return to the heart

↓
↓ cardiac output

↓
Inadequate cellular & tissue perfusion

↓
Septic shock

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In severe sepsis



persistent hypotension despite
adequate fluid resuscitation



Septic shock

- C/F :- peripheral vasodilatation &
ventricular dysfunction
 - generalised tissue hypoperfusion
may persist despite adequate fluid
resuscitation.
- T/t :- Removal of septic focus is an essential
step. e.g. resection of gangrenous
bowels, closure of perforat.ⁿ,
appendicectomy.
 - Antibiotic therapy
 - supportive care
 - Intravenous fluid :- crystalloids such
as isotonic saline/
Ringer's lactate.
 - Vasoconstrictors.

B. Anaphylactic shock :-

- It is a severe hypersensitivity reaction resulting in massive systemic vaso-dilation throughout the body.

- The precipitating factors include drugs, insect bite, blood transfusion, some food, vaccine, pollen.

- causes :-
 - Bronchospasm
 - laryngeal oedema
 - Respiratory distress & hypoxia

- C/F :-
 - hypotension
 - loss of consciousness
 - Anxiety

- pathophysiology :-
 - Due to entry of allergen / due to precipitating factors.

↓
 - hypersensitivity reaction of antigen Ab reaction starts.

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dilation of arterioles & capillaries
& ↑ se capillary permeability of
blood vessels

↓

microvascular leakage throughout body

↓

↓ se blood volume & circulatⁿ in body

↓

↓ se cellular & tissue perfusion

↓

Anaphylactic shock

• T/t :- i) primary

- O₂

- Adrenaline

- IV fluids - isotonic saline /
Ringer's lactate

- Leg end elevatⁿ of bed

ii) secondary

- cpm (chlorpheniramine maleate)

- Hydrocortisone 100mg IV

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Cardiogenic shock

- It is inability of nervous s. to control dilatation of blood vessels.
- It is an uncommon & transitory disorder caused by massive vaso-constrictor control in vascular smooth muscle & imp. of autonomic function.

• cause :- high spinal injury
- vasovagal shock

• C/F :- BP low
- heart rate rapid

• pathophysiology :-

- due to etiology like damage to spinal cord etc

↓
massive vasodilatation

↓
pooling of blood in venous vascular

↓
↓ venous return to the heart.

↓
↓ cardiac output

Inadequate cellular & tissue perfusion



neurogenic shock

- T/t :- Iv fluid
vasopressure drugs
tiedlenberg position

D. Acute Adrenal insufficiency

- cause :- infection, trauma / surgery
Adrenal injury

- s/f :- Headache
weakness

fatigue

lathargy

gt pain

(ABC)

- T/t :- care of airway, breathing & circⁿ
- Iv fluid
- hydrocortisone 100-300mg Iv
- Antibiotics as necessary

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* Management of shock

A. Pharmacological management :-

1. Iv fluids - Ringer's solⁿ & normal saline
2. Blood transfusion
3. Inotropic agents & symptomatic drugs - like dopamine, dobutamine & epinephrine
4. vasodilators - nitroglycerine
5. diuretics - furosemide
6. Antibiotics - ciprofloxacin, amoxicillin, clavulanic acid
7. Anti histamine - epinephrine used in anaphylactic shock
8. corticosteroid - dexamethasone, hydrocortisone
9. Sodium Bicarbonate - used to treat metabolic acidosis
10. Bronchodilators - like atropine, aminophylline etc.

B. non-pharmacological management :-

- maintain patency of the airway
- O₂ administration & breathing support as needed.
- modify Trendelenburg position.

- Assessment of vital signs & consciousness
- Assessment of intake & output of Pt
- oral, enteral / parenteral nutrition support
- provide active & passive exercise
- maintain skin integrity

* surgical management

- wound debridement & wound closure
- Angioplasty for obstructive shock
- valvular repair
- tracheostomy

* T/t :-

1st line management :- fluid

stop blood loss using
cotton at the place
of injury.

2nd line management :-

- Vasopressor
- Inotrops.

Blood Transfusion

* Indications :-

1. Haemorrhage

external

internal
2. Haemorrhagic shock
3. major surgeries

open heart surgery

gastrectomy
4. pre-operatively surgery for severe anaemia
5. excessive bleeding during operation
6. In anaemic pt. if Hb level below 6-7 gm
7. Erythroblastosis foetalis
8. Thallasemia
9. Few blood disorders like :-

i) Leukaemia

ii) Aplastic anaemia

iii) Hemolytic Dz

10. Few coagulative disorders like :-

- i) Thrombo-cytopaenia
- ii) Haemophilia.

11. Burn :- severe burn (class - 3 & 4)

12. During chemotherapy in malignant dz

* collection of blood for blood transfusion :-

NOTE - Before collecting blood from donor, one has to make sure that donor isn't suffering from any dz & may be transmitted through the blood.

• procedure :-

- A sphygmomanometer cuff is applied & inflated to a pressure of 80 mm Hg.



- 0.5 ml local anesthetic solution is injected subcutaneously in the ante-cubital fossa through a 15 gauge needle is introduced into median cubital vein



Blood from the donor is allowed to come out & run into sterile bag & already contains 75 ml of anticoagulant solution & prevent clotting.

- About 410 ml of blood is taken in single bag.

• 2 types of anticoagulant solutions are usually used to mix donor's blood :-

i) CPD solution :- citrate, citric acid, sodium dihydrogen phosphate.

ii) Adenosine

• Blood storage :-

- Bloods are stored in blood bank in special refrigerator at controlled temperature of 4°C.

• Types of Blood transfusion

i) Whole blood transfusion.

- cross blood
- Auto transfusion
- Exchange / replacement transfusion

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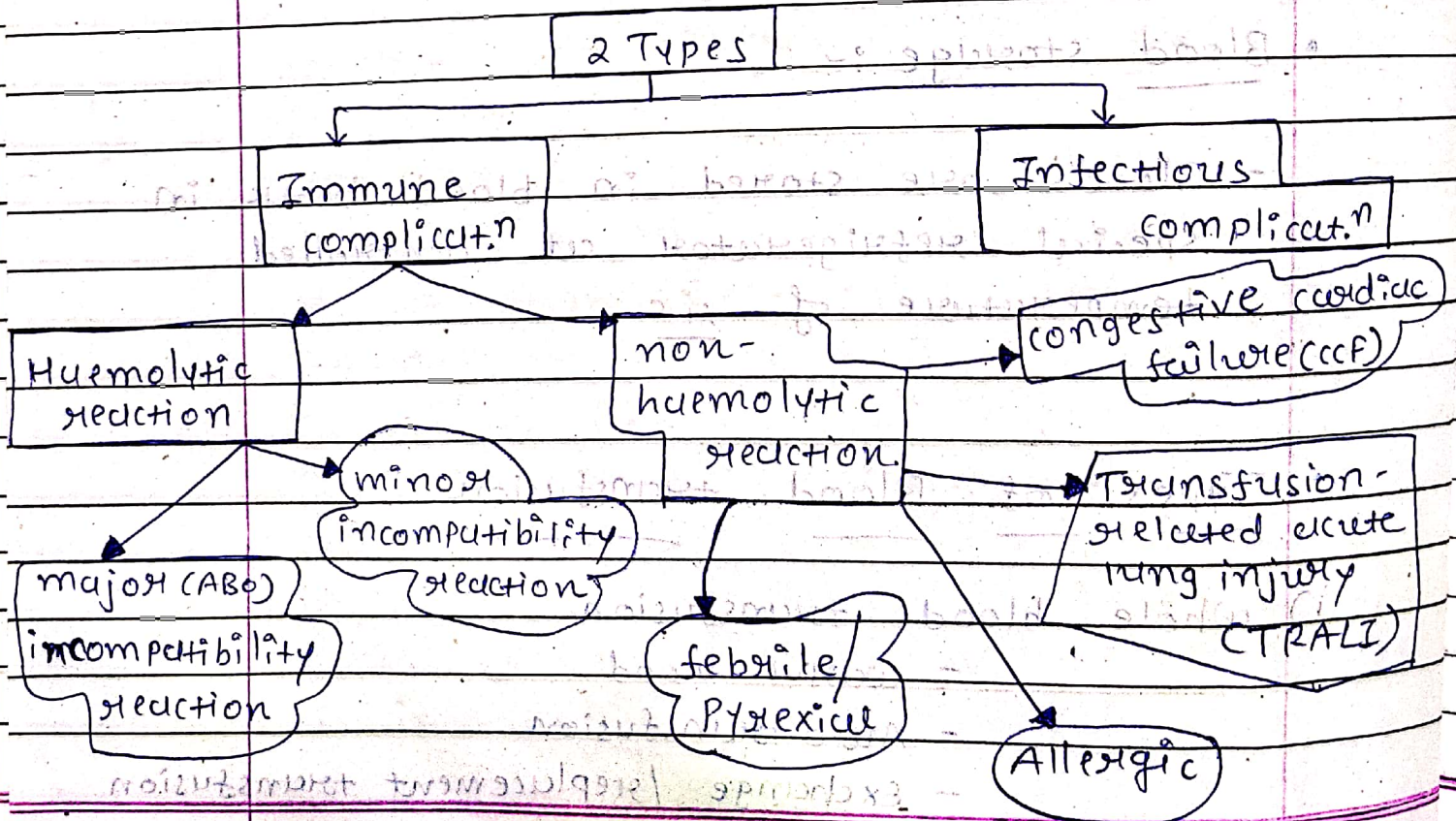
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- 2) packed red cells.
- prepared by centrifugatⁿ of whole blood & removing plasma.
 - used to correct chronic anaemia.
 - To avoid ccf in cardiac pt., stencil
 - Economical & safe.

3) fresh frozen plasma (FFP)

- After massive blood transfusion
- severe liver failure

* complications of blood transfusion :-



I) Immune complications

I) Haemolytic reactn :-

→ A. Major (ABO) Incompatibility reaction :-

• cause :- It occurs due to mis-matched blood transfusion.

- Expired blood

- hemolysed blood.

- mostly this occurs due to

Technical errors like

- sampling

- labelling

- dispatching

- This causes intravascular

hemolysis.

• C/f :- Haematuria

- urine output reduce

- fever

- Nausea, vomiting & headache

- pain in body

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- T/t :- Transfusion should be stopped immediately
 - Administration of IV fluid
 - Anti-histamine & hydrocortisone injection may prescribe.
 - hemodialysis may be use.
 - furosemide 20-40 mg IV.
 - Inj. mannitol 20% + 100 mL IV.

↓

should have for flush the kidney.

→ B. Minor Incompatibility Reactⁿ :-

• cause :- usually mild

↓

occur due to extra-vascular hemolysis

↓

2-21 day.

- occur due to Antibodies to minor Ag.

2) Non-Haemolytic Reactⁿ :-

A. febrile reactⁿ (pyrexial reactⁿ) :-

• cause :- 1) Improperly sterilised transfusion set.

2) presence of pyrogen in donor's blood.

3) Transfusion of infected blood

- T/t :- stop transfusion immediately.
 - Anti-pyretic Injⁿ
 - Anti-histamine given.

- clinical :- pyrexia (fever), chill, Rigor, Restlessness, headache, nausea, vomiting.

B. Allergic reactⁿ :-

- cause :- It occurs due to Allergy to plasma product in donor's blood.

- T/t :- stop blood transfusion immediately.
 - Anti-histamine drugs.
 - sometime calcium & hydrocortisone given.

C. congestive cardiac failure :-

- cause :- CCF can occur, if whole blood is

Transfused rapidly in pt.

∴ pt suffer chronic anaemia

- T/t :- slow blood transfusion
 - Inj. furosemide 20 mg - IV

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II) Infectious complications

• cause :- Dz can be transmitted by blood.

↓
from one pt. to another pt.

↓
Through blood transfusion

• Dz :- serum hepatitis

- AIDS

- Bacterial infections :-

It is due to faulty storage technique

III) Reactions caused by massive transfusion :-

1. Acid base imbalance
2. hyperkalemia
3. citrate toxicity & hypocalcaemia & hypomagnesaemia
4. hypothermia
5. failure of coagulation
↳ clotting factors deficiency

⇒ Excessive Blood transfusion causes ↑

- if short notes of complicatⁿ of BT there is elaborate write in this topic, describe below,
↓

[II]

i) serum hepatitis :-

- most common D₂ c is transmitted during BT.
- Thus, Blood donor should be carefully tested before BT.
- But about 1% of cases remain undetected.
- symptoms of hepatitis develop usually after 3 month.

ii) AIDS :- Now, it is clear that AIDS can be transmitted by it.

iii) Bacterial infection :-

- not commonly occur
- Bacterial Infection can acquire when blood (donor blood) left in warm room.

↓

warm room promote Bact. growth

[III]

i) Acid-base imbalance :-

- Normal blood pH = $7.4 \pm$
- Excessive transfusion $\Rightarrow$

metabolic
Alkalosis

ii) Hyperkalemia :-

- generally not seen

- Excessive transfusion of low Temp. storage blood

↓

- causes Escaption of K from RBC

↓

Hyperkalemia

iii) Hypothermia :-

- Refrigerative blood directly transfer into paralytic pt.

↓ causes

drooping of Temp.

iv) Citrate toxicity :-

- citrate is anticoagulant used in blood products.

- Rapid administration of large quantity of stored blood may cause hypocalcemia & hypomagnesemia when citrate binds Ca & Mg.

↓

- myocardial depression / coagulopathy

v) Failure of coagulation :-

- occurs due to dilution of platelet & various coagulative factors.