الجراحه
المحاظر13
NEOPLASIA

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Nomenclature

Neoplasia
  .new growth
Tumor
  .swelling
Oncology
  .study of neoplasms
Cancer
  .common term for malignant neoplasm
Neoplasm

abnormal mass of tissue, growth of which exceeds & is uncoordinated with that of normal tissues & persists in the same excessive manner after cessation of stimuli which evoked the change
neoplasms have 2 components
- parenchyma (neoplastic cells)
- stroma (supportive connective tissue)
Types of Neoplasms Based on Behavior

Nomenclature of tumors & their biologic behavior are based on parenchymal component

- benign
- malignant
Benign Neoplasms

- have innocent microscopic & gross features
- remain localized
- amenable to local surgical removal
- patient generally survives

In mesenchymal neoplasms, *oma* is attached to cell of origin (*fibroma, chondroma, osteoma, leiomyoma, rhabdomyoma*)
Leiomyoma
In epithelial neoplasms nomenclature depends on
- cell of origin, (adenoma)
- microscopic pattern &/or macroscopic architecture (adenoma, papilloma, cystadenoma, papillary cystadenoma)
Colon, adenoma
Colon, adenoma
Ovary, *cystadenoma*
Malignant Neoplasms

can invade & destroy adjacent tissues  
can spread to distant sites *(metastasize)*  
cause death of patient

malignant neoplasms of mesenchyme  
are called *sarcomas*

- fibrosarcoma *(connective tissue)*
- liposarcoma *(adipose tissue)*
- leiomyosarcoma *(smooth muscle)*
- rhabdomyosarcoma *(skeletal muscle)*
malignant neoplasms of epithelium are called **carcinomas**

adenocarcinoma (*glandular pattern*) - squamous cell carcinoma (*squamous-differentiation*)

malignant tumors with undifferentiated cells of unknown tissue origin are called **undifferentiated malignant tumors** (*anaplastic*)
Polyp

benign or malignant neoplasm with macroscopically visible projection above a mucosal surface projecting into gastric or colonic lumen
Colon, polyp (adenoma)
Colon, polyp (adenoma)
Mixed Neoplasm
divergent differentiation of a single neoplastic clone along two lineages

Salivary gland, mixed tumor
Teratoma

neoplasm arising from more than one germ layer (totipotent cell)

Ovary, teratoma
الجراحة
محاضرة 1
Cell Injury & Cell Death

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If the adaptation capacity is exceeded or if the cell cannot undergo adaptation, the cell injury will occur, and up to certain limits, these changes are reversible, and then the cell can return to normal if the causative agent is removed. However, if the stress is severe or persistent, this leads to irreversible cell injury.
General Principles related to cell injury:

- Cellular response to injurious stimuli depend on type of injury, its duration & its severity.
- Consequences of an injurious depend on type, status & genetic make up of the cell.
- Four intracellular systems are vulnerable to injury include: mitochondria (ATP), protein synthesis, integrity of genetic apparatus & cell membrane.
Stages of the cellular response to stress and injurious stimuli
Causes of cell injury:

1- Hypoxia
   - Ischemia
   - Cardiac & respiratory diseases
   - Low O2 carrying capacity (anemia)

2- Physical injury (temp, radiation, electrical shock, trauma, change in atmospheric pressure)

3- Chemical agents & drugs

4- Microbial agents (virus, bacterial, .....etc)

5- Immunologic reactions (hypersensitivity reaction, anaphylactic shock & autoimmune diseases)

6- Genetic derangement (Down’s syndrome & sickle cell anemia)

7- Nutritional imbalance (vitamin deficiency, protein deficiency, increase fat, alcoholism)

8- Aging
Reversible cell injury:

It is a common type of cell injury usually due to ischemia (hypoxia) in this type of injury the cellular changes will regress and disappear when the injurious agent is removed; the cell will return to normal both morphologically and functionally.

Morphology of reversible cell injury:
The two main morphologic correlates of reversible cell injury are cellular swelling and fatty change.
1- **Cellular swelling**: It is the first manifestation seen in all forms of cell injury & it happens when the cell fail to maintain water & ion homeostasis.

- it is difficult to be shown by light microscope.
- it is more apparent when the whole organ is involved in that case the organ become pale & increase in weight.

**Microscopically**: At the beginning the cytoplasm show many vacuoles & this is called (vacuolar changes), if the vacuoles combine with each other the cell appear as balloon & in that case the condition is called (hydropic changes)
2-Fatty changes (steatosis):

- Is abnormal accumulation of fat of triglyceride type within parenchymal cells rather than adipocytes.
- It is an example of reversible cell injury,
- Seen often in the liver in which fat centrally metabolized & to less extent in the heart & kidney
Fatty changes (steatosis) in liver
Irreversible cell injury:

Irreversible cell injury: occurs when the injury persist or when it is severe from the start. Here the cell reaches the point of no return and progression to cell death is inevitable.

It is of 2 types:

1) Necrosis 2) Apoptosis

Necrosis: it is a sequence of morphologic changes that follow cell death in the living tissue or organs due to action of degradative enzymes or protein denaturation on irreversibly injured cells.
Necrosis

- It is a passive process
- Associated with inflammation
- Randomly occurs
- Involve a group of cells
- Always pathologic
- Causes: ischemia, chemical injury or infarction (cell death due to cut of blood supply), nutritional….etc

Mechanisms of necrosis:

- Denaturation of proteins
- Enzymatic digestion of the cell: either by its own enzymes (autolysis) or the cell digested by proteolytic enzymes secreted from invading inflammatory cells, this is called (heterolysis)
Types (Patterns) of necrosis

1. coagulation necrosis
2. liquifactive necrosis
3. fat necrosis
4. caseous necrosis
5. fibrinoid necrosis
6. gangrene
Coagulative necrosis:

- The most common type of necrosis.
- Occurs anywhere in the body except C.N.S
- Usually due to hypoxia

Grossly:

Whitish-gray or red-hemorrhagic firm wedge shape area of infarction, (the occluded vessel at the apex and the periphery of the organ forming the base).
Coagulative necrosis (infarction) - Spleen
infarction

- It is an **ischemic necrosis** caused by occlusion of either the arterial supply or the venous drainage.
- Coagulative necrosis is characteristic of infarcts (areas of ischemic necrosis) in all of the solid organs except the brain.
Coagulative necrosis (infarction) - kidney
Myocardial infarction
Liquefactive necrosis:

- Seen in
  - Ischemic necrosis (*infarction*) of CNS
  - In focal bacterial, occasionally fungal infections or Abscess formation in pyogenic infections in all tissues.

- Enzyme digestion & autolysis > protein denaturation so the area become soft → cystic.

- Grossly: softening & liquefaction of the necrotic tissue (Soft liquid like)
Liquefactive necrosis (Lung Abscess)
Liquefactive necrosis - Brain infarction
Caseous necrosis:

- It is a special form of necrosis usually seen in tuberculosis.

Grossly: it is soft & yellow white appears as cheese-like.
Caseous necrosis (TB), yellow white appears as cheese-like
Fat necrosis

The areas of white chalky deposits represent foci of fat necrosis with calcium soap formation (fat saponification)
Fibrinoid necrosis:

It is characterized by deposition of fibrin like material in the tissue e.g. Fibrinoid necrosis of blood vessels in *malignant hypertension & vasculitis*. 
Gangrenous necrosis (Gangrene)

- Gangrenous necrosis is not a specific pattern of cell death, but the term is commonly used in clinical practice.
- It is usually applied to a limb, generally the lower leg, that has lost its blood supply and has undergone necrosis (typically coagulative necrosis) involving multiple tissue planes.
- Gangrene may be classified as dry or wet.
Dry gangrene

- In dry gangrene, the part becomes dry and shrinks and its color changes to dark brown or black.
- The **spread** of dry gangrene is **slow**.
- **The lesion remains localized.**
- The irritation caused by the dead tissue produces a line of inflammatory reaction (i.e., **line of demarcation**) between the dead tissue of the gangrenous area and the healthy tissue.
- Dry gangrene usually results from **interference** with **arterial blood supply** to a part without interference with venous return and is a form of **coagulation necrosis**.
Wet gangrene

• In wet gangrene, the area is cold, swollen, and pulseless. The skin is moist, black. Blebs form on the surface, liquefaction occurs, and a foul odor is caused by bacterial action.

• There is **no line of demarcation** between the normal and diseased tissues, and the spread of tissue damage is **rapid**.

• The lesion may extend **proximally**.

• Wet gangrene primarily results from **interference with venous return** from the part.

• **Bacterial invasion** plays an important role in the development of wet gangrene.

• Dry gangrene is confined almost exclusively to the extremities, but wet gangrene may affect the internal organs or the extremities.

• If bacteria invade the necrotic tissue, dry gangrene may be converted to wet gangrene.
Gangrenous necrosis

Dry gangrene - Ischemia

Wet gangrene - D.M
Apoptosis: (Programmed cell death)
It is a death of single cell as a result of the activation of a genetically programmed (suicide) pathway through which the cell removed with minimal damage to the tissue containing them.

- Usually involve single cell.
- **physiological or pathological**
- Apoptosis is an active process (need energy or ATP)
- not associated with inflammation.
Differences between apoptosis & necrosis

<table>
<thead>
<tr>
<th>Apoptosis</th>
<th>Necrosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Active process</td>
<td>• Passive process</td>
</tr>
<tr>
<td>• Occur in single cells</td>
<td>• Affects mass of cells</td>
</tr>
<tr>
<td>• Physiological &amp; pathological</td>
<td>• Always pathological</td>
</tr>
<tr>
<td>• No inflammatory reaction</td>
<td>• Stimulate inflammation</td>
</tr>
<tr>
<td>• Programmed process</td>
<td>• Random process</td>
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</table>
Thank You
الجراحه
محاضرة 2
INFLAMMATION

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INFLAMMATION

- It is a protective reaction.
- caused by many internal & external stimuli.
- harmful agents are destroyed, diluted or walled-off
- without inflammation and mechanism of healing organism could not survive
MECHANISMS

• local - in cases of mild injury
• Systemic by :
  1. alteration
• 2. exsudation - inflammatory exudate
  • liquid (exudate)
  • cellular (infiltrate)
• 3. proliferation (formation of granulation and fibrous tissue)
CLASSIFICATION

• length:
  • acute × chronic (+ subacute, hyperacute)

• according to predominant component
  • 1. alterative (predominance of necrosis - diphtheria)
  • 2. exsudative (pleuritis)
  • 3. proliferative (cholecystitis - thickening of the wall by fibrous tissue)
CLASSIFICATION

• according to histological features
  • nonspecific (not possible to trace the etiology) - vast majority
  • specific (e.g. TB)

• according to causative agent
  • aseptic (sterile) - chemical substances, radiation - inflammation has a reparative character
  • septic (caused by living organisms) - inflammation has a protective character
ACUTE INFLAMMATION

• important role in inflammation has microcirculation!
• supply of white blood cells, fibrin, etc.
LOCAL SYMPTOMATOLOGY

• classical 5 symptoms (Celsus 1st c. B.C., Virchow 19th c. A.D.)
  • 1. calor - heat
  • 2. rubor - redness
  • 3. tumor - swelling
  • 4. dolor - pain
  • 5. functio laesa - loss (or impairment) of function
SYSTEMIC SYMPTOMATOLOGY

• fever (irritation of centre of thermoregulation)
  • IL-1 IL-6 – high erythrocyte sedimentation rate ESR
• leucocytosis - increased number of WBC
  • bacteria – neutrophils
  • parasites – eosinophils
  • viruses - lymphocytosis
• leucopenia - decreased
  • viral infections & typhoid
• immunologic reactions - increased level of some substances (C-reactive protein)
VASCULAR CHANGES

• vasodilation
• protein poor transudate (edema)
• protein rich exudate
• Thrombosis
PHAGOCYTOSIS

- adhesion and invagination into cytoplasm
- engulfment
- lysosomes - destruction
- in highly virulent microorganisms can kill leucocyte.
- in highly resistant microorganisms can persist & reactivation.
OUTCOMES OF ACUTE INFLAMMATION

1. resolution - restoration to normal in limited injury

2. healing by scar
   - tissue destruction
   - fibrinous inflammation
   - purulent infl. → abscess formation (pus, pyogenic membrane, resorption & it takes weeks to months)

3. progression into chronic inflammation
CHRONIC INFLAMMATION

- chronic inflammatory cells ("round cell" infiltrate)
  - lymphocytes
  - plasma cells
  - Monocytes → macrophages activation
- lymphocytes → plasma cells (parts of immune system)
- plasma cells - production of Ig
MORPHOLOGIC PATTERNS OF INFLAMMATION

- 1. alterative
- 2. exsudative
  - 2a. serous
  - 2b. fibrinous
  - 2c. suppurative
  - 2d. pseudomembranous
  - 2e. necrotizing, gangrenous
- 3. proliferative
  Usually secondary (cholecystitis)
MORPHOLOGIC PATTERNS OF INFLAMMATION

• 2a. serous - excessive accumulation of fluid, - skin blister, serous membranes - initial phases of inflamm.

• 2b. fibrinous - higher vascular permeability - exsudation of fibrinogen → fibrin - e.g. pericarditis

• fibrinolysis → resolution; organization → fibrosis → scar
• 2c. suppurative (purulent) - accumulation of neutrophillic leucocytes - formation of pus (pyogenic bacteria)
complications of suppurative inflamm.:  
• bacteremia (no clinical symptoms!; danger of formation of secondary foci of inflamm. (endocarditis, meningitis)  
• sepsis (= massive bacteremia) - septic fever, activation of spleen, septic shock  
• thrombophlebitis - secondary inflammation of wall of the vein with subsequent thrombosis - embolization - pyemia - hematogenous abscesses (infected infarctions)  
• lymphangiitis, lymphadenitis
CHRONIC INFLAMMATION

• causes:
  • persisting infection or prolonged exposure to irritants (intracell. surviving of agents - TBC)
  • repeated acute inflammations (otitis, rhinitis)
  • primary chronic inflammation - low virulence, sterile inflammations (silicosis)
  • autoimmune reactions (rheumatoid arthritis, glomerulonephritis)
GRANULOMATOUS INFLAMMATION

• distinctive chronic inflammation type
• cell mediated immune reaction (delayed)
• aggregates of activated macrophages $\rightarrow$ epithelioid cell $\rightarrow$ multinucleated giant cells (of Langhans type x of foreign body type)
• NO agent elimination but walling off
• intracellular agents (TBC)
GRANULOMATOUS INFLAMMATION

1. Bacteria
   - TBC tuberculosis
   - leprosy
   - syphilis (3rd stage)
2. Parasites + Fungi
3. Inorganic metals or dust
   - silicosis
   - berylliosis
4. Foreign body
   - suture (Schloffer „tumor“), breast prosthesis
5. Unknown - sarcoidosis
الجراحه
محافظة 3
Wounds, Tissue repair and Scars

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Definition

Wound: It is a break in the continuity of the skin, mucous membrane or any other tissue caused by physical, chemical or biological insult.
Wounds

Tissue Repair = Wound healing

Scar
Surgeons induce tissue injuries as part of their access,

It is a common casualty complaint.

Is it important?
Wound healing:

an orchestrated biological process initiated by tissue injury and results in restoration of tissue integrity. The end result of repair process is fibrosis and scar formation.

This process is defined theoretically into three phases.
Phases of wound healing

Inflammatory phase (Early): 0 – 3 days.

Proliferative phase (Intermediate): 3\textsuperscript{rd} day – 3\textsuperscript{rd} week.

Remodelling Phase (Late): up to 1 year
Inflammatory phase (Early):

**Hemostasis:**
- Vasoconstriction,
- Platelets aggregation,
- Activation of coagulation system.

**Inflammation:**
- Neutrophils act to sterilize the wound.
- Monocyte will continue phagocytosis and secrete several growth factors.
Proliferative phase (Intermediate):

Fibroplasia:

➢ Deposition of collagen fibers to form the extracellular matrix.

Angiogenesis:

➢ New capillary networks which comes from endothelial cell division and migration.

"Granulation tissues" consist of new blood vessels, macrophages, fibroblasts embedded in a loose matrix of collagen.
Contraction:
➢ wound is pulled circumferentially toward the center by specialized Myofibroblast to decrease the size of the wound.

Epithelialisation:
➢ epidermis migrate over the wound defect thus forming a barrier to further contamination.
Remodelling Phase (Late):

The collagen fibers align with the skin tension lines to increase the tensile strength of the skin.

However, at best it only reach approximately 80% of the normal skin.
Factors affect wound healing:

Local factors:

Site of the wound, structures involved and mechanism of wounding e.g. incision, crush and avulsion.

Ischemia.

Infection.

Foreign bodies and necrotic tissues.

Chronic venous insufficiency and tissue oedema.

Ionizing radiation.
Systemic Factors

- Systemic diseases:

  Malnutrition: Vit C deficiency results in inadequate collagen production.

  Drugs: steroid and chemotherapeutic agents for cancer therapy.
Types of wound healing:

**Primary Healing:** wound edges opposed, normal healing and minimal scar.

**Secondary Healing:** The wound left open to heal by granulation, contraction and epithelialisation. There will be increased inflammation and proliferation with poor scar.

**Tertiary Healing:** wounds initially left open and the edges opposed later when healing conditions favourable.
Classification of wounds

- Acute wounds
- Some specific wounds
- Chronic wounds
Acute wounds

Tidy:
Sharp instrument e.g. surgical incisions or wound by glass or knife,
it contains no devitalized tissue
Little( contamination and tissue loss).
These wounds are usually single & clear cut.
closed immediately with expected good healing and minimal scar.
• Untidy:
• Blunt injuries e.g. crushing, tearing, avulsion.
• It has a dead tissue
• Significant contamination and often has tissue loss.
• They are often multiple and irregular.
• If closed immediately, healing is unlikely and associated with wound complications.
Granulation Tissue
Local wound care

Wound excision:

The process of removal of the dead tissue from the wound.
Primary closure: the edges of the wound are approximated. The wound will heal by primary intention with minimal scaring e.g. Tidy wounds

Healing by secondary intention: In untidy wounds, it require serial wound excision and cannot be closed. These wounds are left open for care and dressing until healing achieved by granulation with significant scaring.

Delayed Primary Closure: Sometimes Untidy wounds can be converted to tidy wounds which can be closed directly.

Tissue Transfer: when there is tissue loss, appropriate tissue need to be transferred into the defective area e.g. Skin graft.
Some specific wounds

Contusion, Bruises:
closed wounds
blunt trauma
bleeding into the tissues with visible discoloration.
 hematoma which can resolve spontaneously or it may need drainage or repeated aspiration.
Bites:

Animal/human bites.

Aerobic and anaerobic organism prophylaxis required as bites wound typically have high virulent bacterial counts and proper wound excision.
Puncture wounds:

open wounds
penetrating injuries e.g. standing on a nail or sharp objects, or needle stick injuries.
foreign bodies and microorganism are likely to be carried deeply into the tissues.
The danger is, it might give rise to deep abscess.
Degloving:

skin and the S.C. fat are stripped by avulsion from its underlying fascia,

It could be open injury e.g. ring avulsion or closed injury e.g. roll-over injury typically caused by motor vehicle over a limb.
Scars:

Is the inevitable consequences of wound repair. "Remodelling": immature collagen fibers replaced by acellular, avascular tissue composed of mature collagen fibers. red, raised, firm area to pale, flat, soft and symptomless mature scar.
Complication of Scar

Hypertrophied scar:
more cellular and vascular with increased collagen production than mature scar.
Clinically it is red, raised, itchy and tender but confined to the wound site.
It tends to occur in wounds whose healing has delayed because of infection or dehiscence.
spontaneous resolution.

• Keloid scar:
• extensive growth of the scar beyond the wound site.
• It is biologically identical to hypertrophied scar.
• They often occur in wounds which healed without complications.
• It rarely subside with time and require active treatment.
الجراحة
محاظرة 4
General Surgery

Surgical infections I

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Types of wound

Clean (nearly 2-3%) like: Incised wounds
Types of wound

Clean contaminated (20-25%) like:

Lacerated wounds
Types of wound

Infected (30% & more) like: Bullet wounds

Inlet

out let
hypertrophic scar

Keloid scar
Surgical infections

It is an infection which follow the operation (wound infection or post. op. abscess) which is unlikely to respond to conservative treatment & must be excised or drained eg: abscess, empyema, gas gangrene.

Factors influencing the body to infection:
2. Local factors: hematoma, crush injury, bone injury, foreign body.
Hospital infection (Nasocomial)

Infections results from transmission of pathologic microorganism (M.O.) to previously uninfected wound such M.O. are resistant bec. the patient receives prolonged antibiotic treatment. The M.O. found in the ward, operation theater, surgeon, assistant, or cross infection from other patient, or autoinfection from the patient himself in which the wound is contaminated by his cough droplet. Most common M.O. is staph. which is found in anterior nares & perineum.
Hospital infection (Nasoocomial)

The most common M.O. are:

1- Staph. : G+ cocci facultative anerobic found in nose & throat, hospital tools, clothes. It produces coagulase, staphylokinase, hyalourinidase, enterotoxin. Lesions: boil, carbuncle, o.m., abscess, wound infection.

2- Strep. : G+ cocci aerobic & facultative anerobic, 3 types (α- hemolytic viridance, β- hemolytic pyogens, γ- hemolytic fecalis) It produces streptokinase, hyalourinidase, streptolysin O & S & exotoxin, found in mouth, pharynx. Lesions: Erysipelas, scarlet fever, RF, tonsillitis, impetigo, glomerulonephritis, Nec. Faci.
3- G-ve bacilli: rods facultative anaerobic produce endotoxin found in the large bowel. Like E. coli, proteus, pseudomonas, klebsiella, bacteriods.


1- Tetanus

occur due to soil or dirty wounds bullet shell piece clothes piece. It has powerful exotoxin cause tissue & CNS damage. Signs: ↑ temperature, stiff jaw, dysphagia, tonic muscle spasm in the face resus sardonicus, difficult breathing, reflex contraction, death from asphyxia.
Treatment of Tetanus

1- prophylactic management:
   a- active immunization: by injection of absorbed toxoid which is safe. (used for prophylaxis)
   b- passive immunization: every patient with crush injury or wound who has no active immunization should take 1500 IU of ATS inj. I.M. or human antitetanus globulin 250 IU I.M.

2- symptomatic treatment of spasm by: anticonvulsant muscle relaxant, artificial respiration, antibiotic penicillin
2- Gas gangrene

Caused by cl. Welchii some times cl. Septicum usually affects wound of the thigh & buttock because these areas are liable for fecal contamination & lead to amputation. The etiology either by trauma, war injury, industrial. Or DM & atherosclerosis or post op. laparotomy or lower limb amputation or septic abortion.

The infection starts as a simple contamination in which the M.O. digest the tissue & seropurulant discharge but no gas or toxin. Treated by AB. If not the condition change to cl. Cellulitis gas crepitation with infection involve subcutaneous tissue but not the muscle which is intact if not treated cl. Myonecrosis or myositis (actual gas gangrene).
2- Gas gangrene

Clinical picture: pain, edema, swelling, crepitus by palpation, toxemia, ↑↑ temperature, ↑↑ pulse rate.

Management:
1- Prevention: a- excision of the whole dead tissue until fresh bleeding appear.
   b- prophylactic AB.
   c- antigas gangrene serum 22500 IU

2- Treatment: a- immediate pos. op. Blood transfusion with adequate excision of dead tissue & muscle & even some times leg amputation if not respond to treatment.
   b- broad spectrum AB. (penicillin, flagyl, garamycin).
   c- AGS       d- hyperbaric oxygen
2- Gas gangrene
2- Gas gangrene
Apportunistic infection

A- reduced host defense (immune decreased) seen in:
1- immune suppressive therapy, 2- cytotoxic & steroid Rx.
3- radiotherapy for neoplasm, 4- severe burn,
5- starvation, 6- long term use of AB.,
7- AIDS 8- very old & very young (premature baby).

B- maintained invasive therapeutic procedure:
1- I.V. cannulation
2- intravesical catheterization
3- trachio-stomy & pulmonary ventilation.
Apportunistic infection

The commonest M.O. are G-ve (E. coli, pseudomonas, klebsiella, proteus. They originate either from the patient own GIT, or by cross infection from other patient in the hospital & spread by hands of the attendants.

G+ve M.O. staph. Epidermidis derived from skin lead to local infection or bacterimia, often associated with IV or CV line prosthesis to the heart or joint, upper renal tract catheterization. Following splenectomy lead to strep. Pneumonia

Viruses ( Herpes, CMV, V-zostar)
Fungal ( Candida, Aspargilosis, mucourmycosis)
Protozoa ( cryptosporidial diarrhea, pneumocystis carinii pneumonia)
General Surgery

Surgical infections II

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Wound infection
Classification of sources of infection

• Primary or endogenous source such as that following perforated peptic ulcer

• Secondary or exogenous source: acquired from hospital
Spread of infection

Spread of infection:
1- Locally: Cellulitis, Abscess
2- Regionally: lymphangitis
3- Systemically:– blood stream – bacterimia, septicemia
- body cavities – peritonitis, meningitis
- lymphatic system
**Cellulitis**

**Cellulitis**: is the non-suppurative infection of the subcutaneous (s.c.) tissue spreading along the s.c. fascial planes & cross the intercellular spaces. caused by β-hemolytic strep. which has entered the tissue through an accidental wound, scratch or following surgical incision.

**C\P**: wide spread swelling, redness, pain without definite localization itching & stiffness of the site. central necrosis & suppuration may occur later on.

**TTT**: bed rest, elevation of the part, treat the underlining condition eg: DM, proper AB, if not cured under general & Surgical care
Abscess

Abscess : a collection of pus caused by pyogenic organisms, predominantly staphylococcus aureus cause tissue necrosis and suppuration. Pus is composed of dead and dying white blood cells that release damaging cytokines, oxygen free radicals. Abscess is surrounded by an acute inflammatory response and a pyogenic membrane composed of a fibrinous exudate and oedema and the cells of acute inflammation.
C\P: symptoms: depend on site, size, tension of abscess, & virulence lead to generalized illness, throbbing pain & swelling.
   
sings: (general) ↑ temp., rigor may occur.
   (local) 5 signs of inflammation heat, redness, swelling, tenderness, loss of function.

TTT: drainage of the pus & culture \sensitivity AB
Abscesses
Bacterimia

Bacterimia: presence of bacteria in the blood as proved by blood culture without indication of toxemia or clinical features.

   causes: follow dental procedure, o.m., pyelonephritis, major traumatic wound.

Complications: multiple metastatic abscess in the distal organs which need treatment.
Bacteremia is the presence of viable bacteria in the bloodstream.
Septicemia

Septicemia: bacterial proliferation in the blood & their toxins are present in the blood.

Routes by which the bacteria reach the blood:
1. Direct through the blood vessels.
2. Release of infected thrombi following thrombosis of blood vessel in the area of inflammation.
3. Discharge of infected lymph node in to the blood following lymphangitis
Pyemia

Pyemia: circulation of infected emboli composed of mass of M.O. or infected clot lead to multiple focal abscess in many parts of the body.
C\P: rigor & intermittent fever with abscess formation such as in the brain, bone, liver.
Diagnosis of infection

1- C\P
2- Laboratory investigation: CBC – leucocytosis, blood culture, biopsy in case of granulomatus lesion eg: TB.
3- Imaging: - conventional X-ray (fluid level)
   - radioisotopes study (Technetium, gallium scan)
   - Ultrasound (U/S): for liver, spleen, kidney, biliary abscess.
   - C.T scan for brain abscess.
Prevention of infections

1- Antibiotic prophylaxis: when instrumentation or surgery is performed upon a site with normal flora or when infection already exist eg: cystoscopy. AB prophylaxis may be given IV as bolus dose after induction of anesthesia or IM. AB must be given one hour before the surgery eg.: patient with heart valve disease or prosthetic graft of cardiovascular system or with a history of RF & the patient undergo dental surgery or for urethral catheterization give Amoxicillin 3gm orally or Erythromycin 1.5 gm or Clindamycin 600 mg.
Prevention of infections

2- control of hospital infection:
   A- preventing of infection at operation (op. theater cross infection)

a- theater design & architecture:
   • the theater best separated from the ward
   • sterilization center also away from the theater
   • Walls smooth easy to wash & use UV light
   • good ventilation to prevent air borne infection
   • chemical antiseptic are used often to clean the op. room
Prevention of infections

b- the surgeon & attendances:

- no body with overt infection should be allowed into the theater
- using sterile gowns, masks, boots, caps.
- 3-4 min. washing hands by liquid contain chlorhexidine or iodine
- spectators should wear caps, mask, gown, little movement as possible, & number as small as possible.
- patient preop. Shower, & shaving op. area, wear bed clothes.
- all surgical equipments disinfected
Prevention of infections

B- preventing & control of inf. In the surgical ward:
1- isolation policy.
2- good dressing technique
3- disposable articles
4- urine drainage Foleys catheter should be avoided
5- general cleaning & disinfect the ward:
- Cotton blanket changed after 2 weeks or when p. discharged
- sheets changed every 2 days or when p. discharged
- bed covers changed weekly or when p. discharged
- walls, floor, furniture, cleaned by chemical
Sterilization & disinfection

Sterilization: a process of killing all the M.O. & spores including the bacteria, viruses, fungi, parasites.

Disinfection: a process of killing only the vegetative forms of M.O. but leaves the spores intact.

It is achieved either by bactericidal or bacteriostatic agents.

Antisepsis: a process of prevention of contamination with M.O. to the normal tissue.
Methods of sterilization

1- physical:
- heat – dry (hot air, infrared radiation)
  -- moist (pasteurization, boiling.)
  -- autoclave
- light
- ionizing radiation
- filtration

2- chemical:
- inorganic (halogens eg. I, Cl...)
- organic (alcohol, aldehydes, phenol)
- gaseous agents (formaldehydes, ethylene oxide)
Practical sterilization

dressing & gloves by autoclave
surgical instrument by autoclave or chemicals
cystoscopies by – pasteurization 75cº
  --chlorhexidine 0.5% in 70% ethanol
  --ethylene oxide gas (best one)
syringes – glasses by hot air oven
  --disposable by ionizing radiation
rooms by hot formalin vapors
skin by povidone iodine (best) or chlorhexidine
الجراحة
محافظة 6
INFECTIOUS DISEASES

DR. SIDDEEQ BAKR
M.B;CH.B. C.A.B.S
CONS. URO.

SURGERY
LAB6
Infection: is defined as the invasion of living tissue by m.o. or its products (commensals or pathogenic) followed by local reaction which may be associated with general reaction.

Commensals microorganisms: are those can infect human body and be harmless to the body (some are present normally within the human body). When commensal microorganisms cause diseases at that time they called opportunistic infections.

The microorganisms that cause diseases are called pathogens.

The outcome of infection depends on the complex balance between the aggressive mechanisms of the m.o. & the defense mechanisms of the host.
FACTORS INFLUENCING THE INFECTION:

1- Microorganism factors:

1. Dose: number of m.o entering the body

2. Virulence: diseases. their capacity to cause

3. Invasiveness: ability of m.o to multiply & spread in the body.

4. Transmission: ability of m.o to pass to another suitable host (microbes enter the host through: inhalation, ingestion, sexual intercourse, inoculation, bite and injection).

2- Host Factors: (defense mechanisms)

A- Nonspecific defense mechanism:
1. Mechanical barriers: e.g. intact skin & mucous membrane of GIT, respiratory system.....

2. Glandular secretion: acidity of gastric juices, acidity of sweat, secretory IgA antibodies found in saliva, tear, intestinal contents & milk.


**B- Specific defense (Immune response): by cell-mediated & humeral immunity.**

3- Other factors:

**Local factors:** e.g. ischemia & foreign body promote infection.
**Systemic factors**: e.g., malnutrition, diabetes mellitus, chronic alcoholism & malignancy.

**Age**: both very young & very old have increasing risk of infection.

**Drugs**: e.g. appropriate antimicrobial eradicate many susceptible microorganism.
RESULTS OF INFECTION:

1. Eradication: most infections end by total eradication at the site of entry.

2. Persistence of infection: either in form of carrier state or mild chronic forms.

3. Spread: to other parts of the body: -direct spread as in cellulitis. -lymphatic spread -blood spread

4. Host death: in severe infection e.g. tetanus & diphtheria.

TISSUE RESPONSE (MICROSOPOICAL) RESPONSES TO INFECTIONS
There are 5 major histological patterns of tissue reaction in infections:

1- Suppurative inflammation: characterized by production of pus (pyogenic bacteria).

2- Chronic inflammation & scarring, is the final common pathway of many infections as in viruses, schistosomiasis & tuberculosis.

3- Mononuclear & Granulomatous characterized by formation of granuloma as TB, chronic abscess.

4- Cytopathic- Cytoproliferative inflammation., usually produced by viruses.

5- Necrotizing inflammation caused by powerful toxins e.g. Cl. Perfringens lead to gangrene & parasite (Entamoeba histolytica).
BACTERIAL INFECTIONS

CLASSIFICATION OF BACTERIA Bacteria are classified on several criteria:

• Gram stain: bacteria are either gram (-) or gram(+)

• Shape: bacteria are classified as cocci, bacilli (rods), vibrios, spirochetes.

• Growth requirements: bacteria are classified as: 1-Aerobic 2-Anaerobic

MECHANISM OF BACTERIAL INJURY PATHOGENESIS

Bacteria damage the tissue through several mechanisms:

1- Release toxins that kill cells. (exotoxin & Endotoxin)

2- Release lytic enzymes, includes proteases, hyaluronidase, coagulase & fibrinolysins that destroy the tissue & facilitate the spread of bacteria
3- Elicit an inflammatory reaction that may destroy not only the bacteria but also the infected tissue.

4- Elicit an immune reaction that may damage the tissues carrying the same antigen as the bacterium (“cross reactivity”).

**Bacterial infection is divided into:**

1- Acute. 2- Chronic.

**ACUTE BACTERIAL INFECTION:**

1- Catarrhal: affect mucus membrane.

2- Serous: Affect serous cavities and produce serous fluid

3- Pseudomembranous: Characterized by formation of pseudomembrane as in diphtheria.
4-Pyogenic or suppurative (Pus producing) e.g. abscess. M.O stimulate the secretion of IL 1 & TNF which stimulate complement, this attract neutrophil which secrets lytic enzyme, destroy tissue & form abscess.

Localization of pus leads to abscess formation which appear here as red congested Swelling at the side of the neck

**Perimandibular abscess in the chin region (redness, swelling)**
This is an example of pseudomembranous of the colon. The mucosal surface is hyperemic and is partially covered by a thick membrane like yellow-green exudate.

Bacterial infection of the blood

* Classified into:
  - Bacteraemia
  - Septicaemia
  - Pyaemia
  - Toxaemia: presence of toxin in Diphtheria blood e.g.
1-BACTERAEMIA

Presence of small numbers of bacteria in the blood without multiplication.

Patients have sub clinical or minor symptoms & lesion.

E.g., Strept. viridans in blood after vigorous brushing of teeth with dental sepsis. These bacteria (low virulent) are destroyed rapidly in blood because of antibodies, complement, & circulating macrophages.

It is important because it may settle in various parts of the body & cause localized lesion e.g. infective endocarditis.
2- SEPTICEMIA

Multiplication of bacteria in the blood of highly pathogenic bacteria

Serious infection with profound toxemia in which bacteria have overwhelmed the host defenses. It results in serious consequences which may end in death.

Clinically: Tachycardia, hypotension, multiple small hemorrhages due to capillary endothelial damage & shock

3-PYAEMIA (PUS IN THE BLOOD)

* Bacteria invade & multiply in a thrombus which then becomes heavily infiltrated by neutrophils & broken down by their digestive enzymes.

* Small fragments of the soften septic thrombus may then break away & be carried off in the blood Results in the developing of multiple & wide spread abscesses in the affected organs.
Abscess containing pus
Wall of abscess compose of fibrous tissue

THANK YOU

Tofe.radoparder
الجراحة
محافظة 7
General Surgery I

Fluid Therapy

doktor Chadidq Bkr Mraui

C.A.B.S.     CONS. URO.
Paraenteral administration

Is the administration of the fluid by any route other than alimentary tract.

Indications:
- In patient unable to take fluid for any reason or is not enough.
- In post operative period.
Rule of Approximate Thirds

2/3 of body weight is water (lean person)

Body water
- 2/3 intracellular
- 1/3 extracellular

Extracellular water
- 2/3 extravascular
- 1/3 intravascular
Input and output

Input means fluid taken by the patient

Output = urine output + insensible loss

Normal urine output 1 ml /kg /hour (adult)

Normal daily requirement about 2 liters Fluid should be isotonic (not hyper or hypotonic) First 24hrs no Na is needed bec. ADH→↑Aldost.

Best one is 5% Dextrose solution
Normal Water Exchange

Average daily \( ml \)

Sensible urine \( 800-1500 \)
intestineal up to \( 10,000 \)
sweat up to liters

Insensible lungs/skin \( 600-900 \)

70 kg Patient

Mosul university- College of dentistry-oral & maxillofacial surgery department
Total body water - 42 L
Intracellular - 28 L
Extracellular - 14 L
- Intravascular - 3 L
- Interstitial - 11 L
Causes of Fluid Loss

- Gastrointestinal loss
- Fever
- Blood loss
- Burns

- Peritonitis
- Fluid shifts
- Diuretics
- Inhalation of dry gases
Signs of Hypovolemia

- Tachycardia
- Orthostatic hypotension
- Flat neck veins when supine
- Decreased CVP
- Decreased urine output
- Dry membranes
CV collapse

Ringer’s Lactate

Most commonly used solution in the O.R.

Slightly hypotonic - 100 ml free water / liter

Most physiologic solution when large volumes are needed
Lactate metabolizes in liver to bicarbonate

Normal Saline

Large volumes cause dilutional hyperchloremic acidosis

Preferred solution for:

- Hypochloremic metabolic alkalosis
Diluting packed cells
5% Dextrose in Water

- Dextrose is metabolized leaving a large volume of free water
- Used for patients on sodium restriction
- Some dextrose needed when insulin is given
24 Hour Formula

- 100 ml for 1st 10 kg
- 50 ml for 2nd 10 kg
- 20 ml for remaining kg

1 ml of fluid = 15 drops
24 Hour Formula

Formula = volume of fluid per CC * 15/number of hours * 60
Example

- 82 kg patient
  - 100 ml for 1st 10 kg = 1000 ml
  - 50 ml for 2nd 10 kg = 500 ml
  - 20 ml for 62 kg = 1240 ml

Total = 2740 ml

2740 * 15/ 24 * 60 ≈ 28 drop per minute (one drop every two seconds)
Post operative fluid therapy

Immediate post op. period: assessment of vital signs, maintenance fluid, unnecessary to give potassium during 1st 24 hr.

Late post op. period: replace the sensible & insensible loss in fever every 1 degree, increased loss by 250 ml/day.
Fluids needed post op.

1\textsuperscript{st} day

2\textsuperscript{nd} day

day

3\textsuperscript{rd} day
Complications

1. volume excess (over hydration)
2. post op. hyponatremia
3. post op. hypernatremia
4. high out put renal failure
الجراحه
محاظرة 8
Subject
1. Shock
2. Types of shock
3. Hypovolemic shock
4. Vasovagal shock
5. Neurogenic shock
6. Cardiogenic shock
7. Anaphylactic shock
8. Septic shock
9. Pathophysiology of shock
Shock

**Shock**: is a life threaten situation of low cellular oxygen perfusion, manifested by sweating, restlessness, tense thirst, coldness, peripheral cyanosis, rapid pulse thready & low blood pressure & later there is clouding of the consciousness & sometimes lead to death.

**Types of shock**

according to the cause, commonly there is decrease in the effective circulating blood volume.

1- hypovolemic shock
2- vasovagal shock
3- neurogenic shock
4- cardiogenic shock
5- anaphylactic shock
6- septic shock
Hypovolemic shock
due to decrease actual blood volume, causes:

1- hemorrhage (hemorrhagic shock): due to loss of large amount of blood.

2- acute sever dehydration eg: repeated continuous vomiting, diarrhea as in cholera.

3- loss of plasma protein eg: sever burn Body response by shifting of fluid to the circulation, increase heart rate, venous constriction to increase heart rate

Treatment :

1- lower the head & elevate the feet to increase the venous return.

2- stop the bleeding by first aid or surgery
3- replace the lost amount by blood transfusion or by colloid (Dextran or plasma) or crystalloid (Normal saline, Ringer ....) until the blood is prepared.

4- sedation to relief the pain & restlessness by using morphine.

**Vasovagal shock**

Or psychogenic shock due to sudden severe pain or severe emotional reaction. It is due to increase of vagus nerve stimulation which lead to decrease heart rate & vasodilatation of veins in the muscles & veins supplying the splanichnic area lead to pooling of the blood & decrease the blood pressure lead to decrease the venous return & decrease cardiac output. TTT: lower the head & elevate the limbs to increase the blood flow to the brain.
Neurogenic shock

Due to sympathetic tone interruption lead to generalized vasodilatation eg: spinal cord transsection. TTT: treat the cause

Cardiogenic shock

due to ineffective pumping of the blood from the heart, venous return is normal eg: primary pump failure

( extensive MI, CMP, arrhythmia) & mechanical factors (pneumothorax, temponade). TTT: positive inotropic drugs eg: Dopamine & treat the cause.
Anaphylactic shock

mainly due to penicillin others include Dextran, serum, stings. The antigens combined with IgE on the mast cells & basophile lead to release of Histamine lead to bronchospasm & laryngeal edema, hypotension, massive vasodilatation lead to shock. TTT: steroid hydrocortisone 100mg amp. antihistamine allergine amp. Or chlorpheniramine bronchodilator adrenaline 1:1000 amp.
Septic shock due to G-ve septicemia & toxemia & mostly seen in the surgical conditions like peritonitis or liver abscess could occur in any patient (especially in hospitalized), in any age. Toxins liberated extensively lead to dilatation & post capillary sphincteric constriction lead to increase capillary permeability & loss of the intravascular fluid into the extravascular compartment lead to poor perfusion & decrease oxygenation & metabolism at cellular level with normal or increase cardiac output.

Predisposing factors:

DM, alcoholism blood disease, steroid & cytotoxic drugs & immune compromised patient.
Clinical picture:

1- intermittent pyrexia & rigor
2- jaundice due to liver damage
3- acute tubular necrosis of the kidneys
4- peripheral vasomotor paralysis & some times DIC

TTT:

1- isolate the causative agent (blood culture) & treat it.
2- broad spectrum AB.
3- fresh blood transfusion if needed
4- oxygen with adequate ventilation
5- high dose of steroids 1-2 gm hydrocortisone in single dose or methyl prednisolon IV, the effect is to decrease the edema of the capillary wall & so decrease
the amount of fluid leaving to extravascular compartment & improves O2 transport across the capillary wall to the tissues.

6- vasodilators to relax the post capillary sphincter &

Pathophysiology of shock

Shock lead to ↓ VR, ↓ CO& ↓ BP, lead to carotid sinus stimulation lead to ↑ HR & reflex vasoconstriction of the coetaneous & splanichnic vessels ( a physiologic process to ↓ blood to the unimportant organs like skin , limbs GIT.

So keep the perfusion to the vital organs as long as possible so all these sequences lead to:
1- ↓ BP (↓ CO)

2- rapid pulse (reflex carotid ↑)

3- pale cold skin (vasoconstriction)

If the condition prolonged lead to ↓ blood supply to vital organs:

1- brain: deterioration & then loss of consciousness lead to brain death then death

2- heart: cardiac ischemia cardiogenic shock in addition to the main one.

3- lungs: shock lung syndrome (pulmonary edema lead to ↓ O2 transport to the alveoli so in the blood.

4- kidneys: ↓ renal perfusion lead to acute tubular necrosis & anuria.

Tofe.rad
الجراحه
محافظة 9
General Surgery I

LAB 9

Blood Transfusion

Siddeeq B. Marie
C.A.B.S.Cons.Uro.
Types of blood

1-Banked whole blood: 25% of RBC died after 24hrs, another 25% die in 2 weeks old blood, another 25% die in 4 weeks old blood. So older blood:

A-more hemolysis

B-poor platelets

C-poor factor 8

D-higher PH

2-Fresh whole blood: should be given within 6 hrs. rich with factors 8&9.
3- **Packed RBCs**: useful in children, elderly patients.

4- **Frozen RBCs**: less risk of hepatitis, less antigenisity.

5- **platelets**: used for thrombocytopenia, DIC, massive blood loss.

6- **fresh frozen plasma FFP**: at -20 c° or -40 c° used as plasma expander.

7- **purified protein fraction PPF**: good expander allergy free safe for burn.

8- **concentrated human albumin**: good expander, can be stored for long time.
9- CPPt: white sediment collected by warming FFP

10- antihemophlic concentrate: factor 8.

11- dissociated human fibrinogen: stored in dry form & when used mixed with distilled water used for DIC & afibrinogenemia.

Methods of blood transfusion

1- intravenous IV:

a- Auto transfusion: there is a time to collect blood from the patient & given to him when needed.
b- **Isotransfusion:** usual way from donor to patient we can give up 1 liter in short time without warming, blood given in 3-4 hrs. warming is needed in massive blood transfusion.

2- intraperitoneal, intramedullary:

**Technique of blood transfusion**

1- Blood aspirated from a healthy donor with normal Hb. 500cc taken in a plastic bag with liquid anticoagulant then tests for HIV & hepatitis virus B&C done, then stored in blood bank for 4 weeks at 4°C.

2- Blood grouping & Rh. For patient & donor + cross
**matching done by:**

- long method: it takes 1-2 hrs which is the best.

- short method: it takes 5-15 min. used in emergency cases.

- blood group O- blindly could be used for very extreme emergency cases.

**3- blood substitutes:** can be used until cross matching done (colloid: Dextran, Hemacele, ..) When giving blood - check name carefully.

- use sterile sets.

- don't warm the blood.

  If warmed give the blood in 1-2 hrs So t1\2 of the blood at room temp. 5-6 hrs
Indication of blood transfusion

1- replacement of blood loss due to:
- trauma
- hemorrhagic condition
- major surgery with excessive blood loss

2- improvement of oxygen carrying capacity

3- replacement of clotting factors, in multifactorial cases give fresh blood or FFP,

4- pre-operative correction

5- miscellaneous: anemia, debilitation, sepsis,…
Complications of blood transfusion

1- hemolytic reaction

A- major incompatibility reaction: due to giving mismatched blood (different gp.) (ABO incomp.)

B- minor incompatibility reaction: due to error in minor gp. (Rh. Incomp.) There is intravascular destruction of RBCs lead to libration of heam from Hb & this will be deposited in the renal tubules lead to acute tubular necrosis & there will be a collapse of circulation

Clinical picture: fever, rigor, chills, loin pain, hematuria, later anuria.
Treatment:

-stop the blood transfusion immediately.

-large dose of Mannitol to enhance diuresis & prevent renal shut down.

- IV fluid.

-NaHCO₃ to alkalinization of urine & dissolve heam from renal tubules

-some times dialysis needed if above failed.

2- allergic reaction: less dangerous due to any Ag in plasma or WBCs C\P: fever, itching, urticarial rash. Rx: IM antihistamine , IV hydrocortisone , SC adrenaline.
3- **pyrexial reaction**: due to pyrogens in the transfused blood causes fever & rigor, blood is good culture media of bacteria & sepsis.

4- **bacterial sepsis**: when blood kept outside refrigerator for long time.

5- **infections**: syphilis malaria, HIV, HBV.

6- **thrombophlebitis**: from cannula

7- **air embolism**: rare bec. It needs 80ml to cause embolism.
الجراحة المحافظة 10
Preoperative Preparation

د. صديق بكر مرعي
The goals of preoperative evaluation are:

1. To screen for and properly manage comorbid conditions.
2. To assess the risk of anesthesia and surgery and lower it.
3. To identify patients who may require special anesthetic techniques or postoperative care.
4. To educate patients and families about anesthesia and the anesthesiologist’s role.
5. To obtain informed consent.
Cardiovascular

• Patients with ischemic heart disease are at risk for myocardial ischaemia or infarction in the perioperative period.

• A thorough history should ascertain whether angina is new or has recently changed from a previously stable pattern.

• A description of the patient's exercise tolerance.

• Hx of hypertension, heart failure; valvular heart disease; arrhythmias; etc.
Respiratory

• **Cigarette** smoke has several adverse effects, including alteration of mucus secretion, clearance, and decrease in small airway calibre. The chronic smoker should be encouraged to stop smoking for at least 8 weeks prior to the operation,' but stopping smoking for even 24 h. is of benefit.

• Patients with chronic **obstructive pulmonary disease (COPD)** are at increased risk of perioperative respiratory complications. Anaesthesia, surgery and postoperative analgesia all predispose the patient with **COPD** to respiratory depression, atelectasis, retained secretions, pneumonia and respiratory insufficiency or failure.

• The patient with **asthma** is at particular risk as manipulation of the airway and cold dry anaesthetic gases are potent triggers of intraoperative bronchospasm.
• Determine the presence of cough and the colour and amount of sputum. Ensure that there is no acute upper respiratory infection

• **Restrictive lung disease** will be worsened by upper abdominal or thoracic surgery, and place the patient at increased risk for perioperative failure and may require postoperative monitoring in a critical care setting
Endocrine

Patients with diabetes mellitus require careful management in the perioperative period, as the stress of surgery and perioperative fasting can cause marked swings in blood glucose. Diabetics frequently have organ damage involving the cardiovascular, nervous and renal systems.

Patients with thyroid disease may experience difficulties under anaesthesia. Profound hypothyroidism is associated with myocardial depression and exaggerated responses to sedative medications. Hyperthyroid patients are at risk for perioperative thyroid storm.

Patients at risk for adrenal suppression (history of exogenous steroid therapy) may not be able to increase their own corticosteroid production to match the imposed stress of surgery.
GI-Hepatic

• Patients with hepatic disease frequently present problems with fluid and electrolyte imbalance, coagulopathies and altered drug metabolism

• Patients with gastroesophageal reflux (GER) are prone to regurgitation of gastric contents and aspiration pneumonitis during the perioperative period

Renal

• Generally all fluid and electrolyte disorders should be corrected prior to elective surgery
Haematologic

• Anemias  A minimum haemoglobin level of 10 gm/dL is required for elective surgery
• Coagulopathies involving clotting factors and platelets, both congenital and acquired, require careful management
Medications and Allergies

• As a general rule, all cardiac and pulmonary medications and most other necessary medications should be taken with sips of water at the usual time, up to and including the day of surgery. Possible exceptions to this include warfarin, NSAID's, insulin (adjustment of the dose is needed on the day of surgery), oral hypoglycemics and antidepressants.

Prior Anaesthetics

The patient undergoing anaesthesia and surgery should be carefully questioned on their response to previous anaesthetics and a family history of problems with anaesthesia.
Physical Examination

• The physical examination should focus on evaluation of the airway, the cardiovascular system, the respiratory system, and any other systems identified as having symptoms or disease from the history.

• General
A general assessment of the patient's physical and mental status is performed. Note whether the patient is alert, calm, and cooperative.
<table>
<thead>
<tr>
<th>ASA</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Healthy patient without organic, biochemical, or psychiatric disease</td>
</tr>
<tr>
<td>2</td>
<td>A patient with mild systemic disease, e.g., mild asthma or well-controlled hypertension. No significant impact on daily activity. Unlikely to have an impact on anesthesia and surgery</td>
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<tr>
<td>3</td>
<td>Significant or severe systemic disease that limits normal activity, e.g., renal failure on dialysis or class 2 congestive heart failure. Significant impact on daily activity. Probable impact on anesthesia and surgery</td>
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<tr>
<td>4</td>
<td>Severe disease that is a constant threat to life or requires intensive therapy, e.g., acute myocardial infarction, respiratory failure requiring mechanical ventilation. Serious limitation of daily activity. Major impact on anesthesia and surgery</td>
</tr>
<tr>
<td>5</td>
<td>Moribund patient who is equally likely to die in the next 24 hours with or without surgery</td>
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<tr>
<td>6</td>
<td>Brain-dead organ donor</td>
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</tbody>
</table>

“E” added to the classification indicates emergency surgery
<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urinalysis</td>
<td>All patients: for sugar, blood, and protein</td>
</tr>
<tr>
<td>ECG</td>
<td>Age &gt; 50 years&lt;br&gt;History of heart disease, hypertension or chronic lung disease</td>
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<tr>
<td>FBC</td>
<td>Males &gt; 40 years&lt;br&gt;All females&lt;br&gt;All major surgery&lt;br&gt;Whenever anaemia is suspected</td>
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<tr>
<td>Test</td>
<td>Indications</td>
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<tr>
<td>Creatinine and electrolytes</td>
<td>Age &gt; 60 years, All major surgery, Diuretic drugs, Suspected renal disease</td>
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<tr>
<td>Blood glucose</td>
<td>Diabetic patients, Glycosuria</td>
</tr>
<tr>
<td>Coagulation screen</td>
<td>History of bleeding tendency (some units measure before major surgery)</td>
</tr>
<tr>
<td>Pregnancy test</td>
<td>Whenever there is any chance of pregnancy</td>
</tr>
<tr>
<td>Chest radiograph</td>
<td>Not routine, Acute cardiac or chest disease, Chronic cardiac or chest disease that has worsened in the last year, Risk of pulmonary TB (recent arrival from the developing world or immunocompromise), Malignant disease</td>
</tr>
</tbody>
</table>
### Fasting guidelines

<table>
<thead>
<tr>
<th>Ingested material</th>
<th>Minimum fast</th>
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<tbody>
<tr>
<td>Clear liquids</td>
<td>2 h</td>
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<tr>
<td>Breast milk</td>
<td>4 h</td>
</tr>
<tr>
<td>Infant formula milk</td>
<td>4–6 h</td>
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<tr>
<td>Non-human milk</td>
<td>6 h</td>
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<tr>
<td>Light meal</td>
<td>6 h</td>
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<tr>
<td>المعنى</td>
<td>الملاحظات</td>
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الجراحة
المجازفة 11
Postoperative care

Post operative note and orders

The patient should be discharged to the ward with comprehensive orders for the following:
- Vital signs
- Pain control
- Rate and type of intravenous fluid
- Urine and gastrointestinal fluid output
- Other medications
- Laboratory investigations

The patient’s progress should be monitored and should include at least:
- A comment on medical and nursing observations
- A specific comment on the wound or operation site
- Any complications
- Any changes made in treatment

Aftercare: Prevention of complications

- Encourage early mobilization:
  - Deep breathing and coughing
  - Active daily exercise
  - Joint range of motion
  - Muscular strengthening
  - Make walking aids such as canes, crutches and walkers available and provide instructions for their use
- Ensure adequate nutrition
- Prevent skin breakdown and pressure sores:
  - Turn the patient frequently
  - Keep urine and faeces off skin
- Provide adequate pain control

Discharge note

On discharging the patient from the ward, record in the notes:
- Diagnosis on admission and discharge
- Summary of course in hospital
- Instructions about further management, including drugs prescribed.

Ensure that a copy of this information is given to the patient, together with details of any follow-up appointment.
Postoperative Management

If the patient is restless, something is wrong.

Look out for the following in recovery:

- Airway obstruction
- Hypoxia
- Haemorrhage: internal or external
- Hypotension and/or hypertension
- Postoperative pain
- Shivering, hypothermia
- Vomiting, aspiration
- Falling on the floor
- Residual narcosis

The recovering patient is fit for the ward when:

- Awake, opens eyes
- Extubated
- Blood pressure and pulse are satisfactory
- Can lift head on command
- Not hypoxic
- Breathing quietly and comfortably
- Appropriate analgesia has been prescribed and is safely established
Post operative pain relief

- Pain is often the patient’s presenting symptom. It can provide useful clinical information and it is your responsibility to use this information to help the patient and alleviate suffering.

- Manage pain wherever you see patients (emergency, operating room and on the ward) and anticipate their needs for pain management after surgery and discharge.

- Do not unnecessarily delay the treatment of pain; for example, do not transport a patient without analgesia simply so that the next practitioner can appreciate how much pain the person is experiencing.

Pain management is our job.

Pain Management and Techniques

- Effective analgesia is an essential part of postoperative management.

- Important injectable drugs for pain are the opiate analgesics. Nonsteroidal anti-inflammatory drugs (NSAIDs), such as diclofenac (1 mg/kg) and ibuprofen can also be given orally and rectally, as can paracetamol (15 mg/kg).

- There are three situations where an opiate might be given:
  - Preoperatively
  - Intraoperatively
  - Postoperatively

- Opiate premedication is rarely indicated, although an injured patient in pain may have been given an opiate before coming to the operating room.

- Opiates given pre- or intraoperatively have important effects in the postoperative period since there may be delayed recovery and respiratory depression, even necessitating mechanical ventilation.

- Short acting opiate fentanyl is used intra-operatively to avoid this prolonged effect.

- Naloxone antagonizes (reverses) all opiates, but its effect quickly wears off.

- Commonly available inexpensive opiates are pethidine and morphine.

- Morphine has about ten times the potency and a longer duration of action than pethidine. (continued next page)
Post operative pain relief (continued)

- Ideal way to give analgesia postoperatively is to:
  - Give a small intravenous bolus of about a quarter or a third of the maximum dose (e.g. 25 mg pethidine or 2.5 mg morphine for an average adult)
  - Wait for 5–10 minutes to observe the effect: the desired effect is analgesia, but retained consciousness
  - Estimate the correct total dose (e.g. 75 mg pethidine or 7.5 mg morphine) and give the balance intramuscularly.
  - With this method, the patient receives analgesia quickly and the correct dose is given
- If opiate analgesia is needed on the ward, it is most usual to give an intramuscular regimen:
  - Morphine:
    - Age 1 year to adult: 0.1–0.2 mg/kg
    - Age 3 months to 1 year: 0.05–0.1 mg/kg
  - Pethidine: give 7–10 times the above doses if using pethidine

- **Opiate analgesics should be given cautiously if the age is less than 1 year. They are not recommended for babies aged less than 3 months unless very close monitoring in a neonatal intensive care unit is available.**

Anaesthesia & Pain Control in Children

- Ketamine anaesthesia is widely used for children in rural centres (see pages 14–14 to 14–21), but is also good for pain control.
- Children suffer from pain as much as adults, but may show it in different ways.
- Make surgical procedures as painless as possible:
  - Oral paracetamol can be given several hours prior to operation
  - Local anaesthetics (bupivacaine 0.25%, not to exceed 1 ml/kg) administered in the operating room can decrease incisional pain
  - Paracetamol (10–15 mg/kg every 4–6 hours) administered by mouth or rectally is a safe and effective method for controlling postoperative pain
  - For more severe pain, use intravenous narcotics (morphine sulfate 0.05–0.1 mg/kg IV) every 2–4 hours
  - Ibuprofen 10 mg/kg can be administered by mouth every 6–8 hours
  - Codeine suspension 0.5–1 mg/kg can be administered by mouth every 6 hours, as needed.
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الجراحه
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SKIN TUMOURS

DR. SIDDEEKK B. MARIE
The epidermis is composed of keratinised, stratified, squamous epithelium and can be subdivided further into five layers: the stratum basale (deepest), the stratum spinosum, the stratum granulosum the stratum lucidum and the stratum corneum (superficial). It accounts for 5% of the total skin.

The dermis constitutes 95% of the skin and is structurally divided into two layers. The superficial papillary layer is composed of delicate collagen and elastin fibres in ground substance, into which a capillary and lymphatic network ramifies. The deeper reticular layer is composed of coarse branching collagen, layered parallel to the skin surface. Adnexal structures such as hair follicles and sebaceous and sweat glands span both the epidermal and dermal layers and contain some keratinocytes.
Lipoma:
- Lipomas are the most common tumor in man. Lipoma is a dermal or subcutaneous collection of benign adipose tissue. Most lesions are solitary, discrete nodules that remain asymptomatic and commonly occur in the trunk and extremities. Often the lesions may have a fibrous capsule that surrounds the collection of adipose cells.

Dercum syndrome: is a rare condition characterized by multiple, painful lipomas. These lipomas mainly occur on the trunk, the upper arms, and upper legs. Treatment: usually removed for cosmetic reasons or on occasion, as a result of a neural or vascular component (angioliopoma) or when they compress an adjacent nerve.

Large lipomas (>10cm) as well as deep-seated ones should arouse the suspicion of malignant change. The most frequent malignant counterpart is the liposarcoma.
Sebaceous cyst (epidermal cyst) :
- Are the most common form of cysts, they can occur at any age and any body location except in the palm and soles. Most commonly, they occur in areas of increased pilosebaceous activity on the head and upper trunk. The lesion occurs when the duct of sebaceous gland becomes blocked, it distends with its own secretion and ultimately becomes a sebaceous cyst. It is a firm swelling, adherent to skin and has punctum. It may get infection and ulceration.

Complications: infection, ulceration, calcification, keratin horn and malignant change which is very rare

Treatment: if infected treated by drainage the abscess and later on surgical excision
Basal cell carcinoma:

It is the most common malignancy of whites. It arise from A-basal layer epithelial cells. B-external root sheath of hair follicle. It is slowly growing tumor, it doesn’t metastasize but can infiltrate the adjacent tissues. commonly found in face above line drawn from angle of mouth to lobe of ear. It could occur anywhere, but particularly in the skin of scalp, face, arm and hands i.e sun exposed area.

Microscopic appearance: Characteristic finding is of ovoid cells in nest with a single outer (palisading) layer.
Risk factors:
1- Sun exposure.
2- Advancing age.
3- Faint complexion.
4- Long term exposure to psoralene and UVA therapy (for psoriasis).
5- Arsenic exposure.
6- Immune deficiency.
7- Radiation
8- Viral infection e.g. HPV 16, HIV.

Type of basal cell carcinoma:
1- Nodular ulcerative type.
2- Superficial spreading.
3- Sclerosing type.
4- Pigmented type.
5- Basal cell naevoid syndrome (Gorlin syndrome)
Treatment of basal cell carcinoma:

- Curettage and desiccation.
- Surgical excision with safety margin (0.5 cm).
- Cryotherapy.
- Radiation therapy.
- Mohs fresh frozen section.
- Dermabrasion and chemical peel.
- Interferon alpha.
- Co2 LASER.
- Local chemotherapy e.g. 5-fluorouracil
**Sequamous cell carcinoma:**

- It originates from the keratinising cells of epidermis or its appendages. It also arises from stratum basale of epidermis but, unlike BCC, it expresses cytokeratins 1 and 10.

**Types of sequamous cell carcinoma:**

A- verrucous SCC: slow growing, exophytic, and less likely to metastasize.  B- ulcerative SCC: grows rapidly and is locally invasive.

**Microscopic appearances:**

Characteristic irregular masses of squamous epithelium are noted to proliferate and invade the dermis from the germinal layer. This tumor is stains positive for cytokeratin 1 and 10.
Treatment:
as for basal cell carcinoma but with exception is safe margin are more than that of basal cell carcinoma (1-2 cm)
Malignant melanoma:
It is neoplasm of melanocyte which is neural crest derived cells that have differentiated toward melanocytes. It occur in skin but can occur anywhere in body where the melanocytes exist, such as bowel mucosa, retina... etc.

Microscopic appearances:
Atypical melanocyte invades from the dermis-epidermis junction down into the dermis.

Clinical features:
appeared as change in pre-existing naevus or as new lesion.
Assess lesion for ABCDE:
Asymmetry
Border irregularity
Colour change
Diameter more than 6mm
Extra features (itch, bleeding...).
Melanoma thickness grading:

Clark level:
1- In situ
2- Papillary dermis
3- Papillary reticular dermis
4- Reticular dermis
5- Subcutaneous

Breslow depth (mm):
1- <1.00
2- 1.01-2.00
3- 2.01-4.00
4- >4.00
5- More depth in mm means more aggressive tumours
Treatment of malignant melanoma

Surgical excision of primary tumour is the mainstay of treatment

In situ: 0.5 cm safety margin

mm to 4 mm: 2 cm

mm 2-3 cm: 4 <

With lymph node dissection (in case of lymph nodes metastasis)

For advanced melanoma number of therapeutic modalities which included: systemic chemotherapy, immunotherapy, isolated limb perfusion, and radiotherapy
Prognosis:

1- Breslow depth.
2- Type of lesion: superficial type better prognosis.
3- The anatomical site: tumor on the trunk and scalp have poor prognosis.
4- Lymph node metastasis.
5- Sex: good prognosis in female than male

Prevention:

1- Avoid sun exposure by using physical and chemical barrier
2- Prophylactic removal of suspicious naevus
3- Periodic self examination in front of mirror
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